Steven D. Waldman

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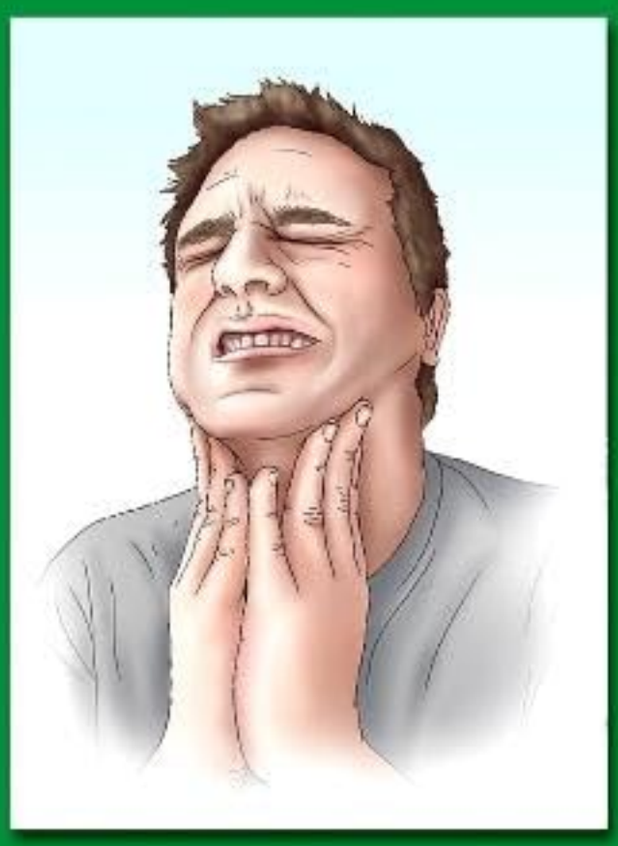
Atlas of COMMON PAIN SYNDROMES



5th EDITION

ELSEVIER

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To my mentor and friend Michael Houston, PhD.
1957–2023

PREFACE

Recently, one of my medical students told me that after several weeks of being really sick and being treated for myriad respiratory ills, she was finally diagnosed with pertussis. Now keep in mind that we are located in Kansas City, USA, not a developing country and cases of pertussis are few and far between. In trying to figure out what went wrong in the care of my student, I asked her several questions: "Were you immunized as a child?" Yes. "Had you recently traveled abroad?" No. "What was the pertussis like?" Horrible! Having never seen a case of pertussis, I then asked the question that really had me puzzled. "How did they come up with the diagnosis of pertussis?"

The student told me that initially she thought that she had picked up a bad case of bronchitis while on her pediatrics rotation. She took a Z-Pak without improvement and then completeda course of moxifloxacin. She did not see much improvement from the antibiotics so she went to the student health service on two separate occasions. Both times the physician concurred with the working diagnosis of bronchitis or early pneumonia. A subsequent trip to the local emergency department yielded the same diagnosis and she was discharged tohome. As the evening progressed, she found that she was having difficulty breathing and returned to the emergency room. She was admitted to the intensive care unit with a working diagnosis of respiratory failure. Antibiotics were given, and breathing treatments administered, yet diagnosis remained elusive. The next morning on rounds, a second-year medical student suggested that perhaps all this coughing was the result of whooping cough, which the student had just read about in her medical microbiology class. At first, everyone laughed and rolled their eyes... two beats... silence, and then... the "ah ha" moment....and the correct diagnosis was finally made.

You may be wondering why I include this story in the preface to a book about pain management. It seems to me that as medical practitioners, we tend to limit ourselves to specific, personalized constructs that we devise to simplify the diagnosis of painful conditions. Within these constructs are the frequent admonitions against hunting for zebras whenever we hear hoof beats. These constructs pressure us to move toward the center of the diagnostic bell-shaped curve, to cleave to evidence-based medicine, etc. However, if taken to extremes, these parameters have the potential to severely limit how we process our patients' histories and come to a diagnosis. It is my hope that this fifth edition of *Atlas of Common Pain Syndromes* will help clinicians recognize, diagnose, and treat painful conditions they otherwise would not have even thought of.....and as a result..... provide more effective care for their patients who are in pain.

CONTENTS

1

Acute Herpes Zoster of the First Division of the Trigeminal Nerve

ICD-10 CODE B02.22

THE CLINICAL SYNDROME

Herpes zoster is an infectious disease caused by the varicella-zoster virus (VZV). Primary infection with VZV in a non-immune host manifests clinically as the childhood disease chickenpox (varicella). Investigators have postulated that during the course of this primary infection, the virus migrates to the dorsal root or cranial ganglia, where it remains dormant and produces no clinically evident disease. In some individuals, the virus reactivates and travels along the sensory pathways of the first division of the trigeminal nerve, where it produces the characteristic pain and skin lesions of herpes zoster, or shingles.

Why reactivation occurs in some individuals but not in others is not fully understood, but investigators have theorized that a decrease in cell-mediated immunity may play an important role in the evolution of this disease by allowing the virus to multiply in the ganglia, spread to the corresponding sensory nerves, and produce clinical disease. Patients who are suffering from malignant disease (particularly lymphoma) or chronic disease and those receiving immunosuppressive therapy (chemotherapy, steroids, radiation) are generally debilitated and thus are much more likely than the healthy population to develop acute herpes zoster. These patients all have in common a decreased cell-mediated immune response, which may also explain why the incidence of shingles increases dramatically in patients older than 60 years and is relatively uncommon in those younger than 20 years. There is anecdotal evidence that there has been an increased incidence of herpes zoster infection associated with COVID-19.

The first division of the trigeminal nerve is the second most common site for the development of acute herpes zoster, after the thoracic dermatomes. Rarely, the virus attacks the geniculate ganglion and results in hearing loss, vesicles in the ear, and pain (Fig. 1.1). This constellation of symptoms is called Ramsay Hunt syndrome and must be distinguished from acute herpes zoster involving the first division of the trigeminal nerve.

SIGNS AND SYMPTOMS

As viral reactivation occurs, ganglionitis and peripheral neuritis cause pain that may be accompanied by flu-like symptoms. The pain generally progresses from a dull, aching sensation to dysesthetic or neuritic pain in the distribution of the first division of the trigeminal nerve. In most patients, the pain of acute herpes zoster precedes the eruption of rash by 3–7 days, and this delay often leads to an erroneous diagnosis (see "Differential Diagnosis"). However, in most patients, the clinical diagnosis of shingles is readily made when the characteristic rash appears. As with chickenpox, the rash of herpes zoster appears in crops of macular lesions that rapidly progress to papules and then to vesicles (Fig. 1.2). Eventually, the vesicles coalesce, and crusting occurs (Fig. 1.3). In some patients, the appearance of vesicular lesions on the nose indicating the involvement of the nasociliary nerve is an indicator of subsequent ocular inflammation and complications including corenal ulceration and exposure keratitis (Fig. 1.4). The affected area can be extremely painful, and the pain tends to be exacerbated by any movement or contact (e.g., with clothing or sheets). As the lesions heal, the crust falls away, leaving pink scars that gradually become hypopigmented and atrophic.

In most patients, the hyperesthesia and pain resolve as the skin lesions heal. In some patients, however, pain persists beyond lesion healing. This common and feared complication of acute herpes zoster is called postherpetic neuralgia, and older persons are affected at a higher rate than is the general population suffering from acute herpes zoster (Fig. 1.5). The symptoms of postherpetic neuralgia can vary from a mild, self-limited condition to a debilitating, constantly burning pain that is exacerbated by light touch, movement, anxiety, or temperature change. This unremitting pain may be so severe that it completely devastates the patient's life; ultimately, it can lead to suicide. To avoid this disastrous sequela to a usually benign, self-limited disease, the clinician must use all possible therapeutic efforts in patients with acute herpes zoster of the trigeminal nerve.

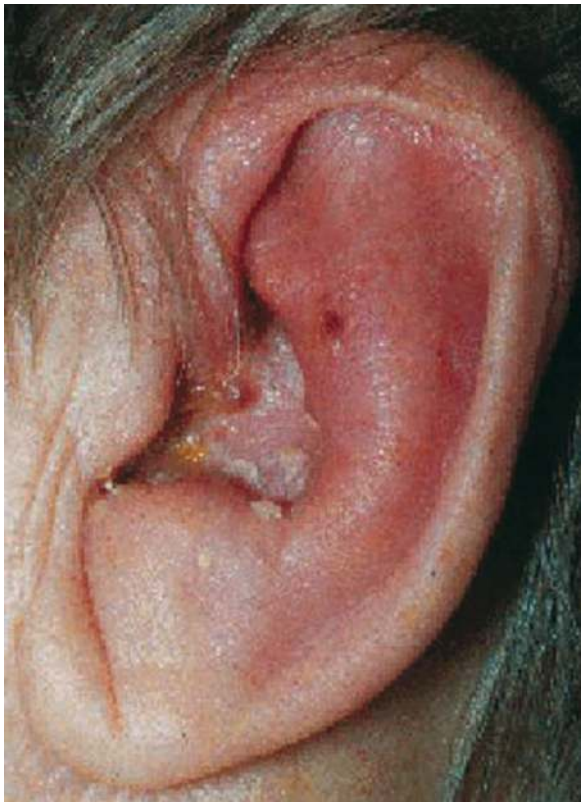

FIG 1.1 Ramsay Hunt syndrome.

FIG 1.2 The pain of acute herpes zoster of the trigeminal nerve often precedes the characteristic vesicular rash.

TESTING

Although in most instances the diagnosis is easily made on clinical grounds, confirmatory testing is occasionally required. Such testing may be desirable in patients with other skin lesions that confuse the clinical picture, such as in patients with acquired immunodeficiency syndrome who are suffering from Kaposi sarcoma. In such patients, polymerase chain reaction testing and immunofluorescent antibody testing can rapidly identify herpes zoster virus and distinguish it from herpes simplex infections (Fig. 1.6). In uncomplicated cases, the diagnosis of acute herpes zoster may be strengthened by obtaining a Tzanck smear from the base of a fresh vesicle; this smear reveals multinucleated giant cells and eosinophilic inclusions (Fig. 1.7). However, this inexpensive bedside test does not have the ability to distinguish between lesions caused by the VZV and herpes simplex infections.

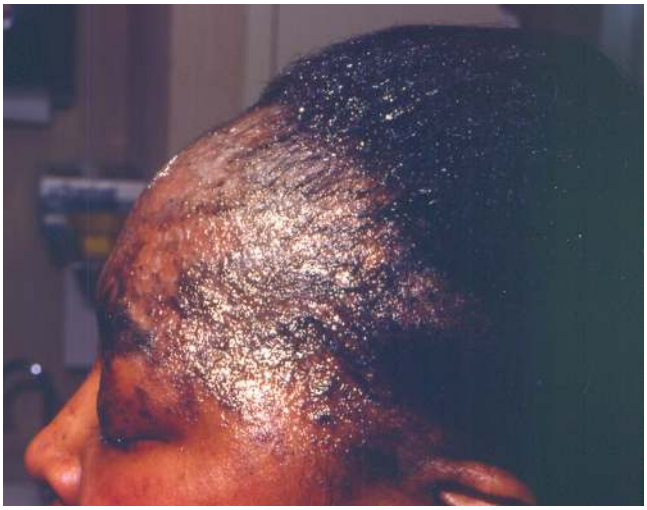

FIG 1.3 Acute herpes zoster involving the ophthalmic division of the left trigeminal nerve. (From Waldman SD. *Pain management*. Philadelphia: Elsevier; 2007.)

DIFFERENTIAL DIAGNOSIS

A careful initial evaluation, including a thorough history and physical examination, is indicated in all patients suffering from acute herpes zoster of the trigeminal nerve. The goal is to rule out occult malignant or systemic disease that may be responsible for the patient's immunocompromised state. A prompt diagnosis allows early recognition of changes in clinical status that may presage the development of complications, including myelitis or dissemination of the disease. Other causes of pain in the distribution of the first division of the trigeminal nerve include trigeminal neuralgia, sinus disease, glaucoma, retro-orbital tumor, inflammatory disease (e.g., Tolosa–Hunt syndrome), and intracranial disease, including tumor.

TREATMENT

The therapeutic challenge in patients presenting with acute herpes zoster of the trigeminal nerve is twofold: (1) the immediate relief of acute pain and symptoms and (2) the prevention of complications, including postherpetic neuralgia. Most pain specialists agree that the earlier treatment is

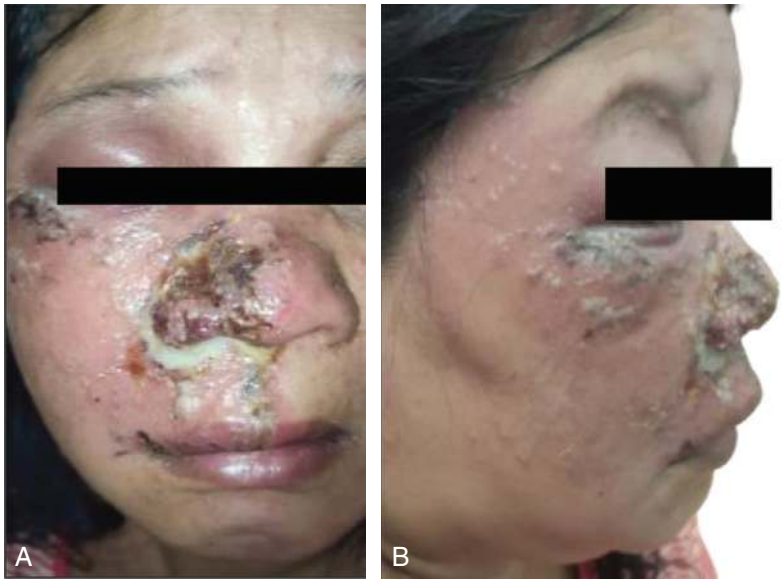

FIG 1.4 Hutchinson sign. Herpes zoster rashes along right ophthalmic and mandibular divisions, with positive. Hutchinson sign as seen on anterior **(A)** and lateral **(B)** views. (From Selvi RT, Loi CW, Mohamad I. Hutchinson sign in herpes zoster with COVID-19. *Vis J Emerg Med*. 2022;27:101356.)

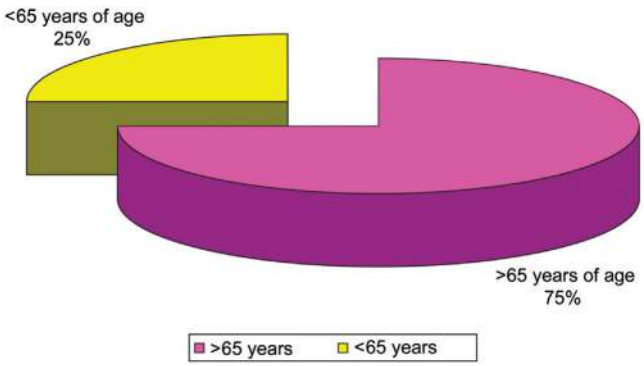

FIG 1.5 Age of patients suffering from acute herpes zoster.

initiated, the less likely it is that postherpetic neuralgia will develop. Further, because older individuals are at the highest risk for developing postherpetic neuralgia, early and aggressive treatment of this group of patients is mandatory.

Nerve Block

Sympathetic neural blockade with local anesthetic and steroid through stellate ganglion block is the treatment of choice to relieve the symptoms of acute herpes zoster of the trigeminal nerve, as well as to prevent postherpetic neuralgia. As vesicular crusting occurs, the steroid may also reduce neural scarring. Sympathetic nerve block is thought to achieve these goals by blocking the profound sympathetic stimulation caused by viral inflammation of the nerve and gasserian ganglion. If untreated, this sympathetic hyperactivity can cause ischemia secondary to decreased blood flow of the intraneural capillary bed. If this ischemia is allowed to persist, endoneural edema forms, thus increasing endoneural pressure and causing a further reduction in endoneural blood flow, with irreversible nerve damage.

These sympathetic blocks should be continued aggressively until the patient is pain-free and should be reimplemented if the pain returns. Failure to use sympathetic neural blockade immediately and aggressively, especially in older patients, may sentence the patient to a lifetime of suffering from postherpetic neuralgia. Occasionally, some patients do not experience pain relief from stellate ganglion block but do respond to blockade of the trigeminal nerve.

Opioid Analgesics

Opioid analgesics can be useful to relieve the aching pain that is common during the acute stages of herpes zoster, while sympathetic nerve blocks are being implemented. Opioids are less effective in relieving neuritic pain, which is also common. Careful administration of potent, long-acting opioid analgesics (e.g., oral morphine elixir, methadone) on a time-contingent rather than an as-needed basis may be a beneficial adjunct to the pain relief provided by the sympathetic neural blockade. Because many patients suffering from acute herpes zoster are older or have severe multisystem disease, close monitoring for the potential side effects of potent opioid analgesics (e.g., confusion or dizziness, which may cause a patient to fall) is warranted. Daily dietary fiber supplementation and milk of magnesia should be started along with opioid analgesics to prevent constipation.

Adjuvant Analgesics

The anticonvulsant gabapentin represents a first-line treatment for the neuritic pain of acute herpes zoster of the trigeminal nerve. Studies suggest that gabapentin may also help prevent postherpetic neuralgia. Treatment with

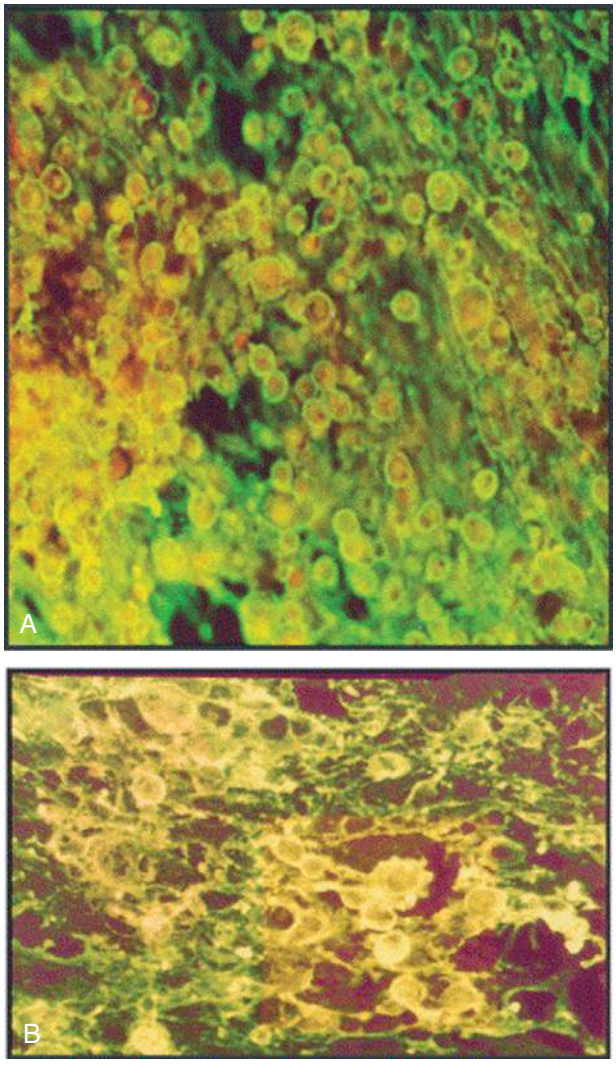

FIG 1.6 Detection of anti-varicella-zoster virus IgG by the fluorescent antibody to membrane antigen assay. Positive result **(A)** and negative control **(B)**. (From Sauerbrei A, Färber I, Brandstädt A, Schacke M, Wutzler P. Immunofluorescence test for sensitive detection of varicella-zoster virus-specific IgG: an alternative to fluorescent antibody to membrane antigen test. *J Virol Methods*. 2004;19(1):15–30.)

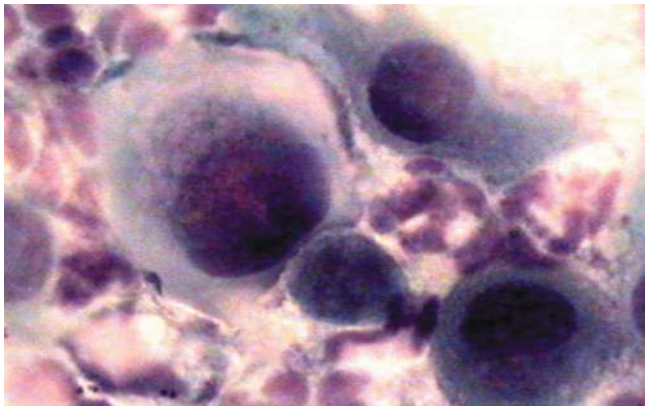

FIG 1.7 Tzanck smear showing giant multinucleated cell. (Courtesy Dr. John Minarcik.)

gabapentin should begin early in the course of the disease; this drug may be used concurrently with neural blockade, opioid analgesics, and other adjuvant analgesics, including antidepressants, if care is taken to avoid central nervous system side effects. Gabapentin is started at a bedtime dose of 300 mg and is titrated upward in 300-mg increments to a maximum of 3600 mg given in divided doses, as side effects allow. Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine should be considered in patients suffering from severe neuritic pain who fail to respond to nerve blocks and gabapentin. If this drug is used, strict monitoring of hematologic parameters is indicated, especially in patients receiving chemotherapy or radiation therapy. Phenytoin may also be beneficial to treat neuritic pain, but it should not be used in patients with lymphoma; the drug may induce a pseudolymphoma-like state that is difficult to distinguish from the actual lymphoma.

Antidepressants may also be useful adjuncts in the initial treatment of patients suffering from acute herpes zoster. On a short-term basis, these drugs help alleviate the significant sleep disturbance that is commonly seen. In addition, antidepressants may be valuable in ameliorating the neuritic component of the pain, which is treated less effectively with opioid analgesics. After several weeks of treatment, antidepressants may exert a mood-elevating effect, which may be desirable in some patients. Care must be taken to observe closely for central nervous system side effects in this patient population. In addition, these drugs may cause urinary retention and constipation, which may mistakenly be attributed to herpes zoster myelitis.

Antiviral Agents

A few antiviral agents, including valacyclovir, famciclovir, and acyclovir, can shorten the course of acute herpes zoster and may even help prevent the development of postherpetic neuralgia. They are probably useful in attenuating the disease in immunosuppressed patients. These antiviral agents can be used in conjunction with the aforementioned treatment modalities. Careful monitoring for side effects is mandatory.

Adjunctive Treatments

The application of ice packs to the lesions of acute herpes zoster may provide relief in some patients. The application of heat increases pain in most patients, presumably because of the increased conduction of small fibers; however, it is beneficial in an occasional patient and may be worth trying if the application of cold is ineffective. Transcutaneous electrical nerve stimulation and vibration may also be effective in a limited number of patients. The favorable risk-to-benefit ratio of these modalities makes them reasonable alternatives for patients who cannot or will not undergo sympathetic neural blockade or cannot tolerate pharmacologic interventions.

Topical application of aluminum sulfate as a tepid soak provides excellent drying of the crusting and weeping lesions of acute herpes zoster, and most patients find these soaks soothing. Zinc oxide ointment may also be used as a protective agent, especially during the healing phase, when temperature sensitivity is a problem. Disposable diapers can be used as absorbent padding to protect healing lesions from contact with clothing and sheets.

COMPLICATIONS AND PITFALLS

In most patients, acute herpes zoster of the trigeminal nerve is a self-limited disease. In older patients and in immunosuppressed patients, however, complications may occur. Cutaneous and visceral dissemination may range from a mild rash resembling chickenpox to an overwhelming, life-threatening infection in those already suffering from severe multisystem disease. Myelitis may cause bowel, bladder, and lower extremity paresis. Ocular complications of trigeminal nerve involvement may range from severe photophobia to keratitis with loss of sight.

CLINICAL PEARLS

Because the pain of herpes zoster usually precedes the eruption of skin lesions by 3–7 days, some other painful conditions (e.g., trigeminal neuralgia and glaucoma) may erroneously be diagnosed. In this setting, an astute clinician should advise the patient to call immediately if a rash appears, because acute herpes zoster is a possibility. If the patient is wearing a face mask, it is advisable to remove it in order to identify the presence of Hutchinson sign given its prognostic value in identifying the potential for ocular involvement. Some pain specialists believe that in a few immunocompetent patients, when reactivation of VZV occurs, a rapid immune response attenuates the natural course of the disease and the characteristic rash of acute herpes zoster may not appear. In this case, pain in the distribution of the first division of the trigeminal nerve without an associated rash is called zoster sine herpete and is, by necessity, a diagnosis of exclusion. Therefore other causes of head pain must be ruled out before this diagnosis is invoked.

SUGGESTED READINGS

Au SCL. Herpes zoster ophthalmicus: the importance of face mask removal examination under the COVID-19 pandemic. *Vis J Emerg Med*. 2021;24:101083.

Bandaranayake T, Shaw AC. Host resistance and immune aging. *Clin Geriatr Med*. 2016;32(3):415–432.

Lee HY, Kim MG, Park DC, et al. Zoster sine herpete causing facial palsy. *Am J Otolaryngol*. 2012;33(5):565–571.

Niederer RL, Meyer JJ, Liu K, Danesh-Meyer HV. Herpes zoster ophthalmicus clinical presentation and risk factors for loss of vision. *Am J Ophthalmol*. 2021;226:83–89.

O'Connor KM, Paauw DS. Herpes zoster. *Med Clin North Am*. 2013;97(4):503–522.

Pradhan RR. Ramsay Hunt syndrome. *IDCases*. 2020;21:e00850.

Schmader K. Zoster herpes. *Clin Geriatr Med*. 2016;32(3):539–553.

Selvi RT, Loi CW, Mohamad I. Hutchinson sign in herpes zoster with COVID-19. *Vis J Emerg Med*. 2022;27:101356.

Staikov I, Neykov N, Marinovic B, et al. Herpes zoster as a systemic disease. *Clin Dermatol*. 2014;32(3):424–429S.

Waldman D. Ocular pain. In: *Pain review*. 2nd ed. Philadelphia: Saunders; 2016:211–213.

Yawn BP, Wollan PC, St. Sauver JL, et al. Herpes zoster eye complications: rates and trends. *Mayo Clin Proc*. 2013;88(6):562–570.

2

Migraine Headache

ICD-10 CODE G43.109

THE CLINICAL SYNDROME

Migraine headache is a periodic unilateral headache that may begin in childhood but almost always develops before the age of 30 years. Attacks occur with variable frequency, ranging from every few days to once every several months. More frequent migraine headaches are often associated with a phenomenon called "analgesic rebound." Between 60% and 70% of patients who suffer from migraine are female, and many report a family history of migraine headache. The personality type of migraineurs has been described as meticulous, neat, compulsive, and often rigid. They tend to be obsessive in their daily routines and often find it hard to cope with the stresses of everyday life. Migraine headache may be triggered by changes in sleep patterns or diet or by the ingestion of tyramine-containing foods, monosodium glutamate, nitrates, chocolate, wine, or citrus fruits. Changes in endogenous and exogenous hormones, such as with the use of birth control pills, can also trigger migraine headache as can the ingestion of nitroglycerine for angina. The typical migraine headache is characterized by four phases: (1) the prodrome; (2) the aura; (3) the headache; and (4) the postdrome (Fig. 2.1). Some migraineurs will experience a premonition or warning that a migraine may be on the horizon. This premonition or warning is known as a prodrome and may manifest as mood changes, food cravings, frequent yawning, changes in libido, and constipation. Approximately 20% of patients suffering from migraine headache also experience a neurologic event before the onset of pain called an aura. The aura most often takes the form of a visual disturbance, but it may also manifest as an alteration in smell or hearing; these are called olfactory and auditory auras, respectively. Following a migraine headache, some patients will experience a period of confusion, dizziness, weakness, or elation known as a postdrome.

SIGNS AND SYMPTOMS

Migraine headache is, by definition, a unilateral headache. Although the headache may change sides with each episode, the headache is never bilateral at its onset. The pain of migraine headache is usually periorbital or retro-orbital. It is pounding, and its intensity is severe. The time from the onset to peak of migraine pain is short, ranging from 20 minutes to 1 hour. In contradistinction to tension-type headache, migraine headache is often associated with systemic symptoms, including nausea and vomiting, photophobia, and sonophobia, as well as alterations in appetite, mood, and libido. Menstruation is a common trigger of migraine headache.

As mentioned, in approximately 20% of patients, migraine headache is preceded by an aura (called migraine with aura). The aura is thought to be the result of ischemia of specific regions of the cerebral cortex. A visual aura often occurs 30–60 minutes before the onset of headache pain; this may take the form of blind spots, called scotoma, or a zigzag disruption of the visual field, called fortification spectrum (Fig. 2.2). Occasionally, patients with migraine lose an entire visual field during the aura. Auditory auras usually take the form of hypersensitivity to sound, but other alterations of hearing, such as sounds perceived as farther away than they actually are, have also been reported. Olfactory auras may take the form of strong odors of substances that are not actually present or extreme hypersensitivity to otherwise normal odors, such as coffee or copy machine toner. Migraine that manifests without other neurologic symptoms is called migraine without aura.

Rarely, patients who suffer from migraine experience prolonged neurologic dysfunction associated with headache pain. Such neurologic dysfunction may last for more than 24 hours and is termed migraine with prolonged aura. These patients are at risk for the development of permanent neurologic deficits, and risk factors such as hypertension, smoking, and oral contraceptives must be addressed. Even less common than migraine with prolonged aura is migraine with complex aura. Patients suffering from migraine with complex aura experience significant neurologic dysfunction that may include aphasia or hemiplegia. As with migraine with prolonged aura, patients suffering from migraine with complex aura may develop permanent neurologic deficits.

Patients suffering from all forms of migraine headache appear systemically ill (Fig. 2.3). Pallor, tremulousness, diaphoresis, and light sensitivity are common physical findings. The temporal artery and the surrounding area may be tender. If an aura is present, the results of the neurologic examination will be abnormal; the neurologic examination is usually within normal limits before, during, and after migraine without aura.

TESTING

No specific test exists for migraine headache. Testing is aimed primarily at identifying occult pathologic processes or other

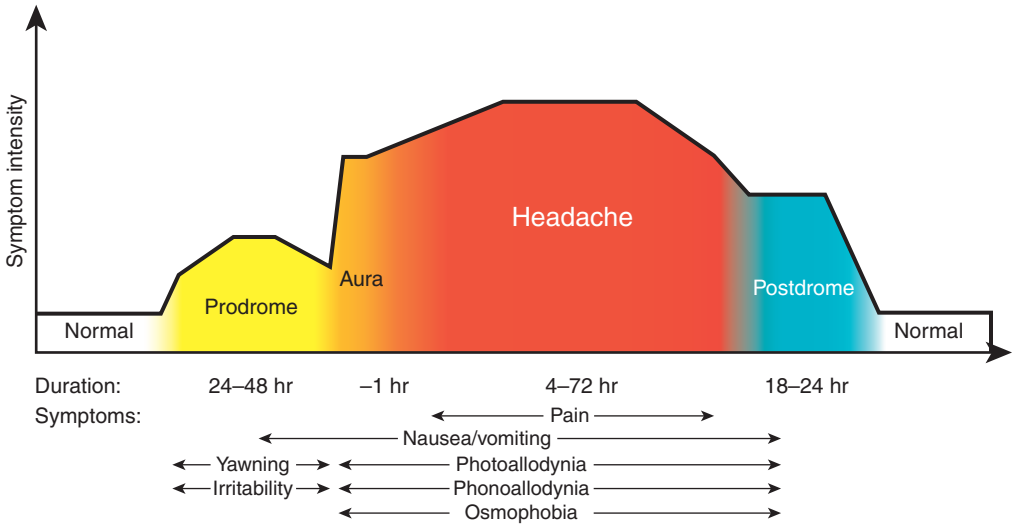

FIG 2.1 The four phases of migraine. (Redrawn from Burgos-Vega C, Moy J, Dussor G. Meningeal afferent signaling and the pathophysiology of migraine. *Prog Mol Biol Transl Sci*. 2015; 131:537–564.)

FIG 2.2 Artist's depiction of zigzag fortification spectrum visual aura. (From Podoll K, Ayles D. Sarah Raphael's migraine with aura as inspiration for the foray of her work into abstraction. *Int Rev Neurobiol*. 2006;74:109–118.)

diseases that may mimic migraine headache (see "Differential Diagnosis"). All patients with a recent onset of headache thought to be migraine should undergo magnetic resonance imaging (MRI) of the brain. If neurologic dysfunction accompanies the patient's headache symptoms, MRI should be performed with and without gadolinium contrast medium (Fig. 2.4); magnetic resonance angiography should be considered as well. MRI should also be performed in patients with previously stable migraine headaches who experience an inexplicable change in symptoms. Screening laboratory tests, including an erythrocyte sedimentation rate, complete blood count, and automated blood chemistry, should be performed if the diagnosis of migraine is in question. Ophthalmologic evaluation is indicated in patients who experience significant ocular symptoms.

FIG 2.3 Migraine headache is an episodic, unilateral headache that occurs most commonly in female patients.

DIFFERENTIAL DIAGNOSIS

The diagnosis of migraine headache is usually made on clinical grounds by obtaining a targeted headache history. Tension-type headache is often confused with migraine headache, and this misdiagnosis can lead to illogical treatment plans because these two headache syndromes are

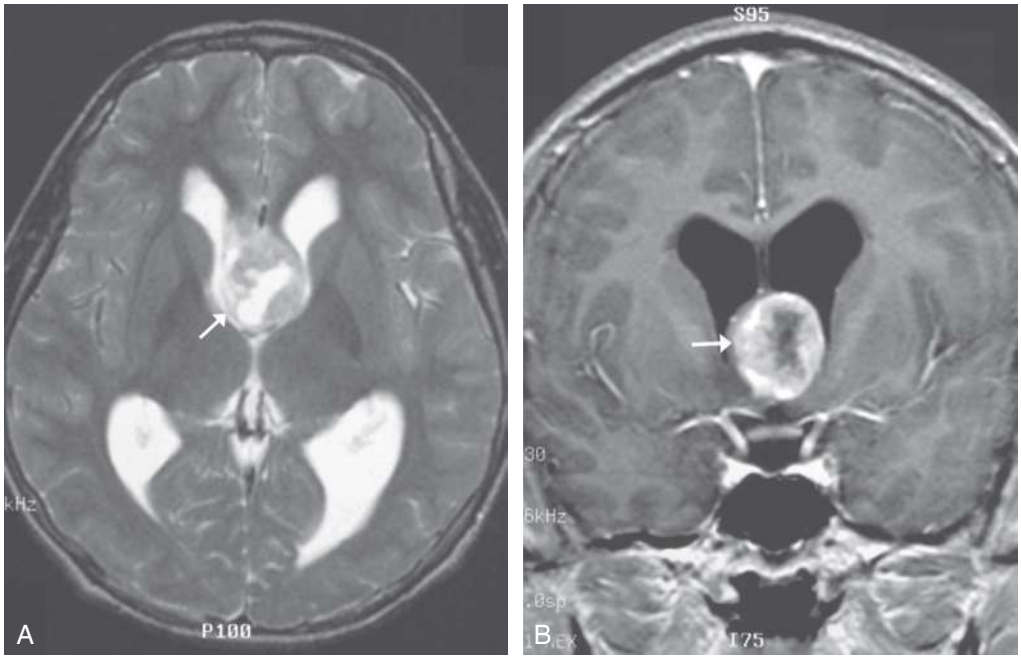

FIG 2.4 Glioblastoma multiforme involving the septum pellucidum. **A,** Axial T2-weighted magnetic resonance imaging (MRI) through the inferior aspect of the frontal horns of the lateral ventricles. An ovoid, heterogeneously hyperintense mass (*arrow*) arising from the inferior aspect of the septum pellucidum indents and partially occludes the frontal horns bilaterally. Note the irregularly marginated intratumoral hyperintensity, suggesting central necrosis. **B,** Following intravenous administration of gadolinium, coronal T1-weighted MRI demonstrates intense contrast enhancement (*arrow*) of the thick peripheral rind, with nonenhancement of the central cavity. (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:140.)

TABLE 2.1 Comparison of Migraine Headache and Tension-type Headache

	Migraine Headache	Tension-type Headache
Onset-to-peak interval	Minutes to 1 hr	Hours to days
Frequency	Rarely >1/wk	Often daily or continuous
Location	Temporal	Nuchal or circumferential
Character	Pounding	Aching, pressure, bandlike
Laterality	Always unilateral	Usually bilateral
Aura	May be present	Never present
Nausea and vomiting	Common	Rare
Duration	Usually <24 hr	Often days

managed quite differently. Table 2.1 distinguishes migraine headache from tension-type headache and should help clarify the diagnosis.

Diseases of the eyes, ears, nose, and sinuses may also mimic migraine headache. The targeted history and physical examination, combined with appropriate testing, should allow the clinician to identify and properly treat any underlying diseases of these organ systems. The following conditions may all mimic migraine and must be considered when treating patients with headache: glaucoma temporal arteritis; sinusitis; intracranial disease, including chronic subdural hematoma, tumor (see Fig. 2.4), brain abscess, hydrocephalus, and pseudotumor cerebri; and inflammatory conditions, including sarcoidosis.

TREATMENT

When deciding how best to treat a patient suffering from migraine, the clinician should consider the frequency and severity of the headaches, their effect on the patient's lifestyle, the presence of focal or prolonged neurologic disturbances, the results of previous testing and treatment, any history of previous drug abuse or misuse, and the presence of other systemic diseases (e.g., peripheral vascular or coronary artery disease) that may preclude the use of certain treatment modalities.

If the patient's migraine headaches occur infrequently, a trial of abortive therapy may be warranted. However, if the headaches occur with greater frequency or cause the patient to miss work or be hospitalized, prophylactic therapy is warranted.

Abortive Therapy

For abortive therapy to be effective, it must be initiated at the first sign of headache. This is often difficult because of the short interval between the onset and peak of migraine headache, coupled with the problem that migraine sufferers often experience nausea and vomiting that may limit the use of oral medications. By altering the route of administration to parenteral or transmucosal, this situation can be avoided.

Abortive medications that can be considered in patients with migraine headache include compounds that contain isometheptene mucate (e.g., Midrin); the nonsteroidal anti-inflammatory drug (NSAID) naproxen; ergot alkaloids; the triptans including sumatriptan, rizatriptan, almotriptan, naratriptan, zolmitriptan, frovatriptan, and eletriptan; and intravenous lidocaine combined with antiemetic compounds. Unlike the triptans, the 5-HT_{1F} receptor agonist lasmiditan has no vasoactive effects but can be sedating. Calcitonin gene-related peptide (CGRP) receptor antagonists including ubrogepant and rimegepant are rapidly becoming the abortive therapy of choice for migraine headache given their efficacy and low side effect profile. The inhalation of 100% oxygen may abort migraine headache, and sphenopalatine ganglion block with local anesthetic may be effective. Caffeine-containing preparations, barbiturates, ergotamines, triptans, and opioids have a propensity to cause a phenomenon called analgesic rebound headache, which may ultimately be more difficult to treat than the original migraine. The ergotamines and triptans should not be used in patients with coexistent peripheral vascular disease, coronary artery disease, or hypertension.

Prophylactic Therapy

For most patients with migraine headache, prophylactic therapy is a better option than abortive therapy. The mainstay of prophylactic therapy is beta-blocking agents. Propranolol, metoprolol, timolol, and most other drugs in this class can control or decrease the frequency and intensity of migraine headache and help prevent auras. An 80-mg daily dose of the long-acting formulation is a reasonable starting point for most patients with migraine. Propranolol should not be used in patients with asthma or other reactive airway diseases.

Valproic acid, calcium channel blockers (e.g., verapamil), clonidine, tricyclic antidepressants, the angiotensin-converting enzyme inhibitor lisinopril, and NSAIDs have also been used for the prophylaxis of migraine headache. Onabotulinum toxin A may also provide migraine prophylaxis in selected patients. The CGRP antagonists including togepant, eptinezumab, erenumab, fremanezumab, and galcanezumab have shown demonstrable utility in the prophylactic treatment of migraine and have favorable side effect profiles. Lasmiditan has also been used to prevent migraine but causes sedation and is a controlled substance. Each of these drugs has advantages and disadvantages, and the clinician should tailor a treatment plan that best meets the needs of the individual patient.

COMPLICATIONS AND PITFALLS

In most patients, migraine headache is a painful but not life-threatening disease. However, patients who suffer from migraine with prolonged aura or migraine with complex aura are at risk for the development of permanent neurologic deficits. Such patients are best treated by headache specialists who are familiar with these unique risks and are better equipped to deal with them. Occasionally, prolonged nausea and vomiting associated with severe migraine headache may result in dehydration that necessitates hospitalization and treatment with intravenous fluids.

CLINICAL PEARLS

The most common reason for a patient's lack of response to traditional treatment for migraine headache is that the patient is actually suffering from tension-type headache, analgesic rebound headache, or a combination of headache syndromes. The clinician must be sure that the patient is not taking significant doses of over-the-counter headache preparations containing caffeine or other vasoactive drugs, such as barbiturates, ergots, or triptans, that may cause analgesic rebound headache. Until these drugs are withdrawn, the patient's headache will not improve.

SUGGESTED READINGS

Aguilar-Shea AL, Membrilla JA, Diaz-de-Teran J. Migraine review for general practice. *Aten Primaria*. 2022;54(2):1–8.

Mascarella D, Matteo E, Pensato U, et al. Chronic migraine with medication overuse headache: is detoxification still necessary in the era of new prophylaxes? *J Neurol Sci*. 2021;429(suppl): 23–24.

Spindler BL, Ryan M. Medications approved for preventing migraine headaches. *Am J Med*. 2020;133(6):664–667.

Waldman SD. Migraine headache. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2016:200–203.

Wilbanks C. Migraine in adult primary care. *J Nurse Pract*. 2022; 18(4):366–369.

Younger DS. Epidemiology of migraine. *Neurol Clin*. 2016;34(4): 849–861.

3

Tension-type Headache

ICD-10 CODE G44.209

THE CLINICAL SYNDROME

Tension-type headache, formerly known as muscle contraction headache, is the most common type of headache that afflicts humankind. It can be episodic or chronic, and it may or may not be related to muscle contraction. Significant sleep disturbance usually occurs. Patients with tension-type headache are often characterized as having multiple unresolved conflicts surrounding work, marriage, and social relationships, and psychosexual difficulties. Testing with the Minnesota Multiphasic Personality Inventory in large groups of patients with tension-type headache revealed not only borderline depression but somatization as well. Most researchers believe that this somatization takes the form of abnormal muscle contraction in some patients; in others, it results in simple headache.

SIGNS AND SYMPTOMS

Tension-type headache is usually bilateral but can be unilateral; it often involves the frontal, temporal, and occipital regions (Fig. 3.1). It may present as a bandlike, nonpulsatile ache or tightness in the aforementioned anatomic areas (Fig. 3.2). Associated neck symptoms are common. Tension-type headache evolves over a period of hours or days and then tends to remain constant, without progression. It has no associated aura, but significant sleep disturbance is usually present. This disturbance may manifest as difficulty falling asleep, frequent awakening at night, or early awakening. These headaches most frequently occur between 4 and 8 AM and 4 and 8 PM. Although both sexes are affected, female patients predominate. No hereditary pattern to tension-type headache is found, but this type of headache may occur in family clusters because children mimic and learn the pain behavior of their parents.

The triggering event for acute, episodic tension-type headache is invariably either physical or psychological stress. This may take the form of a fight with a coworker or spouse or an exceptionally heavy workload. Physical stress, such as a long drive, working with the neck in a strained position, acute cervical spine injury resulting from whiplash, or prolonged exposure to the glare from a cathode ray tube, may precipitate a headache. A worsening of preexisting degenerative cervical spine conditions, such as cervical spondylosis, can also trigger a tension-type headache. The pathologic process responsible for the development of tension-type headache can produce temporomandibular joint dysfunction as well.

TESTING

No specific test exists for tension-type headache. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic tension-type headache (see "Differential Diagnosis"). All patients with a recent onset of headache that is thought to be tension type should undergo magnetic resonance imaging (MRI) of the brain and, if significant occipital or nuchal symptoms are present, of the cervical spine. MRI should also be performed in patients with previously stable tension-type headaches who have experienced a recent change in symptoms. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry should be performed if the diagnosis of tension-type headache is in question.

DIFFERENTIAL DIAGNOSIS

Tension-type headache is usually diagnosed on clinical grounds by obtaining a targeted headache history. Despite their obvious differences, tension-type headache is often incorrectly diagnosed as migraine headache. Such misdiagnosis can lead to illogical treatment plans and poor control of headache symptoms. Table 3.1 helps to distinguish tension-type headache from migraine headache and should aid the clinician in making the correct diagnosis.

Diseases of the cervical spine and surrounding soft tissues may also mimic tension-type headache. Arnold–Chiari malformations may manifest clinically as tension-type headache, but these malformations can easily be identified on images of the posterior fossa and cervical spine (Fig. 3.3). Occasionally, frontal sinusitis is confused with tension-type headache, although individuals with acute frontal sinusitis appear systemically ill. Temporal arteritis, chronic subdural hematoma, and other intracranial diseases such as tumor may be incorrectly diagnosed as tension-type headache.

TREATMENT

Abortive Therapy

In determining the best treatment, the physician must consider the frequency and severity of the headaches, their effect on the patient's lifestyle, the results of any previous

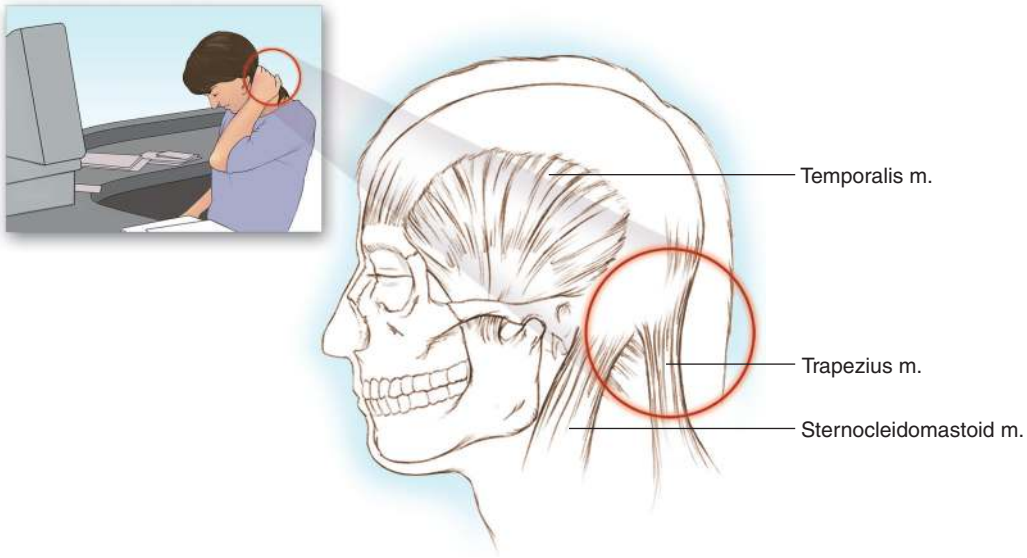

FIG 3.1 Mental or physical stress is often the precipitating factor in tension-type headache.

FIG 3.2 Tension-type headache presents as a bandlike, nonpulsatile ache or tightness in the forehead, temples, neck, and occipital regions. (Redrawn from Kaufman DM. *Kaufman's clinical neurology for psychiatrists*. 7th ed. Philadelphia: Elsevier; 2013:F9-1.)

TABLE 3.1 Comparison of Tension-type Headache and Migraine Headache

	Tension-type Headache	Migraine Headache
Onset-to-peak interval	Hours to days	Minutes to 1 hr
Frequency	Often daily or continuous	Rarely >1/wk
Location	Nuchal or circumferential	Temporal
Character	Aching, pressure, and bandlike	Pounding
Laterality	Usually bilateral	Always unilateral
Aura	Never present	May be present
Nausea and vomiting	Rare	Common
Duration	Often days	Usually <24 hr

therapy, and any prior drug misuse or abuse. If the patient suffers an attack of tension-type headache only once every 1 or 2 months, the condition can often be managed by teaching the patient to reduce or avoid stress. Analgesics or nonsteroidal antiinflammatory drugs (NSAIDs) can provide symptomatic relief during acute attacks. Combination analgesic drugs used concomitantly with barbiturates or opioid analgesics have no place in the treatment of patients with headache. The risk of abuse and dependence more than outweighs any theoretic benefit. The physician should also avoid an abortive treatment approach in patients with a prior history of drug misuse or abuse. Many drugs, including simple analgesics and NSAIDs, can produce serious consequences if they are abused.

Prophylactic Therapy

If the headaches occur more frequently than once every 1 or 2 months or are so severe that the patient repeatedly misses work or social engagements, prophylactic therapy is indicated.

Antidepressants

Antidepressants are generally the drugs of choice for the prophylactic treatment of tension-type headache. These drugs not only help to decrease the frequency and intensity

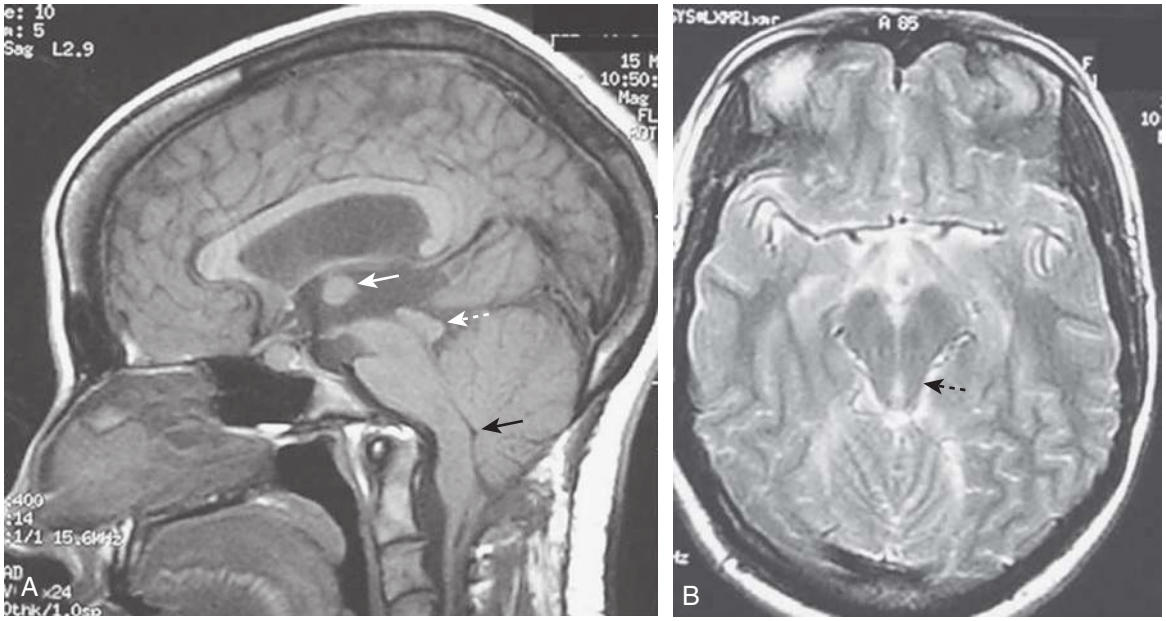

FIG 3.3 A, Sagittal T1-weighted magnetic resonance imaging (MRI) of an adult patient with Arnold–Chiari type II deformity. The posterior fossa is small with a widened foramen magnum. Inferior displacement of the cerebellum and medulla with elongation of the pons and fourth ventricle (*black arrow*) is evident. The brainstem is kinked as it passes over the back of the odontoid. An enlarged massa with intermedia (*white arrow*) and beaking of the tectum (*broken white arrow*) are visible. B, Axial T2-weighted MRI shows the small posterior fossa with beaking of the tectum (*broken black arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011:30.)

of headaches but also normalize sleep patterns and treat any underlying depression. Patients should be educated about the potential side effects of this class of drugs, including sedation, dry mouth, blurred vision, constipation, and urinary retention. Patients should also be told that relief of headache pain generally takes 3–4 weeks. However, normalization of sleep occurs immediately, and this may be enough to provide a noticeable improvement in headache symptoms.

Amitriptyline, started at a single bedtime dose of 25 mg, is a reasonable initial choice. The dose may be increased in 25-mg increments as side effects allow. Other drugs that can be considered if the patient does not tolerate the sedative and anticholinergic effects of amitriptyline include trazodone (75–300 mg at bedtime) or fluoxetine (20–40 mg at lunchtime). Because of the sedating nature of these drugs (with the exception of fluoxetine), they must be used with caution in older patients and in others who are at risk for falling. Care should also be exercised when using these drugs in patients who are susceptible to cardiac arrhythmias, because these drugs may be arrhythmogenic. Simple analgesics or longer acting NSAIDs may be used with antidepressant compounds to treat exacerbations of headache pain.

Biofeedback

Monitored relaxation training combined with patient education about coping strategies and stress reduction techniques may be of value in some tension-type headache sufferers who are adequately motivated. Patient selection is of paramount importance if good results are to be achieved. If the patient is significantly depressed, it may be beneficial to treat the depression before trying biofeedback. The use of biofeedback may allow the patient to control the headaches while avoiding the side effects of medications.

Cervical Epidural Nerve Block

Multiple studies have demonstrated the efficacy of cervical epidural nerve block with steroids in providing long-term relief of tension-type headaches in patients for whom all other treatment modalities have failed. This treatment can also be used while waiting for antidepressant compounds to become effective. Cervical epidural nerve block can be performed on a daily to weekly basis, depending on clinical symptoms.

COMPLICATIONS AND PITFALLS

A few patients with tension-type headache have major depression or uncontrolled anxiety states in addition to a chemical dependence on opioid analgesics, barbiturates, minor tranquilizers, or alcohol. Attempts to treat these patients in the outpatient setting are disappointing and frustrating. Inpatient treatment in a specialized headache unit or psychiatric setting results in more rapid amelioration of the underlying and coexisting problems and allows the concurrent treatment of headache. Monoamine oxidase inhibitors can often reduce the frequency and severity of tension-type headache in this

subset of patients. Phenelzine, at a dosage of 15 mg three times a day, is usually effective. After 2–3 weeks, the dosage is tapered to an appropriate maintenance dose of 5–10 mg three times a day. Monoamine oxidase inhibitors can produce life-threatening hypertensive crises if special diets are not followed or if these drugs are combined with some commonly used prescription or over-the-counter medications. Therefore their use should be limited to highly reliable and compliant patients. Physicians prescribing this potentially dangerous group of drugs should be well versed in how to use them safely.

CLINICAL PEARLS

Although tension-type (muscle contraction) headache occurs frequently, it is commonly misdiagnosed as migraine headache. By obtaining a targeted headache history and performing a targeted physical examination, the physician can make a diagnosis with a high degree of certainty. The avoidance of addicting medications, coupled with the appropriate use of pharmacologic and nonpharmacologic therapies, should result in excellent palliation and long-term control of pain in most patients suffering from this headache syndrome.

SUGGESTED READINGS

Faraji F. Tension-type headache. In: Togha M, Jafari E, Mohammadianinejad SE, et al., eds. *Headache and migraine in practice*. New York: Academic Press; 2022:chap 3, 75–83.

Jafari E. Headache in women. In: Togha M, Jafari E, Mohammadianinejad SE, et al., eds. *Headache and migraine in practice*. New York: Academic Press; 2022:chap 10, 265–300.

Mohammadianinejad SE, Ansari B. Medication overuse headache. In: Togha M, Jafari E, Mohammadianinejad SE, et al., eds. *Headache and migraine in practice*. New York: Academic Press; 2022:chap 6(e), 161–168.

Rangel TC, Raposo MCF, Rocha-Filho PAS. The prevalence and severity of insomnia in university students and their associations with migraine, tension-type headache, anxiety and depression disorders: a cross-sectional study. *Sleep Med*. 2021;88:241–246.

Waldman SD. Targeted headache history. *Med Clin North Am*. 2013;97(2):185–195.

Waldman SD. Cervical epidural block: translaminar approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:188–192.

Waldman SD. Tension-type headache. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:198–200.

4

Cluster Headache

ICD-10 CODE G44.009

THE CLINICAL SYNDROME

Cluster headache derives its name from the headache pattern—that is, headaches occur in clusters, followed by headache-free remission periods. Cluster headache is a primary headache that is included in the group of headaches known as the trigeminal autonomic cephalgias. Unlike other common headache disorders that affect primarily female patients, cluster headache is much more common in male patients, with a male-to-female ratio of 5 : 1. Much less common than tension-type headache or migraine headache, cluster headache is thought to affect approximately 0.5% of the male population. Cluster headache is most often confused with migraine by clinicians who are unfamiliar with the syndrome; however, a targeted headache history allows the clinician to distinguish between these two distinct headache types easily (Table 4.1).

The onset of cluster headache occurs in the late third or early fourth decade of life, in contradistinction to migraine, which almost always manifests by the early second decade. Unlike migraine, cluster headache does not appear to run in families, and cluster headache sufferers do not experience auras. Attacks generally occur approximately 90 minutes after the patient falls asleep. This association with sleep is reportedly maintained when a shift worker changes from nighttime to daytime hours of sleep. Cluster headache also appears to follow a distinct chronobiologic pattern that coincides with seasonal changes in the length of the day. This pattern results in an increased frequency of cluster headache in the spring and fall.

During a cluster period, attacks occur two or three times a day and last for 45 minutes to 1 hour. Cluster periods usually last for 8–12 weeks, interrupted by remission periods of less than 2 years. In rare patients, the remission periods become shorter and shorter, and the frequency may increase up to 10-fold. This situation is termed chronic cluster headache and differs from the more common episodic cluster headache described earlier.

SIGNS AND SYMPTOMS

Cluster headache is characterized as a unilateral headache that is retro-orbital and temporal in location. The pain has a deep burning or boring quality. Physical findings during an attack of cluster headache may include Horner's syndrome, consisting of ptosis, abnormal pupil constriction, facial flushing, and conjunctival injection (Fig. 4.1). Additionally, profuse lacrimation and rhinorrhea are often present. The ocular changes may become permanent with repeated attacks. Peau d'orange skin over the malar region, deeply furrowed glabellar folds, and telangiectasia may be observed.

Attacks of cluster headache may be provoked by small amounts of alcohol, nitrates, histamines, and other vasoactive substances, as well as occasionally by high altitude. When the attack is in progress, the patient may be unable to lie still and may pace or rock back and forth in a chair. This behavior contrasts with that characterizing other headache syndromes, during which patients seek relief by lying down in a dark, quiet room.

The pain of cluster headache is said to be among the worst pain a human being can suffer. Because of the severity of the pain, the clinician must watch closely for medication overuse or misuse. Suicide has been associated with prolonged, unrelieved attacks of cluster headache.

TESTING

No specific test exists for cluster headache. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic cluster headache (see "Differential Diagnosis"). All patients with a recent onset of headache thought to be cluster headache should undergo magnetic resonance imaging (MRI) of the brain. If neurologic dysfunction accompanies the patient's headache symptoms, MRI should be performed with and without gadolinium contrast medium (Fig. 4.2); magnetic resonance angiography should be considered as well. MRI should also be performed in patients with previously stable cluster headache who experience an inexplicable change in symptoms. Screening laboratory tests, including an erythrocyte sedimentation rate, complete blood count, and automated blood chemistry, should be performed if the diagnosis of cluster headache is in question. Ophthalmologic evaluation, including measurement of intraocular pressures, is indicated in patients who experience significant ocular symptoms.

DIFFERENTIAL DIAGNOSIS

Cluster headache is usually diagnosed on clinical grounds by obtaining a targeted headache history. Migraine headache is often confused with cluster headache, and this misdiagnosis can lead to illogical treatment plans because the management of these two headache syndromes is quite different. Table 4.1

TABLE 4.1 Comparison of Cluster Headache and Migraine Headache

	Cluster Headache	Migraine Headache
Gender	Male 5:1	Female 2:1
Age of onset	Late 30s to early 40s	Menarche to early 20s
Family history	No	Yes
Aura	Never	May be present (20% of the time)
Chronobiologic pattern	Yes	No
Onset-to-peak interval	Seconds to minutes	Minutes to 1 hr
Frequency	2 or 3/day	Rarely >1/wk
Duration	45 min	Usually <24 hr

FIG 4.1 Horner's eye findings. Classic clinical eye findings are demonstrated in this patient with a right Horner syndrome (ptosis of the upper eyelid, elevation of the lower eyelid, and miosis). (From Reede DL, Garcon E, Smoker WR, et al. Horner's syndrome: clinical and radiographic evaluation. *Neuroimaging Clin N Am.* 2008;18(2):369–385.)

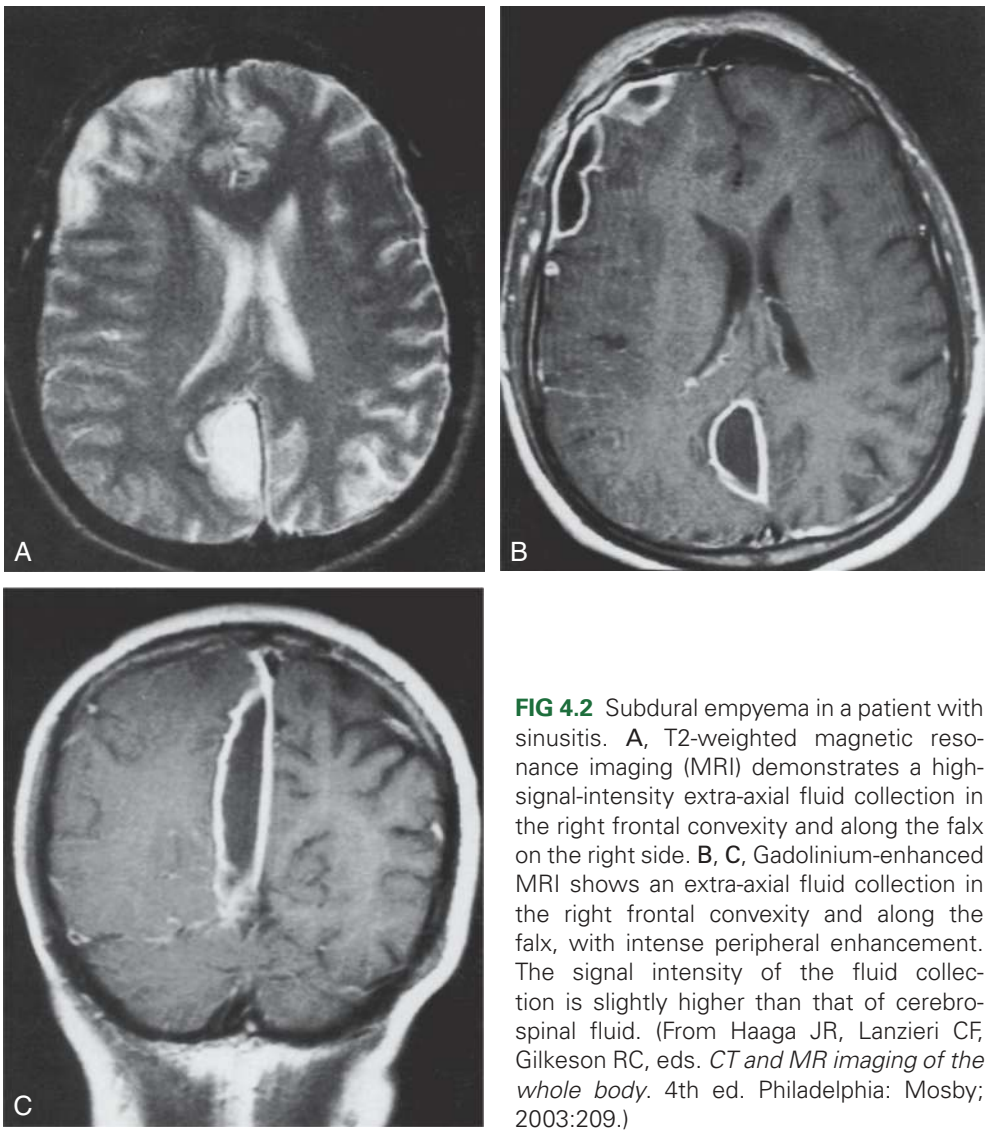

FIG 4.2 Subdural empyema in a patient with sinusitis. **A**, T2-weighted magnetic resonance imaging (MRI) demonstrates a high-signal-intensity extra-axial fluid collection in the right frontal convexity and along the falx on the right side. **B**, **C**, Gadolinium-enhanced MRI shows an extra-axial fluid collection in the right frontal convexity and along the falx, with intense peripheral enhancement. The signal intensity of the fluid collection is slightly higher than that of cerebrospinal fluid. (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:209.)

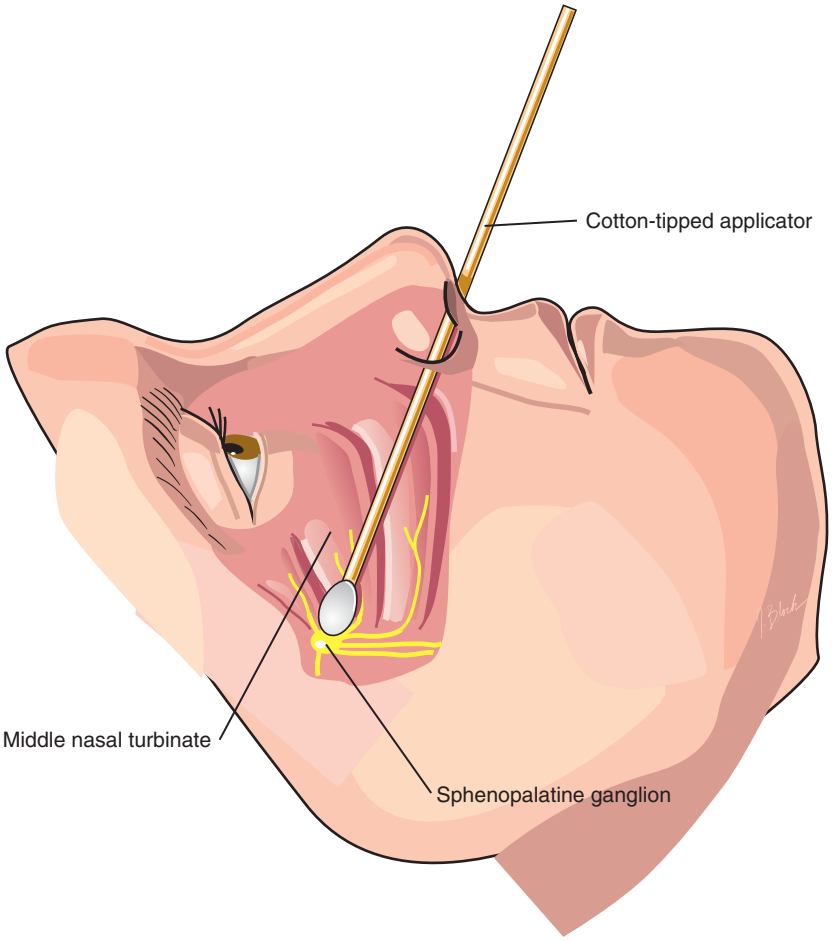

FIG 4.3 Sphenopalatine ganglion block is a useful treatment in the management of cluster headache. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2015.)

distinguishes cluster headache from migraine headache and should help clarify the diagnosis.

Diseases of the eyes, ears, nose, and sinuses may also mimic cluster headache. The targeted history and physical examination, combined with appropriate testing, should help an astute clinician identify and properly treat any underlying diseases of these organ systems. The following conditions may all mimic cluster headache and must be considered in patients with headache: glaucoma; temporal arteritis; sinusitis (see Fig. 4.2); intracranial disease, including chronic subdural hematoma, tumor, brain abscess, hydrocephalus, and pseudotumor cerebri; and inflammatory conditions, including sarcoidosis.

TREATMENT

Whereas most patients with migraine headache experience improvement with beta-blocker therapy, patients suffering from cluster headache usually require more individualized therapy. Initial treatment is commonly prednisone combined with daily sphenopalatine ganglion blocks with local anesthetic (Fig. 4.3). A reasonable starting dose of prednisone is 80 mg given in divided doses and tapered by 10 mg/dose per day. If headaches are not rapidly brought under control, inhalation of 100% oxygen through a close-fitting mask is added. Octreotide, a synthetic form of somatostatin, may also be useful in aborting acute attacks of cluster headache (Fig. 4.4).

If headaches persist and the diagnosis of cluster headache is not in question, a trial of lithium carbonate may be considered. The therapeutic window of lithium carbonate is small, however, and this drug should be used with caution. A starting dose of 300 mg at bedtime may be increased after 48 hours to 300 mg twice a day. If no side effects are noted after 48 hours, the dose may be increased again to 300 mg three times a day. The patient should stay at this dosage for a total of 10 days, after which the drug should be tapered over a 1-week period. Other medications that can be considered if these treatments are ineffective include methysergide and sumatriptan and sumatriptan-like drugs as well as octreotide and the calcitonin gene-related peptide blocker galcanezumab.

In rare patients, the aforementioned treatments are ineffective. In this setting, given the severity of the pain of cluster headache and the risk of suicide, more aggressive treatment is indicated. Destruction of the gasserian ganglion either by

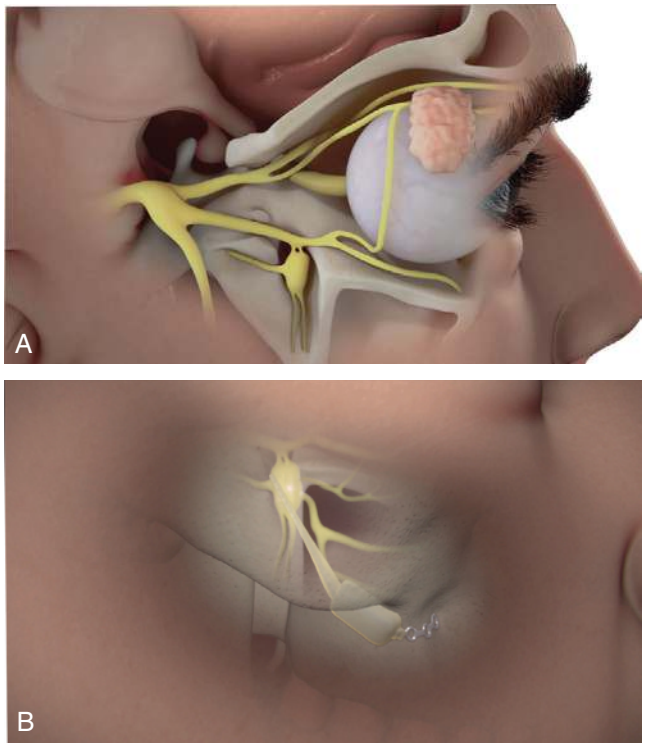

FIG 4.4 A, Anatomy relevant to implantation of the sphenopalatine ganglion stimulation system. B, Proper positioning for sphenopalatine ganglion stimulation electrode. (From Goadsby PJ, Sahai-Srivastava S, Kezirian EJ, et al. Safety and efficacy of sphenopalatine ganglion stimulation for chronic cluster headache: a double-blind, randomised controlled trial. *Lancet Neurol.* 2019;18(12):1081–1090.)

injection of glycerol or by radiofrequency lesioning may be a reasonable next step. Case studies suggest that deep brain stimulation, vagus nerve stimulation, occipital nerve stimulation, sphenopalatine ganglion stimulation, and surgical section of the petrosal nerves may play a role in the treatment of intractable cluster headache.

COMPLICATIONS AND PITFALLS

The major risk in patients suffering from uncontrolled cluster headache is that they may become despondent owing to the unremitting, severe pain and commit suicide. Therefore, if the clinician has difficulty controlling the patient's pain, hospitalization should be considered.

CLINICAL PEARLS

Cluster headache represents one of the most painful conditions encountered in clinical practice and must be viewed as a true pain emergency. In general, cluster headache is more difficult to treat than migraine headache and requires more individualized therapy. Given the severity of the pain associated with cluster headache, multiple modalities should be used early in the course of an episode of cluster headache. The clinician should beware of patients presenting with a classic history of cluster headache who request opioid analgesics.

SUGGESTED READINGS

Belvis R, Rodríguez R, Guasch M, et al. Efficacy and safety of surgical treatment of cluster headache. *Med Clín (English Edition)*. 2020;154(3):75–79.

Brandt RB, Haan J, Terwindt GM, Fronczek R. Cluster headache and pain: features and treatments. In: Rajendram R, Patel VB, Preedy VR, Martin CR, eds. *Features and assessments of pain, anaesthesia, and analgesia*. New York: Academic Press; 2022:chap 9, 93–104.

Burish MJ, Rozen TD. Trigeminal autonomic cephalalgias. *Neurol Clin*. 2019;37(4):847–869.

Fontaine D, Lanteri-Minet M. Occipital nerve stimulation for chronic cluster headache. *Lancet Neurol*. 2021;20(7):498–499.

Goadsby PJ, Sahai-Srivastava S, Kezirian EJ, et al. Safety and efficacy of sphenopalatine ganglion stimulation for chronic cluster headache: a double-blind, randomised controlled trial. *Lancet Neurol*. 2019;18(1):1081–1090.

Kasimu M, Paerhati H, Lei J, et al. Surgical treatment for the cluster headache: clinical experience. *World Neurosurg*. 2021;149:e1134–e1139.

Prasad S, Mehadi A, Kaka N, et al. Diagnostic protocols and newer treatment modalities for cluster headache. *Dis Mon*. 2022;101316.

Vyas DB, Ho AL, Dadey DY, et al. Deep brain stimulation for chronic cluster headache: a review. *Neuromodulation*. 2019;22(4):388–397.

Waldman SD. Cluster headache. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:208–209.

Waldman SD. Sphenopalatine ganglion block: transnasal approach. In: *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2021:13–16.

5

Swimmer's Headache

ICD-10 CODE G50.8

THE CLINICAL SYNDROME

Swimmer's headache is seen with increasing frequency owing to the growing number of people who are swimming as part of a balanced program of physical fitness. Although an individual suffering from swimmer's headache most often complains of a unilateral frontal headache that occurs shortly after he or she begins to swim, this painful condition is more correctly characterized as a compressive mononeuropathy. Swim goggles that are either too large or too tight compress the supraorbital nerve as it exits the supraorbital foramen and cause swimmer's headache (Fig. 5.1). The onset of symptoms is insidious in most patients, usually after the patient has been swimming for a while and is caused by prolonged compression of the supraorbital nerve. Several reported cases of acute-onset swimmer's headache have a common history of the patient's suddenly tightening one side of the goggles after experiencing a leak during his or her swim. In most cases, symptoms abate after the use of the offending goggles is discontinued. However, with chronic compression of the supraorbital nerve, permanent nerve damage may result.

SIGNS AND SYMPTOMS

Swimmer's headache is usually unilateral and involves the skin and scalp subserved by the supraorbital nerve (Fig. 5.2). Swimmer's headache usually manifests as cutaneous sensitivity above the affected supraorbital nerve that radiates into the ipsilateral forehead and scalp. This sensitivity may progress to unpleasant dysesthesias and allodynia, and the patient often complains that his or her hair hurts. With prolonged compression of the supraorbital nerve, a "woody" or anesthetized feeling of the supraorbital region and forehead may occur. Physical examination may reveal allodynia in the distribution of the compressed supraorbital nerve or, rarely, anesthesia. An occasional patient may present with edema of the eyelid resulting from compression of the soft tissues by the tight goggles. Rarely, purpura may be present, secondary to damage to the fragile blood vessels in the loose areolar tissue of the eyelid.

TESTING

No specific test exists for swimmer's headache. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic swimmer's headache (see "Differential Diagnosis"). All patients with the recent onset of headache thought to be swimmer's headache should undergo magnetic resonance imaging (MRI) of the brain, and strong consideration should be given to obtaining computed tomography (CT) scanning of the sinuses, with special attention to the frontal sinuses, given the frequency of sinusitis in swimmers. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry should be performed if the diagnosis of swimmer's headache is in question.

DIFFERENTIAL DIAGNOSIS

Swimmer's headache is usually diagnosed on clinical grounds by obtaining a targeted headache history. Despite their obvious differences, swimmer's headache is often misdiagnosed as migraine headache. Such misdiagnosis leads to illogical treatment plans and poor control of headache symptoms. Table 5.1 distinguishes swimmer's headache from migraine headache and should aid the clinician in making the correct diagnosis.

As mentioned earlier, diseases of the frontal sinuses may mimic swimmer's headache and can be differentiated with MRI and CT scanning. Rarely, temporal arteritis may be confused with swimmer's headache, although individuals with temporal arteritis appear systemically ill. Intracranial disease such as tumor may also be incorrectly diagnosed as swimmer's headache (Fig. 5.3).

TREATMENT

The mainstay of treatment of swimmer's headache is removal of the offending goggles. The patient should be instructed to use care when putting on his or her goggles and to avoid placing them directly over the supraobrbital nerve. Often, simply substituting a new pair of goggles made of softer rubber does the trick, but occasionally, custom-fitted goggles with a smaller area of seal around the eyes that do not compress the supraorbital nerve but are large enough to avoid compressing the globe may be required. Analgesics or nonsteroidal antiinflammatory drugs can provide symptomatic relief. However, even these drugs can lead to serious consequences if they are abused.

If the symptoms persist after the removal of the offending goggles, gabapentin may be considered. Baseline blood tests should be obtained before starting therapy with 300 mg of gabapentin at bedtime for two nights. The patient should be

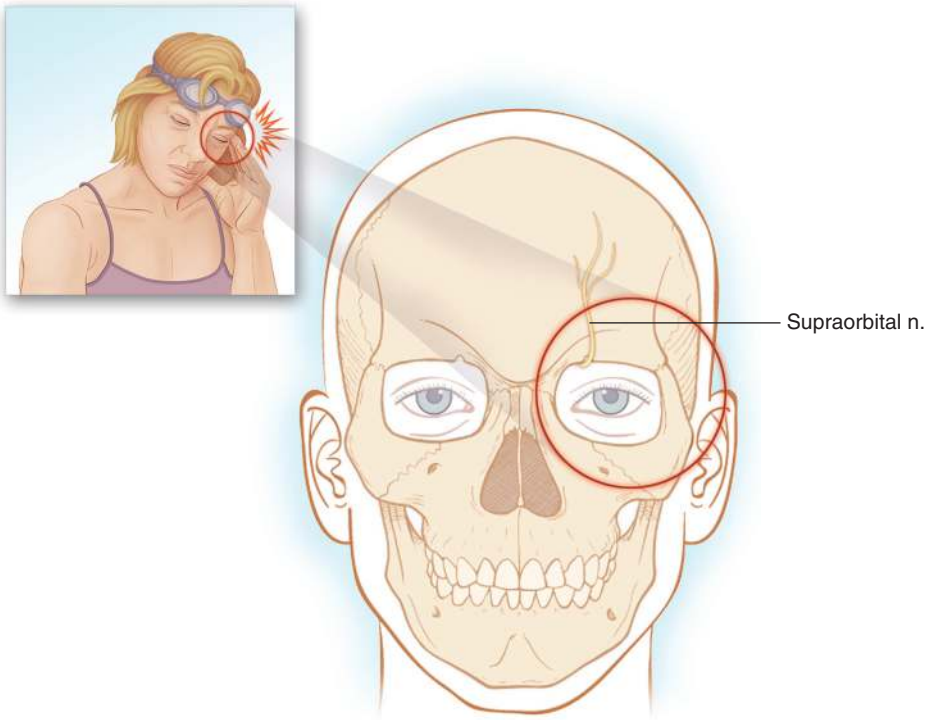

FIG 5.1 Swim goggles that are too tight can compress the supraorbital nerve and cause swimmer's headache.

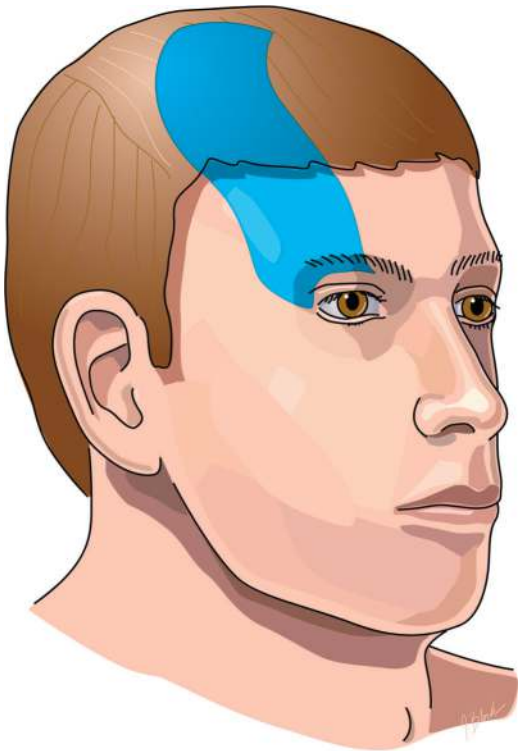

FIG 5.2 Sensory distribution of the supraorbital nerve. (From Waldman SD. *Atlas of interventional pain management*. 2nd ed. Philadelphia: Saunders; 2004:40.)

TABLE 5.1 Comparison of Swimmer's Headache and Migraine Headache

	Swimmer's Headache	Migraine Headache
Onset-to-peak interval	Minutes	Minutes to 1 hr
Frequency	With swimming	Rarely >1/wk
Localization	Supraorbital radiating into the ipsilateral forehead and scalp	Temporal
Character	Cutaneous and scalp sensitivity progressing to painful dysesthesias and numbness	Pounding
Laterality	Usually unilateral	Always unilateral
Aura	Never present	May be present
Nausea and vomiting	Rare	Common
Duration	Usually subsides with removal of goggles, but may become chronic	Usually <24 hr

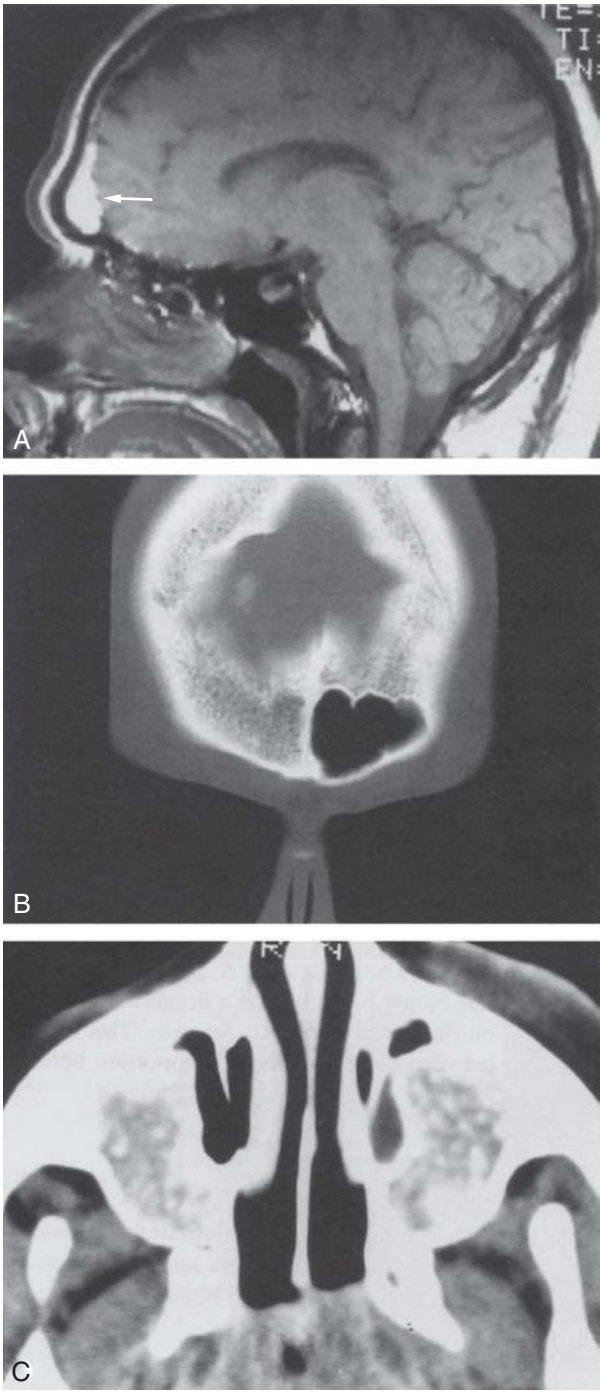

FIG 5.3 Intracranial disease that may mimic swimmer's headache. **A,** Sagittal T1-weighted (TR 500, TE 32) magnetic resonance image in the midline. Increased signal is seen overlying the frontal sinus (*arrow*). This may represent fat, hemorrhage, or a paramagnetic substance in a metastatic tumor such as melanoma. **B,** Accompanying coronal computed tomography (CT) scan shows a nonpneumatized and nondeveloped right frontal sinus. The marrow signal from this right frontal sinus was thought to produce the abnormal signal in the study in **A. C,** Noncontrast-enhanced axial CT scan through the maxillary sinuses in a patient with sickle cell disease. The speckled pattern overlying the maxillary sinuses proved to be hyperactive marrow. (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:565.)

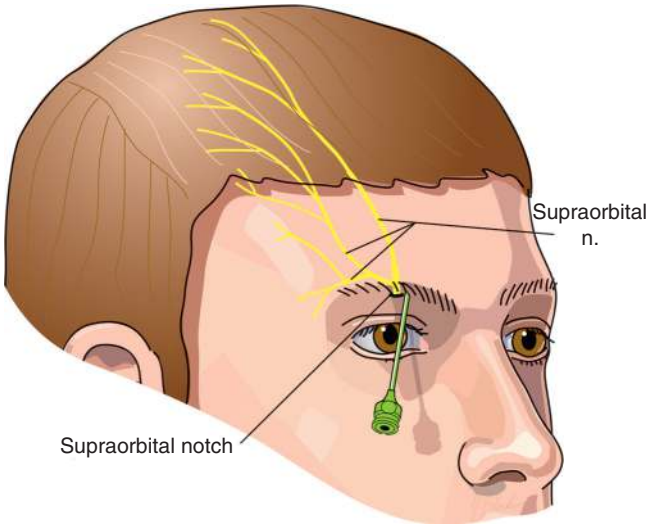

FIG 5.4 Correct needle placement for supraorbital nerve block. (From Waldman SD. *Atlas of interventional pain management*. 2nd ed. Philadelphia: Saunders; 2004:40.)

cautioned about potential side effects, including dizziness, sedation, confusion, and rash. The drug is then increased, as side effects allow, in 300-mg increments given in equally divided doses over 2 days, until pain relief is obtained or a total dose of 2400 mg/day is reached. At this point, if the patient has experienced partial pain relief, blood values are measured and the drug is carefully titrated upward using 100-mg tablets. Rarely is more than 3600 mg/day required. If significant sleep disturbance is present, amitriptyline at an initial bedtime dose of 25 mg and titrated upward, as side effects allow, may be beneficial.

In rare patients with persistent symptoms, supraorbital nerve block with local anesthetic and steroid may be a reasonable next step. To perform supraorbital nerve block, the patient is placed supine with the head in the neutral position. The skin is prepared with povidone-iodine solution, with care taken to avoid spilling solution into the eye. The supraorbital notch is identified by palpation. A 1½-inch, 25-gauge needle is advanced perpendicularly to the skin at the level of the supraorbital notch. Then, 3–4 mL of preservative-free local anesthetic and 40 mg of depot methylprednisolone are injected in a fan configuration to anesthetize the peripheral branches of the nerve (Fig. 5.4). To block the supratrochlear nerve, the needle is directed medially from the supraorbital notch toward the apex of the nose. Paresthesias are occasionally elicited.

COMPLICATIONS AND PITFALLS

In most cases, swimmer's headache is a painful but self-limited condition that is easily managed once it is diagnosed. Failure to remove the offending goggles promptly may result in permanent nerve damage with associated dysesthesias and numbness. Failure to recognize coexistent intracranial disease or systemic diseases such as frontal sinusitis or tumor can have disastrous results.

CLINICAL PEARLS

Although swimmer's headache is occurring with greater frequency owing to the increased interest in physical fitness, it is often misdiagnosed as sinus headache or occasionally migraine. By obtaining a targeted headache history and performing a targeted physical examination, the physician can make a diagnosis with a high degree of certainty. Avoidance of potentially addictive medications, coupled with the appropriate use of pharmacologic and nonpharmacologic therapies, should result in excellent palliation and long-term control of pain in most patients suffering from this headache syndrome.

SUGGESTED READINGS

Bandary DE, Lustofin SV, McLaren JR, Dillon JK. Surgical management of post-traumatic trigeminal neuralgia—case report and review of the literature. *J Maxillofac Surg*. 2022;80(2):214–222.

Krymchantowski AV. Headaches due to external compression. *Curr Pain Headache Rep*. 2010;14(4):321–324.

Pruksapong C, Kawichai W, Attainsee A, Sawani A. The anatomical variations of the emergence routes of supraorbital nerve: a cadaveric study and systematic review. *Asian J Surg*. 2022;45(1):220–225.

Waldman SD. Supraorbital nerve block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:76–78.

6

Analgesic Rebound Headache

ICD-10 CODE G44.10

THE CLINICAL SYNDROME

Analgesic rebound headache, which is also known as analgesic overuse headache, is a recently identified headache syndrome that occurs in headache sufferers who overuse abortive medications to treat their symptoms. The overuse of these medications results in increasingly frequent headaches that become unresponsive to both abortive and prophylactic medications. Over a period of weeks, the patient's episodic migraine or tension-type headache becomes more frequent and transforms into a chronic daily headache. This daily headache becomes increasingly unresponsive to analgesics and other medications, and the patient notes an exacerbation of headache symptoms if abortive or prophylactic analgesic medications are missed or delayed (Fig. 6.1). Although the exact underlying pathophysiology responsible for the evolution of analgesic rebound headache has not been fully elucidated, it has been postulated that dysfunction of the trigeminal modulating system and central sensitization may play a role. Analgesic rebound headache is probably underdiagnosed by healthcare professionals, and its frequency is on the rise owing to the heavy advertising of over-the-counter headache medications containing caffeine.

SIGNS AND SYMPTOMS

Clinically, analgesic rebound headache manifests as a transformed migraine or tension-type headache and may assume the characteristics of both these common headache types, thus blurring their distinctive features and making diagnosis difficult. Common to all analgesic rebound headaches is the excessive use of any of the following medications: simple analgesics, such as acetaminophen; sinus medications, including simple analgesics; combinations of aspirin, caffeine, and butalbital (Fiorinal); nonsteroidal antiinflammatory drugs; opioid analgesics; ergotamines; and triptans, such as sumatriptan (Box 6.1). As with migraine and tension-type headache, the physical examination is usually within normal limits, although psychiatric comorbidities may be more prevalent in this group of headache sufferers.

TESTING

No specific test exists for analgesic rebound headache. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic tension-type or migraine headaches (see "Differential Diagnosis"). All patients with the recent onset of chronic daily headaches thought to be analgesic rebound headaches should undergo magnetic resonance imaging (MRI) of the brain and, if significant occipital or nuchal symptoms are present, of the cervical spine. MRI should also be performed in patients with previously stable tension-type or migraine headaches who have experienced a recent change in headache symptoms. Recent research has suggested that MRI diffusion tensor imaging may be useful in the diagnosis of patients suffering with medication overuse headache with migraine as the primary underlying headache. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry should be performed if the diagnosis of analgesic rebound headache is in question.

DIFFERENTIAL DIAGNOSIS

Analgesic rebound headache is usually diagnosed on clinical grounds by obtaining a targeted headache history. Because analgesic rebound headache assumes many of the characteristics of the underlying primary headache, diagnosis can be confusing in the absence of a careful medication history, including specific questions regarding over-the-counter headache medications and analgesics. Any change in a previously stable headache pattern needs to be taken seriously and should not automatically be attributed to analgesic overuse without a careful reevaluation of the patient.

TREATMENT

Treatment of analgesic rebound headache consists of discontinuation of the overused or abused drugs and then complete abstention of the overused or abused drug or drugs for at least 3 months. The addition of an appropriate prophylactic headache treatment (e.g., propranolol, botulinum toxin, or calcitonin gene-related peptide antibodies for the migraineur) may further decrease the incidence of headaches in patients suffering from analgesic rebound headache (Fig. 6.2). Nonpharmacologic approaches including avoiding triggering factors, cognitive-behavioral therapy, and biofeedback may serve an adjunctive role in the treatment of analgesic rebound headache.

Care should be taken to avoid abrupt discontinuation of medications such as barbiturates and/or opioids because significant side effects, including seizures and acute

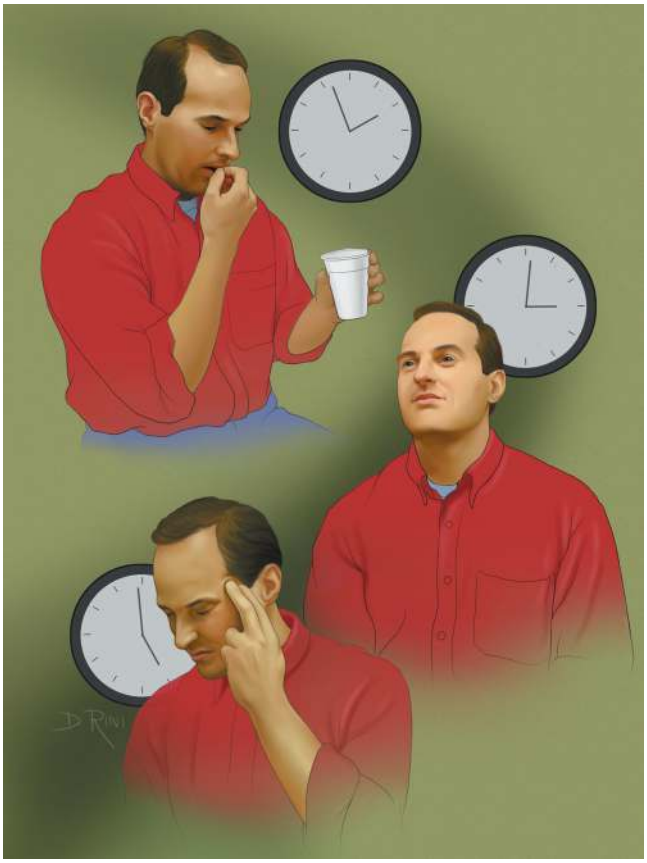

FIG 6.1 Classic temporal relationship between the taking of abortive medications and the onset of analgesic rebound headache.

abstinence syndrome, may occur. In this setting, tapering of the offending medication is necessary and may require hospitalization (Fig. 6.3). Many patients cannot tolerate outpatient discontinuation of these medications and ultimately require hospitalization in a specialized headache unit. If outpatient treatment is being considered, the following points should be carefully explained to the patient:

- The headaches and associated symptoms will get worse before they get better.
- Any use, no matter how small, of the offending medications will result in continued analgesic rebound headaches.
- The patient cannot self-medicate with over-the-counter drugs.
- The significant overuse of opioids or combination medications containing butalbital or ergotamine can result in physical dependence, and discontinuation of such drugs must be done under the supervision of a physician familiar with the treatment of physical dependencies.
- If the patient follows the physician's orders regarding discontinuation of the offending medications, he or she can expect the headaches to improve.

BOX 6.1 Drugs Implicated in Analgesic Rebound Headache

Simple analgesics
Nonsteroidal antiinflammatory drugs
Opioid analgesics
Sinus medications
Ergotamines
Combination headache medications that include butalbital
Triptans (e.g., sumatriptan)

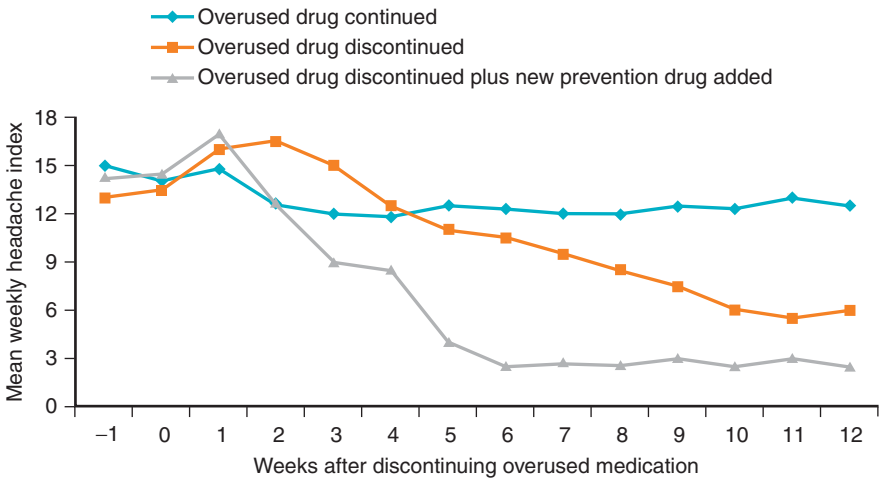

FIG 6.2 Treatment of analgesic rebound headache consists of discontinuation of the overused or abused drugs and complete abstention of the overused or abused drug or drugs for at least 3 months. The addition of an appropriate prophylactic headache treatment (e.g., propranolol for the migraineur) may further decrease the incidence of headaches in patients suffering from analgesic rebound headache. (Redrawn from Mathew NT, Kurman R, Perez F. Drug induced refractory headache—clinical features and management. *Headache*. 1990;30(10):634–638, Fig. 1.)

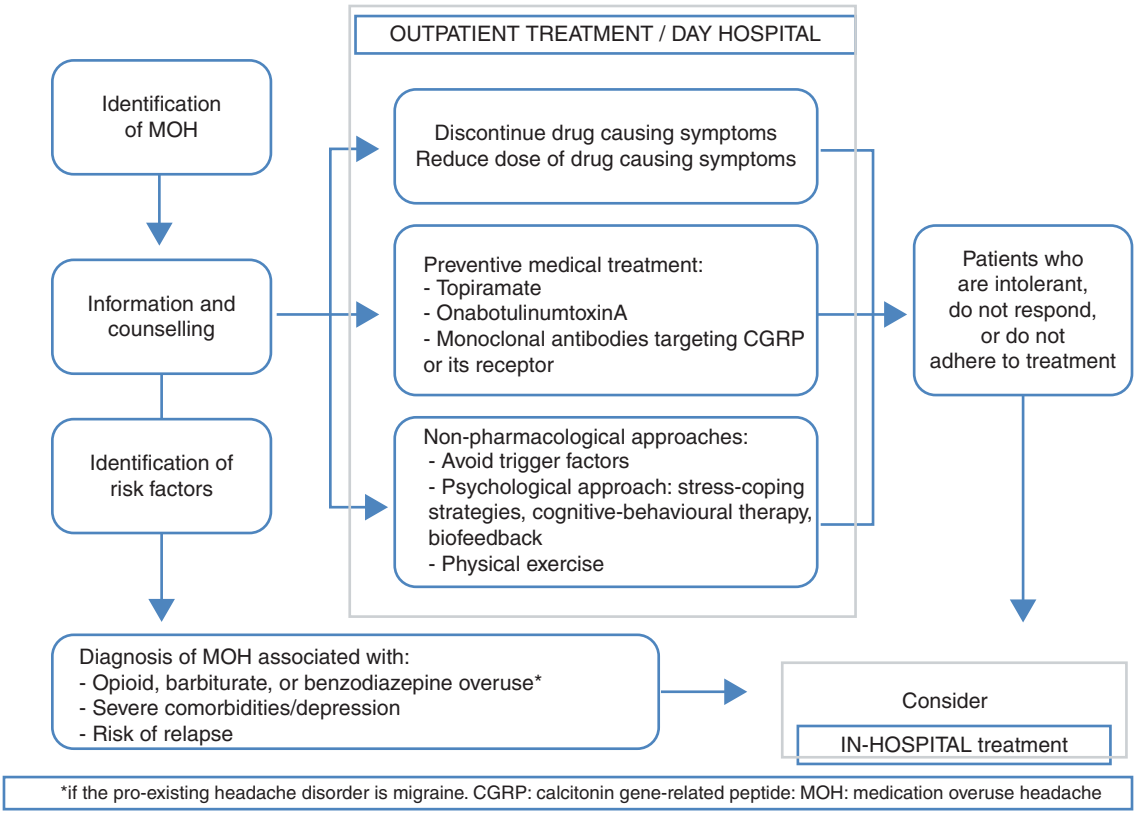

FIG 6.3 Management of medication overuse (analgesic rebound) headache. (From González-Oria, Belvís R, Cuadrado ML, et al. Document of revision and updating of medication overuse headache (MOH). *Neurología (English Edition)*. 2021;36(3):229–240, ISSN 2173-5808, https://doi.org/10.1016/j.nrleng.2020.04.021. (https://www.sciencedirect.com/science/article/pii/S2173580821000249.)

COMPLICATIONS AND PITFALLS

Patients who overuse or abuse medications, including opioids, ergotamines, and butalbital, develop a physical dependence on these drugs, and their abrupt cessation results in a drug abstinence syndrome that can be life threatening if it is not properly treated. Therefore most of these patients require inpatient tapering in a controlled setting.

CLINICAL PEARLS

Analgesic rebound headache occurs much more commonly than was previously thought. The occurrence of analgesic rebound headache is a direct result of the overprescribing of abortive headache medications in patients for whom they are inappropriate. When in doubt, the clinician should avoid abortive medications altogether and treat most headache sufferers prophylactically.

SUGGESTED READINGS

Abrams BM. Medication overuse headaches. *Med Clin North Am*. 2013;97(2):337–352.

Calabresi P, Cupini LM. Medication-overuse headache: similarities with drug addiction. *Trends Pharmacol Sci*. 2005;26(2):62–68.

Corand V, Moisset X, Radat F, Lucas C. Medication overuse headache: updating of the French recommendations regarding the treatment strategies. *Rev Neurol*. 2021;177(7):760–764.

González-Oria C, Belvís R, Cuadrado ML, et al. Document of revision and updating of medication overuse headache (MOH). *Neurología (English Edition)*. 2021;36(3):229–240.

Michultka DM, Blanchard EB, Appelbaum KA, et al. The refractory headache patient. II. High medication consumption (analgesic rebound) headache. *Behav Res Ther*. 1989;27(4):411–420.

Ready DM. Too much of a good thing: medication overuse headache. In: Diamond S, ed. *Headache and migraine biology and management*. San Diego: Academic Press; 2015:253–266.

Rojo E, Pedraza MI, Muñoz I, et al. Chronic migraine with and without medication overuse: experience in a hospital series of 434 patients. *Neurología (English Edition)*. 2015;30(3):153–157.

Russell MB. Epidemiology and management of medication-overuse headache in the general population. *Neurol Sci*. 2019;40(suppl 1):S23–S26.

Waldman SD. Analgesic rebound headache. In: *Pain review*. Philadelphia: Elsevier; 2017:219–220.

Westergaard ML, Glümer C, Hansen EH, et al. Prevalence of chronic headache with and without medication overuse: associations with socioeconomic position and physical and mental health status. *Pain*. 2014;155(10):2005–2013.

Occipital Neuralgia

ICD-10 CODE M53.82

THE CLINICAL SYNDROME

Occipital neuralgia is usually the result of blunt trauma to the greater and lesser occipital nerves (Fig. 7.1). The greater occipital nerve arises from fibers of the dorsal primary ramus of the second cervical nerve and, to a lesser extent, from fibers of the third cervical nerve. The greater occipital nerve pierces the fascia just below the superior nuchal ridge, along with the occipital artery. It supplies the medial portion of the posterior scalp as far anterior as the vertex (Fig. 7.2). The lesser occipital nerve arises from the ventral primary rami of the second and third cervical nerves (see Fig. 7.2). It passes superiorly along the posterior border of the sternocleidomastoid muscle and divides into cutaneous branches that innervate the lateral portion of the posterior scalp and the cranial surface of the pinna of the ear.

Less commonly, repetitive microtrauma from working with the neck hyperextended (e.g., painting ceilings) or looking for prolonged periods at a computer monitor whose focal point is too high, thus extending the cervical spine, may also cause occipital neuralgia. Occipital neuralgia is characterized by persistent pain at the base of the skull with occasional sudden, shocklike paresthesias in the distribution of the greater and lesser occipital nerves. Tension-type headache, which is much more common, occasionally mimics the pain of occipital neuralgia.

SIGNS AND SYMPTOMS

A patient suffering from occipital neuralgia experiences neuritic pain in the distribution of the greater and lesser occipital nerves when the nerves are palpated at the level of the nuchal ridge. Some patients can elicit pain with rotation or lateral bending of the cervical spine.

FIG 7.1 Occipital neuralgia is caused by trauma to the greater and lesser occipital nerves.

FIG 7.2 The sensory distribution of the greater and lesser occipital nerves. *n*, Nerve. (From Waldman S. *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021: Fig. 7.2.)

TESTING

No specific test exists for occipital neuralgia. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic occipital neuralgia (see "Differential Diagnosis"). All patients with the recent onset of headache thought to be occipital neuralgia should undergo magnetic resonance imaging (MRI) of the brain and cervical spine. The MRI should also be performed in patients with previously stable occipital neuralgia who have experienced a recent change in headache symptoms (Fig. 7.3). Computed tomography scanning of the brain and cervical spine may also be useful in identifying intracranial diseases that may mimic the symptoms of occipital neuralgia (Fig. 7.4). Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry should be performed if the diagnosis of occipital neuralgia is in question.

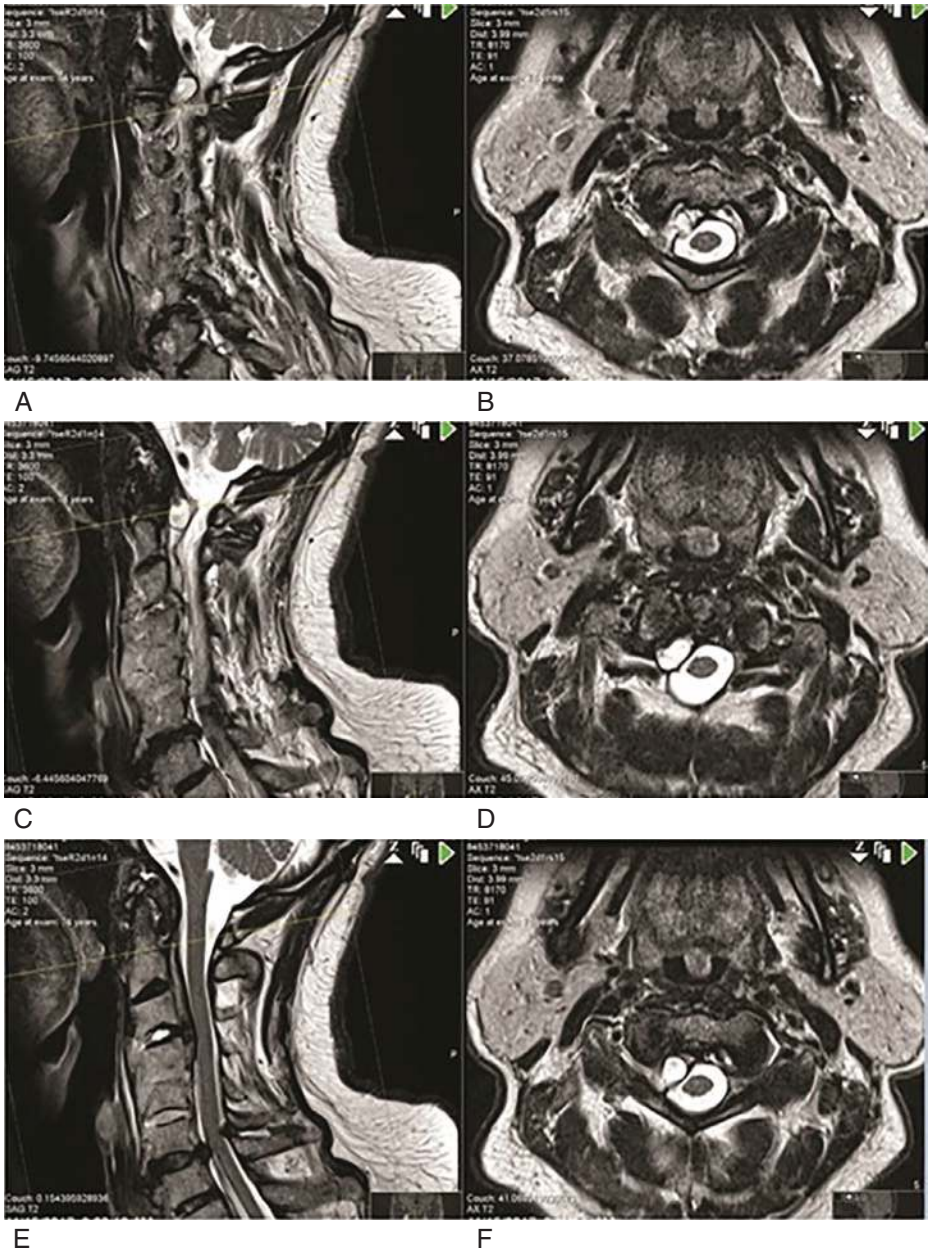

FIG 7.3 A, F, Magnetic resonance imaging scan of a patient with a clinical diagnosis of occipital neuralgia. T2 sequences with sagittal **(A, C, E)** and axial **(B, D, F)** views of the cervical spine depicting the T2 hyperintense cyst (synovial) of right-sided C1–C2 facet joint with compression of the C2 nerve root and its ganglion. **E,** There appears to be C6–C7 focal disk herniation with mild compression at the index level. However, the patient was not symptomatic from this level. (From Janjua MJ, Reddy S, El Ahmadieh TY, et al. Occipital neuralgia: a neurosurgical perspective. *J Clin Neurosci*. 2020;71:263–270.)

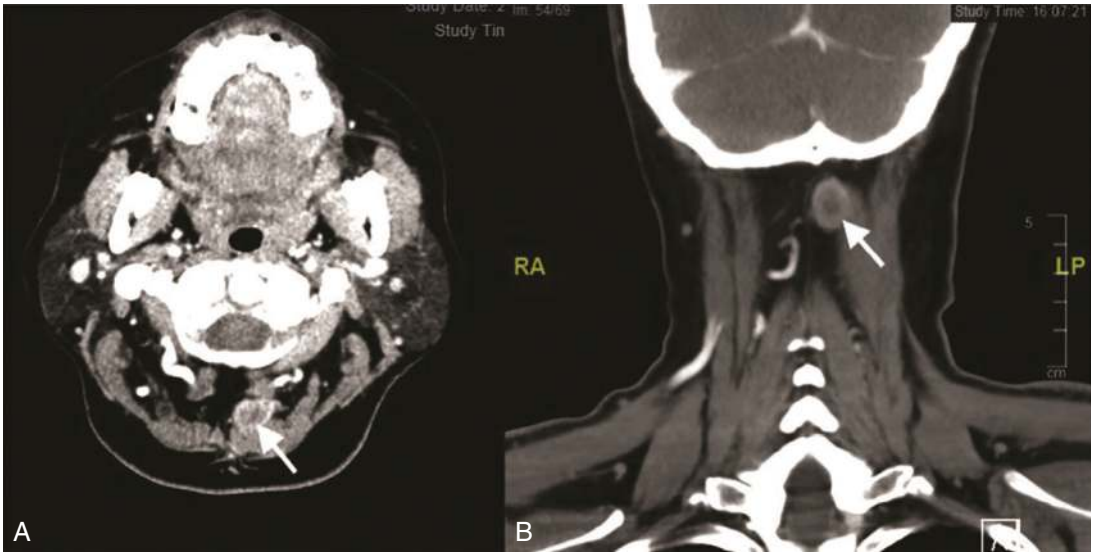

FIG 7.4 Computed tomography neck axial (A) and coronal (B) images showing a hypodense rim enhancing lesion in the left occipital region. (From Lee SYC, Lim MY, Loke SC, et al. Greater occipital nerve schwannoma—a rare cause of occipital neuralgia. *Otolaryngol Case Rep*. 2020;14:100–143.)

Neural blockade of the greater and lesser occipital nerves can help confirm the diagnosis and distinguish occipital neuralgia from tension-type headache. The greater and lesser occipital nerves can easily be blocked at the nuchal ridge.

DIFFERENTIAL DIAGNOSIS

Occipital neuralgia is an infrequent cause of headache and rarely occurs in the absence of trauma to the greater and lesser occipital nerves. More often, patients with headaches involving the occipital region are suffering from tension-type headache. Tension-type headache does not respond to occipital nerve blocks but is amenable to treatment with antidepressants such as amitriptyline, in conjunction with the cervical epidural nerve block. Therefore the clinician should reconsider the diagnosis of occipital neuralgia in patients whose symptoms are consistent with occipital neuralgia but who fail to respond to greater and lesser occipital nerve blocks (Box 7.1).

BOX 7.1 Differential Diagnosis of Occipital Neuralgia

Tension-type headache
Rheumatoid arthritis involving the upper cervical facet joints
Osteoarthritis involving the upper cervical facet joints
Arnold–Chiari malformation
Acceleration–deceleration injuries
Iatrogenic damage to the occipital nerves
Atlantoaxial subluxation
Atlantoaxial lateral masses
C2–C3 radiculopathy
C2–C3 subluxation or arthropathy
Cervical myelopathy
Posterior fossa tumor
Acromegaly
Neurofibromatosis type 1
Paget disease
Giant cell arteritis
Hemicrania continua
Herpes zoster
Pott disease
Synovial facet cyst
Neuritis
Neurosyphilis

TREATMENT

The treatment of occipital neuralgia consists primarily of neural blockade with local anesthetic and steroid, combined with the judicious use of nonsteroidal antiinflammatory drugs, muscle relaxants, tricyclic antidepressants, and physical therapy.

To perform neural blockade of the greater and lesser occipital nerves, the patient is placed in a sitting position with the cervical spine flexed and the forehead on a padded bedside table. A total of 8 mL of local anesthetic is drawn up in a 12-mL sterile syringe. For the treatment of occipital neuralgia or other painful conditions involving the greater and lesser occipital nerves, a total of 80 mg methylprednisolone is added to the local anesthetic with the first block and 40 mg of depot steroid is added with subsequent blocks. The occipital artery is palpated at the level of the superior nuchal ridge. After the skin is prepared with an antiseptic solution, a 1½-inch, 22-gauge needle is inserted just medial to the artery and is advanced perpendicularly until the needle approaches the periosteum of the underlying occipital bone. Paresthesias may be elicited, and the patient should be warned of this possibility. The needle is then redirected superiorly, and after gentle aspiration, 5 mL of solution is

injected in a fanlike distribution, with care taken to avoid the foramen magnum, which is located medially (Fig. 7.5). The lesser occipital nerve and several superficial branches of the greater occipital nerve are then blocked by directing the needle laterally and slightly inferiorly. After gentle aspiration, an additional 3–4 mL of solution is injected (see Fig. 7.5). Ultrasound needle guidance may improve the accuracy of needle placement (Figs. 7.6–7.8). Should the patient experience a recurrence of symptoms after initial relief from a trial of occipital nerve blocks, radiofrequency lesioning of the affected occipital nerves is a reasonable next step (Fig. 7.9). For patients suffering from occipital neuralgia that fails to respond to the foregoing treatment modalities, a trail of injection of type A botulinum toxin or occipital nerve stimulation should be considered (Fig. 7.10).

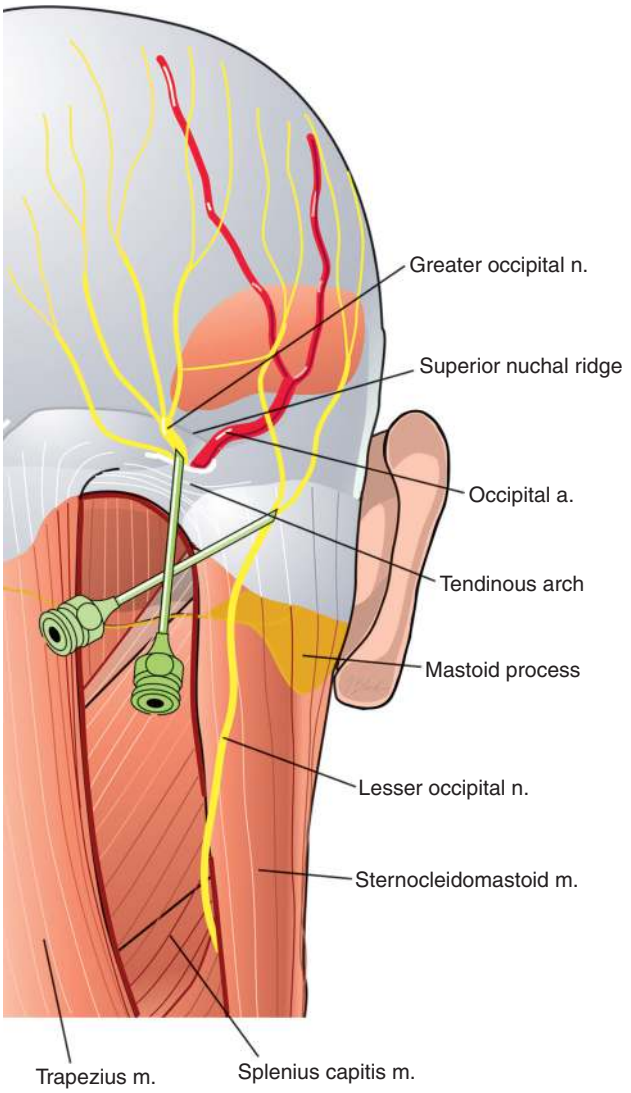

FIG 7.5 Proper needle placement for the greater and lesser occipital nerve block. (From Waldman SD. *Atlas of interventional pain management*. 2nd ed. Philadelphia: Saunders; 2004:25.)

COMPLICATIONS AND PITFALLS

The scalp is highly vascular. This vascularity, coupled with the close proximity to arteries of both the greater and lesser occipital nerves, means that the clinician must carefully calculate the total dose of local anesthetic that can be safely given, especially if bilateral nerve blocks are being performed. This vascularity and the proximity to the arterial supply give rise to an increased incidence of postblock ecchymosis and hematoma formation. These complications can be decreased

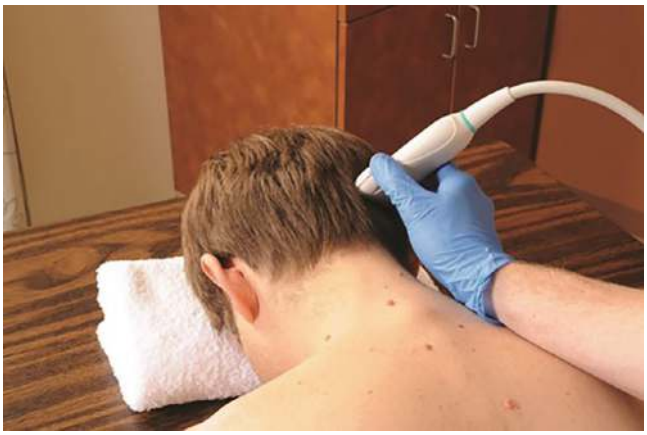

FIG 7.6 Proper ultrasound transducer placement for greater and lesser occipital block. (Courtesy Steven D. Waldman, MD.)

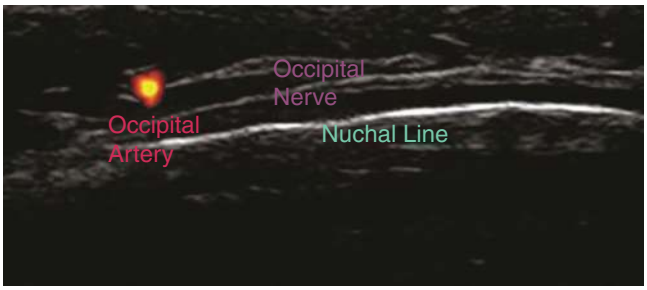

FIG 7.7 Color Doppler image demonstrating the relationship of the occipital artery and occipital nerve. (Courtesy Steven D. Waldman, MD.)

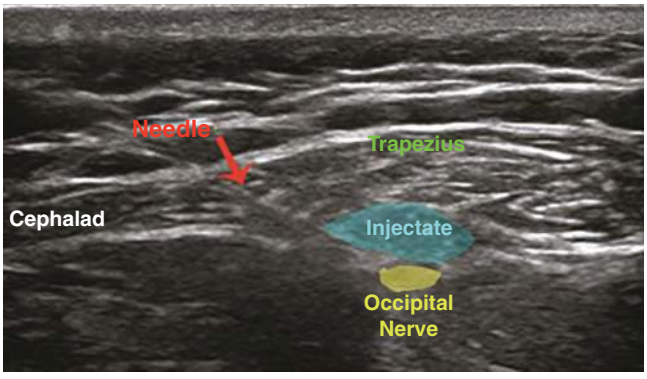

FIG 7.8 Ultrasound-guided greater occipital nerve block. (Courtesy Steven D. Waldman, MD.)

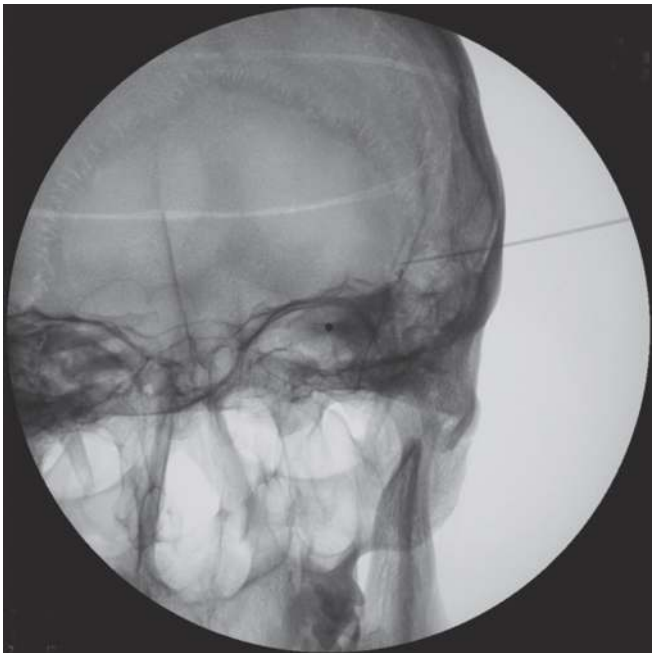

FIG 7.9 Radiofrequency lesioning of the greater occipital nerve.

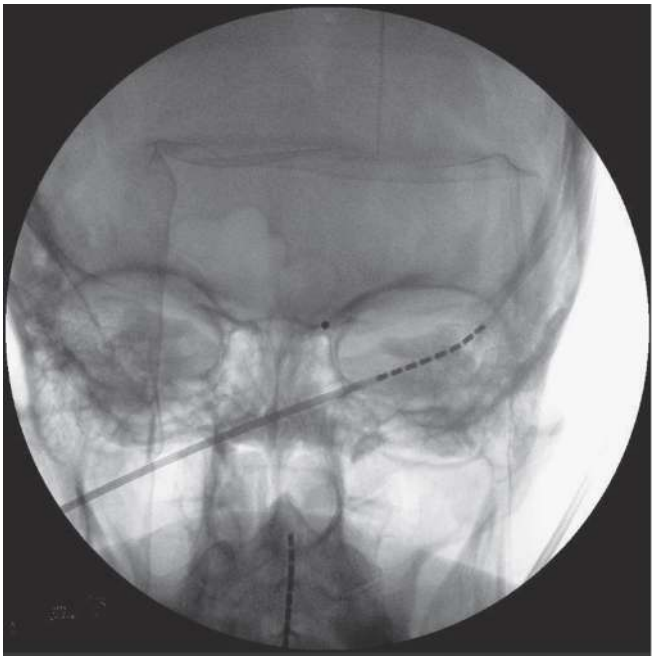

FIG 7.10 Occipital nerve stimulator lead in the correct position.

if manual pressure is applied to the area of the block immediately after injection. Application of cold packs for 20 minutes after the block can also decrease the amount of pain and bleeding. Care must be taken to avoid inadvertent needle placement into the foramen magnum, because the subarachnoid administration of local anesthetic in this region results in immediate total spinal anesthesia.

As with other headache syndromes, the clinician must be sure that the diagnosis is correct and that the patient has no coexistent intracranial disease or disease of the cervical spine that may be erroneously attributed to occipital neuralgia.

CLINICAL PEARLS

The most common reason that greater and lesser occipital nerve blocks fail to relieve headache pain is that the patient has been misdiagnosed. Any patient with headaches so severe that they require neural blockade should undergo MRI of the head to rule out unsuspected intracranial disease. Further, cervical spine radiographs should be considered to rule out congenital abnormalities such as Arnold–Chiari malformations that may be the hidden cause of the patient's occipital headaches.

SUGGESTED READINGS

Finiels P-J, Batifol D. The treatment of occipital neuralgia: review of 111 cases. *Neurochirurgie*. 2016;62(5):233–240.

Janjua MB, Reddy S, El Ahmadieh TY, et al. Occipital neuralgia: a neurosurgical perspective. *J Clin Neurosci*. 2020;71:263–270.

Lee SYC, Lim MY, Loke SC, et al. Greater occipital nerve schwannoma—a rare cause of occipital neuralgia. *Otolaryngol Case Rep*. 2020;14:100–143.

Waldman SD. Greater and lesser occipital nerve block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:27–30.

Waldman SD. Occipital neuralgia. In: *Pain review*. 2nd ed. Philadelphia: Saunders; 2017:256–257.

8

Pseudotumor Cerebri

ICD-10 CODE G93.2

THE CLINICAL SYNDROME

An often missed diagnosis, pseudotumor cerebri is a relatively common cause of headache. It has an incidence of 2.2 per 100,000 patients, approximately the same incidence as cluster headache. Also known as idiopathic intracranial hypertension, pseudotumor cerebri is seen most frequently in overweight women between the ages of 20 and 45 years. If epidemiologic studies look only at obese women, the incidence increases to approximately 20 cases per 100,000 patients. An increased incidence of pseudotumor cerebri is also associated with pregnancy. The exact cause of pseudotumor cerebri has not been elucidated, but the common denominator appears to be a defect in the absorption of cerebrospinal fluid (CSF). Predisposing factors include ingestion of various medications including tetracycline, vitamin A, corticosteroids, and nalidixic acid (Box 8.1). Other implicating factors include blood dyscrasias, anemias, endocrinopathies, and chronic respiratory insufficiency. In many patients, however, the exact cause of pseudotumor cerebri remains unknown.

SIGNS AND SYMPTOMS

More than 90% of patients suffering from pseudotumor cerebri present with the complaint of headache are female and have headaches that increase with the Valsalva maneuver. Associated nonspecific central nervous system, signs and symptoms such as dizziness, diplopia, tinnitus, photophobia, nausea and vomiting, and ocular pain, can often obfuscate what should otherwise be a reasonably straightforward diagnosis, given that basically all patients suffering from pseudotumor cerebri (1) have papilledema on fundoscopic examination (Fig. 8.1), (2) are female, and (3) are obese. The extent of papilledema varies from patient to patient and may be associated with subtle visual field defects including an enlarged blind spot and inferior nasal visual field defects (Fig. 8.2). If the condition is untreated, blindness may result (Fig. 8.3).

BOX 8.1 Medications Reportedly Associated With Intracranial Hypertension

Vitamins
Vitamin A
Retinol
Retinoids

Antibiotics
Tetracycline and derivatives
Nalidixic acid
Nitrofurantoin
Penicillin

Protein Kinase C Inhibitors
Lithium carbonate

Histamine (H_2)-Receptor Antagonists
Cimetidine

Steroids
Corticosteroid withdrawal
Levonorgestrel
Danazol
Leuprolide acetate
Tamoxifen
Growth hormone
Oxytocin
Anabolic steroids

Nonsteroidal Antiinflammatory Drugs
Ketoprofen
Indomethacin
Rofecoxib

Antiarrhythmics
Amiodarone

Anticonvulsants
Phenytoin

Dopamine Precursors
Levodopa
Carbidopa

TESTING

By convention, the diagnosis of primary pseudotumor cerebri (idiopathic intracranial hypertension) is made when four criteria are identified: (1) signs and symptoms suggestive of increased intracranial pressure including papilledema; (2) magnetic resonance imaging (MRI) or computed tomography (CT) of the brain reveals normal brain parenchyma with no evidence of hydrocephaly, masses, structural lesions, or meningeal enhancements;

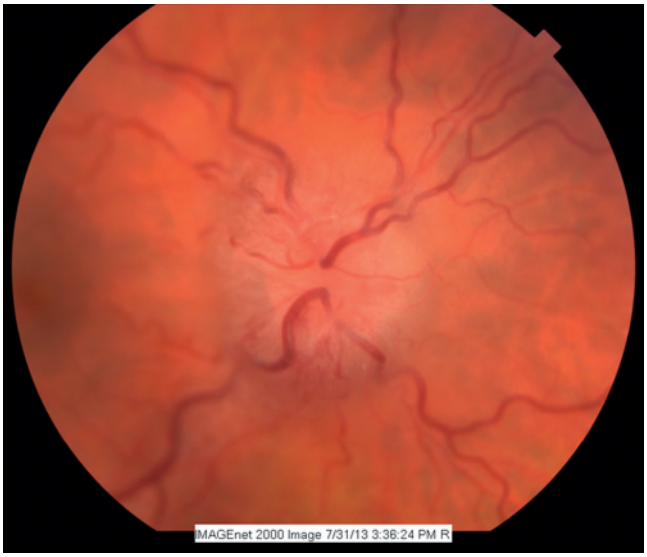

FIG 8.1 Severe papilledema in a patient suffering from pseudotumor cerebri. (Courtesy Corey W. Waldman, MD.)

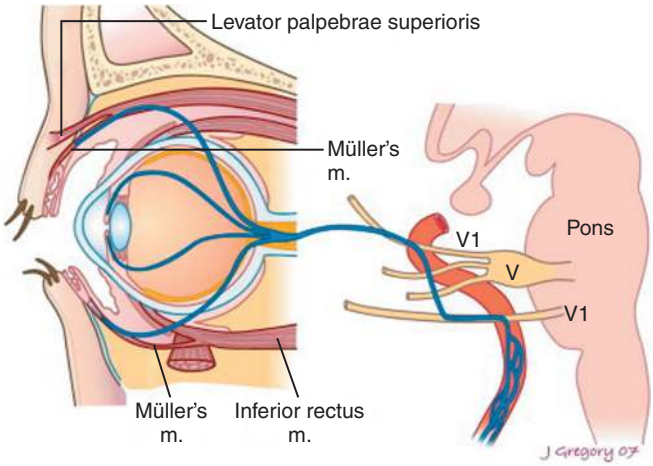

FIG 8.3 Müller's muscles. Müller's muscle in the upper eyelid arises from the undersurface of the levator palpebrae superioris muscle. Interruptions of the sympathetic innervation to this muscle cause ptosis of the upper eyelid. Müller's muscle in the lower lid will elevate the lower eyelid slightly in Horner's syndrome ("upside-down ptosis"). (From Reede DL, Garcon E, Smoker WR, et al. Horner's syndrome: clinical and radiographic evaluation. *Neuroimaging Clin N Am.* 2008;18(2):369–385.)

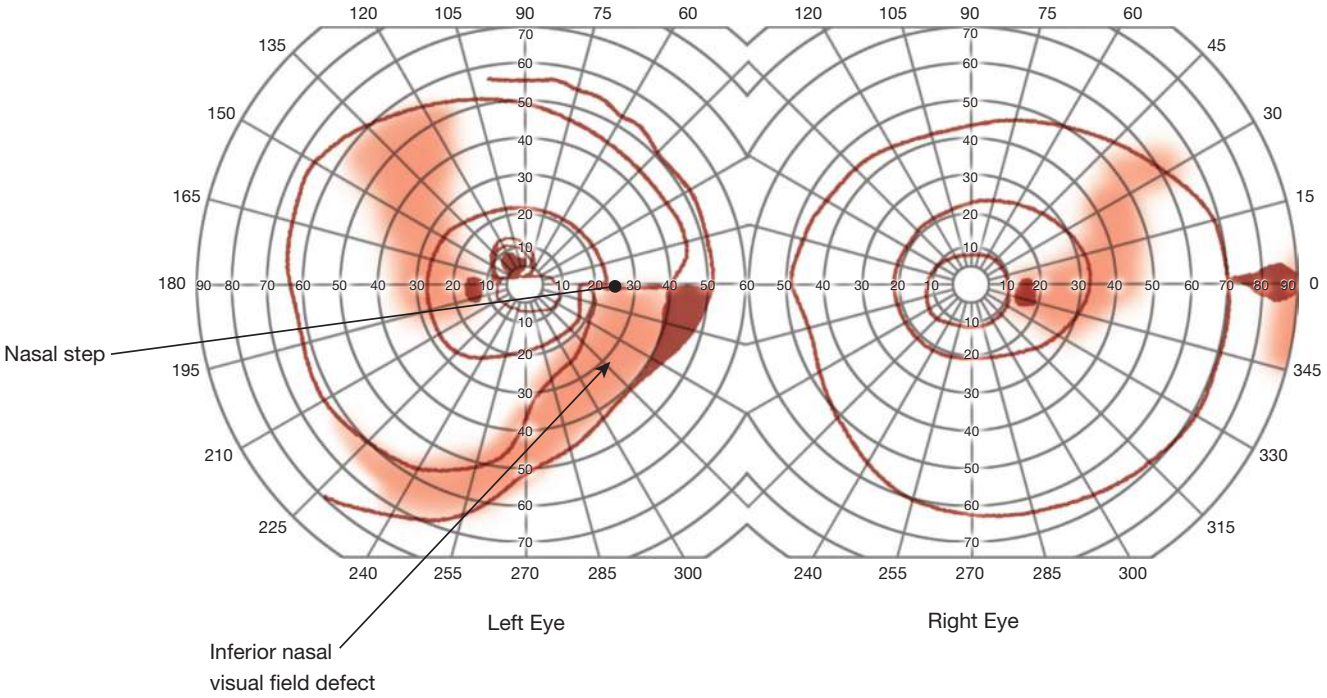

FIG 8.2 The most common visual field defects associated with pseudotumor cerebri are an abnormally enlarged blind spot and a nasal step defect affecting the inferior quadrants of the visual field.

(3) increased CSF pressure documented by lumbar puncture; and (4) normal CSF chemistry, cultures, and cytology (Box 8.2). An urgent MRI and CT scanning of the brain with contrast media should be obtained on all patients suspected of having increased intracranial pressure to rule out intracranial mass, vascular abnormalities, and infection, among other disorders (Fig. 8.4). Once the absence of space-occupying lesions or dilated ventricles is confirmed on neuroimaging, it is safe to proceed with lumbar puncture to measure the CSF pressure and obtain the fluid for chemistry, cultures, and cytology. Optical coherence tomography may also be useful in monitoring the efficacy

of interventions aimed at lowering the CSF pressure. MRI findings that support the diagnosis of primary pseudotumor cerebri include (1) abnormalities of the sella turcica including empty sella syndrome; (2) orbital abnormalities including flattening of the posterior sclera of the globe and tortuosity of the optic nerves; and (3) bilateral optic disk protrusion (Figs. 8.5–8.7). The CT scanning may reveal the unique finding of an increase in the area of the foramen ovale (Fig. 8.8).

BOX 8.2 Diagnostic Criteria for Pseudotumor Cerebri

1. Signs and symptoms suggestive of increased intracranial pressure, including papilledema
2. Normal magnetic resonance imaging or computed tomography of the brain performed with and without contrast media
3. Increased cerebrospinal fluid pressure documented by lumbar puncture
4. Normal cerebrospinal fluid chemistry, cultures, and cytology

DIFFERENTIAL DIAGNOSIS

If a specific cause is found for a patient's intracranial hypertension, it is by definition not idiopathic but rather a specific secondary type of intracranial hypertension. Causes of secondary intracranial hypertension that should be considered before diagnosing a patient with idiopathic intracranial hypertension are listed in Box 8.3. These include the various forms of intracranial hemorrhage, intracranial tumor, and cranial or cervical spine abnormalities such as Arnold–Chiari malformation, cerebral venous sinus thrombosis, abnormalities of the ventricular system, hepatic failure, and intracranial

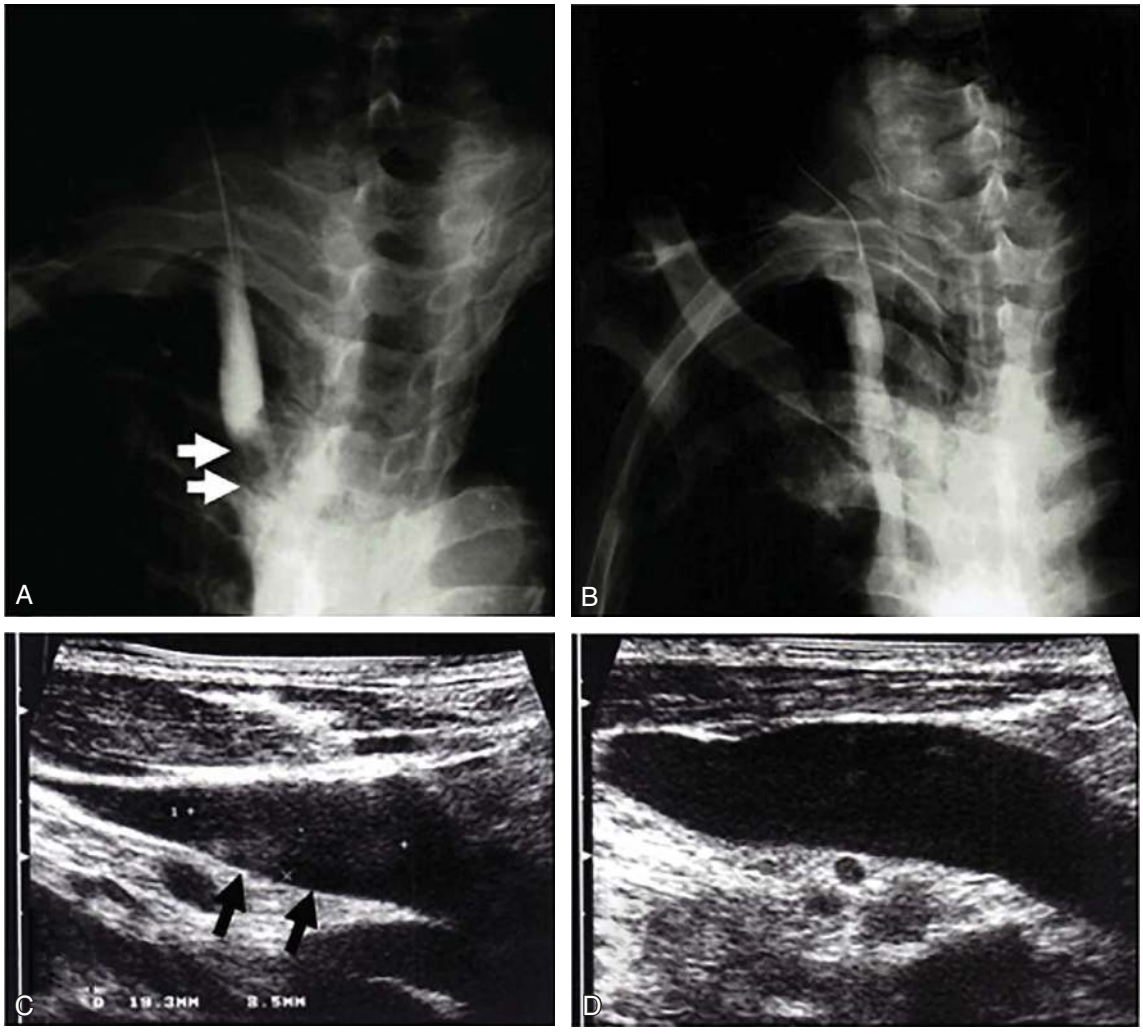

FIG 8.4 **A,** The pretherapeutic venogram of the right internal jugular vein (IJV), in frontal projection, shows the obstruction of the lower IJV. The *arrows* indicate the filling defect of the jugular vein thrombosis (JVT). **B,** A posttherapeutic venogram obtained after the anticoagulation therapy using warfarin shows the resolution and recanalization of the JVT. **C,** A pretherapeutic ultrasonogram of the right IJV shows the JVT. The *arrows* indicate the thrombus in the right IJV. **D,** A posttherapeutic ultrasonogram after the anticoagulation therapy using warfarin shows the resolution and recanalization of the JVT. (From Matsuda W, Noguchi S, Fujiyama F. Pseudotumor cerebri and lung cancer-associated jugular vein thrombosis: role of anatomical variations of torcular herophili. *eNeurologicalSci*. 2018;13:18–20.)

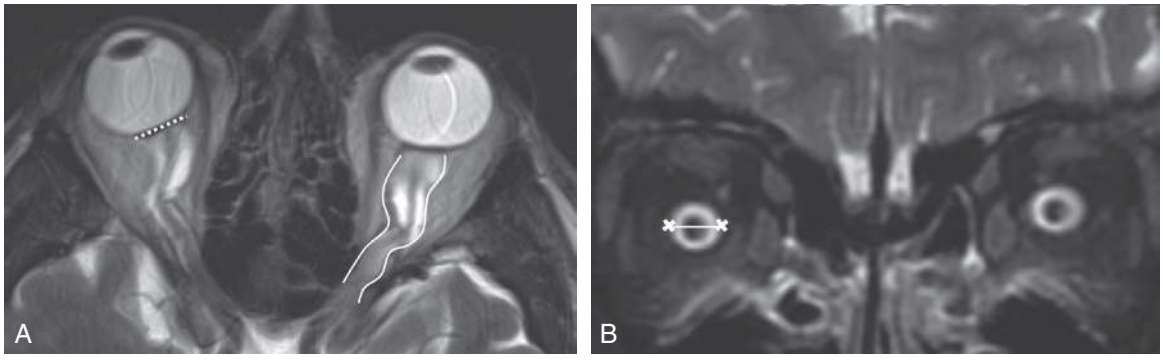

FIG 8.5 Orbital findings of pseudotumor cerebri on magnetic resonance imaging (MRI). **A,** MRI, fast spin echo (FSE) T2 sequence on the transverse plane, showing distortion of the normal morphology of the eyeball with bilateral flattening of the posterior sclera (*dotted line*) as well as tortuosity of the optic nerves on the horizontal plane (*solid lines*). **B,** MRI, short TI inversion recovery (STIR) sequence on the coronal plane, showing distension of the optic nerve sheath behind the eyeball (*line*). (From Veiga-Canuto D, Carreres-Polo J. Role of imaging in pseudotumor cerebri syndrome. *Radiología (English Edition)*. 2020;62(5):400–410. http://www.sciencedirect.com/science/article/pii/S2173510720300677.)

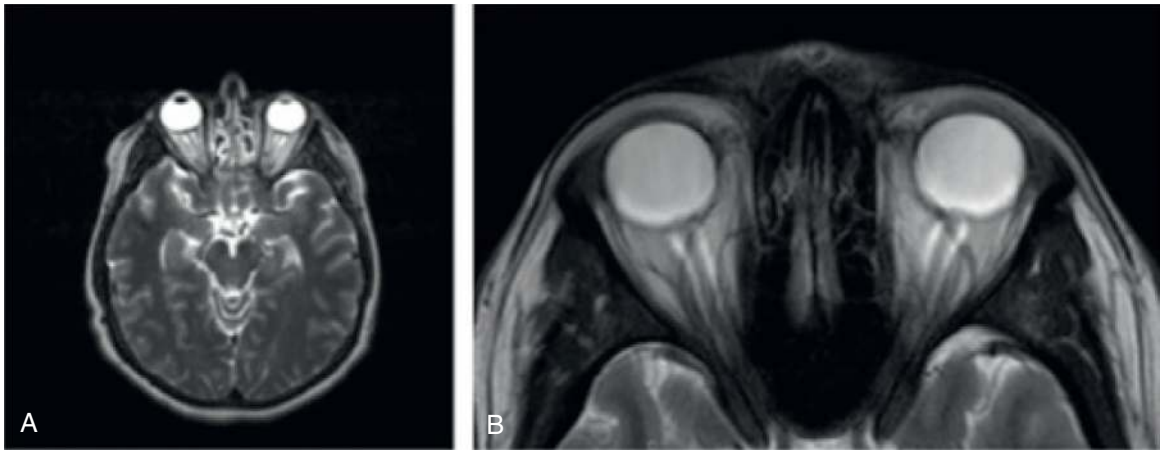

FIG 8.6 Orbital findings associated with papilloedema. (**A**) Magnetic resonance imaging (MRI), FSE T2 sequence on the transverse plane. Bilateral intraocular protrusion of the optic disc (*black arrows*) is identified. (**B**) Same patient as in Image A. MRI, transverse 3D gradient T1 sequence with fat saturation following administration of intravenous contrast, showing bilateral, symmetrical enhancement in the prelaminar region of the optic nerves (*white arrows*). (From Akhter A, Schulz L, Inger HE, McGregor JM, Current Indications for Management Options in Pseudotumor Cerebri, *Neurologic Clinics*. 2022;40(2):391–404.)

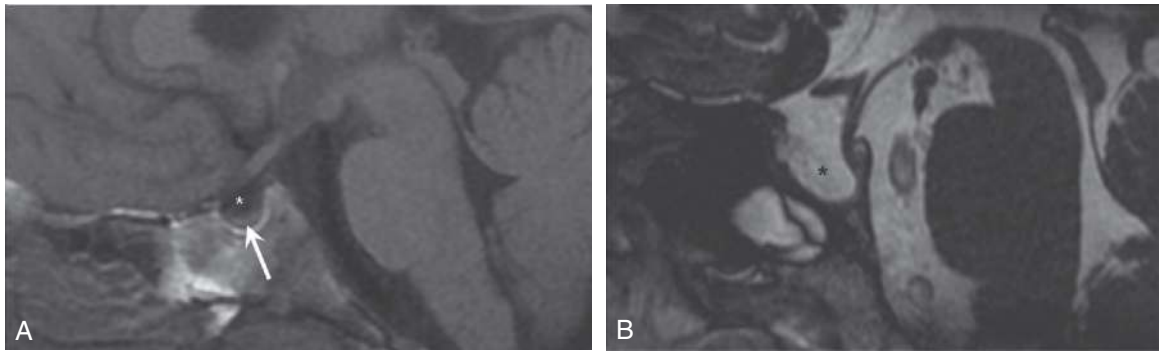

FIG 8.7 Radiological findings in the sella turcica in patients suffering from pseudotumor cerebri. **A,** Partially empty sella turcica, seen in sagittal turbo spin echo (TSE) T1 sequence. The typical characteristics are seen with the sella turcica almost completely filled with cerebrospinal fluid (CSF) (*) and the pituitary gland narrowed (*arrow*). **B,** Empty sella turcica visualized in sagittal TSE T2 sequence. The sella turcica is seen to be remodeled, expanded, and completely filled with CSF (*), with the pituitary gland not visible. (From Veiga-Canuto D, Carreres-Polo J. Role of imaging in pseudotumor cerebri syndrome. *Radiología (English Edition)*. 2020;62(5):400–410.)

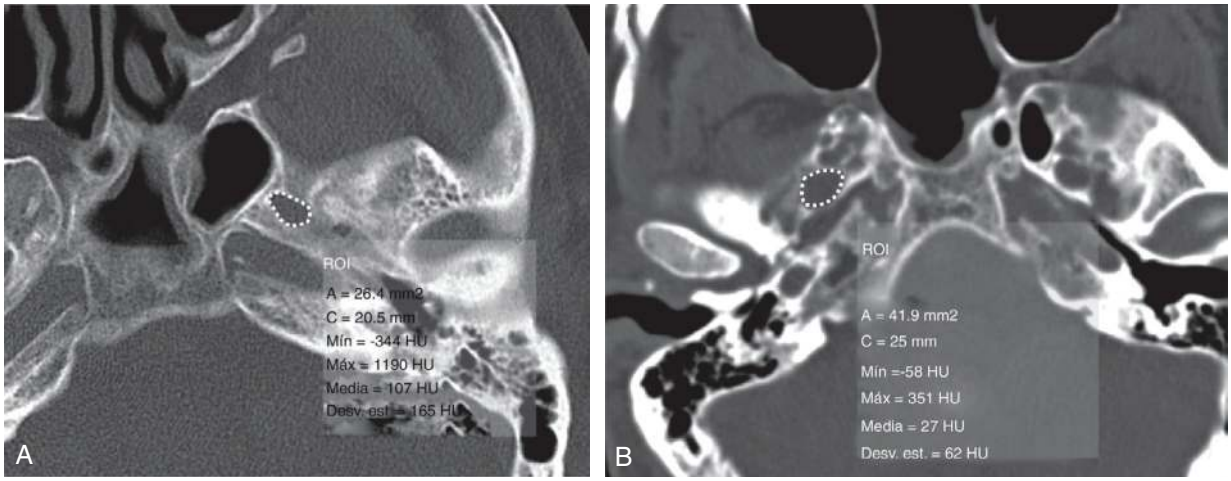

FIG 8.8 Increased area of the foramen ovale. **A,** Area of the foramen ovale on a computed tomographic scan of the head without contrast in a healthy patient, with a value of 26.4 mm^2. Compare this area measurement to a patient with idiopathic intracranial hypertension **(B)** in which the mean area of the foramen ovale is 41.9 mm^2, exceeding 40 mm^2. (From Veiga-Canuto D, Carreres-Polo J. Role of imaging in pseudotumor cerebri syndrome. *Radiología (English Edition)*. 2020;62(5):400–410.)

BOX 8.3 Common Causes of Secondary Intracranial Hypertension

- Intracranial Hemorrhage
  - Intraventricular hemorrhage
  - Subarachnoid hemorrhage
  - Intraparenchymal hemorrhage
  - Subdural hematoma
  - Epidural hematoma
- Intracranial Tumor
  - Primary brain tumors
  - Meningiomas
  - Pineal tumors
  - Pituitary tumors
  - Posterior fossa tumors
  - Hamartomas
- Cranial or Cervical Spine Abnormalities
  - Arnold–Chiari malformation
  - Craniosynostosis
  - Craniofacial dysostosis
- Cerebral Venous Sinus Thrombosis
- Abnormalities of the Ventricular System
  - Aqueductal stenosis
  - Dandy–Walker syndrome
- Intracranial Infections
  - Meningitis
  - Encephalitis
  - Intracranial abscess
  - Intracranial parasites
  - Epidural abscess
- Intracranial Granulomas
  - Eosinophilic granuloma
  - Wegener's granulomatosis
  - Sarcoidosis
- Lead Poisoning

infections. A failure to diagnose a potentially treatable cause of intracranial hypertension may result in significant mortality and morbidity.

TREATMENT

A reasonable first step in the treatment of patients who exhibit all four criteria necessary for the diagnosis of pseudotumor cerebri is the initiation of oral acetazolamide. If poorly tolerated, the use of furosemide or chlorthalidone can be considered. A short course of systemic corticosteroids such as dexamethasone may also be used if the patient does not respond to diuretic therapy. The addition of weight loss strategies in combination with the use of acetazolamide may hasten the resolution of the symptoms of pseudotumor cerebri. Early clinical reports suggest that octreotide, a synthetic somatostatin analogue, may be beneficial in the management of treatment-resistant pseudotumor cerebri. For resistant cases, neurosurgical interventions including CSF shunt procedures are a reasonable next step. If papilledema persists, decompression procedures on the optic nerve sheath have been advocated as has the use of bariatric surgery.

COMPLICATIONS AND PITFALLS

As mentioned earlier, untreated pseudotumor cerebri can result in permanent visual loss and significant morbidity. Furthermore, a failure to diagnose and treat properly the secondary causes of increased intracranial hypertension can lead to disastrous results for the patient, including potentially avoidable death.

CLINICAL PEARLS

Pseudotumor cerebri is predominately a disease that affects women. It is a relatively straightforward diagnosis if one thinks of it. Patients suffering from pseudotumor cerebri have papilledema on fundoscopic examination and are invariably obese. Visual field defects can be subtle and include an enlarged blind spot and associated inferior nasal visual field defects. Often, medications are found to be the causative agent in the evolution of this headache syndrome and should be diligently sought. As with all headache syndromes, other causes of increased intracranial pressure, such as tumor or hemorrhage, must be ruled out.

SUGGESTED READINGS

Akhter A, Schulz L, Inger HE, McGregor JM. Current indications for management options in pseudotumor cerebri. *Neurol Clin*. 2022;40(2):391–404.

Aylward SC, Reem RE. Pediatric intracranial hypertension. *Pediatr Neurol*. 2017;66:32–43.

Ball AK, Clarke CE. Idiopathic intracranial hypertension. *Lancet Neurol*. 2006;5(5):433–442.

Beres SJ, Digre KB, Friedman DI, Liu GT. Pseudotumor cerebri syndrome is the best term for this condition. *Pediatr Neurol*. 2018;87:9–10.

Elder BD, Sankey EW, Goodwin CR, et al. Outcomes and experience with lumbopleural shunts in the management of idiopathic intracranial hypertension. *World Neurosurg*. 2015;84(2):314–319.

Eldweik L, McClelland C, Stein JD, et al. Association between cycline antibiotic and development of pseudotumor cerebri syndrome. *J Am Acad Dermatol*. 2019;81(2):456–462.

Handley JD, Baruah BP, Williams DM, et al. Bariatric surgery as a treatment for idiopathic intracranial hypertension: a systematic review. *Surg Obes Relat Dis*. 2015;11(6):1396–1403.

House PM, Stodieck SRG. Octreotide: the IIH therapy beyond weight loss, carbonic anhydrase inhibitors, lumbar punctures and surgical/interventional treatments. *Clin Neurol Neurosurg*. 2016;150:181–184.

Mancera N, Murr MM, Drucker M. Bariatric surgery and its impact on pseudotumor cerebri: a case report. *Am J Ophthalmol Case Rep*. 2018;10:68–70.

Matsuda W, Noguchi S, Fujiyama F. Pseudotumor cerebri and lung cancer-associated jugular vein thrombosis: role of anatomical variations of torcular herophili. *eNeurologicalSci*. 2018;13:18–20.

Thurtell M. Update in the management of idiopathic intracranial hypertension. *Neurol Clin*. 2021;39(1):147–161. https://www.sciencedirect.com/science/article/pii/S073386192030075X.

Veiga-Canuto D, Carreres-Polo J. Role of imaging in pseudotumor cerebri syndrome. *Radiología (English Edition)*. 2020;62(5):400–410.

Waldman SD. Pseudotumor cerebri. In: *Pain review*. 2nd ed. Philadelphia: Saunders Elsevier; 2017:204–205.

Wall M. Update on idiopathic intracranial hypertension. *Neurol Clin*. 2017;35(1):45–57.

9

Intracranial Subarachnoid Hemorrhage

ICD-10 CODE 160.9

THE CLINICAL SYNDROME

Subarachnoid hemorrhage (SAH) represents one of the most neurologically devastating forms of cerebrovascular accident. Fewer than 60% of patients suffering from the malady will recover cognitively and functionally to their premorbid state. From 65% to 70% of all SAH results from rupture of intracranial berry aneurysms. Arteriovenous malformations, neoplasm, and angiomas are responsible for most of the remainder (Fig. 9.1). Berry aneurysms are prone to rupture because of their lack of a fully developed muscular media and collagen-elastic layer. Systemic diseases associated with an increased incidence of berry aneurysm include Marfan's syndrome, Ehlers–Danlos syndrome, sickle cell disease, coarctation of the aorta, alpha-1-antitrypsin deficiency, polycystic kidney disease, fibromuscular vascular dysplasia, and pseudoxanthoma elasticum (Box 9.1). Hypertension, alcohol and caffeine consumption, smoking, and cocaine use, which are modifiable risk factors, and cerebral atherosclerosis increase the risk of SAH. Blacks are more than twice as likely to suffer SAH when compared with Whites. Female patients are affected more often than male patients, and the mean age of patients suffering from SAH is 50 years. Even with modern treatment, the mortality associated with significant SAH is approximately 25%.

SIGNS AND SYMPTOMS

Massive SAH is often preceded by a warning in the form of what is known as sentinel or thunderclap headache. This headache is thought to be the result of leakage from an aneurysm that is preparing to rupture. The sentinel headache is of sudden onset, with a temporal profile characterized by a rapid onset to peak in intensity. The sentinel headache may be associated with photophobia and nausea and vomiting. Of patients, 90% with intracranial SAH will experience a sentinel headache within 3 months of significant SAH.

Patients with significant SAH experience the sudden onset of severe headache, which the patient often describes as the

BOX 9.1 Systemic Diseases Associated With an Increased Incidence of Berry Aneurysm

Marfan's syndrome
Ehlers–Danlos syndrome
Sickle cell disease
Polycystic kidneys
Coarctation of the aorta
Fibromuscular vascular dysplasia
Pseudoxanthoma elasticum

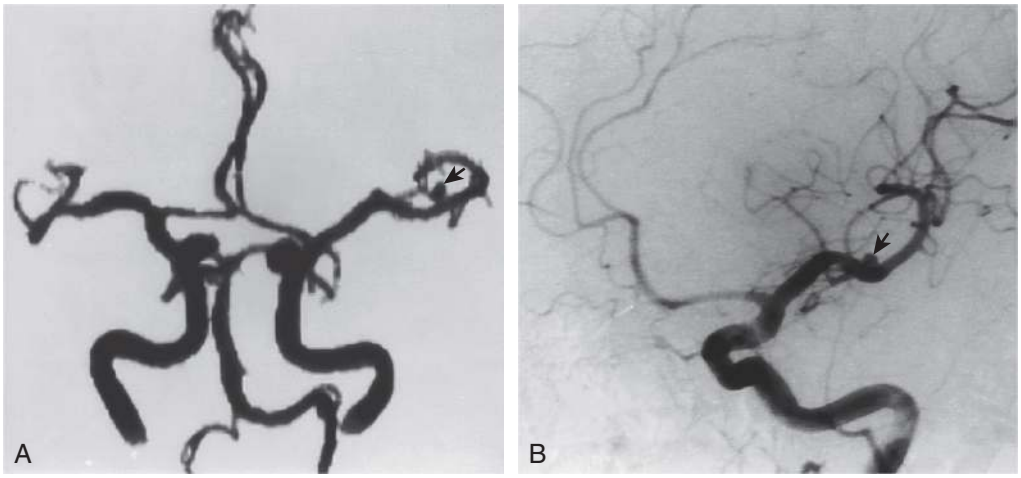

FIG 9.1 Berry aneurysm in a patient with autosomal-dominant polycystic kidney disease. **A,** A three-dimensional time-of-flight magnetic resonance angiogram with a vessel-tracking postprocessing algorithm discloses a left middle cerebral artery bifurcation aneurysm (*arrow*). **B,** Catheter angiogram shows the same lesion (*arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging.* 3rd ed. Philadelphia: Saunders; 2006:1420.)

FIG 9.2 The headache associated with subarachnoid hemorrhage is often described as the worst headache the patient has ever experienced.

BOX 9.2 Symptoms Associated With Subarachnoid Hemorrhage

Severe headache
Nausea and vomiting
Photophobia
Vertigo
Lethargy
Confusion
Nuchal rigidity
Neck and back pain

worst headache of his or her life (Fig. 9.2). This headache is usually associated with nausea and vomiting, photophobia, vertigo, lethargy, confusion, nuchal rigidity, and neck and back pain (Box 9.2). The patient experiencing acute SAH appears acutely ill, and up to 50% will lose consciousness as the intracranial pressure rapidly rises in response to unabated hemorrhage. Cranial nerve palsy, especially of the abducens nerve, may also occur as a result of increased intracranial pressure. Focal neurologic signs, paresis, seizures, subretinal hemorrhages, and papilledema are often present on physical examination.

TESTING

Testing in patients suspected of suffering with SAH has two immediate goals: (1) to identify an occult intracranial pathologic process or other diseases that may mimic SAH and may be more amenable to treatment (see "Differential Diagnosis") and (2) to identify the presence of SAH. All patients with a recent onset of severe headache thought to be secondary to SAH should undergo emergency computed tomography (CT) scanning of the brain (Fig. 9.3). Modern multidetector CT scanners have a diagnostic accuracy approaching 100% for SAH if CT angiography of the cerebral vessels is part of the scanning protocol. Cerebral angiography may also be required if surgical intervention is being considered and the site of bleeding cannot be accurately identified.

Magnetic resonance imaging (MRI) of the brain and magnetic resonance angiography may be useful if an aneurysm is not identified on CT studies and may be more accurate in the diagnosis of arteriovenous malformations (Fig. 9.4). Screening laboratory tests, including an erythrocyte sedimentation rate, complete blood count, coagulation studies, and automated blood chemistry, should be performed in patients suffering from SAH. Blood typing and crossmatching should be considered in any patient in whom surgery is being contemplated or who has preexisting anemia. Careful serial ophthalmologic examination should be performed on all patients suffering from SAH, to chart the course of papilledema.

Lumbar puncture may be useful in revealing blood in the spinal fluid, but its utility may be limited by the presence of increased intracranial pressure, which makes lumbar puncture too dangerous. Electrocardiographic abnormalities are common in patients suffering from SAH and are thought to result from abnormally high levels of circulating catecholamines and hypothalamic dysfunction.

DIFFERENTIAL DIAGNOSIS

For the most part, the differential diagnosis of SAH can be thought of as the diagnosis of the lesser of two evils because most of the diseases that mimic SAH are also associated with significant mortality and morbidity. Box 9.3 lists diseases that may be mistaken for SAH. Prominent among them are stroke, collagen vascular disease, infection, neoplasm, hypertensive crisis, spinal fluid leaks, and various more benign causes of headache.

TREATMENT

Medical Management

The treatment of SAH begins with careful acute medical management, with an eye to minimizing the sequelae of both the cerebral insult and the morbidity associated with a severe illness. Bed rest with the head of bed elevated to

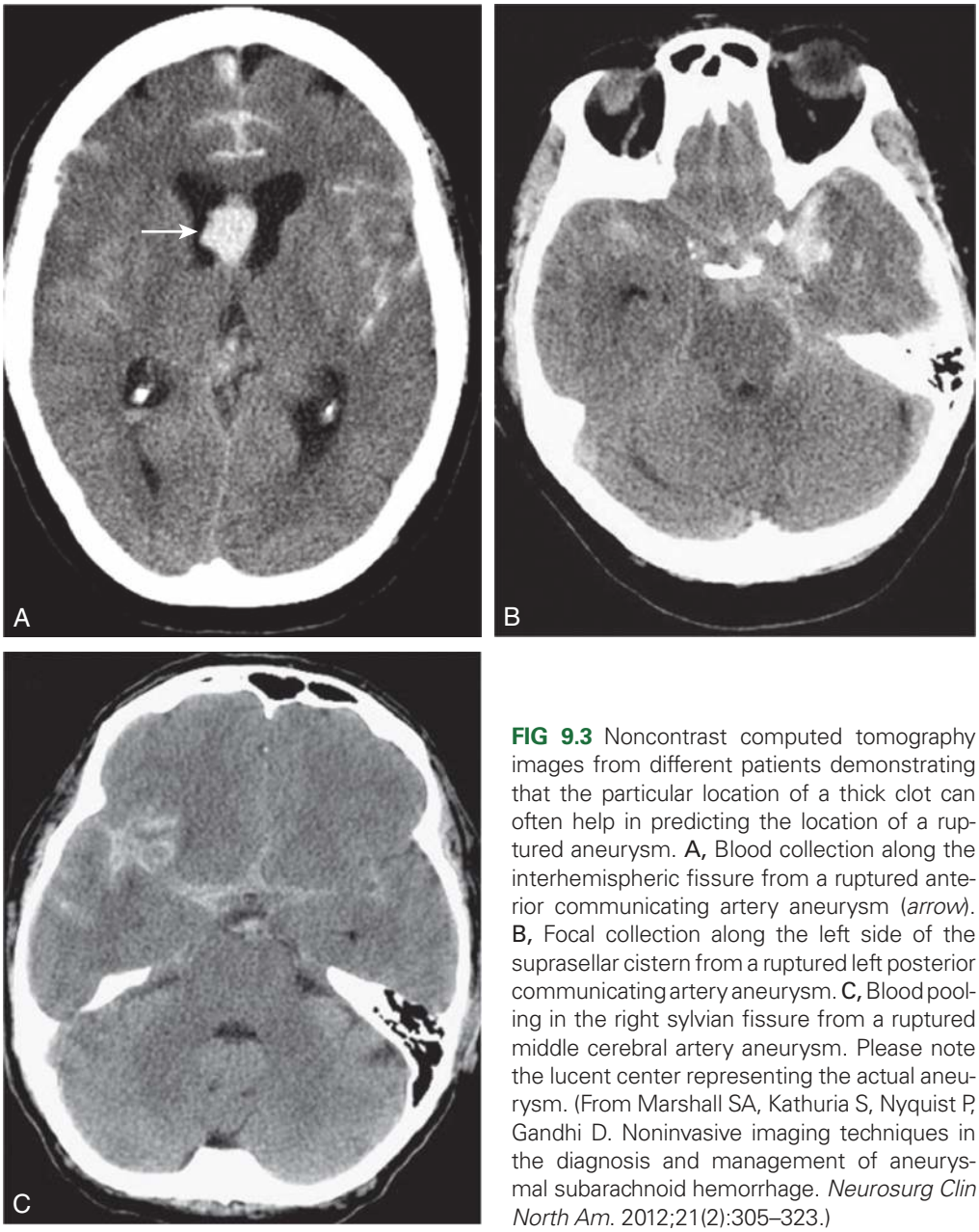

FIG 9.3 Noncontrast computed tomography images from different patients demonstrating that the particular location of a thick clot can often help in predicting the location of a ruptured aneurysm. **A,** Blood collection along the interhemispheric fissure from a ruptured anterior communicating artery aneurysm (*arrow*). **B,** Focal collection along the left side of the suprasellar cistern from a ruptured left posterior communicating artery aneurysm. **C,** Blood pooling in the right sylvian fissure from a ruptured middle cerebral artery aneurysm. Please note the lucent center representing the actual aneurysm. (From Marshall SA, Kathuria S, Nyquist P, Gandhi D. Noninvasive imaging techniques in the diagnosis and management of aneurysmal subarachnoid hemorrhage. *Neurosurg Clin North Am.* 2012;21(2):305–323.)

30–35 degrees to promote good venous drainage is a reasonable first step in the treatment of the patient suffering from SAH. Accurate intake and output determinations, as well as careful management of hypertension and hypotension, are also essential during the initial management of SAH, and invasive cardiovascular monitoring should be considered sooner rather than later in this setting. Pulse oximetry and end-tidal carbon dioxide monitoring should be initiated early in the course of treatment to identify respiratory insufficiency. Avoidance of overuse of opioids and sedatives is important, to prevent hypoventilation with its attendant increase in intracranial pressure and cerebral ischemia. Seizure precautions and aggressive treatment of seizures are also required. Nimodipine, the calcium antagonist, to prevent delayed cerebral ischemia, is also recommended. Vomiting should be controlled to avoid the increase in intracranial pressure associated with the Valsalva maneuver. Prophylaxis of gastrointestinal bleeding, especially if steroids are used to treat increased intracranial pressure, and the use of pneumatic compression devices to avoid thrombophlebitis are also worth considering. If unconsciousness occurs, endotracheal intubation using techniques to avoid increases in intracranial pressure should be performed, and hyperventilation to decrease blood carbon dioxide levels should be considered.

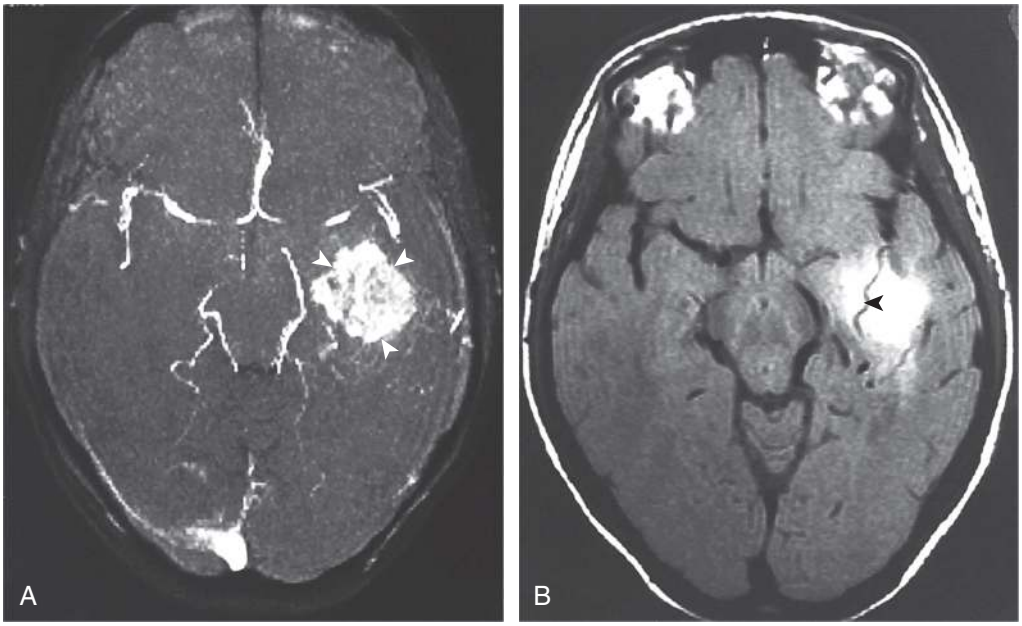

FIG 9.4 Left temporal hemorrhage from an arteriovenous malformation. **A,** On gradient-echo magnetic resonance imaging (MRI), the hematoma appears bright because of methemoglobin (*arrowheads*), and no abnormal vessel is visualized. **B,** On spin-echo MRI with flow presaturation below the section to be imaged, flow voids of abnormal vessels posterior to the hematoma and an abnormal vessel running through the hematoma (*arrowhead*) are visible. (From Mattle H, Edelman RR, Atkinson DJ. Zerebrale angiographie mittels kernspintomographie. *Schweiz Med Wochenschr.* 1992;122:323–333.)

BOX 9.3 Diseases That May Mimic Subarachnoid Hemorrhage

- Stroke hemorrhagic
  - Ischemic
- Neoplasm
- Infection
  - Meningitis
  - Encephalitis
  - Abscess
  - Parasitic
- Hypertensive crisis
- Loss of spinal fluid
  - Postdural puncture headache
  - Spontaneous spinal fluid leak
- Collagen vascular disease
  - Lupus cerebritis
  - Vasculitis
  - Polymyositis
- Headache
  - Cluster headache
  - Thunderclap headache
  - Migraine
  - Ice-pick headache
  - Sexual headache

Treatment of increased intracranial pressure with dexamethasone, the osmotic agent mannitol, and furosemide may be required. Calcium channel blockers, cilostazol, and magnesium may be beneficial to reduce cerebrovascular spasm and decrease the zone of ischemia. Studies showed that statins may also be useful in this setting. Antifibrinolytics, such as epsilon-aminocaproic and tranexamic acid, may be useful to decrease the incidence of rebleeding in selected patients.

Surgical Treatment

Surgical treatment of hydrocephalus with ventricular drainage may be required to treat highly elevated intracranial pressure, with the caveat that too rapid a decrease in intracranial pressure in this setting may result in an increased incidence of rebleeding. Surgical treatment with clipping of the aneurysm or interventional radiologic endovascular occlusive coil treatment of continued bleeding or rebleeding carries a high risk of morbidity and mortality, but it may be necessary if more conservative treatments fail (Fig. 9.5).

COMPLICATIONS AND PITFALLS

Complications and pitfalls in the diagnosis and treatment of SAH generally fall into three categories. The first category involves the failure to recognize a sentinel hemorrhage and to evaluate and treat the patient before significant SAH occurs. The second category involves misdiagnosis, which results in treatment delays that ultimately cause an increase in mortality and morbidity. The third category involves less than optimal medical management, which results in avoidable mortality and morbidity. Examples are pulmonary embolus from thrombophlebitis and aspiration pneumonia from failure to protect the patient's airway.

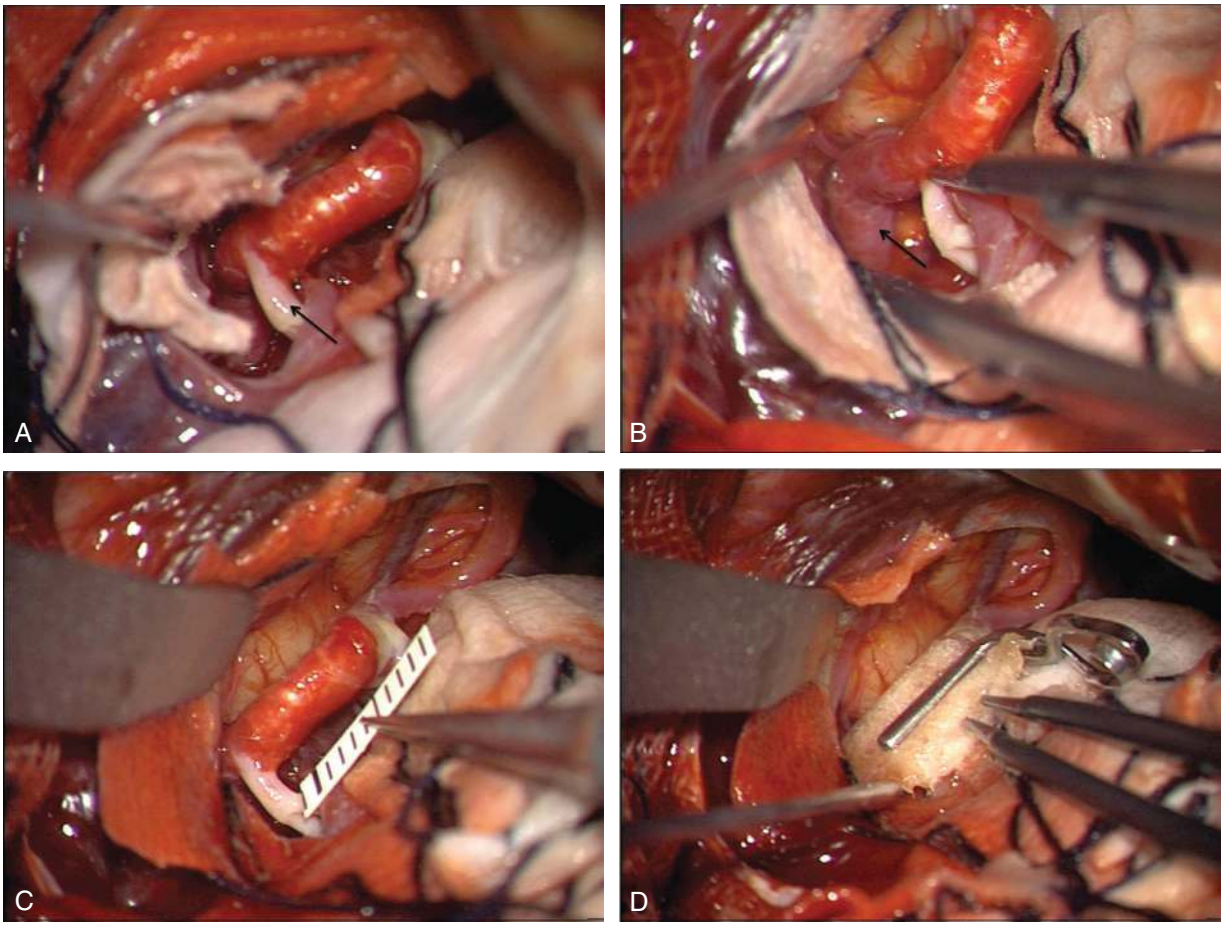

FIG 9.5 Intraoperative views showed the dissecting aneurysm in the left MCA. **A,** Dissected MCA aneurysm without blood flow, and a small branch (*arrow*) with scant blood flow. **B,** TCD applied to the distal segment (*arrow*) of the dissecting MCA; there is no blood flow. **C,** The photo shows a 9 mm dissected MCA aneurysm with atherosclerotic change. **D,** Dissected aneurysm was wrapped, and clip reinforcement was done. (From Lee J-M. Subarachnoid hemorrhage due to middle cerebral artery dissection mimicking aneurysm—case report. *Radiol Case Rep.* 2022;17(7):2537–2541.)

CLINICAL PEARLS

The identification of sentinel headache and subsequent aggressive treatment before significant SAH occurs to give the patient his or her best chance of a happy outcome. Treatment of significant SAH is difficult, and ultimately results are disappointing. Careful attention to initial and ongoing medical management, with aggressive monitoring and treatment of associated hypertension and hypotension and respiratory abnormalities, is crucial to prevent avoidable complications.

SUGGESTED READINGS

Abraham MK, Chang WW. Subarachnoid hemorrhage. *Emerg Med Clin North Am*. 2016;34(4):901–916.

Edlow JA, Abraham MK. Neurologic emergencies—making the diagnosis and treating the life threats. *Emerg Med Clin North Am*. 2016;34(4):xvii–xviii.

Howard RS. The management of haemorrhagic stroke. *Anaesth Intensive Care Med*. 2016;17(12):596–601.

Larson AS, Brinjikji W. Subarachnoid hemorrhage of unknown cause: distribution and role of imaging. *Neuroimaging Clin North Am*. 2021;31(2):167–175.

Lee J-M. Subarachnoid hemorrhage due to middle cerebral artery dissection mimicking aneurysm—case report. *Radiol Case Rep*. 2022;17(7):2537–2541.

Manhas A, Nimjee SM, Agrawal A, et al. Comprehensive overview of contemporary management strategies for cerebral aneurysms rategies for cerebral aneurysms. *World Neurosurg*. 2015;84(4):1147–1160.

Perry JJ, Stiell IG, Sivilotti MA, et al. Clinical decision rules to rule out subarachnoid hemorrhage for acute headache. *JAMA*. 2013;310(12):1248–1255.

10

Trigeminal Neuralgia

ICD-10 CODE G50.0

THE CLINICAL SYNDROME

Trigeminal neuralgia occurs in many patients because of tortuous blood vessels that compress the trigeminal root as it exits the brainstem (Fig. 10.1). Acoustic neuromas, cholesteatomas, aneurysms, angiomas, and bony abnormalities may also lead to compression of the nerve. The severity of the pain produced by trigeminal neuralgia is rivaled only by that of cluster headache. Uncontrolled pain has been associated with suicide and should therefore be treated as an emergency. Attacks can be triggered by daily activities involving contact with the face, such as brushing the teeth, shaving, and washing (Fig. 10.2). Pain can be controlled with medication in most patients. Approximately 2–3% of patients with trigeminal neuralgia also have multiple sclerosis. Trigeminal neuralgia is also called tic douloureux.

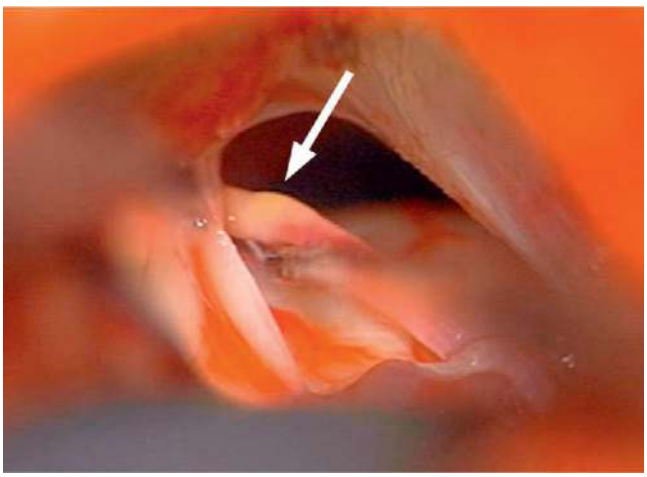

FIG 10.1 Left-sided microvascular decompression in a patient after two prior radiosurgery procedures. Note radiation-related atherosclerotic changes (indicated by *arrow*) in conflicting superior cerebellar artery held away from the nerve by a microdissection tool. (From Chen JCT. Microvascular decompression for trigeminal neuralgia in patients with and without prior stereotactic radiosurgery. *World Neurosurg.* 2012;78(1–2):149–154, Fig. 1.)

SIGNS AND SYMPTOMS

Trigeminal neuralgia causes episodic pain afflicting the areas of the face supplied by the trigeminal nerve. The pain is unilateral in 97% of cases; when it does occur bilaterally, the same division of the nerve is involved on both sides. The second or third division of the nerve is affected in most patients, and the first division is affected less than 5% of the time. The pain develops on the right side of the face in 57% of unilateral cases. A rare familial form of trigeminal neuralgia has been identified. The pain is characterized by paroxysms of electric shock–like pain lasting from several seconds to less than 2 minutes. The progression from onset to peak is essentially instantaneous.

Patients with trigeminal neuralgia go to great lengths to avoid any contact with trigger areas. In contrast, persons with other types of facial pain, such as temporomandibular joint dysfunction, tend to rub the affected area constantly or apply heat or cold to it. Patients with uncontrolled trigeminal neuralgia frequently require hospitalization for rapid control of pain. Between attacks, patients are relatively pain-free. A dull ache remaining after the intense pain subsides may indicate persistent compression of the nerve by a structural lesion. This disease is hardly ever seen in persons younger than 30 years unless it is associated with multiple sclerosis.

Patients with trigeminal neuralgia often have severe depression (sometimes to the point of being suicidal), with high levels of superimposed anxiety during acute attacks. Both these problems may be exacerbated by the sleep deprivation that often accompanies painful episodes. Patients with coexisting multiple sclerosis may exhibit the euphoric dementia characteristic of that disease. Physicians should reassure persons with trigeminal neuralgia that the pain can almost always be controlled.

TESTING

All patients with a new diagnosis of trigeminal neuralgia should undergo magnetic resonance imaging (MRI) of the brain and brainstem, with and without gadolinium contrast medium, to rule out posterior fossa or brainstem lesions and demyelinating disease (Fig. 10.3). Magnetic resonance

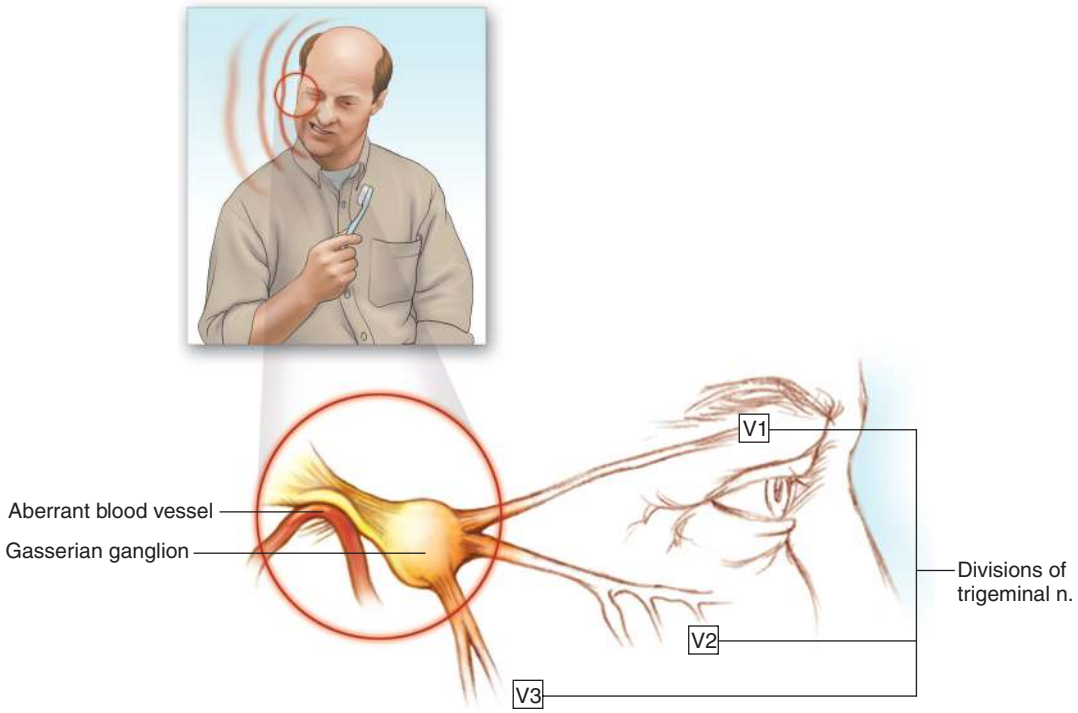

FIG 10.2 Paroxysms of pain triggered by brushing the teeth.

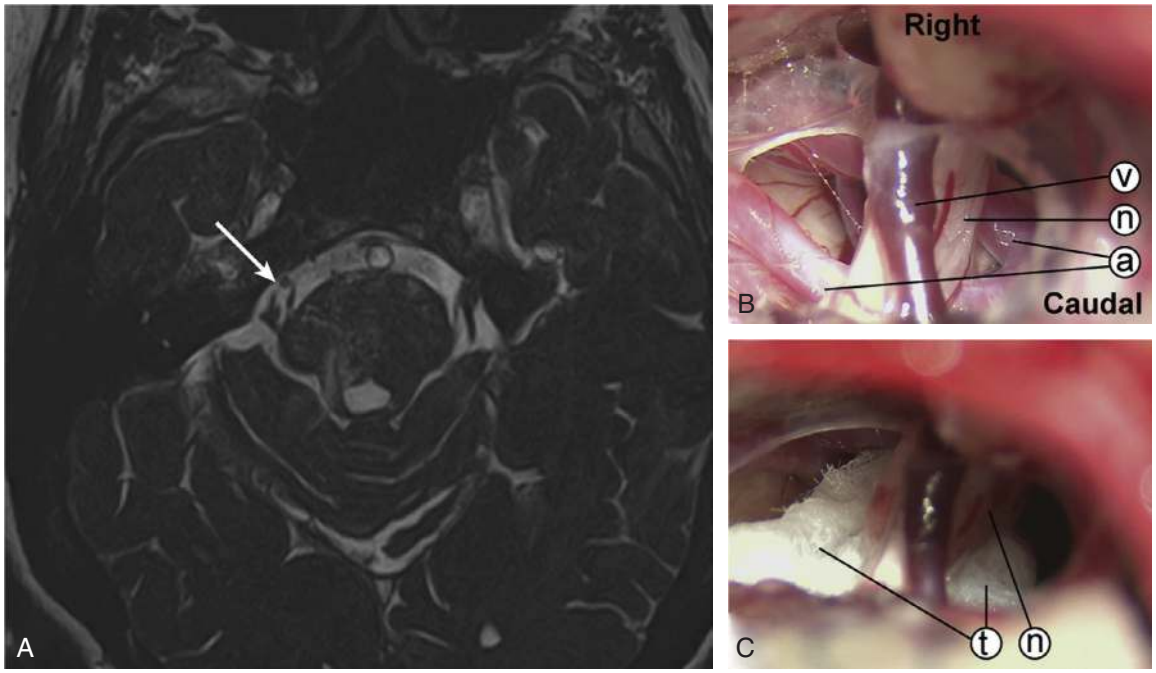

FIG 10.3 Example of concordant (true positive) magnetic resonance imaging (MRI) and intraoperative findings. The patient is an elderly woman with right trigeminal neuralgia (TN) for the past 2 years unresponsive to medication. **A,** MRI reveals a neurovascular conflict on the right side (*arrow*). **B,** Intraoperative photograph of the supracerebellar artery (a) ventral to the trigeminal nerve (n), with the petrosal vein (v) lying posterior to the nerve. **C,** Teflon sponge barrier (polytetrafluoroethene) (t) is inserted ventral to the nerve (n) from medial to lateral. The patient is relieved of TN. (From Hitchon PW, Bathla G, Moritani T, et al. Predictability of vascular conflict by MRI in trigeminal neuralgia. *Clin Neurol Neurosurg.* 2019;182:171–176.)

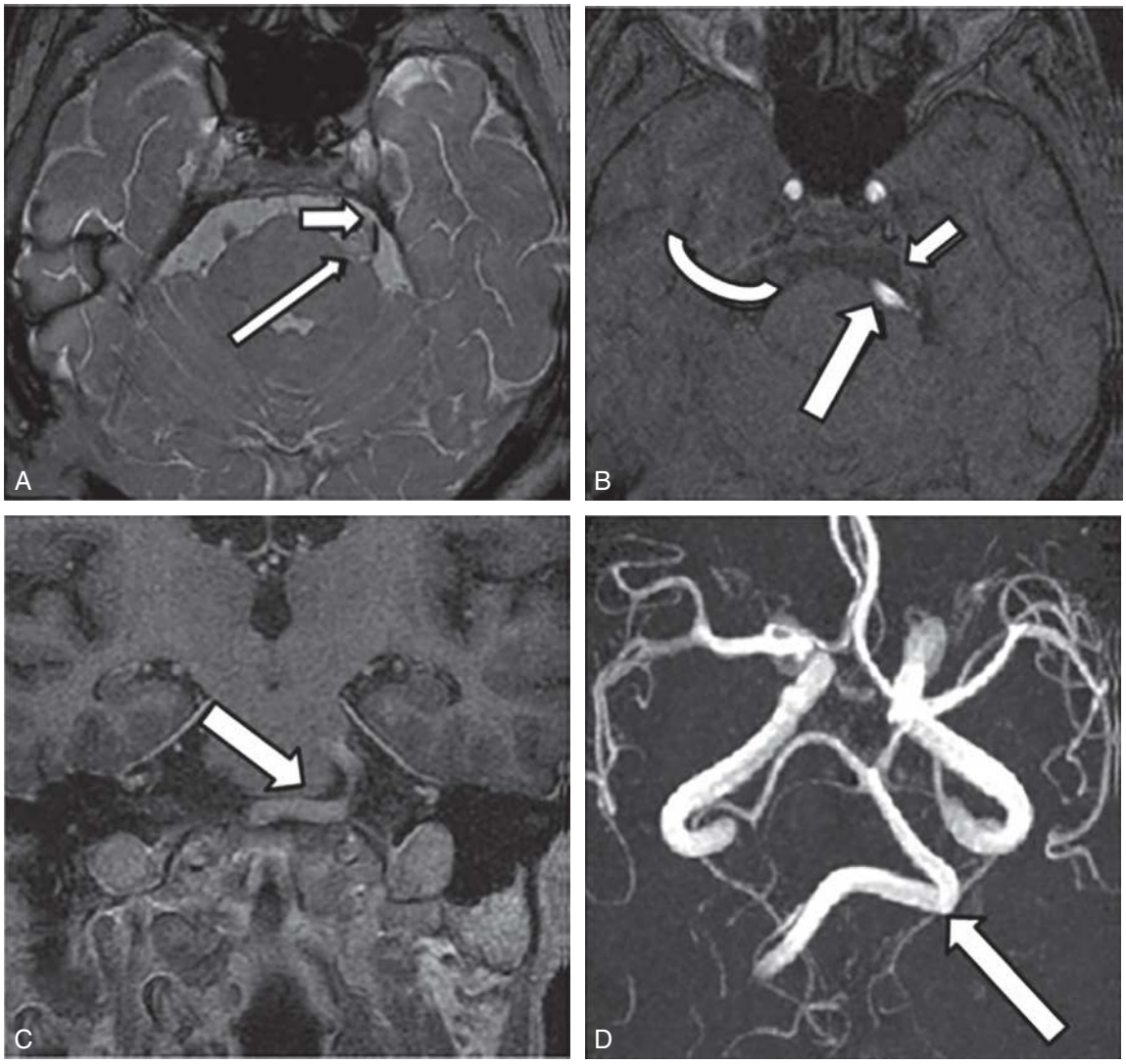

FIG 10.4 A 54-year-old male with left trigeminal neuralgia. Magnetic resonance imaging (MRI) of the brain trigeminal protocol showing dolichoectatic basilar artery (*long arrows*), touching the trigeminal nerve (*short arrows*). **A,** Axial constructive interference in steady state (CISS) of the cisteral portion. **B,** Magnetic resonance angiography (MRA); note the relatively diminished caliber of the trigeminal nerve when compared to the contralateral side (*curved arrow*). **C,** Postcontrast series in coronal plane and **(D)** MRA clearly showing the dolichoectatic course of the basilar artery (*arrows*). (From Geneidi EAS, Ali HI, Ghany WAA, et al. Trigeminal pain: potential role of MRI. *Egypt J Radiol Nucl Med*. 2016;47(4):1549–1555.)

angiography is also useful to confirm vascular compression of the trigeminal nerve by aberrant blood vessels (Fig. 10.4). Computed tomographic angiography may also be useful to clarify the role of aberrant blood vessels in the evolution of the patient's pain symptomatology (Fig. 10.5). Additional imaging of the sinuses should be considered if occult or coexisting sinus disease is a possibility. If the first division of the trigeminal nerve is affected, ophthalmologic evaluation to measure intraocular pressure and rule out intraocular disease is indicated. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry should be performed if the diagnosis of trigeminal neuralgia is in question. A complete blood count is required for baseline comparisons before starting treatment with carbamazepine (see "Treatment").

DIFFERENTIAL DIAGNOSIS

Trigeminal neuralgia is generally a straightforward clinical diagnosis that can be made on the basis of a targeted history and physical examination. Diseases of the eyes, ears, nose, throat, and teeth may all mimic trigeminal neuralgia or may coexist and confuse the diagnosis. Atypical facial pain is sometimes confused with trigeminal neuralgia, but it can be distinguished by the character of the pain: atypical facial

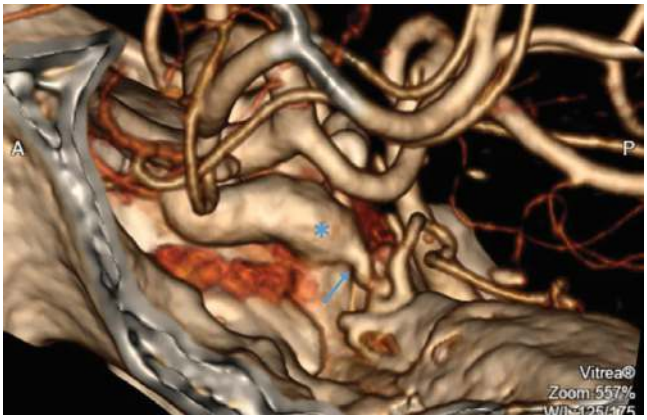

FIG 10.5 Lateral three-dimensional reformatted computed tomography angiography image demonstrating origin of persistent trigeminal artery variant (PTAV) (*arrow*) from the cavernous segment of the inferior carotid artery (ICA) (*asterisk*), proximal to the posterior genu. The tortuous PTAV is seen coursing posteriorly and eventually supplies the anterior ICA territory. (From Ling MM, Gupta M, Acharya J. Trigeminal neuralgia associated with a variant of persistent trigeminal artery. *Radiol Case Rep.* 2020;15(11):2225–2228, Fig. 3. ISSN 1930-0433.)

pain is dull and aching, whereas the pain of trigeminal neuralgia is sharp and neuritic. Additionally, the pain of trigeminal neuralgia occurs in the distribution of the divisions of the trigeminal nerve, whereas the pain of atypical facial pain does not follow any specific nerve distribution. Multiple sclerosis should be considered in all patients who present with trigeminal neuralgia before the fifth decade of life.

TREATMENT

Drug Therapy

Fosphenytoin

Fosphenytoin is a water-soluble phenytoin prodrug that can be given intravenously. It has been used in the management of intractable seizures and has been shown to be useful in the management of severe, uncontrolled pain of trigeminal neuralgia that has responded to other treatment modalities. The drug is diluted with normal saline at a ratio of 15 mg/mL of normal saline and administered at a rate of 50 mg/min with a dosage not to exceed a total of 750 mg. Dizziness is the most common reported side effect of this treatment modality.

Carbamazepine

Carbamazepine is considered the first-line treatment for trigeminal neuralgia. In fact, a rapid response to this drug essentially confirms the clinical diagnosis. Despite the safety and efficacy of carbamazepine, some confusion and anxiety have surrounded its use. This medication, which may be the patient's best chance for pain control, is sometimes discontinued because of laboratory abnormalities erroneously attributed to it. Therefore baseline measurements consisting of a complete blood count, urinalysis, and automated blood chemistry profile should be obtained before starting the drug.

Carbamazepine should be initiated slowly if the pain is not out of control, with a starting dose of 100–200 mg at bedtime for 2 nights. The patient should be cautioned about side effects, including dizziness, sedation, confusion, and rash. The drug is increased in 100- to 200-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 1200 mg/day is reached. Careful monitoring of laboratory parameters is mandatory to avoid the rare possibility of a life-threatening blood dyscrasia. At the first sign of blood count abnormality or rash, this drug should be discontinued. Failure to monitor patients who are taking carbamazepine can be disastrous, because aplastic anemia can occur. When pain relief is obtained, the patient should be kept at that dosage of carbamazepine for at least 6 months before considering tapering the medication. The patient should be informed that under no circumstances should the drug dosage be changed or the drug refilled or discontinued without the physician's knowledge.

Gabapentin

In the uncommon event that carbamazepine does not adequately control a patient's pain, gabapentin may be considered. As with carbamazepine, baseline blood tests should be obtained before starting therapy and the patient should be cautioned about potential side effects, including dizziness, sedation, confusion, and rash. The initial dose of gabapentin is 300 mg at bedtime for 2 nights. The drug is then increased in 300-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 2400 mg/day is reached. At this point, if the patient has experienced only partial pain relief, blood values are measured and the drug is carefully titrated upward using 100-mg tablets. Rarely is a dosage greater than 3600 mg/day required.

Baclofen

Baclofen may be of value in some patients who fail to obtain relief from carbamazepine or gabapentin. As with those drugs, baseline laboratory tests should be obtained before beginning baclofen therapy and the patient should be warned about the same potential adverse effects. The patient starts with a 10-mg dose at bedtime for 2 nights; then, the drug is increased in 10-mg increments given in equally divided doses over 7 days, as side effects allow, until pain relief is obtained or a total dose of 100 mg/day is reached. This drug has significant hepatic and central nervous system side effects, including weakness and sedation. As with carbamazepine, careful monitoring of laboratory values is indicated when using baclofen.

When treating individuals with any of these drugs, the physician should make sure that the patient knows that premature tapering or discontinuation of the medication may lead to the recurrence of pain, which will be more difficult to control.

Invasive Therapy

Trigeminal Nerve Block

The use of trigeminal nerve block with local anesthetic and steroid is an excellent adjunct to drug treatment of trigeminal neuralgia. This technique rapidly relieves pain while medications are being titrated to effective levels. The initial block is carried out with preservative-free bupivacaine combined with methylprednisolone. Subsequent daily nerve blocks are performed in a similar manner but using a lower dose of methylprednisolone. This approach may also be used to control breakthrough pain.

Retrogasserian Injection of Glycerol

The injection of small quantities of glycerol into the area of the gasserian ganglion can provide long-term relief for patients suffering from trigeminal neuralgia who have not responded to optimal drug therapy. This procedure should be performed only by a physician well versed in the problems and pitfalls associated with neurodestructive procedures (Fig. 10.6).

Radiofrequency Destruction of the Gasserian Ganglion

The gasserian ganglion can be destroyed by creating a radiofrequency lesion under biplanar fluoroscopic guidance. This procedure is reserved for patients in whom all the previously mentioned treatments for intractable trigeminal neuralgia have failed and who are not candidates for microvascular decompression of the trigeminal root.

Balloon Compression of the Gasserian Ganglion

The insertion of a balloon by a needle placed through the foramen ovale into Meckel's cave under radiographic guidance is a straightforward technique (Fig. 10.7). Once the balloon is

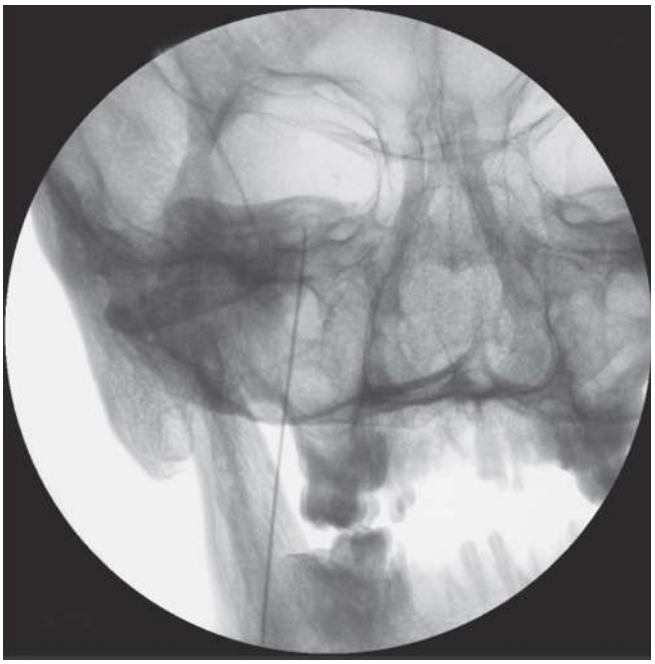

FIG 10.6 Fluoroscopic image demonstrating a needle placed through the foramen ovale into Meckel's cave.

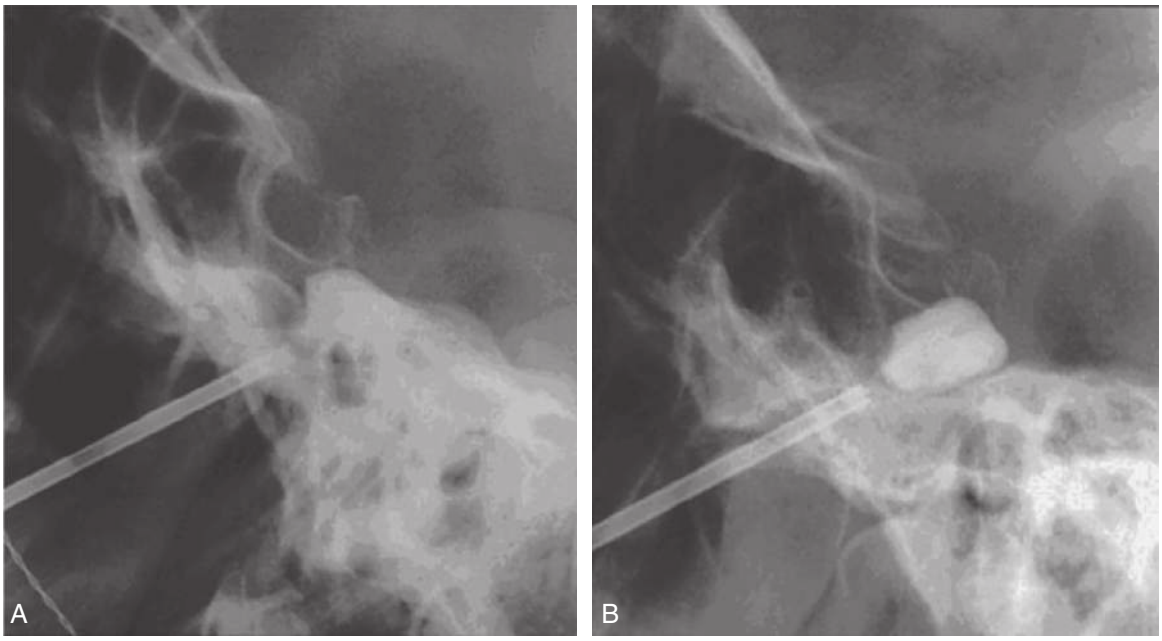

FIG 10.7 Balloon compression of the gasserian ganglion. Lateral fluoroscopic view demonstrating inflated balloon within Meckel cave. (From Goerss SJ, Atkinson JL, Kallmes DF. Variable size percutaneous balloon compression of the gasserian ganglion for trigeminal neuralgia. *Surg Neurol.* 2009;71[3]:388–390.)

in proximity to the gasserian ganglion, it is inflated to compress the ganglion. This technique has been shown to provide palliation of trigeminal neural pain in selected candidates in whom medication management has failed and who are not candidates for more invasive procedures.

Microvascular Decompression of the Trigeminal Root

This technique, which is also called Jannetta's procedure, is the major neurosurgical treatment of choice for intractable trigeminal neuralgia (Fig. 10.8). It is based on the theory that trigeminal neuralgia is in fact a compressive mononeuropathy. The operation consists of identifying the trigeminal root close to the brainstem and isolating the compressing blood vessel. A sponge is then interposed between the vessel and the nerve, to relieve the compression and thus the pain.

Gamma Knife

Gamma knife is a painless, outpatient procedure that uses the focused emission of gamma rays from a cobalt source to destroy the area anterior to the junction of the trigeminal nerve and the pons, the trigeminal nerve entry site immediately adjacent to the pons, the midposterior

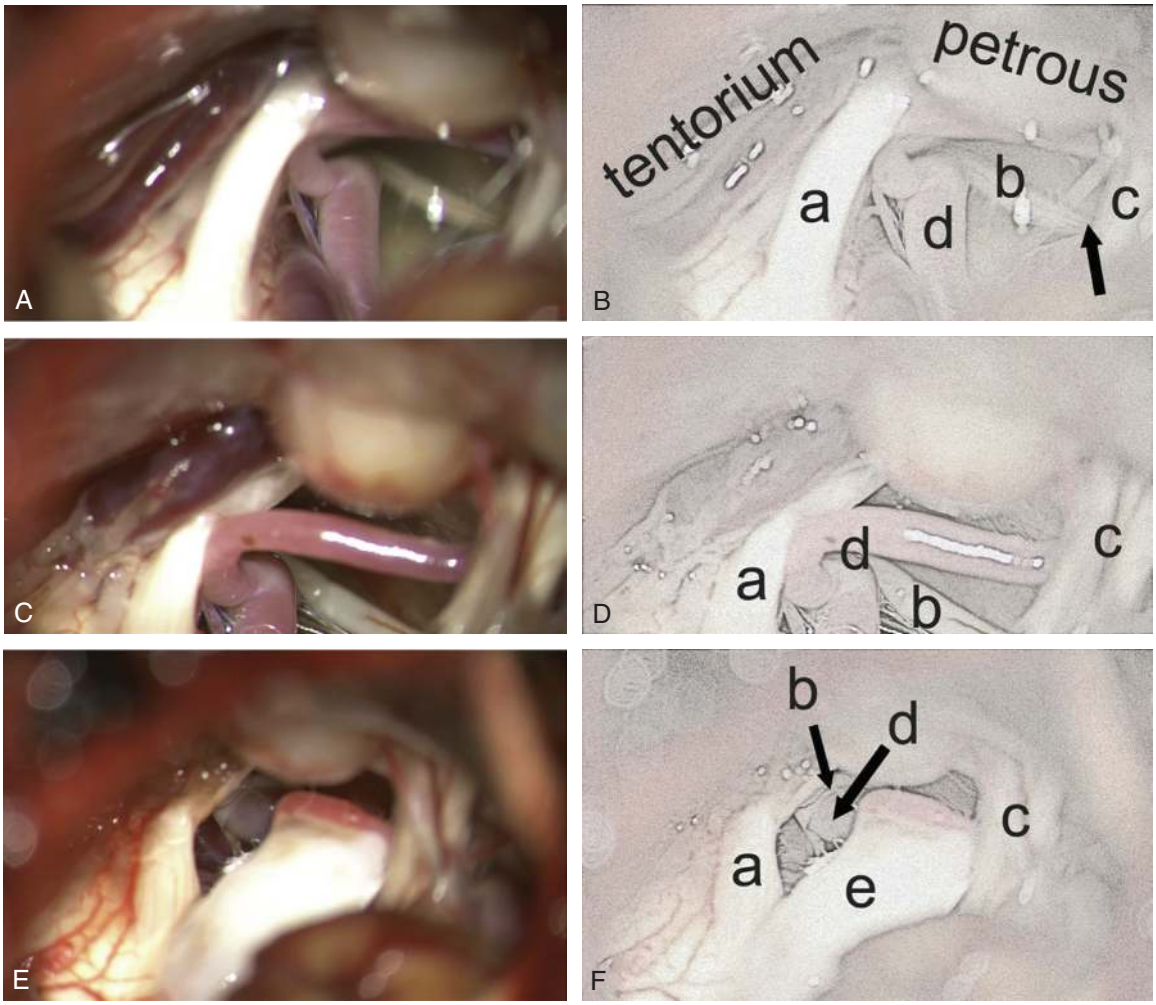

FIG 10.8 Intraoperative microscope photographs during microvascular decompression of the right trigeminal nerve. (Left column) Three intraoperative microscope photographs at various stages of the procedure. (Right column) Identical faded photos to assist with labeling of structures. (Top row) The superior aspect of the right cerebellopontine angle has been exposed revealing the tentorium, petrous, and trigeminal nerve (a) that is clearly compressed between a vein superiorly and a tortuous anterior inferior cerebellar artery (AICA) (d) inferiorly. The seventh and eighth nerve complex (c) can be seen inferiorly, toward the right of the frame. The sixth nerve (b) can be seen through the arachnoid after looping around the tortuous AICA as it descends inferiorly toward the Dorello canal (*arrow*). (Middle row) The sixth nerve (b) is more clearly seen after arachnoid release and severe compression of the trigeminal nerve (a) by the AICA (d) is more readily appreciated. (Bottom row) The AICA and its branches (d) have been mobilized inferiorly and stuck to the pons below the trigeminal nerve with Teflon and fibrin glue (e). The loop of the abducent nerve around the AICA can be seen deep to the severely dented but now well-decompressed trigeminal nerve (a). (From Borg A, Zrinzo L. Aberrant abducent nerve during microvascular decompression for trigeminal neuralgia. *World Neurosurg*. 2020;138:454–456.)

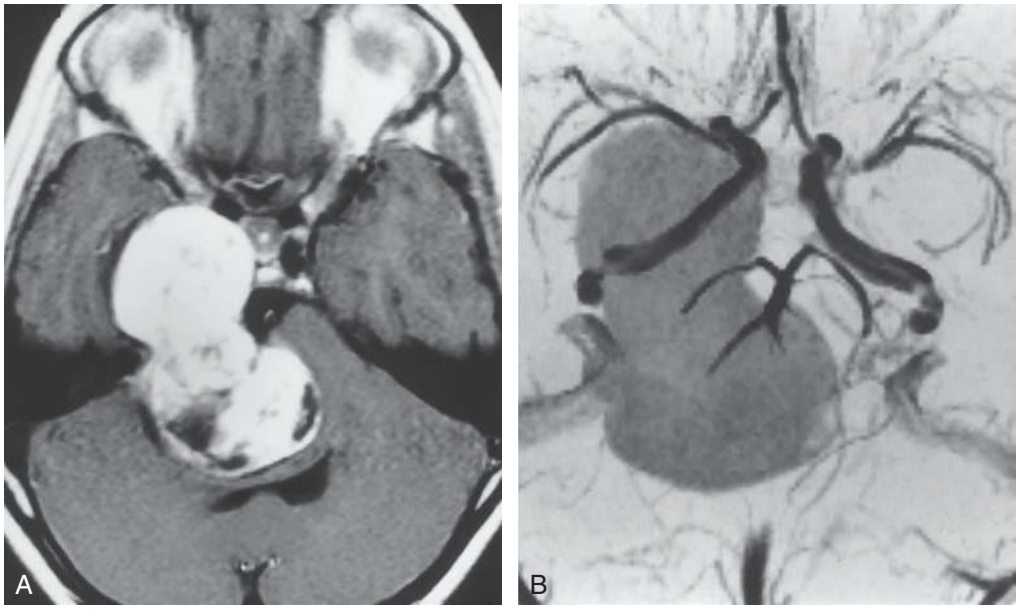

FIG 10.9 Cystic and solid schwannoma of the right trigeminal nerve and ganglion. **A,** Axial enhanced magnetic resonance imaging (MRI) showing a dumbbell-shaped tumor extending across the incisura from the posterior fossa into the medial portion of the right middle fossa. Note the heterogeneous enhancement of the tumor that suggests areas of decreased cellularity and cystic change and a more solid component. **B,** Axial magnetic resonance angiogram performed after the MRI examination showing near-homogeneous enhancement of the tumor because of the delay in imaging. Note the exquisite demonstration of the tumor in the skull base, including the displaced right petrous carotid artery. (From Stark DD, Bradley WG Jr, eds. *Magnetic resonance imaging*. Vol 3. 3rd ed. St Louis: Mosby; 1999:1218.)

portion of the trigeminal nerve, or the cisternal segment of the trigeminal nerve. Complications include facial numbness and sensory deficit.

COMPLICATIONS AND PITFALLS

The pain of trigeminal neuralgia is severe and can lead to suicide. Therefore it must be considered a medical emergency, and strong consideration should be given to hospitalizing such patients. If a dull ache remains after the intense pain of trigeminal neuralgia subsides, this is highly suggestive of persistent compression of the nerve by a structural lesion such as a brainstem tumor or schwannoma (Figs. 10.9 and 10.10). Trigeminal neuralgia is hardly ever seen in persons younger than 30 years unless it is associated with multiple sclerosis, and all such patients should undergo MRI to identify demyelinating disease.

CLINICAL PEARLS

Trigeminal nerve block with local anesthetic and steroid is an excellent stopgap measure for patients suffering from the uncontrolled pain of trigeminal neuralgia while waiting for drug treatments to take effect. This technique may lead to the rapid control of pain and allow the patient to maintain adequate oral hydration and nutrition and avoid hospitalization.

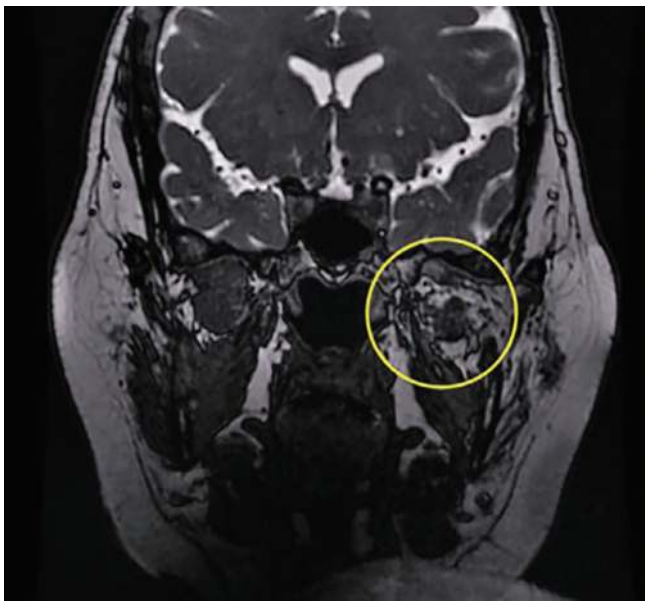

FIG 10.10 Coronal T2-weighted at suppressed magnetic resonance image without contrast showing (*yellow circle*) metastatic infiltration of approximately 20 mm in length, on the prepontine cistern, porous acousticus, and Meckel cave. Moderately enhancing fragmented areas of necrosis are noted. (From Thomas DC, Singer SR, Pitchumani PK, et al. Facial pain and trigeminal neuralgia secondary to metastasis: a case report. *J Am Dent Assoc*. 2022;153(5):484–488.)

SUGGESTED READINGS

Boeddinghaus R, Whyte A. Imaging of trigeminal neuralgia and other facial pain. *Neuroimaging Clin North Am*. 2021;31(4):485–508.

Jiao L, Ye H, Lv J, et al. A systematic review of repeat microvascular decompression for recurrent or persistent trigeminal neuralgia. *World Neurosurg*. 2022;158:226–233.

Lv W, Hu Q, Chi L, Zhang L. Factors that may affect recurrence of trigeminal neuralgia after percutaneous balloon compression. *J Clin Neurosci*. 2022;99:248–252.

Noro S, Seo Y, Honjo K, et al. Intravenous fosphenytoin therapy for rescue of acute trigeminal neuralgia crisis in patients awaiting neurosurgical procedures: a cross-sectional study. *J Clin Neurosci*. 2021;94:59–64.

Sun J, Wang M, Zhang L, Yu Y. A meta-analysis of the effectiveness and safety of microvascular decompression in elderly patients with trigeminal neuralgia. *J Clin Neurosci*. 2022;99:22–34.

Thomas DC, Singer SE, Pitchumani PK, et al. Facial pain and trigeminal neuralgia secondary to metastasis: a case report. *J Am Dent Assoc*. 2022;153(5):484–488.

Waldman SD. Gasserian ganglion block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:33–39.

Waldman SD. Gasserian ganglion block: balloon compression technique. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:44–47.

Waldman SD. Gasserian ganglion block: radiofrequency lesioning. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:40–43.

Temporomandibular Joint Dysfunction

ICD-10 CODE M26.60

THE CLINICAL SYNDROME

Temporomandibular joint (TMJ) dysfunction (also known as myofascial pain dysfunction of the muscles of mastication) is characterized by pain in the joint itself that radiates into the mandible, ear, neck, and tonsillar pillars. The TMJ is a true joint that is divided into upper and lower synovial cavities by a fibrous articular disk (Fig. 11.1). Internal derangement of this disk may result in pain and TMJ dysfunction, but extracapsular causes of TMJ pain are much more common (Box 11.1). The TMJ is innervated by branches of the mandibular nerve. The muscles involved in TMJ dysfunction often include the temporalis, masseter, and external and internal pterygoids; the trapezius and sternocleidomastoid may be involved as well.

SIGNS AND SYMPTOMS

Headache often accompanies the pain of TMJ dysfunction and is clinically indistinguishable from tension-type headache. Stress is often the precipitating factor or an exacerbating factor in the development of TMJ dysfunction (Fig. 11.2). A history of bruxism or jaw clenching is often present (Fig. 11.3).

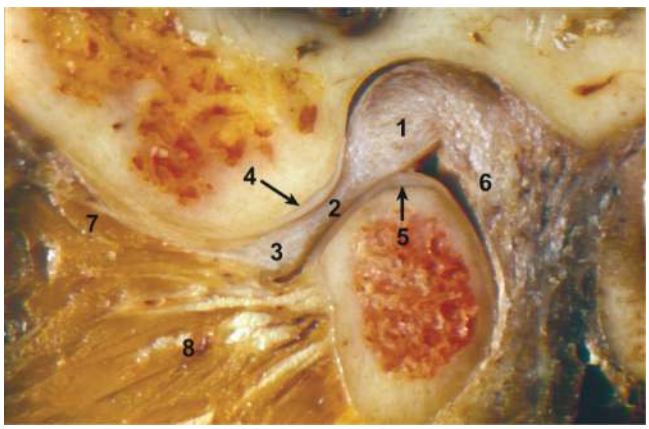

FIG 11.1 Anatomic section through a temporomandibular joint. **1,** Pars posterior; **2,** pars intermediate; **3,** pars anterior; **4, 5,** fibrocartilage covering the condyle and fossa; **6,** inferior lamina of the posterior attachment; **7, 8,** superior and inferior head of the lateral pterygoid muscle. (From Klineberg I, Eckert S. *Functional occlusion in restorative dentistry and prosthodontics*. St Louis: Mosby; 2016:67–85.)

Dental malocclusion may also play a role in its evolution. Internal derangement and arthritis of the TMJ may manifest as clicking or grating when the mouth is opened and closed. If the condition is untreated, the patient may experience increasing pain in the aforementioned areas, as well as limitation of jaw movement and mouth opening.

Trigger points may be identified when palpating the muscles involved in TMJ dysfunction. Crepitus on range of motion of the joint suggests arthritis rather than dysfunction of myofascial origin. In severe cases, deviation of the mandible may occur (Fig. 11.4).

TESTING

Radiographs of the TMJ are usually within normal limits in patients suffering from TMJ dysfunction, but they may be useful to help identify inflammatory or degenerative

BOX 11.1 Causes and Mimics of Temporomandibular Joint Pain

- Intraarticular disorders
- Osteoarthritis
- Rheumatoid arthritis
- Psoriatic arthritis
- Ankylosing spondylitis
- Reiter syndrome
- Crystal arthropathies
- Intraarticular disk derangement
- Disk displacement
- Disk tearing
- Trauma
- Intraarticular hemorrhage
- Contusion
- Bruising
- Temporal arteritis
- Tumor
- Infection
- Congenital disorders
- Postradiation fibrosis
- Trismus
- Reflex sympathetic dystrophy of the face
- Disorders involving muscles of mastication
- Fibromyalgia
- Myositis
- Tenosynovitis
- Muscle spasm
- Bruxism

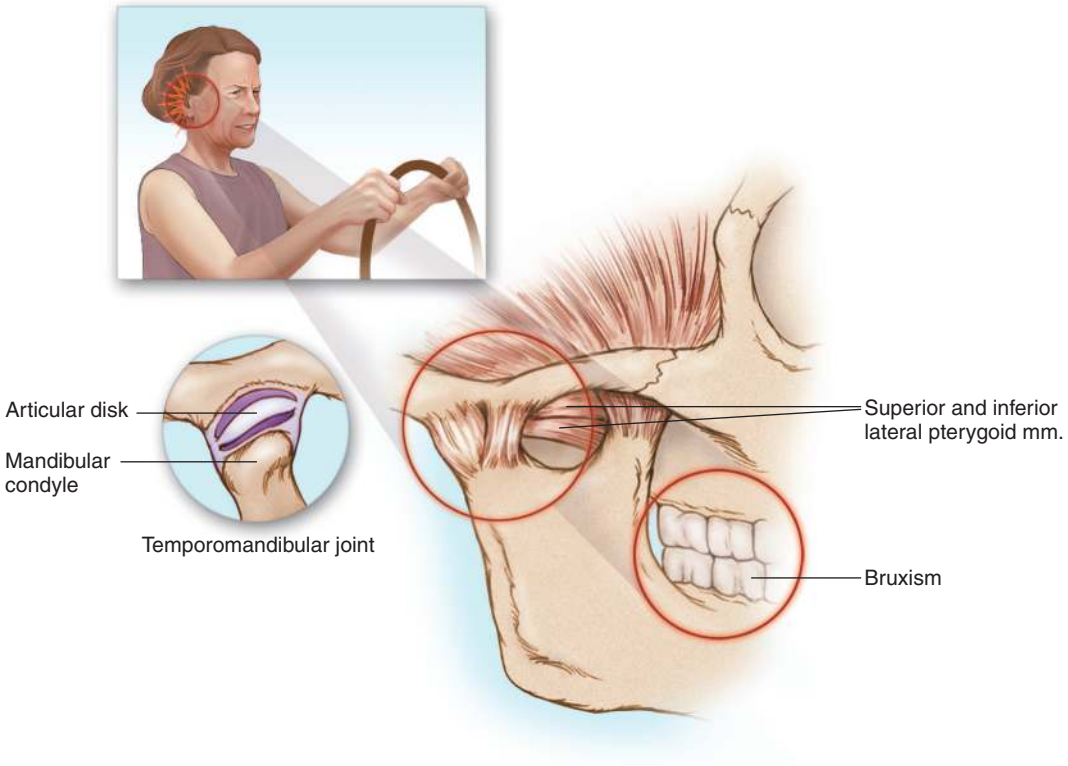

FIG 11.2 Stress is often a trigger for temporomandibular joint dysfunction.

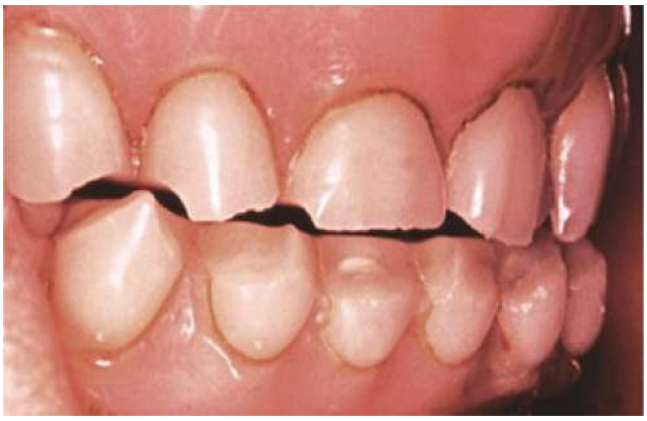

FIG 11.3 Damage to the occlusal surfaces secondary to bruxism. (From Johansson A, Omar R, Carlsson GE. Bruxism and prosthetic treatment: a critical review. *J Prosthodont Res*. 2011;55(3):127–136.)

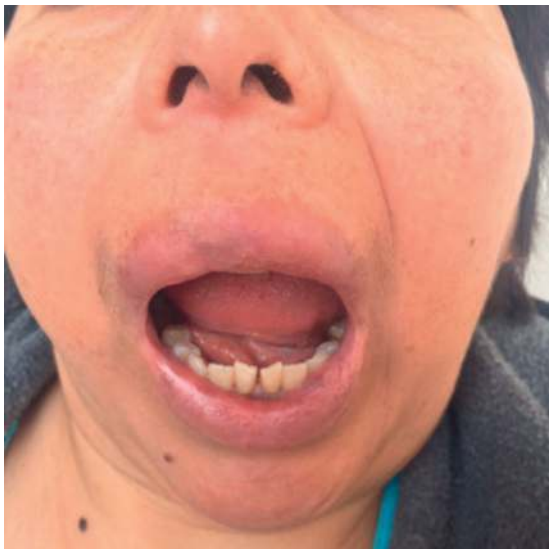

FIG 11.4 Deviation of the mandible in a patient with temporomandibular joint dysfunction secondary to rheumatoid arthritis. (Published with permission of the Publisher. Original source: Ibáñez-Mancera NG, Vinitzky-Brener I, Muñoz-López S, et al. Disfunción de la articulación temporomandibular en pacientes con artritis reumatoide. *Esp Cir Oral Maxilofac*. 2017;39(2):85–90.)

arthritis of the joint as well as crystal deposition diseases (Fig. 11.5). Arthroscopy, arthography, and computerized tomographic imaging of the joint can help the clinician identify derangement of the disk, as well as other abnormalities of the joint itself (Figs. 11.6–11.8). Magnetic resonance imaging may provide more detailed information regarding the condition of the disk and articular surface and should be considered in complicated cases (Fig. 11.9). A complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing are indicated if inflammatory arthritis or temporal arteritis is suspected. Injection of the joint with small amounts of local anesthetic can serve as a diagnostic maneuver to determine whether the TMJ is in fact the source of the patient's pain (Fig. 11.10).

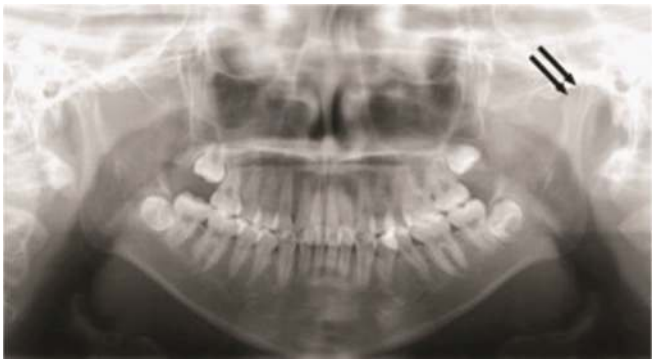

FIG 11.5 Panoramic radiograph. Irregularity of surface (erosion) (*black arrows*) is seen on upper portion of left condyle, indicating osteoarthritis. However, the patient did not complain of pain in left temporomandibular joint. (From Sano T, Yajima A, Otonari-Yamamoto M, et al. Interpretation of images and discrepancy between osteoarthritic findings and symptomatology in temporomandibular joint. *Jpn Dent Sci Rev*. 2008;44(1):83–89.)

DIFFERENTIAL DIAGNOSIS

The clinical symptoms of TMJ dysfunction may be confused with pain of dental or sinus origin or may be characterized as atypical facial pain (see Box 11.1). With careful questioning and physical examination, however, the clinician can usually distinguish these overlapping pain syndromes. Tumors of the zygoma and mandible, as well as retropharyngeal tumors, may produce ill-defined pain attributed to the TMJ, and these potentially life-threatening diseases must be excluded in any patient with facial pain. Reflex sympathetic dystrophy of the face should also be considered in any patient presenting with ill-defined facial pain after trauma, infection, or central nervous system injury. The pain of TMJ dysfunction is dull and aching, whereas the pain of reflex sympathetic dystrophy of the face is burning, with significant allodynia often present. Stellate ganglion block may help distinguish the two pain syndromes, because the pain

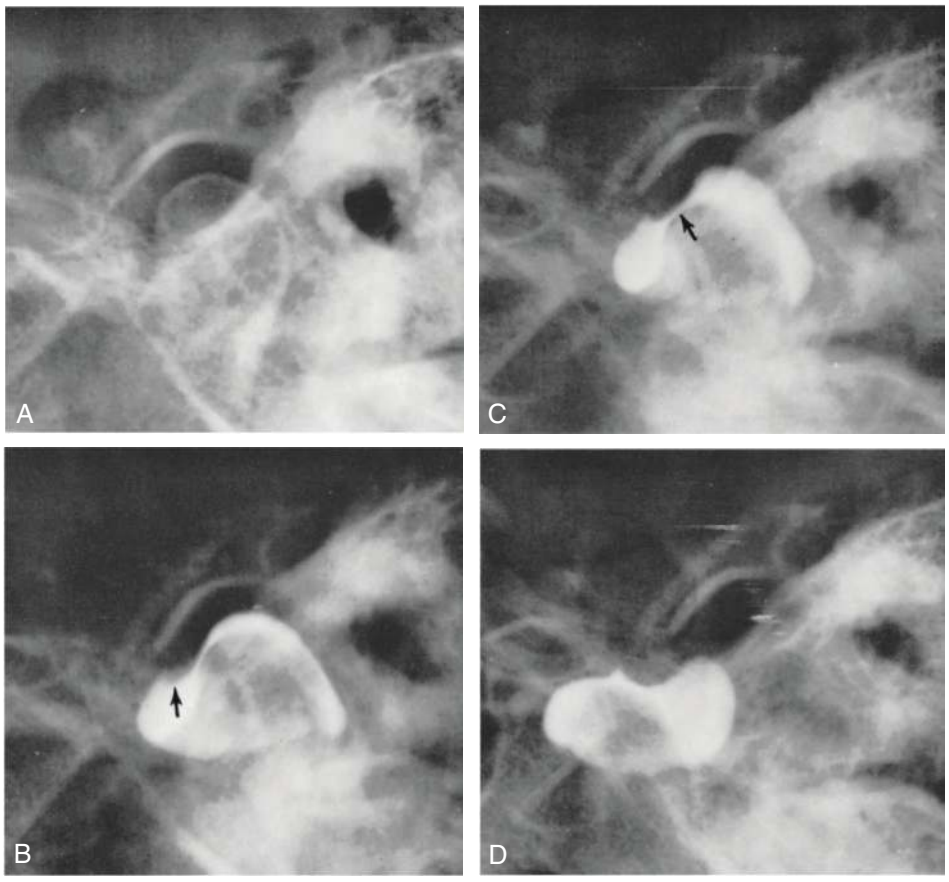

FIG 11.6 Arthrography of an abnormal temporomandibular joint showing disk dislocation with reduction in a 20-year-old woman with clicking and intermittent pain. **A,** Magnification transcranial radiograph with the mouth closed shows normal osseous anatomy and isocentric condyle position in the mandibular fossa. **B,** With the mouth closed, contrast agent fills the inferior joint space and outlines the undersurface of the disk. The posterior band of the disk is located anterior to the condyle (*arrow*) and bulges prominently in the anterior recess. This appearance is diagnostic of anterior dislocation of the disk. **C,** With the mouth half open, contrast agent has been redistributed, and the condyle has moved onto the posterior band (*arrow*), which is now compressed between the condyle and the eminence. **D,** With the mouth fully open, the condyle has translated anterior to the eminence; in so doing, it has crossed the prominent, thick posterior band and is causing a click. The posterior band is now in a normal position posterior to the condyle. (From Resnick D. Diagnosis of bone and joint disorders. 4th ed. Philadelphia: Saunders; 2002:1723.)

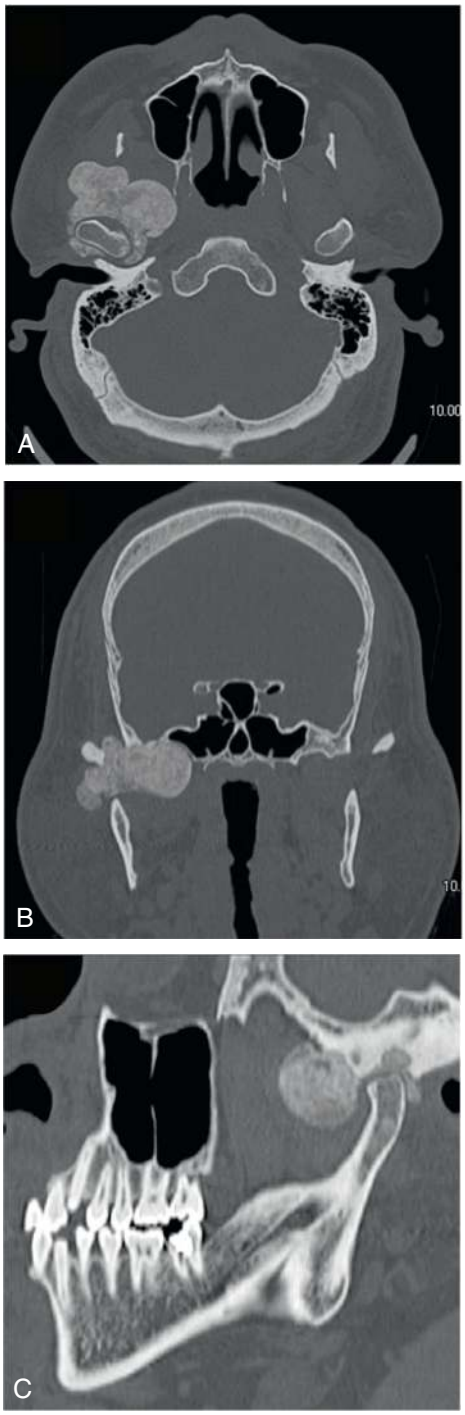

FIG 11.7 Computerized tomography (CT) imaging showing a large calcified mass around the right temporomandibular joint (TMJ). **A,** Axial CT scan showing a ring-shaped calcified mass around the condylar process of the right TMJ; the mass is not continuous with the mandibular condyle. **B,** Coronal CT scan revealing a calcified mass in the joint space; bone resorption and thinning of the middle cranial base are present and the lesion appears to extend into the middle cranial fossa. **C,** Sagittal CT scan of the right TMJ; the calcified mass limits condylar head movement. (From Kudoh K, Kudoh T, Tsuru K, et al. A case of tophaceous pseudogout of the temporomandibular joint extending to the base of the skull. *Int J Oral Maxillofac Surg*. 2017;46(3):355–359, Fig. 1.)

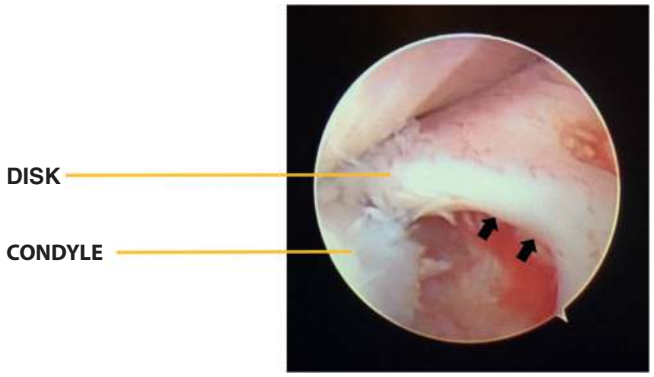

FIG 11.8 Arthroscopic view of left temporomandibular joint in a patient with severe masticatory parafunction. Osteoarthritis with disk perforation and exposed condyle. *Black arrow* indicates disk perforation. (From Israel HA. Internal derangement of the temporomandibular joint: new perspectives on an old problem. *Oral Maxillofac Surg Clin North Am*. 2016;28(3): 313–333, Fig. 4.)

of reflex sympathetic dystrophy of the face readily responds to this sympathetic nerve block, whereas the pain of TMJ dysfunction does not. In addition, the pain of TMJ dysfunction must be distinguished from the pain of jaw claudication associated with temporal arteritis.

TREATMENT

The mainstay of therapy is a combination of drug treatment with tricyclic antidepressants, physical modalities such as oral orthotic devices and physical therapy, and intraarticular injection of the joint with small amounts of local anesthetic and steroid. Antidepressant compounds such as nortriptyline at a single bedtime dose of 25 mg can help alleviate sleep disturbance and treat any underlying myofascial pain syndrome. Orthotic devices help the patient avoid jaw clenching and bruxism, which may exacerbate the clinical syndrome. Intraarticular injection with local anesthetic combined with steroid is useful to palliate acute pain to allow physical therapy, as well as to treat joint arthritis that may contribute to the patient's pain and joint dysfunction. The intraarticular injection of platelet-rich plasma may also be beneficial in the treatment of TMJ dysfunction (see Fig. 11.10). Clinical studies suggest that the injection of type A botulinum toxin into the temporalis and masseter muscles may also provide symptomatic relief. Rarely, surgical treatment of the displaced intraarticular disk is required to restore the joint to normal function and reduce pain.

For intraarticular injection of the TMJ, the patient is placed in the supine position with the cervical spine in the neutral position. The TMJ is identified by asking the patient to open and close the mouth several times and palpating the area just anterior and slightly inferior to the acoustic auditory meatus. After the joint is identified, the patient is asked to hold his or her mouth in the neutral position. A total of 0.5 mL of local anesthetic is drawn up in a 3-mL sterile

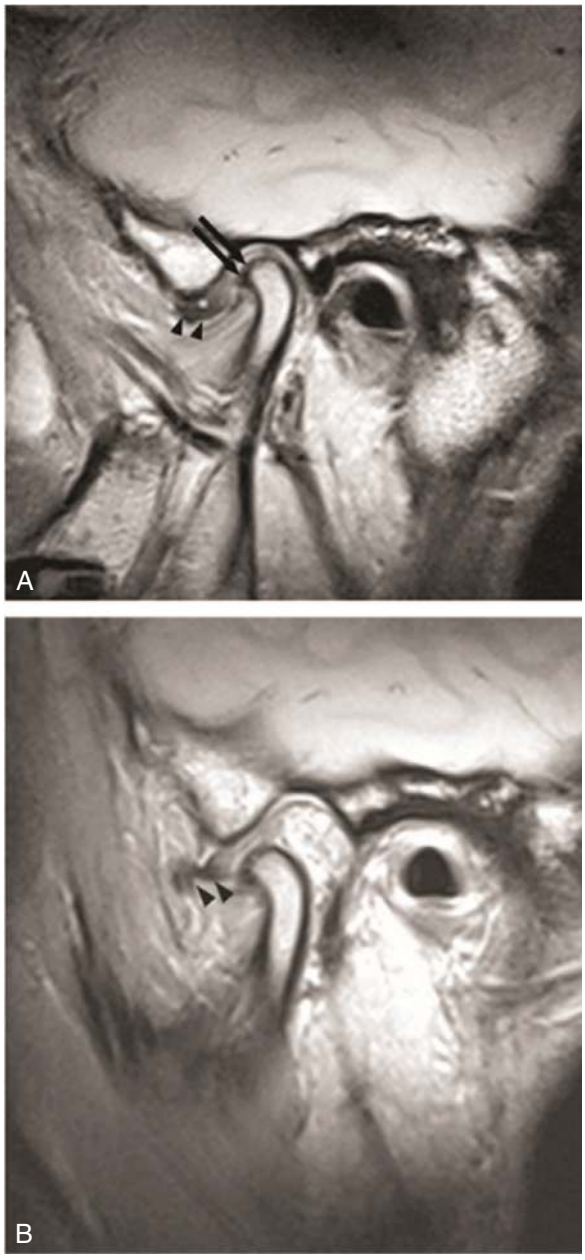

FIG 11.9 Magnetic resonance images. Parasagittal proton density-weighted closed-mouth image **(A)** shows anterior osteophyte of condyle (*black arrows*). Disk (*black arrowheads*) is anterior of condyle. On mouth opening **(B)**, disk (*black arrowheads*) remained anterior. This was consistent with osteoarthritis associated with disk displacement without reduction. (From Sano T, Yajima A, Otonari-Yamamoto M, et al. Interpretation of images and discrepancy between osteoarthritic findings and symptomatology in temporomandibular joint. *Jap Dent Sci Rev*. 2008;44(1):83–89.)

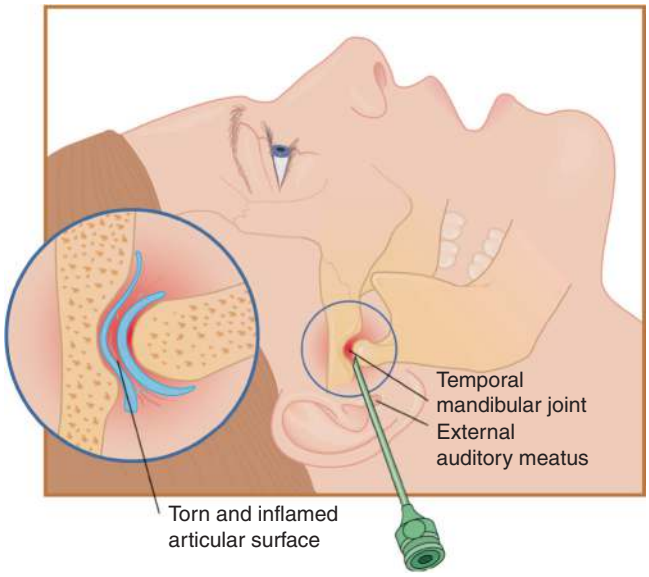

FIG 11.10 Correct needle placement for injections of the temporomandibular joint. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:5.)

syringe. When treating TMJ dysfunction, internal derangement of the TMJ, or arthritis or other painful conditions involving the TMJ, a total of 20 mg methylprednisolone is added to the local anesthetic with the first block; 10 mg methylprednisolone is added to the local anesthetic with subsequent blocks. After the skin overlying the TMJ is prepared with antiseptic solution, a 1-inch, 25-gauge styleted needle is inserted just below the zygomatic arch directly in the middle of the joint space. The needle is advanced approximately ¼–¾ inch in a plane perpendicular to the skull until a pop is felt, indicating that the joint space has been entered (see Fig. 11.10). After careful aspiration, 1 mL of solution is slowly injected. Injection of the joint may be repeated at 5- to 7-day intervals if symptoms persist. Ultrasound needle guidance may improve the accuracy of needle placement when performing injection of the TMJ (Fig. 11.11).

COMPLICATIONS AND PITFALLS

The vascularity of the region and the proximity to major blood vessels lead to an increased incidence of postblock ecchymosis and hematoma formation, and the patient should be warned of this potential complication. Despite the region's vascularity, intraarticular injection can be performed safely (albeit with an increased risk of hematoma formation) in the presence of anticoagulation by using a 25- or 27-gauge needle, if the clinical situation indicates a favorable risk-to-benefit ratio. These complications can be decreased if manual pressure is applied to the area of the block immediately after injection. The application of cold packs for 20 minutes after the block also decreases the amount of postprocedural pain and bleeding. Another complication that occurs with some frequency is inadvertent block of the facial nerve, with associated facial weakness. When this occurs, protection of the cornea with sterile ophthalmic lubricant and patching is mandatory.

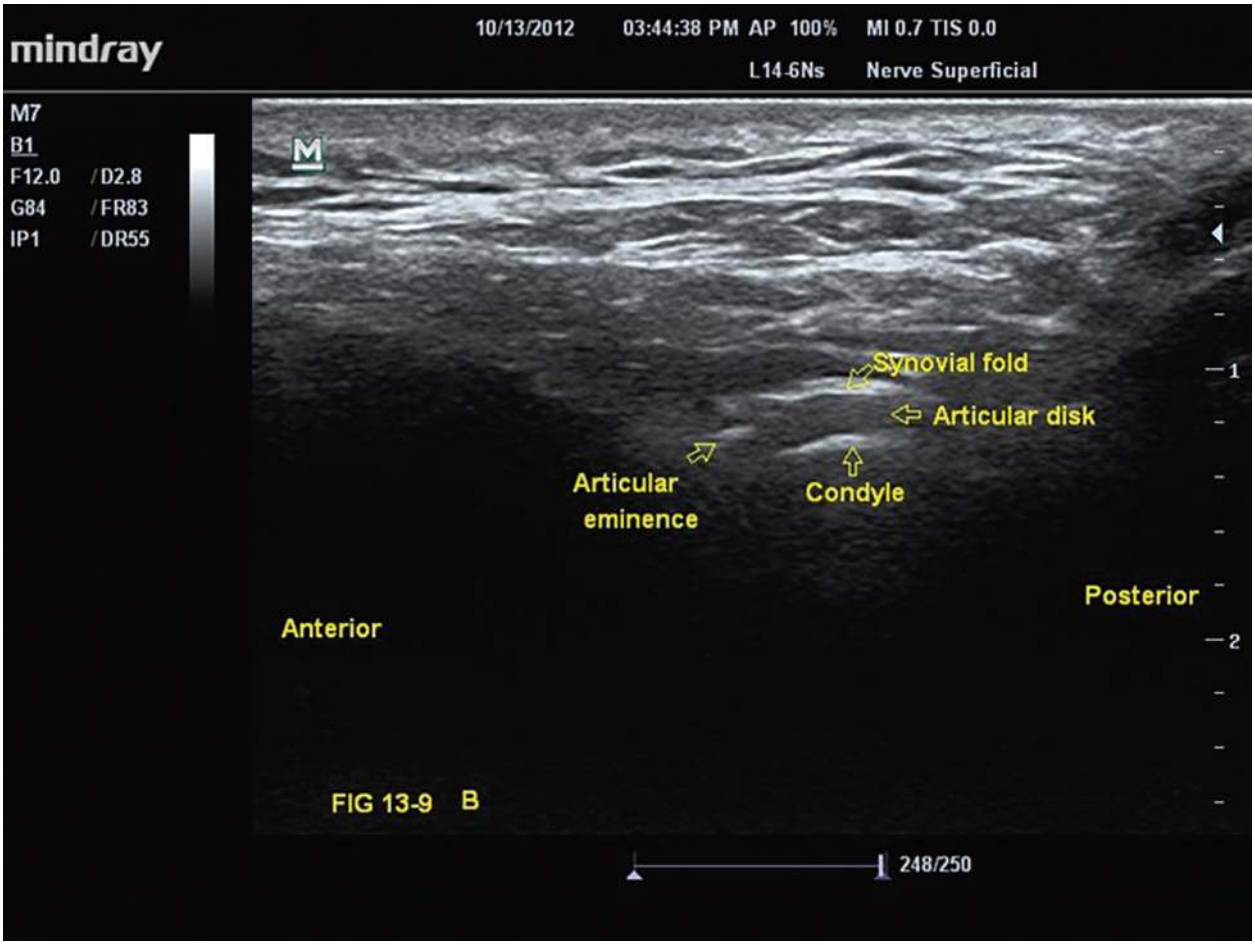

FIG 11.11 Ultrasound image demonstrating ultrasound-guided injection of the temporomandibular joint. (Courtesy Steven Waldman.)

CLINICAL PEARLS

Pain from TMJ dysfunction requires careful evaluation to design an appropriate treatment plan. Infection and inflammatory causes, including collagen vascular diseases, must be excluded. When TMJ pain occurs in older patients, it must be distinguished from the jaw claudication associated with temporal arteritis. Stress and anxiety often accompany TMJ dysfunction, and these factors must be addressed and managed. The myofascial pain component is best treated with tricyclic antidepressants, such as amitriptyline. Dental malocclusion and nighttime bruxism should be treated with an acrylic bite appliance. Opioid analgesics and benzodiazepines should be avoided in patients suffering from TMJ dysfunction.

SUGGESTED READINGS

Andre A, Kang J, Dym H. Pharmacologic treatment for temporomandibular and temporomandibular joint disorders. *Oral Maxillofac Surg Clin North Am*. 2022;34(1):49–59.

Eckstein FM, Wurm MC, Eckstein M, et al. Imaging, histopathological degree of degeneration and clinical findings—do these correlate in patients with temporomandibular joint disorders. *J Stomatol Oral Maxillofac Surg*. 2022;123(3):353–357.

Hosgor H. The relationship between temporomandibular joint effusion and pain in patients with internal derangement. *J Cranio-Maxillofac Surg*. 2019;47(6):940–944.

Jenzer AC, Jackson H, Berry-Cabán CS. Temporomandibular joint pain presentation of myocardial ischemia. *J Oral Maxillofac Surg*. 2018;76(11):2317.e12317.e2.

Promerat A, Constant M, Ferri J, Nicot R. Temporomandibular joint synovial cysts: a systematic review of the literature and a report of two cases. *J Stomatol, Oral Maxillofac Surg*. 2021; S2468–S7855.

Rajapakse S, Ahmed N, Sidebottom AJ. Current thinking about the management of dysfunction of the temporomandibular joint: a review. *Br J Oral Maxillofac Surg*. 2017;55(4):351–356.

Reverand SD, Guerra MM, Campo JR, et al. Correlation between joint effusion and clinical symptoms, magnetic resonance imaging and arthroscopic findings in patients with temporomandibular joint disease. *J Cranio-Maxillofacial Surg*. 2020;48(12):1146–1151.

Waldman SD. Temporomandibular joint injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2021:238–239.

Atypical Facial Pain

ICD-10 CODE G50.1

THE CLINICAL SYNDROME

Atypical facial pain (also known as persistent idiopathic facial pain or atypical facial neuralgia) describes a heterogeneous group of pain syndromes that have in common the fact that the facial pain cannot be classified as trigeminal neuralgia. The pain is continuous but may vary in intensity. It is almost always unilateral and may be characterized as aching or cramping, rather than the shocklike neuritic pain typical of trigeminal neuralgia. Most patients suffering from atypical facial pain are female. The pain is felt in the distribution of the trigeminal nerve but invariably overlaps the divisions of the nerve (Fig. 12.1). In many cases, atypical facial pain is a diagnosis of exclusion.

Headache often accompanies atypical facial pain and is clinically indistinguishable from tension-type headache. Stress is often the precipitating factor or an exacerbating factor in the development of atypical facial pain. Depression and sleep disturbance are also present in many patients. A history of facial trauma, infection, or tumor of the head or neck may be elicited in some patients with atypical facial pain, but in most cases, no precipitating event can be identified. Atypical facial pain is more common in women with a peak incidence in the third to fifth decade.

SIGNS AND SYMPTOMS

Table 12.1 compares atypical facial pain with trigeminal neuralgia. Unlike trigeminal neuralgia, which is characterized by sudden paroxysms of neuritic shocklike pain, atypical facial pain is constant and has a dull, aching quality, but it may vary in intensity. The pain of trigeminal neuralgia is always within the distribution of one division of the trigeminal nerve,

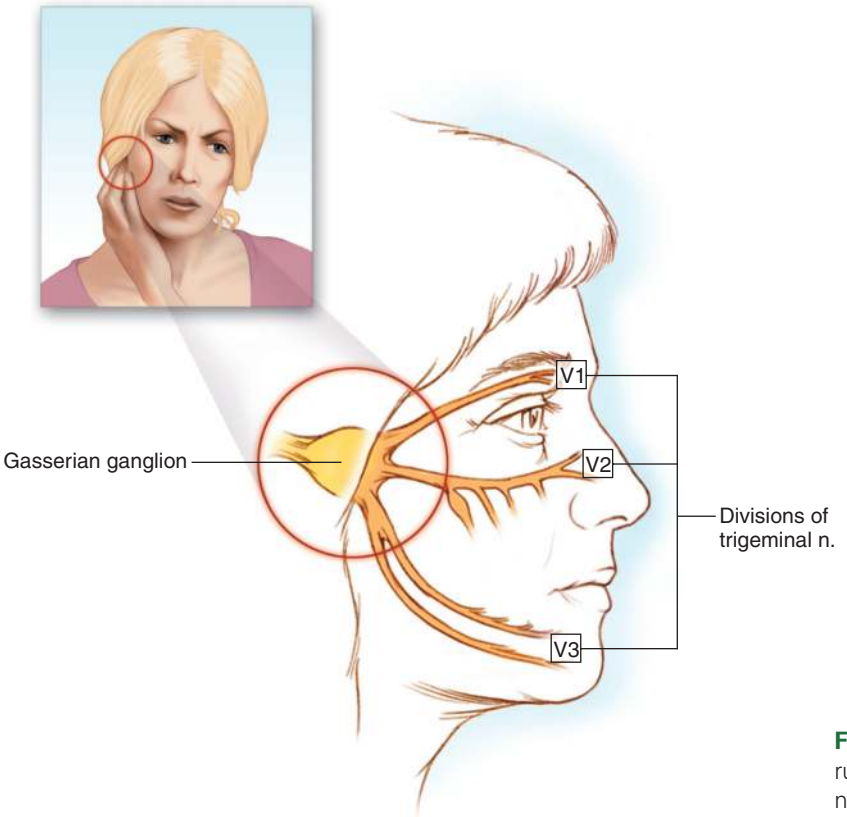

FIG 12.1 Patients with atypical facial pain often rub the affected area; those with trigeminal neuralgia do not.

TABLE 12.1 Comparison of Trigeminal Neuralgia and Atypical Facial Pain

	Trigeminal Neuralgia	Atypical Facial Pain
Temporal pattern of pain	Sudden and intermittent	Constant
Character of pain	Shocklike and neuritic	Dull, cramping, aching
Pain-free intervals	Usual	Rare
Distribution of pain	One division of trigeminal nerve	Overlapping divisions of trigeminal nerve
Trigger areas	Present	Absent
Underlying psychopathology	Rare	Common

whereas atypical facial pain always overlaps these divisional boundaries. The trigger areas characteristic of trigeminal neuralgia are absent in patients suffering from atypical facial pain. By definition, patients with atypical facial pain should have a normal neurologic examination.

TESTING

Radiographs of the head are usually within normal limits in patients suffering from atypical facial pain, but they may be useful to identify a tumor or bony abnormality (Fig. 12.2). Computerized tomographic and/or magnetic resonance imaging (MRI) of the brain and sinuses can help the clinician identify an intracranial disorder such as tumor, sinus disease, and infection (Figs. 12.3 and 12.4). A complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing are indicated if inflammatory arthritis or temporal

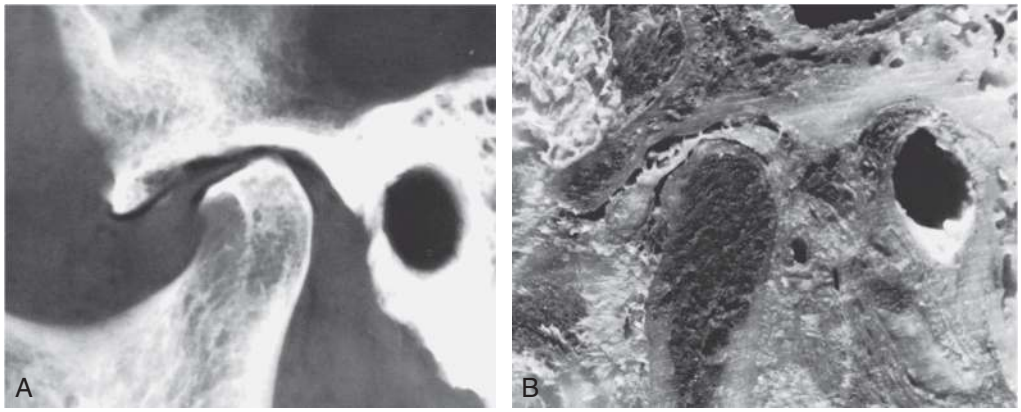

FIG 12.2 Osteoarthritis compared in a specimen radiograph (A) and a photograph (B) of a sagittally sectioned specimen. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:1739.)

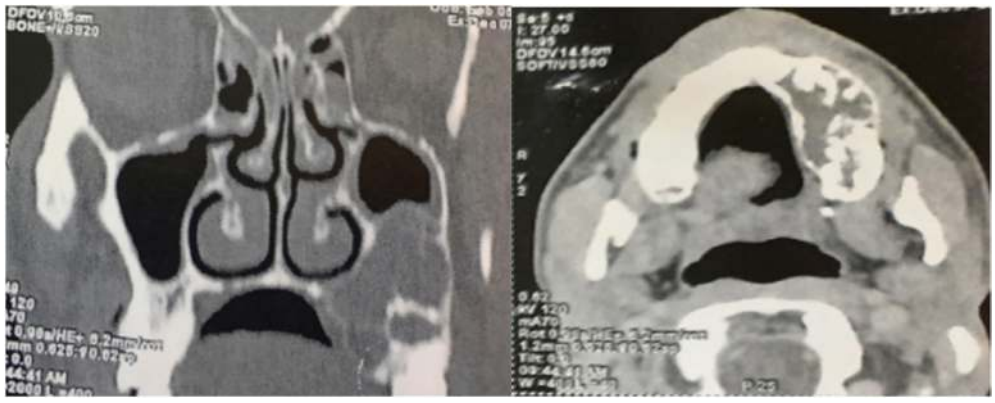

FIG 12.3 Preoperative coronal and axial CT scan shows an expansive soft tissue mass in the left maxilla. The lesion has expanded into the alveolar ridge and maxillary sinus. (From Ngham H, Elkrimi Z, Bijou W, et al. Odontogenic myxoma of the maxilla: a rare case report and review of the literature. *Ann Med Surg*. 2022;77:103575.)

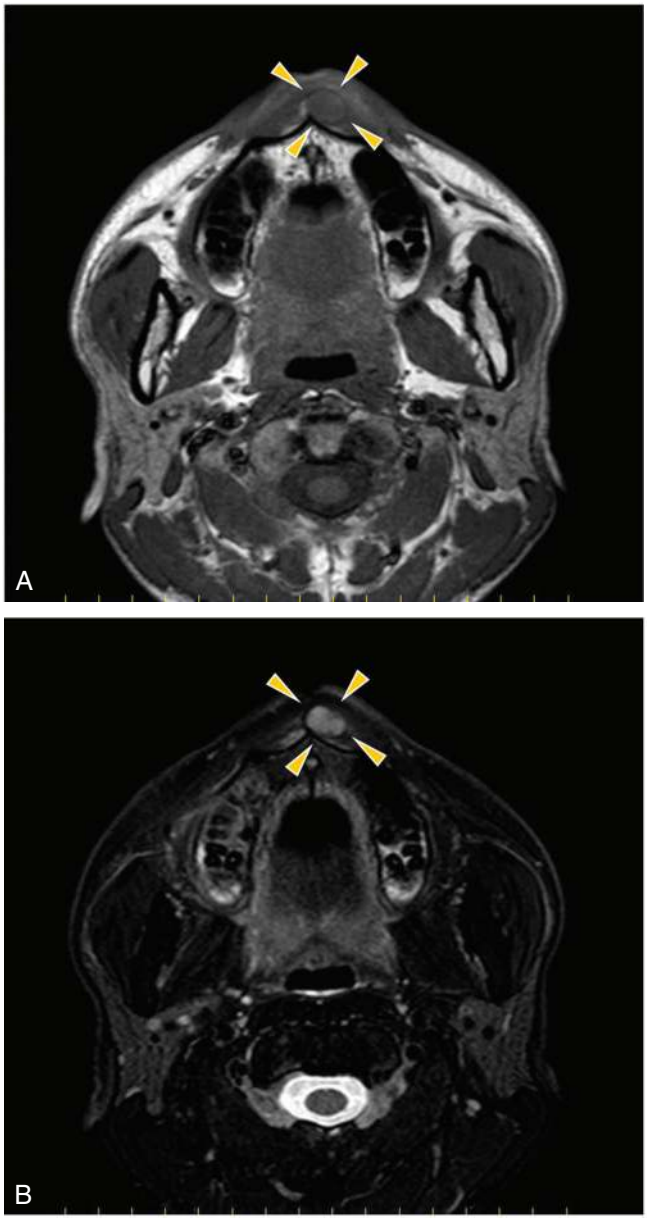

FIG 12.4 Magnetic resonance imaging (MRI) findings. A spherical lesion with regular well-defined borders (indicated by the *arrows*). **A,** T1-weighted image with a low-signal region in the alveolus of the left anterior maxilla. **B,** A high-signal region in the STIR/PAIR-T2-weighted image in the alveolus of the left anterior maxilla. (From Takeuchi S, Yamano Y, Toeda Y, et al. A rare case of basal cell adenoma in the alveolus of the anterior maxilla. *J Oral Maxillofac Surg Med Pathol.* 2022;34(2):185–189.)

arteritis is suspected. Injection of the temporomandibular joint with small amounts of local anesthetic can serve as a diagnostic maneuver to determine whether the temporomandibular joint is the source of the patient's pain. MRI of the cervical spine is also indicated if the patient is experiencing significant occipital or nuchal pain.

DIFFERENTIAL DIAGNOSIS

The clinical symptoms of atypical facial pain may be confused with pain of dental or sinus origin or may be erroneously characterized as trigeminal neuralgia. Careful questioning and physical examination usually allow the clinician to distinguish these overlapping pain syndromes. Tumors of the zygoma and mandible, as well as posterior fossa and retropharyngeal tumors, may produce ill-defined pain that is attributed to atypical facial pain, and these potentially life-threatening diseases must be excluded in any patient with facial pain (Fig. 12.5). Reflex sympathetic dystrophy of the face should also be considered in any patient presenting with ill-defined facial pain after trauma, infection, or central nervous system injury. As noted, atypical facial pain is dull and aching, whereas reflex sympathetic dystrophy of the face causes burning pain, and significant allodynia is often present. Stellate ganglion block may help distinguish these two pain syndromes; the pain of reflex sympathetic dystrophy of the face readily responds to this sympathetic nerve block, whereas atypical facial pain does not. Atypical facial pain must also be distinguished from the pain of jaw claudication associated with temporal arteritis.

TREATMENT

The mainstay of therapy is a combination of drug treatment with tricyclic antidepressants and physical modalities such as oral orthotic devices and physical therapy. Trigeminal nerve block and intraarticular injection of the temporomandibular joint with small amounts of local anesthetic and steroid may also be of value. Antidepressants such as nortriptyline, at a single bedtime dose of 25 mg, can help alleviate sleep disturbance and treat any underlying myofascial pain syndrome. Orthotic devices help the patient avoid jaw clenching and bruxism, which may exacerbate the clinical syndrome. The management of underlying depression and anxiety is also mandatory. The use of topical agents including capsaicin, lidocaine, and an eutectic mixture of local anesthetics as well as the injection of botulinum toxin into the muscle of mastication may also be beneficial. Case reports suggest that a single inhalation of 25 mg of delta-9-tetrahydrocannabinol three times daily may alleviate neuropathic facial pain. Clinical studies suggest that percutaneous stimulation of the trigeminal nerve may be a treatment option for intractable atypical facial pain (Fig. 12.6).

COMPLICATIONS AND PITFALLS

The major pitfall when caring for patients thought to be suffering from atypical facial pain is the failure to diagnose an underlying pathologic process that may be responsible for the patient's pain. Atypical facial pain is essentially a diagnosis of exclusion. If trigeminal nerve block or intraarticular injection of the temporomandibular joint is being considered as part of the treatment plan, the clinician must remember that the

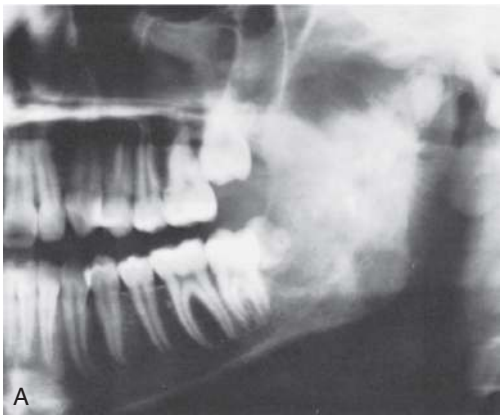

FIG 12.5 Osteosarcoma of the mandible **(A)** and the condylar head and neck **(B)** in a 12-year-old girl. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002: 1726.)

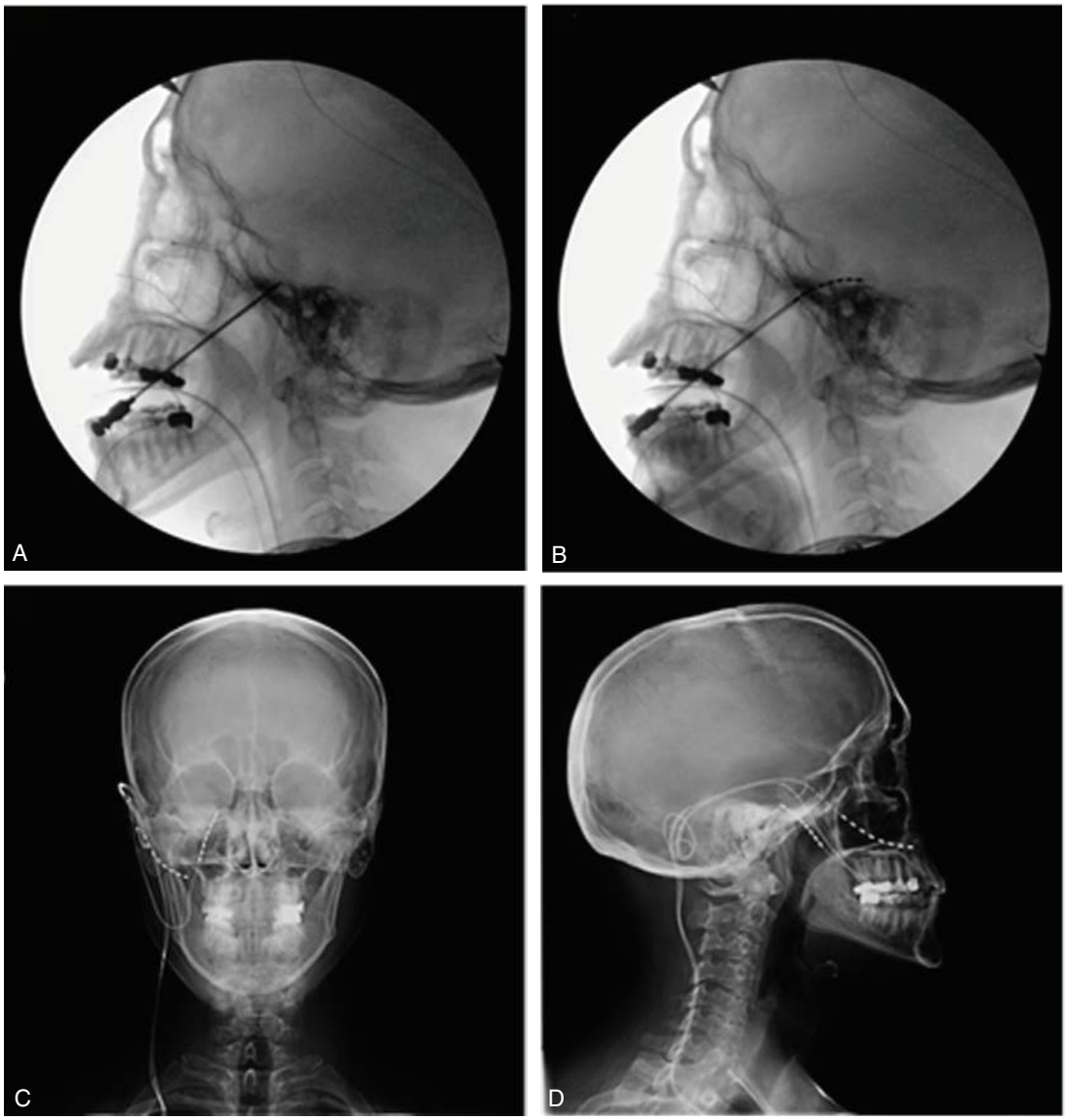

FIG 12.6 Imaging studies showing percutaneous trigeminal nerve stimulator placement. **A,** Intraoperative fluoroscopic image of intracranial lead placement. The stylet has been advanced to cannulate the foramen ovale. **B,** The stylet has been removed, and the lead has been advanced into Meckel's cave. Anteroposterior **(C)** and lateral **(D)** radiographs demonstrating lead placement for the infraorbital nerve and trigeminal nerve. (From Tanner McMahon J, Muhibullah ST, Nicole Bentley J, et al. Percutaneous trigeminal nerve stimulation for persistent idiopathic facial pain: a case series. *World Neurosurg.* 2019(126):e1379–e1386.)

region's vascularity and proximity to major blood vessels can lead to an increased incidence of postblock ecchymosis and hematoma formation, and the patient should be warned of this potential complication.

CLINICAL PEARLS

Atypical facial pain requires careful evaluation to design an appropriate treatment plan. Infection and inflammatory causes, including collagen vascular diseases, must be excluded. Stress and anxiety often accompany atypical facial pain, and these factors must be addressed and treated. The myofascial pain component of atypical facial pain is best treated with tricyclic antidepressants such as amitriptyline. Dental malocclusion and nighttime bruxism should be treated with an acrylic bite appliance. Opioid analgesics and benzodiazepines should be avoided in patients suffering from atypical facial pain.

SUGGESTED READINGS

Clarkson E, Jung E. Atypical facial pain. *Dent Clin North Am*. 2020; 64(1):249–253.

Jay GW, Barkin RL. Trigeminal neuralgia and persistent idiopathic facial pain (atypical facial pain). *Dis Mon*. 2022;68(6):101302.

McMahon JT, Tora MS, Bentley JN, et al. Percutaneous trigeminal nerve stimulation for persistent idiopathic facial pain: a case series. *World Neurosurg*. 2019;126:e1379–e1386.

Penn MC, Choi W, Brasfield K, et al. A review of medical and surgical options for the treatment of facial pain. *Otolaryngol Clin North Am*. 2022;55(3):607–632.

Waldman SD. Atypical facial pain. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:233–234.

Zeng Y-J. Persistent idiopathic facial pain originating from cervical abnormalities. *World Neurosurg*. 2020;133:248–252.

13

Hyoid Syndrome

ICD-10 CODE M65.20

THE CLINICAL SYNDROME

Hyoid syndrome is caused by calcification and inflammation of the attachment of the stylohyoid ligament to the hyoid bone. The styloid process extends in a caudal and ventral direction from the temporal bone from its origin just below the auditory meatus. The stylohyoid ligament's cephalad attachment is to the styloid process, and its caudad attachment is to the hyoid bone. In hyoid syndrome, the stylohyoid ligament becomes calcified at its caudad attachment to the hyoid bone (Fig. 13.1). Tendinitis of the other muscular attachments to the hyoid bone may contribute to this painful condition. Hyoid syndrome also may be seen in conjunction with Eagle's syndrome. Patients suffering from diffuse idiopathic skeletal hyperostosis are thought to be susceptible to the development of hyoid syndrome because of the propensity for calcification of the stylohyoid ligament in this disease (Fig. 13.2).

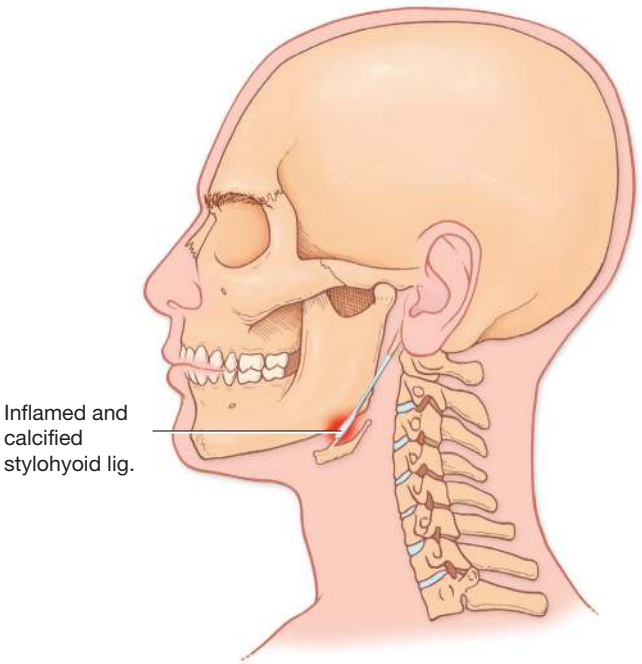

FIG 13.1 In hyoid syndrome, the stylohyoid ligament becomes calcified at its caudad attachment to the hyoid bone.

SIGNS AND SYMPTOMS

The pain of hyoid syndrome is sharp and stabbing; it occurs when moving the mandible, turning the neck, or swallowing. The pain starts below the angle of the mandible and radiates into the anterolateral neck (Fig. 13.3); it is often referred to the ipsilateral ear. Some patients complain of a foreign body sensation in the pharynx. Injection of local anesthetic and steroid into the attachment of the stylohyoid ligament to the greater cornu of the hyoid bone is both a diagnostic and a therapeutic maneuver.

TESTING

No specific test exists for hyoid syndrome. Plain radiography, computed tomography, or magnetic resonance imaging of the neck may reveal calcification of the caudad attachment of the stylohyoid ligament at the hyoid bone or other occult abnormalities responsible for the patient's pain symptomatology (Figs. 13.4 and 13.5). This calcification is highly suggestive of hyoid syndrome in patients suffering from the previously described constellation of symptoms. A complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing are indicated if inflammatory arthritis or temporal arteritis is suspected. As noted earlier, injection of small amounts of anesthetic into the attachment of the stylohyoid ligament to the hyoid bone can help determine whether this is the source of the patient's pain. If difficulty swallowing is a prominent feature of the clinical presentation, endoscopy of the esophagus, with special attention to the gastroesophageal junction, is mandatory to identify esophageal tumors or strictures resulting from gastric reflux.

DIFFERENTIAL DIAGNOSIS

The diagnosis of hyoid syndrome is one of exclusion, and the clinician must first rule out other conditions (Box 13.1). Sternohyoid muscle syndrome may mimic the symptoms of hyoid syndrome and will present as a mass in the lower lateral neck that appears when the patient swallows and then disappears after swallowing is completed (Fig. 13.6). Retropharyngeal infection and tumor may produce ill-defined pain that mimics the pain and other symptoms of hyoid syndrome, and these potentially life-threatening diseases must be excluded (Fig. 13.7).

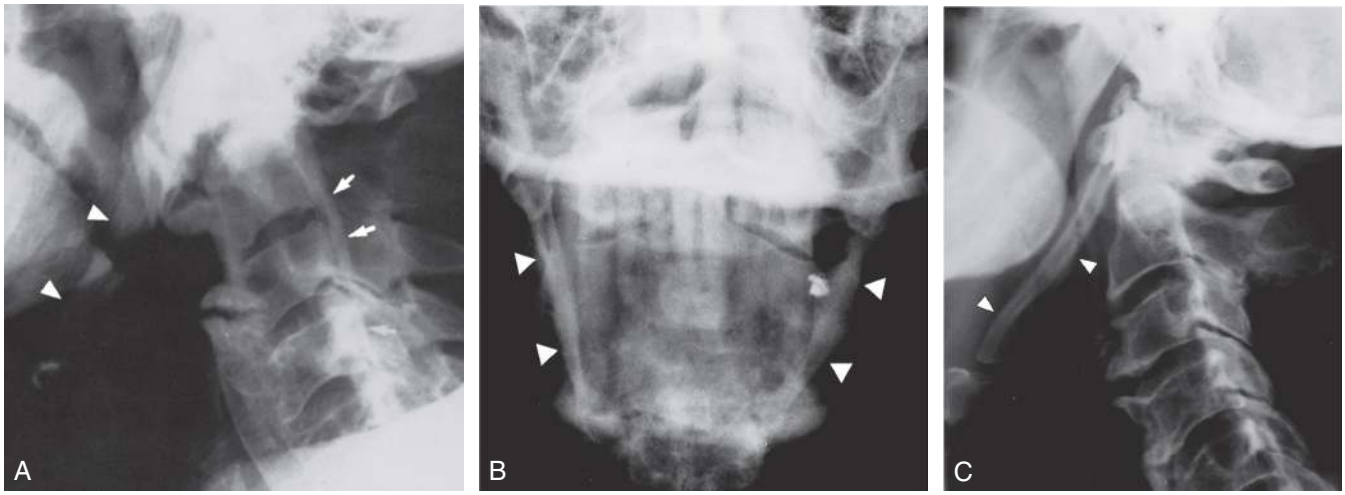

FIG 13.2 Cervical spine abnormalities in diffuse idiopathic skeletal hyperostosis (DISH). **A, B,** Radiographic abnormalities in this patient with DISH include extensive anterior bone formation, ossification of the posterior longitudinal ligament (*arrows*), and ossification of both stylohyoid ligaments (*arrowheads*). **C,** In another patient, note the extensive ossification of the stylohyoid ligament (*arrowheads*) and the changes caused by spinal DISH. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:1483.)

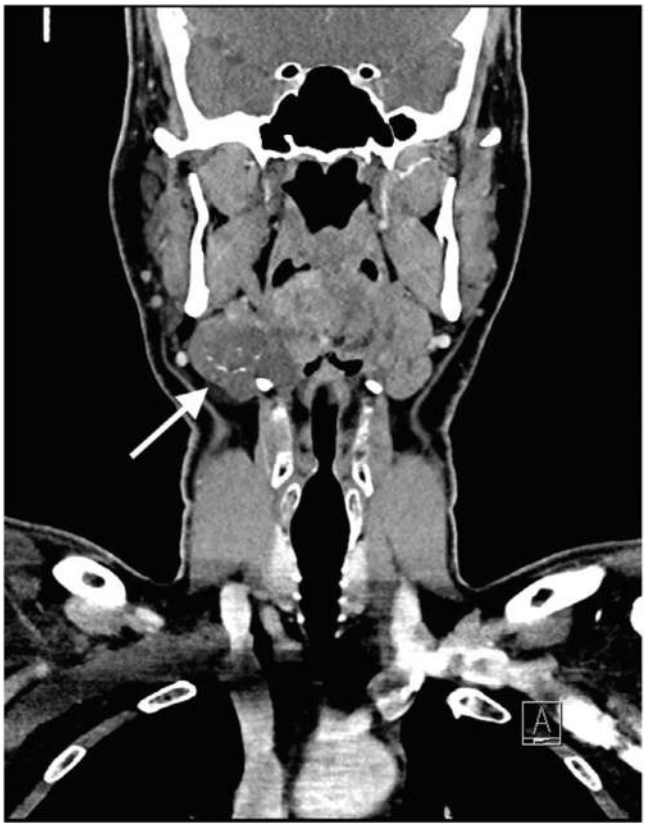

FIG 13.3 Coronal helical CT images with contrast demonstrating a 3.9 × 3.0-cm septated mass (*white arrow*) with multiple foci of calcification without mass effect. (From Shameem MM, Makki FM, Kameh D, Bekeny JR. Osteochondroma of the hyoid bone: a case report and review of the literature. *Otolaryngol Case Rep*. 2020;17:100232.)

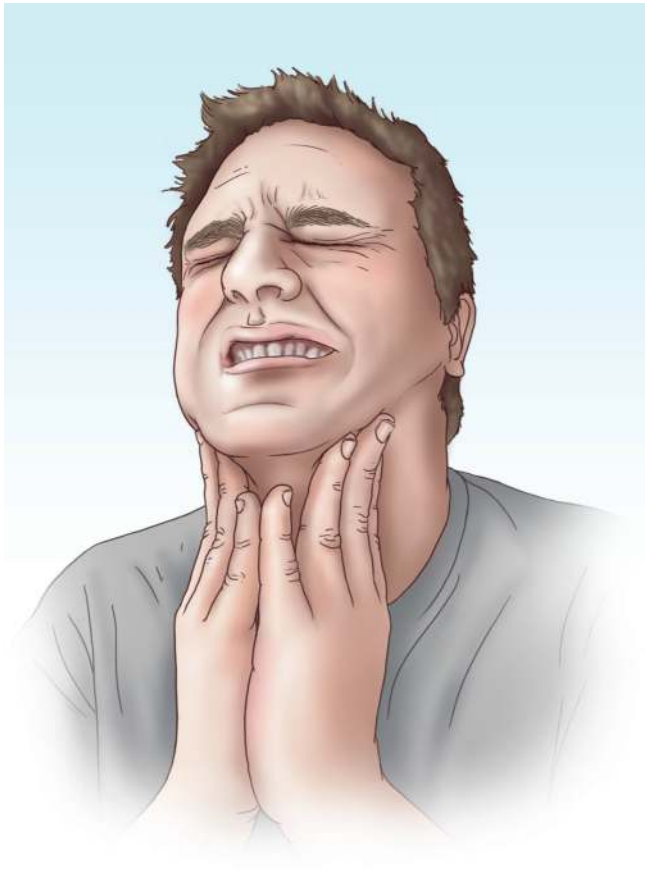

FIG 13.4 The pain of hyoid syndrome is sharp and stabbing and occurs when moving the mandible, turning the neck, or swallowing. The pain starts below the angle of the mandible and radiates to the anterolateral neck.

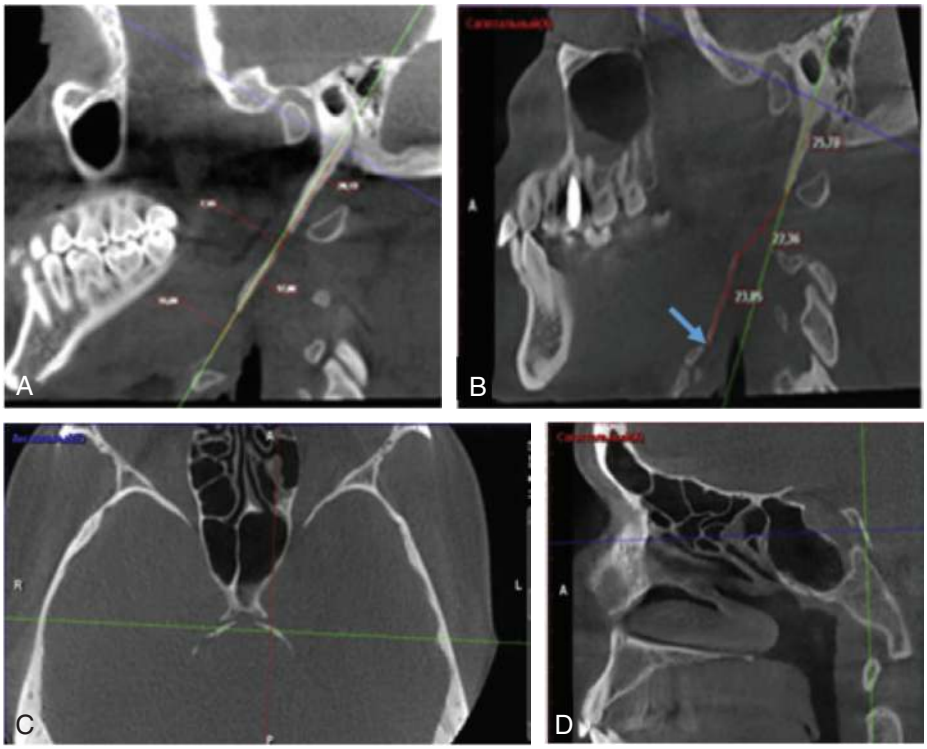

FIG 13.5 Bilateral stylohyoid ligament ossification. Corrected sagittal views show right **(A)** and left **(B)** styloid complex. The *blue arrow* indicates neoarthrosis between the lesser horn of hyoid and the lower end of the left ossified stylohyoid ligament. Axial **(C)** and sagittal **(D)** views illustrate the dural calcifications. (From Kabak S, Savrasova N, Melnichenko Y, et al. Stylohyoid complex syndrome: a report of two cases and review of the literature. *Heliyon*. 2020;6(5).)

BOX 13.1 Conditions That Can Mimic Hyoid Syndrome

Glossopharyngeal neuralgia
Retropharyngeal tumor
Retropharyngeal abscess
Osteomyelitis of the hyoid bone
Atypical facial pain
Mandibular tumor
Esophageal disease
Jaw claudication of temporal arteritis

Osteomyelitis of the hyoid bone, especially in immunocompromised patients, may also mimic hyoid syndrome. Glossopharyngeal neuralgia is another painful condition that can be mistaken for hyoid syndrome. However, the pain of glossopharyngeal neuralgia is similar to the paroxysms of shocklike pain in trigeminal neuralgia, rather than the sharp, shooting pain with movement associated with hyoid syndrome. Because glossopharyngeal neuralgia may be associated with serious cardiac bradyarrhythmias and syncope, the clinician must distinguish between the two syndromes.

TREATMENT

The pain of hyoid syndrome is best treated with local anesthetic and steroid injection of the attachment of the stylohyoid ligament. Owing to the vascularity of this area and the proximity to neural structures, this technique should be performed only by those familiar with the regional anatomy.

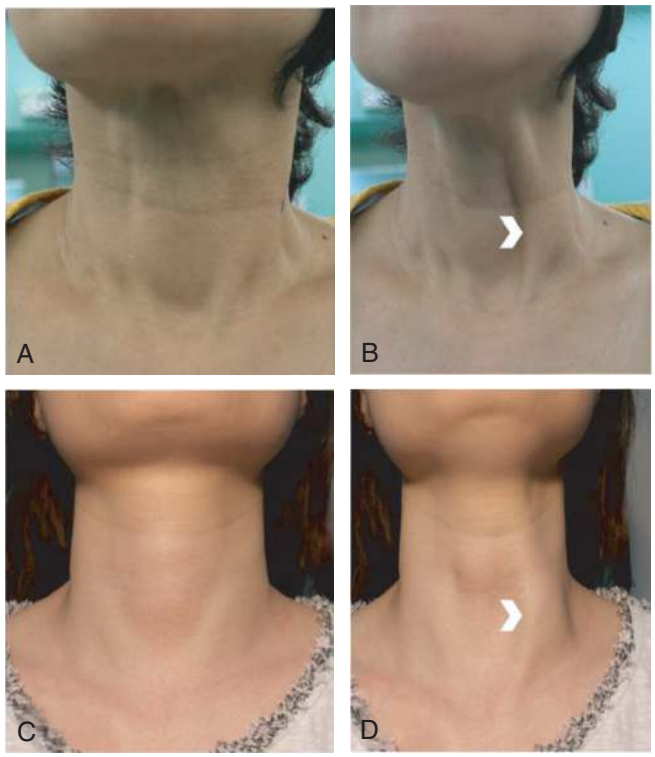

FIG 13.6 Neck photograph of two different patients suffering from sternohyoid muscle syndrome. The resting state is shown in **(A, C)** and the appearance of the abnormal muscle is shown when swallowing **(B, D,** *white arrow head*). Note the abnormal attachment of the muscle to the mid-portion of the clavicle. (From Kim JS, Hong KH, Hong YT, et al. Sternohyoid muscle syndrome. *Am J Otolaryngol*. 2015;36(2):190–194.)

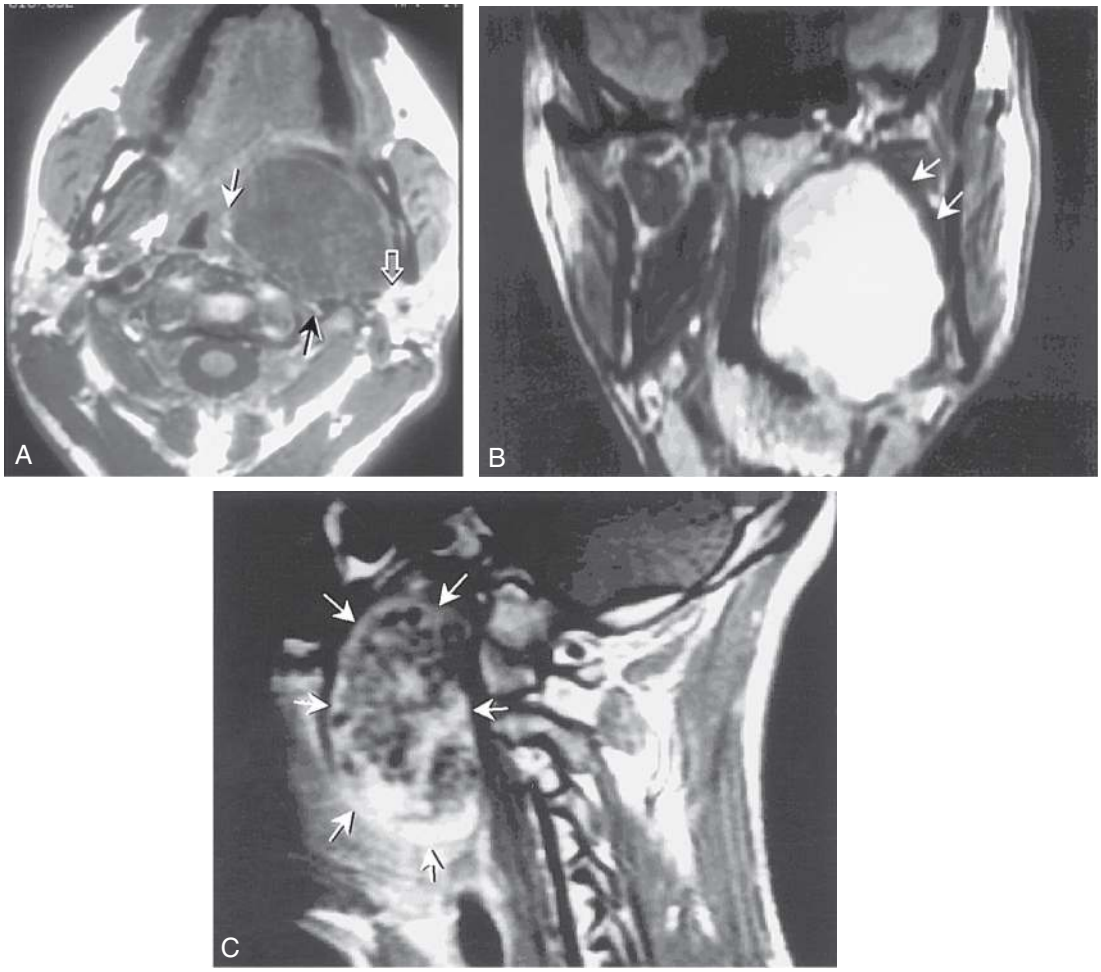

FIG 13.7 Pleomorphic adenoma. **A,** Nonenhanced, T1-weighted axial magnetic resonance imaging (MRI) demonstrates a well-defined mass of lower signal intensity than adjacent muscle. The mass is displacing the prestyloid parapharyngeal fat medially (*solid white arrow*) and the internal carotid artery posteriorly (*solid black arrow*). No intact fat plane can be demonstrated between the lesion and the deep lobe of the parotid gland (*open arrow*). **B,** Intermediate-weighted coronal MRI demonstrates a relatively homogeneous, well-defined mass of increased signal intensity relative to adjacent muscle and lymphoid tissue. The oropharyngeal mucosa is displaced medially. The left medial pterygoid muscle is compressed and displaced superolaterally (*arrows*). **C,** Contrast-enhanced, T1-weighted sagittal MRI demonstrates a markedly heterogeneous mass (*arrows*), with multiple low-signal-intensity regions that may represent areas of calcification or fibrosis. (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:653.)

A trial of nonsteroidal antiinflammatory agents may also be worthwhile in mild cases. Antidepressants such as nortriptyline, at a single bedtime dose of 25 mg, can help alleviate sleep disturbance and treat any underlying myofascial pain syndrome.

COMPLICATIONS AND PITFALLS

The major pitfall when caring for patients thought to be suffering from hyoid syndrome is the failure to diagnose some other underlying disease that may be responsible for the pain. If injection of the caudad attachment of the stylohyoid ligament is being considered as part of the treatment plan, the clinician should remember that the area's vascularity and proximity to major blood vessels can lead to an increased incidence of postblock ecchymosis and hematoma formation, and the patient should be warned of this potential complication.

CLINICAL PEARLS

The clinician should always look for occult malignant disease in patients suffering from pain in this region. Tumors of the larynx, hypopharynx, and anterior triangle of the neck may manifest with clinical symptoms identical to those of hyoid syndrome. Given the low incidence of hyoid syndrome compared with pain secondary to malignant disease, hyoid syndrome must be considered a diagnosis of exclusion.

SUGGESTED READINGS

Jose A, Shakil, Nagori A, Arya S, Roychoudhury A. Hyoid bone syndrome masquerading as temporomandibular joint dysfunction. *Br J Oral Maxillofac Surg*. 2019;57(5):477–478.

Kabak S, Savrasova N, Melnichenko Y, et al. Stylohyoid complex syndrome: a report of two cases and review of the literature. *Heliyon*. 2020;6(5).

Kaur M, Singh C, Siew SF, Lai PS. Isolated hyoid bone fracture in a child due to a fall. *Int J Pediatr Otorhinolaryngol*. 2020;139:1104.

Kim JS, Hong KH, Hong YT, et al. Sternohyoid muscle syndrome. *Am J Otolaryngol*. 2015;36(2):190–194.

Shameem MM, Makki FM, Kameh D, Bekeny JR. Osteochondroma of the hyoid bone: a case report and review of the literature. *Otolaryngol Case Rep*. 2020;17:100232.

Sharif KF, Baik FM, Yue LE, et al. Osteoradionecrosis of the hyoid bone complicated by pharyngocutaneous fistula: a case report and literature review. *Am J Otolaryngol*. 2019;40(2):314–318.

Wei K-C, Hsiao M-Y, Wang T-G. The kinematic features of hyoid bone movement during swallowing in different disease populations: a narrative review. *J Formos Med Assoc*. 2022; 121(10):1892–1899.

Reflex Sympathetic Dystrophy of the Face

ICD-10 CODE G90.59

THE CLINICAL SYNDROME

Reflex sympathetic dystrophy (RSD) is an infrequent cause of face and neck pain. Also known as chronic regional pain syndrome type I, RSD of the face is a classic case in which the clinician must think of the diagnosis to make it. Although the symptom complex in this disorder is relatively constant from patient to patient and although RSD of the face and neck closely parallels its presentation in an upper or lower extremity, the diagnosis is often missed. As a result, extensive diagnostic and therapeutic procedures may be performed in an effort to palliate the patient's facial pain. The common denominator in all patients suffering from RSD of the face is trauma (Fig. 14.1), which may take the following forms: actual injury to the soft tissues, dentition, or bones of the face; infection; cancer; arthritis; or insults to the central nervous system or cranial nerves. There have been several reports of patients who abruptly developed RDS after contracting COVID-19. It has been postulated that COVID-19-related dysautonomia may predispose the group of patients to the development of RSD.

SIGNS AND SYMPTOMS

The hallmark of RSD of the face is burning pain. The pain is frequently associated with cutaneous or mucosal allodynia and does not follow the path of either the cranial or the peripheral nerves. Trigger areas, especially in the oral mucosa, are common, as are trophic skin and mucosal changes in the area affected by RSD (Fig. 14.2). Sudomotor and vasomotor changes may also be identified, but these are often less obvious than in patients suffering from RSD of the extremities. Often, patients with RSD of the face have evidence of previous dental extractions performed in an effort to achieve pain relief. These patients also frequently experience significant sleep disturbance and depression.

TESTING

Although no specific test exists for RSD, a presumptive diagnosis can be made if the patient experiences significant pain relief after stellate ganglion block with a local anesthetic. Given the diverse nature of the tissue injury that can cause RSD of the face, however, the clinician must assiduously search for occult disease that may mimic or coexist with RSD (see "Differential Diagnosis"). All patients with a presumptive diagnosis of RSD of the face should undergo magnetic resonance imaging of the brain and, if significant occipital or nuchal symptoms are present, of the cervical spine. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry

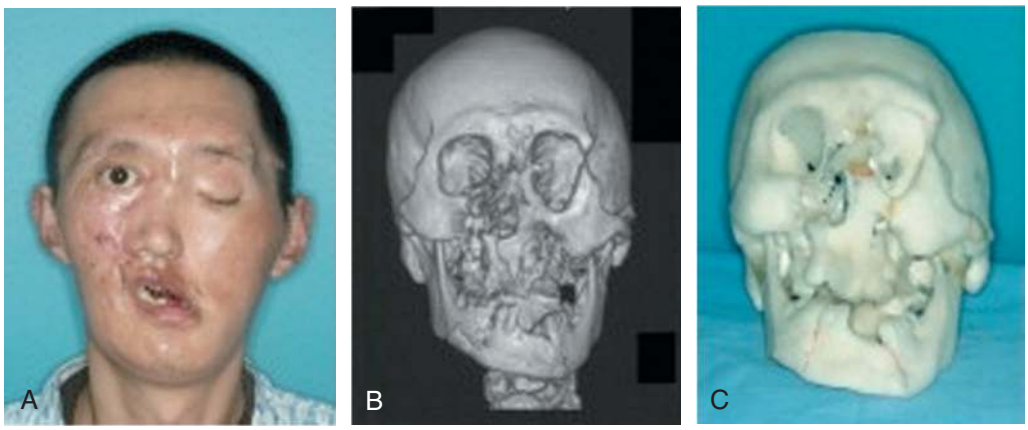

FIG 14.1 Example of severe facial deformity secondary to panfacial fractures before definitive treatment. **A,** Preoperative facial photograph. **B,** Three-dimensional computed tomography (CT) scan showing the mandibular fracture in the tooth-bearing region. The left side of the midface has severely displaced fractures, and the right side has bone defects. **C,** Stereolithic model based on CT data to assist in treatment planning. (From He D, Zhang Y, Ellis E III. Panfacial fractures: analysis of 33 cases treated late. *J Oral Maxillofac Surg*. 2007;65(12):2459–2465.)

should be performed to rule out infection or other inflammatory causes of tissue injury that may serve as a nidus for RSD. Radionuclide bone scanning may help identify boney abnormalities commonly found in RDS. Sudomotor testing may also help confirm asymmetry of sweating in patients suffering from RSD.

DIFFERENTIAL DIAGNOSIS

The clinical symptoms of RSD of the face may be confused with pain of dental or sinus origin or may be erroneously characterized as atypical facial pain or trigeminal neuralgia (Table 14.1). Careful questioning and physical examination usually allow the clinician to distinguish among these overlapping pain syndromes. Stellate ganglion block may help distinguish RSD from atypical facial pain, because RSD readily responds to sympathetic nerve block, whereas atypical facial pain does not. Zygoma and mandible tumors, as well as those of the posterior fossa and retropharyngeal tumors, may produce ill-defined pain attributed to RSD of the face, and these potentially life-threatening diseases must be excluded in any patient with facial pain. RSD of the face must also be distinguished from the pain of jaw claudication associated with temporal arteritis.

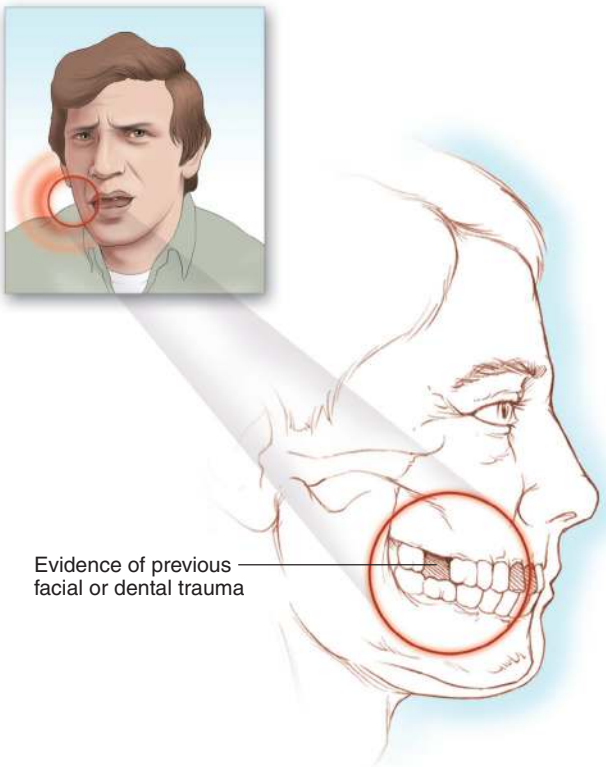

FIG 14.2 Reflex sympathetic dystrophy of the face frequently occurs following trauma, such as dental extractions.

TREATMENT

The successful treatment of RSD of the face requires two phases. First, any nidus of tissue trauma that is contributing to the ongoing sympathetic dysfunction responsible for the symptoms must be identified and removed. Second, interruption of the sympathetic innervation of the face by means of stellate ganglion block with local anesthetic must be implemented. This may require daily stellate ganglion block for a significant period. Occupational therapy consisting of tactile desensitization of the affected skin may also be of value. Underlying depression and sleep disturbance are best treated with a tricyclic antidepressant such as nortriptyline, given as a single 25-mg dose at bedtime. Gabapentin may help palliate any neuritic pain component and is best started slowly with a single bedtime dose of 300 mg, with dosage titration upward in divided doses to a maximum dose of 3600 mg/day. Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function. Although intravenous infusion of mannitol to scavenge free radicals has been used as a last-ditch treatment for refractory reflex dystrophy, larger studies have failed to demonstrate its efficacy. The use of bisphosphonate osteoporosis medications such as alendronate and calcitonin may help prevent the bone loss associated with RSD. Anecdotal reports that intravenous ketamine as well as spinal cord and dorsal root stimulation may help alleviate the pain of RSD.

TABLE 14.1 Differential Diagnosis of Reflex Sympathetic Dystrophy of the Face

	Trigeminal Neuralgia	Atypical Facial Pain	RSD of the Face
Temporal pattern of pain	Sudden and intermittent	Constant	Constant
Character of pain	Shocklike and neuritic	Dull, cramping, aching	Burning with allodynia
Pain-free intervals	Usual	Rare	Rare
Distribution of pain	One division of trigeminal nerve	Overlapping divisions of trigeminal nerve	Overlapping divisions of trigeminal nerve
Trigger areas	Present	Absent	Present
Underlying psychopathology	Rare	Common	Common
Trophic skin changes	Absent	Absent	Present
Sudomotor and vasomotor changes	Absent	Absent	Often present

RSD, Reflex sympathetic dystrophy.

Opioid analgesics and benzodiazepines should be avoided to prevent iatrogenic chemical dependence.

COMPLICATIONS AND PITFALLS

The main complications of RSD of the face are those associated with its misdiagnosis. In this case, chemical dependence, depression, and multiple failed therapeutic procedures are the rule rather than the exception. Stellate ganglion block is a safe and effective technique for pain management, but it also has side effects and risks.

CLINICAL PEARLS

The key to recognize RSD of the face is a high index of clinical suspicion. RSD should be suspected in any patient who has burning pain or allodynia associated with antecedent trauma. Once the syndrome is recognized, blockade of the sympathetic nerves subserving the painful area confirms the diagnosis. Repeated sympathetic blockade, combined with adjunctive therapies, results in pain relief in most cases. The frequency and the number of sympathetic blocks recommended to treat RSD vary among pain practitioners; however, early and aggressive neural blockade is believed to provide more rapid resolution of pain and disability.

SUGGESTED READINGS

Bussa M, Mascaro A, Cuffaro L, Rinaldi S. Adult complex regional pain syndrome type I: a narrative review. *PM R*. 2017;9:707–719.

Eshak N, Abdelnabi M, Ball S. Dysautonomia: an overlooked neurological manifestation in a critically ill COVID-19 patient. *Am J Med Sci*. 2020;360:427–429.

Fotuhi M, Mian A, Meysami S, Raji CA. Neurobiology of COVID19. *J Alzheimers Dis*. 2020;76:3–19.

Petersen PB, Mikkelsen KL, Lauritzen JB, Krogsgaard MR. Risk factors for post-treatment complex regional pain syndrome (CRPS): an analysis of 647 cases of CRPS from the Danish Patient Compensation Association. *Pain Pract*. 2018;18:341–349.

Shehab D, Abdulsalam SJ, Reebye RN. Complex regional pain syndrome as a sequala of COVID-19 pneumonia. *Rev Neurol*. 2022;178(8):865–867.

Urits I, Shen AH, Jones MR, et al. Complex regional pain syndrome, current concepts and treatment options. *Curr Pain Headache Rep*. 2018;22:10.

Waldman SD. Reflex sympathetic dystrophy of the face. In: *Pain review*. 2nd ed. Philadelphia, PA: Elsevier; 2009:253–254.

Waldman SD. Stellate ganglion block: anterior approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia, PA: Elsevier; 2021:156–162.

Waldman SD, Waldman K. Reflex sympathetic dystrophy of the face and neck: report of six patients treated with stellate ganglion block. *Reg Anesth Pain Med*. 1987;12(1):15–17.

15

Cervical Facet Syndrome

ICD-10 CODE M47.812

THE CLINICAL SYNDROME

Cervical facet syndrome is a constellation of symptoms consisting of neck, head, shoulder, and proximal upper extremity pain that radiates in a nondermatomal pattern. The pain is ill defined and dull. It may be unilateral or bilateral and is thought to be the result of a pathologic process of the facet joint. The pain of cervical facet syndrome is exacerbated by flexion, extension, and lateral bending of the cervical spine. It is often worse in the morning after physical activity. Each facet joint receives innervation from two spinal levels; it receives fibers from the dorsal ramus at the corresponding vertebral level and from the vertebra above. This pattern explains the ill-defined nature of facet-mediated pain and explains why the dorsal nerve from the vertebra above the offending level must often be blocked to provide complete pain relief.

SIGNS AND SYMPTOMS

Most patients with cervical facet syndrome have tenderness to deep palpation of the cervical paraspinous musculature; muscle spasm may also be present. Patients exhibit decreased range of motion of the cervical spine and usually complain of pain on flexion, extension, rotation, and lateral bending of the cervical spine (Fig. 15.1). No motor or sensory deficit is present unless the patient has coexisting radiculopathy, plexopathy, or entrapment neuropathy.

If the C1–C2 facet joints are involved, the pain is referred to the posterior auricular and occipital region. If the C2–C3 facet joints are involved, the pain may radiate to the forehead and eyes. Pain emanating from the C3–C4 facet joints is referred superiorly to the suboccipital region and inferiorly to the posterolateral neck, and pain from the C4–C5 facet joints radiates to the base of the neck. Pain from the C5–C6 facet joints is referred to the shoulders and interscapular region, and pain from the C6–C7 facet joints radiates to the supraspinous and infraspinous fossae (Fig. 15.2).

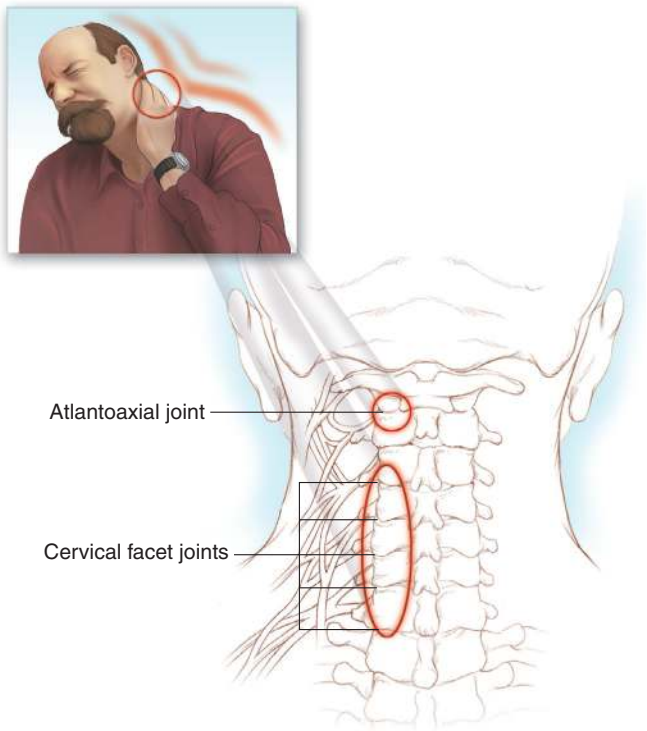

FIG 15.1 The pain of cervical facet syndrome is made worse by flexion, extension, and lateral bending of the cervical spine.

TESTING

By the fifth decade of life, almost all individuals exhibit some abnormality of the facet joints of the cervical spine on plain radiographs (Fig. 15.3). The clinical significance of these findings has long been debated by pain specialists, but it was not until the advent of computed tomography scanning and magnetic resonance imaging (MRI) that the relationship between these abnormal facet joints and the cervical nerve roots and other surrounding structures was clearly understood. MRI of the cervical spine should be performed in all

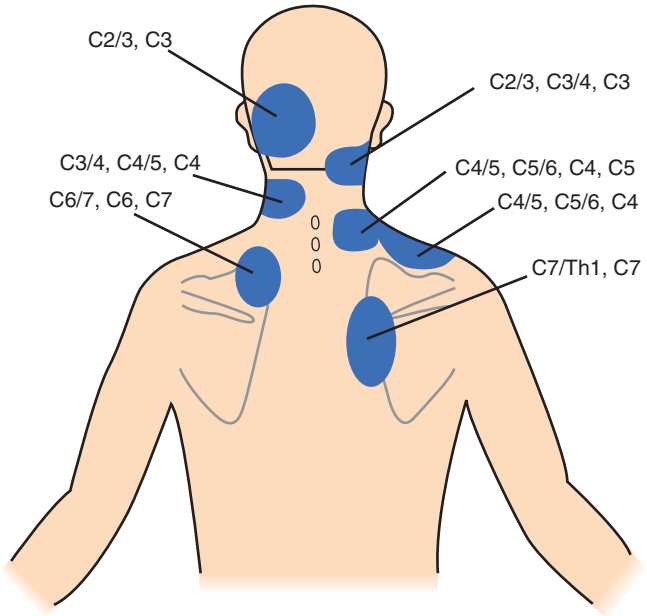

FIG 15.2 Referred pain distributions for the zygapophyseal joints from C1 to T7-Th1 and the dorsal rami C3 to C7. (From Sial KA, Simopoulos TT, Bajwa ZH, et al. Cervical facet syndrome. In: Waldman SD, Bloch JI, eds. *Pain management*. Philadelphia: Saunders; 2007:561–567, Fig. 53.2.)

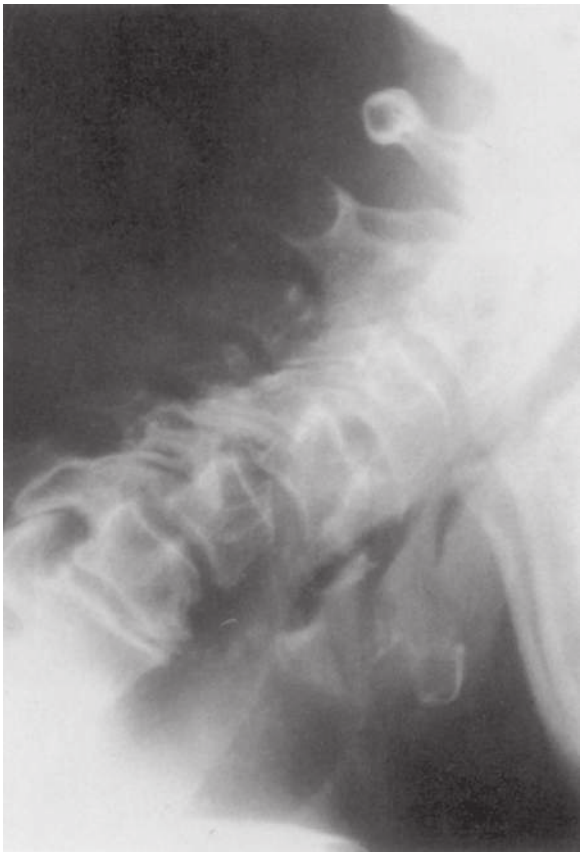

FIG 15.3 Lateral view of the cervical spine showing osteoarthritis of the apophyseal joints of the upper cervical spine, with resultant subluxation of C4 on C5. Additional findings are degenerative disk disease at C5–C6 and C6–C7, associated osteophyte formation at C6–C7, and subluxation of C5 on C6. (From Brower AC, Flemming DJ. *Arthritis in black and white*. 2nd ed. Philadelphia: Saunders; 1997:290.)

patients suspected of suffering from cervical facet syndrome. However, any data gleaned from this sophisticated imaging technique can provide only a presumptive diagnosis. To prove that a specific facet joint is contributing to the patient's pain, a diagnostic intraarticular injection of that joint with a local anesthetic is required. If the diagnosis of cervical facet syndrome is in doubt, screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, human leukocyte antigen (HLA)-B27 antigen screening, and automated blood chemistry should be performed to rule out other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Cervical facet syndrome is a diagnosis of exclusion that is supported by a combination of clinical history, physical examination, radiography, MRI, and intraarticular injection of the suspect facet joint. Pain syndromes that may mimic cervical facet syndrome include cervicalgia, cervical bursitis, cervical fibromyositis, inflammatory arthritis, and disorders of the cervical spinal cord, roots, plexus, and nerves.

TREATMENT

Cervical facet syndrome is best treated with a multimodality approach. Physical therapy consisting of heat modalities and deep sedative massage, combined with nonsteroidal antiinflammatory drugs and skeletal muscle relaxants, is a reasonable starting point. The addition of cervical facet blocks is a logical next step. For symptomatic relief, blockade of the medial branch of the dorsal ramus or intraarticular injection of the facet joint with local anesthetic and steroid is extremely effective (Fig. 15.4). Ultrasound guidance may improve the accuracy of needle placement in selected patients (Fig. 15.5). Anecdotal reports suggest that injection of plate-rich plasma may provide palliation of the pain associated with cervical facet syndrome. Radiofrequency lesioning of the medial branches of the affected facet joints should be considered in patients who have experienced good, but temporary relief of their pain following facet block with local anesthetic and steroid. Underlying sleep disturbance and depression are best treated with a tricyclic antidepressant such as nortriptyline, which can be started at a single bedtime dose of 25 mg.

Cervical facet block is often combined with atlanto-occipital block for the treatment of pain in this area. Although the atlanto-occipital joint is not a true facet joint in the anatomic sense, the technique is analogous to the facet joint block commonly used by pain practitioners and may be viewed as such.

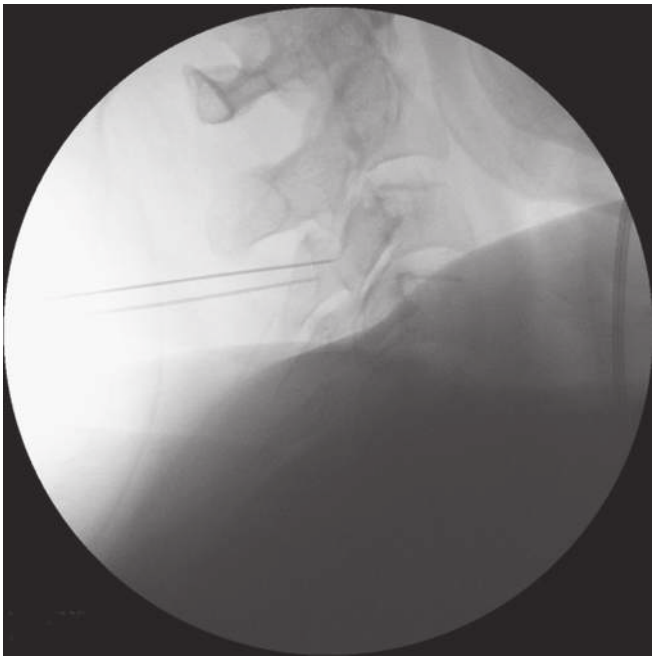

FIG 15.4 Fluoroscopic image of medial branch block for cervical facet syndrome.

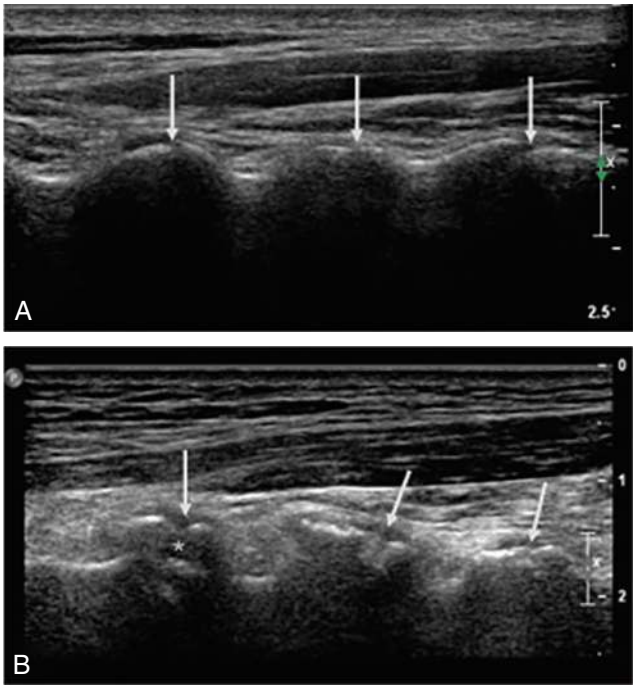

FIG 15.5 The lateral aspects of the C3–C4, C4–C5, and C5–C6 facet joints in an asymptomatic 16-year-old female **(A),** and an asymptomatic 51-year-old male **(B)**. Note the smooth undulating pattern in **(A)** compared to **(B)**. The joint openings are gaps at the top of the undulations (*arrows*), much smaller in **(A)** than in **(B),** where increased joint fluid (*asterisk* at C3–4) can be seen. (From Bodor M, Murthy N, Uribe Y. Ultrasound-guided cervical facet joint injections. *Spine J.* 2022;22(6):983–992.)

COMPLICATIONS AND PITFALLS

The proximity to the spinal cord and exiting nerve roots makes it imperative that cervical facet block be carried out only by those familiar with the regional anatomy and experienced in interventional pain management techniques. The proximity to the vertebral artery, combined with the vascular nature of this region, makes the potential for intravascular injection high, and the injection of even a small amount of local anesthetic into the vertebral artery can result in seizures. Given the proximity of the brain and brainstem, ataxia resulting from vascular uptake of local anesthetic is not uncommon after cervical facet block. Many patients also complain of a transient increase in headache and cervicalgia after injection of the joint.

CLINICAL PEARLS

Cervical facet syndrome is a common cause of neck, occipital, shoulder, and upper extremity pain. It is often confused with cervicalgia and cervical fibromyositis. Diagnostic intraarticular facet block can confirm the diagnosis. The clinician must take care to rule out diseases of the cervical spinal cord, such as syringomyelia, that may initially manifest in a similar manner. Ankylosing spondylitis may also manifest as cervical facet syndrome and must be correctly identified to avoid ongoing joint damage and functional disability.

Many pain specialists believe that cervical facet block and atlanto-occipital block are underused in the treatment of "post-whiplash" cervicalgia and cervicogenic headaches and that they should be considered whenever cervical epidural or occipital nerve blocks fail to provide palliation of headache and neck pain syndromes.

SUGGESTED READINGS

Bodor M, Murthy N, Uribe Y. Ultrasound-guided cervical facet joint injections. *Spine J*. 2022;22(6):983–992.

Burnham TR, Clements N, Conger A, et al. A comparison of the effectiveness of cervical medial branch radiofrequency ablation for chronic facet joint syndrome in patients selected by two common medial branch block paradigms. *Interv Pain Med*. 2022;1(2):100091.

Burnham T, Conger A, Salazar F, et al. The effectiveness of cervical medial branch radiofrequency ablation for chronic facet joint syndrome in patients selected by a practical medial branch block paradigm. *Pain Med*. 2020;21(10):2071–2076.

Candido KD, England B. Cervical spine pain related to the facet joints. *Tech Reg Anesth Pain Manag*. 2015;19(3–4):109–118.

Smith A, Andruski B, Deng G, Burnham R. Cervical facet joint platelet-rich plasma in people with chronic whiplash-associated disorders: a prospective case series of short-term outcomes. *Interv Pain Med*. 2022;1(2):100078.

Waldman SD. Ultrasound guided cervical medial branch block. In: *Comprehensive atlas of ultrasound-guided pain management*

injection techniques. 2nd ed. Philadelphia: Wolters Kluwer, Elsevier; 2020:180–188.
Waldman SD. Ultrasound guided cervical intra-articular facet block. In: *Comprehensive atlas of ultrasound-guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer, Elsevier; 2020:189–196.
Waldman SD. Cervical facet block: medial branch technique block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:188–195.
Waldman SD. Cervical facet joints. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:58–59.

16

Cervical Radiculopathy

ICD-10 CODE M54.12

THE CLINICAL SYNDROME

Cervical radiculopathy is a constellation of symptoms consisting of neurogenic neck and upper extremity pain emanating from the cervical nerve roots. In addition to pain, the patient may experience numbness, weakness, and loss of reflexes. These symptoms are usually unilateral. The C6 and C7 nerve roots are most commonly affected. The causes of cervical radiculopathy include herniated disk, foraminal stenosis, tumor, osteophyte formation, and, rarely, infection. The age-adjusted incidence of cervical radiculopathy is 83 per 100,000 persons with smoking, axial load bearing, female gender, White race, and the arthritides being predisposing factors.

SIGNS AND SYMPTOMS

Patients suffering from cervical radiculopathy complain of pain, numbness, tingling, and paresthesias in the distribution of the affected nerve root or roots (Table 16.1). Patients may also note weakness and lack of coordination in the affected extremity. Muscle spasms and neck pain, as well as pain referred to the trapezius and interscapular region, are common. Decreased sensation, weakness, and reflex changes are demonstrated on physical examination. Patients with C7 radiculopathy commonly place the hand of the affected extremity on top of the head to obtain relief (Fig. 16.1). Spurling test will often exacerbate the pain of cervical radiculopathy. This test is performed by asking the patient to extend

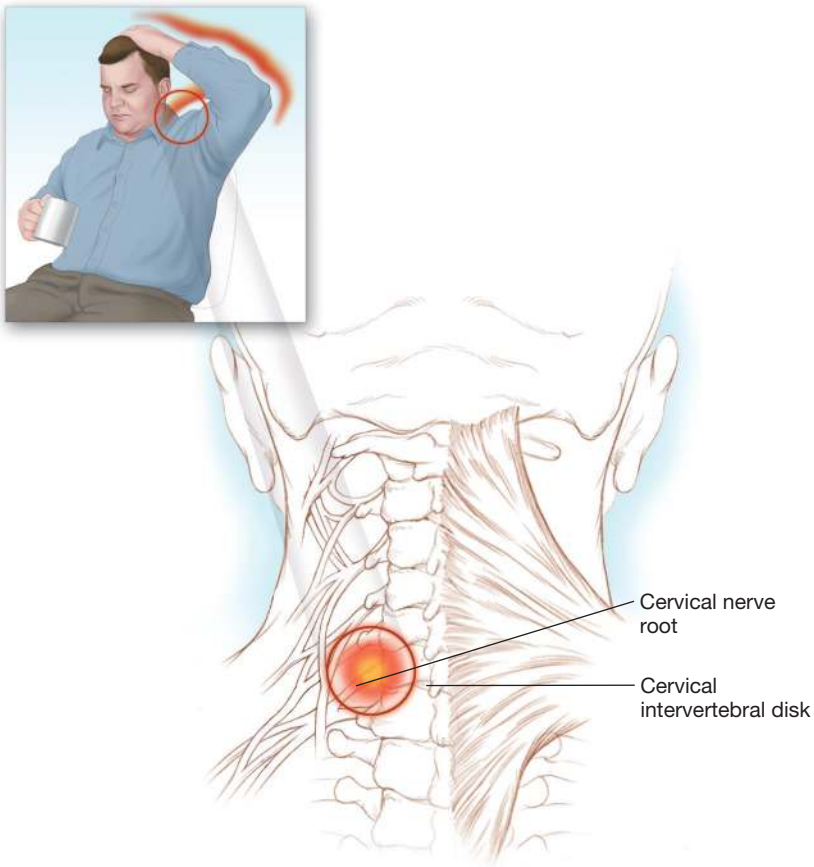

FIG 16.1 Patients with C7 radiculopathy often place the hand of the affected extremity on the head to obtain relief.

TABLE 16.1 Clinical Features of Cervical Radiculopathy

Cervical Root	Pain	Sensory Changes	Weakness	Reflex Changes
C5	Neck, shoulder, anterolateral arm	Numbness in deltoid area	Deltoid and biceps	Biceps reflex
C6	Neck, shoulder, lateral aspect of arm	Dorsolateral aspect of thumb and index finger	Biceps, wrist extensors, pollicis longus	Brachioradialis reflex
C7	Neck, shoulder, lateral aspect of arm, dorsal forearm	Index and middle fingers and dorsum of hand	Triceps	Triceps reflex

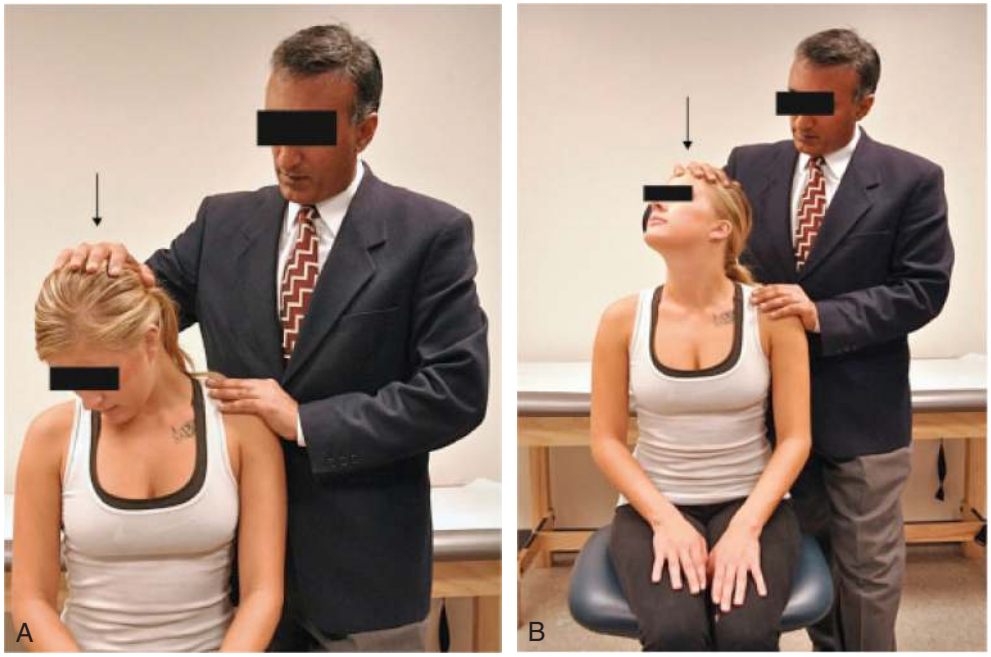

FIG 16.2 Spurling maneuver **(A)** and modified Spurling maneuver **(B)**. (From Mostoufi A. Cervical radiculopathy. In: Frontera WR, Silver JK, Rizzo TD, eds. *Essentials of physical medicine and rehabilitation*. 4th ed. 2020:chap 5, 22–28.)

and laterally rotate the cervical spine while the physician applies an axial load (Fig. 16.2).

Occasionally, patients suffering from cervical radiculopathy experience compression of the cervical spinal cord, with resulting myelopathy. Cervical myelopathy is most commonly caused by a midline herniated cervical disk, spinal stenosis, tumor, or, rarely, infection. Patients suffering from cervical myelopathy may experience decreased manual dexterity of the upper extremities and, occasionally, lower extremity weakness and bowel and bladder symptoms. This condition represents a neurosurgical emergency and should be treated as such.

TESTING

Magnetic resonance imaging (MRI) provides the best information regarding the cervical spine and its contents (Fig. 16.3). MRI is highly accurate and can identify abnormalities that may put the patient at risk for cervical myelopathy (Figs. 16.4 and 16.5). In patients who cannot undergo MRI, such as those with pacemakers, computed tomography or myelography is a reasonable alternative. Provocative discography may also provide useful diagnostic information if the MRI findings are equivocal. Radionuclide bone scanning and plain radiography are indicated if fractures or bony abnormalities such as metastatic disease are being considered.

Although these tests provide the clinician with useful neuroanatomic information, electromyography and nerve conduction velocity testing furnish neurophysiologic information that can determine the actual status of each nerve root and the brachial plexus. Electromyography can also distinguish plexopathy from radiculopathy and can identify a coexistent entrapment neuropathy, such as carpal tunnel syndrome. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, human leukocyte antigen (HLA)-B27

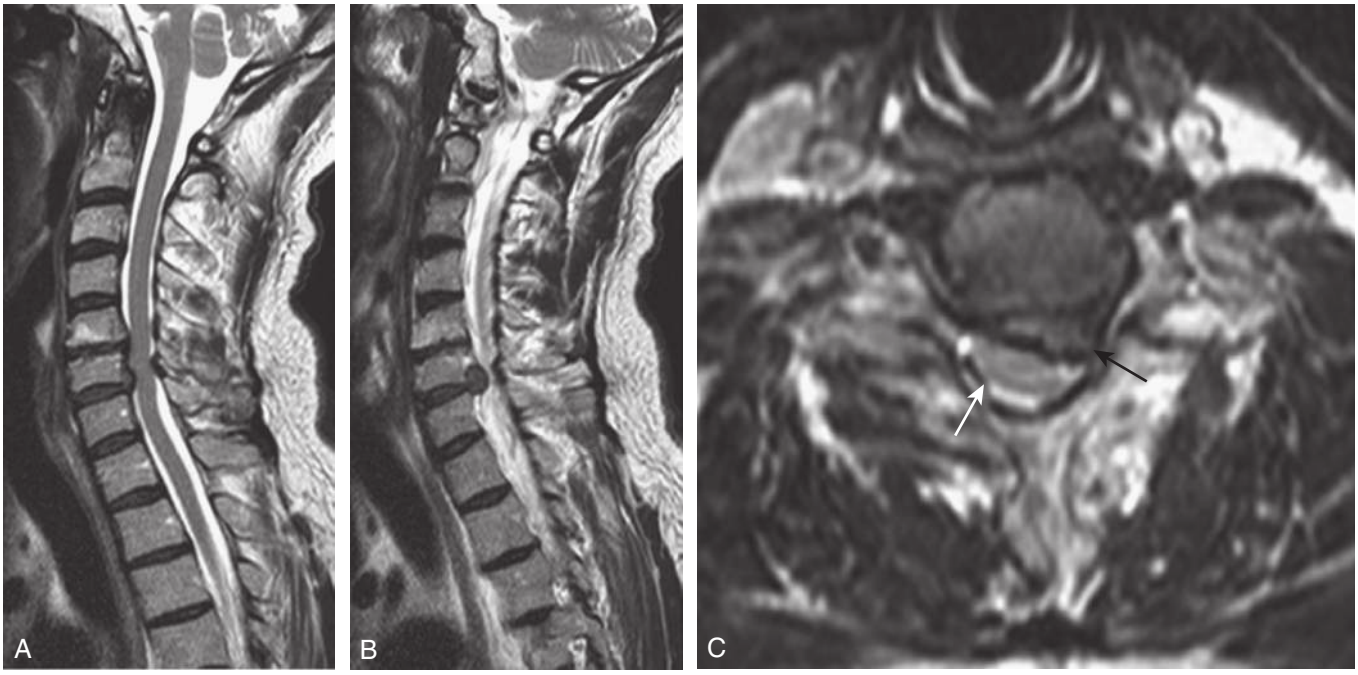

FIG 16.3 Magnetic resonance (MR) images of a patient with left-sided radicular symptoms. **A,** The midline sagittal T2W MR image shows disk degeneration at C5–C6 with disk space narrowing. Less marked disk narrowing is seen at C6–C7, but there is also a posterior disk herniation, which is much more prominent on the parasagittal T2W MR image **(B)**. **C,** The axial T2W MR image demonstrates a large paracentral disk herniation (*black arrow*) that is compressing the cervical cord (*white arrow*). (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Saunders; 2011: Fig. 16.1.)

antigen screening, and automated blood chemistry should be performed if the diagnosis of cervical radiculopathy is in question.

DIFFERENTIAL DIAGNOSIS

Cervical radiculopathy is a clinical diagnosis supported by a combination of clinical history, physical examination, radiography, and MRI. Pain syndromes that may mimic cervical radiculopathy include cervicalgia, cervical bursitis, cervical fibromyositis, inflammatory arthritis, cardiac pain, acute herpes zoster of the cervical dermatomes, entrapment syndromes of the upper extremity, thoracic outlet syndrome, and disorders of the cervical spinal cord, roots, plexus, and nerves (Table 16.2).

TREATMENT

Cervical radiculopathy is best treated with a multimodality approach. Physical therapy, including heat modalities and deep sedative massage, combined with nonsteroidal antiinflammatory drugs and skeletal muscle relaxants, is a reasonable starting point. The addition of cervical epidural nerve blocks is a logical next step. Cervical epidural blocks with local anesthetic and steroid are extremely effective in the treatment of cervical radiculopathy. Underlying sleep disturbance and depression are best treated with a tricyclic antidepressant, such as nortriptyline, which can be started at a single bedtime dose of 25 mg. In patients who fail to respond to epidural steroid injections, a trial of spinal cord stimulation is a reasonable next step if definitive surgical treatment is not an option (Fig. 16.6).

COMPLICATIONS AND PITFALLS

Failure to diagnose cervical radiculopathy accurately may put the patient at risk for the development of cervical myelopathy, which, if untreated, may progress to quadriparesis or quadriplegia.

CLINICAL PEARLS

Carpal tunnel syndrome should be differentiated from cervical radiculopathy involving the cervical nerve roots, which may mimic median nerve compression. Further, cervical radiculopathy and median nerve entrapment may coexist in the double-crush syndrome, which is seen most commonly in patients with carpal tunnel syndrome.

FIG 16.4 Cervical spinal stenosis. **A,** Measurement of the sagittal diameter of the spinal canal is accomplished by calculating the distance between the posterior surface of the vertebral body and the spinolaminar line (*between the arrows*). At the C4–C7 levels, spinal cord compression is unlikely if the diameter of the canal is 13 mm or more. **B,** Photograph of a sagittal section of the cervical spine reveals stenosis of the central canal related to intervertebral (osteo)chondrosis and osteophytes anteriorly and ligamentous laxity and hypertrophy posteriorly. **C,** Sagittal multiple planar gradient recalled magnetic resonance image reveals stenosis of the lower cervical spine related to the presence of osteophytes arising from the posterior surface of the vertebral bodies. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:1655.)

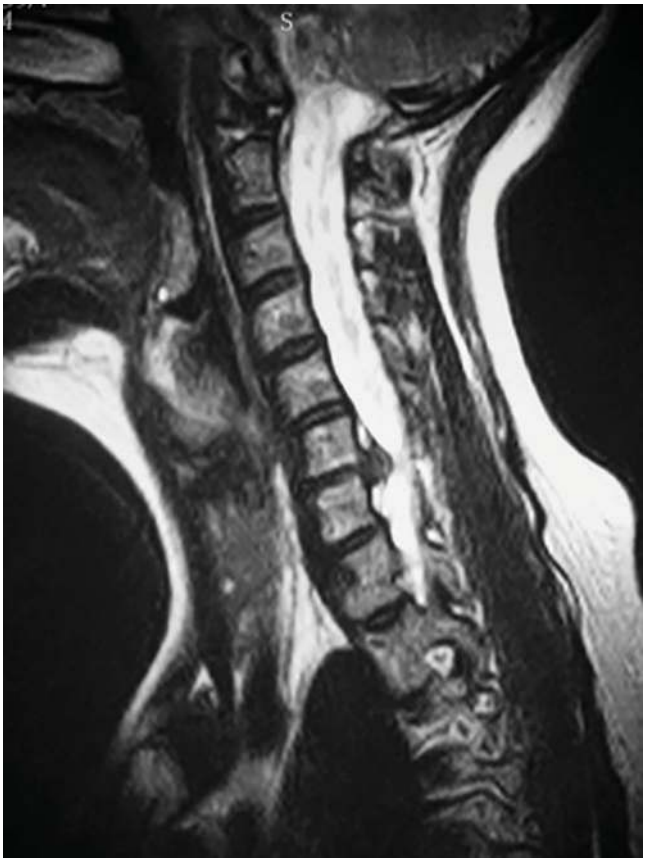

FIG 16.5 Sagittal magnetic resonance image showing C6–C7 disk degeneration and a large herniation. (From Sueki DG, Pablo EV, Delamarter RB, Kim PD. Anterior cervical discectomy and fusion. In: Maxey L, Magnusson J, eds. *Rehabilitation for the postsurgical orthopedic patient*. 3rd ed. Mosby; 2013: chap 14, 256–282.)

TABLE 16.2 Clinical Syndromes That Can Mimic Cervical Radiculopathy

Clinical Condition	Signs and Symptoms
Cervical myelopathy	Decreased manual dexterity, gait changes, bowel or bladder dysfunction, upper motor neuron findings (e.g., Hoffman sign)
Acute herpes zoster	Pain proceeding rash in affected cervical dermatomes
Cardiogenic pain	Radiating pain into the left shoulder and upper extremity
Parsonage–Turner syndrome	Acute onset of upper extremity pain, usually followed by weakness and sensory disturbances
Entrapment of the peripheral nerves of the upper extremities	Sensory deficits and weakness in the distribution of the affected peripheral nerve
Thoracic outlet syndrome	Pain and weakness of the lower brachial plexus secondary to nerve root compression

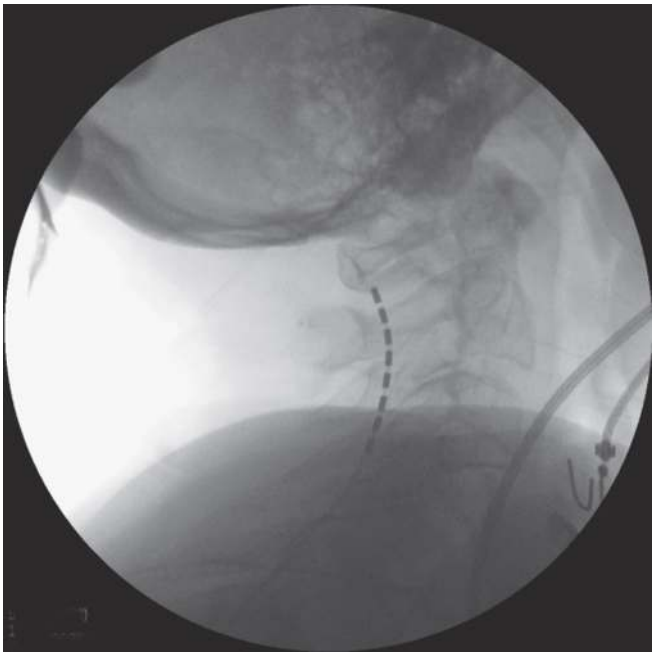

FIG 16.6 Spinal cord stimulator lead within the cervical epidural space.

SUGGESTED READINGS

Corey DL, Comeau D. Cervical radiculopathy. *Med Clin North Am*. 2014;98(4):791–799.

Singh S, Kumar D, Kumar S. Risk factors in cervical spondylosis. *J Clin Orthop Trauma*. 2014;5(4).

Sleijser-Koehorst MLS, Coppieters MW, Epping R, et al. Diagnostic accuracy of patient interview items and clinical tests for cervical radiculopathy. *Physiotherapy*. 2021;111:74–82.

Waldman SD. Cervical epidural nerve block: the translaminar approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:206–214.

Waldman SD. Cervical spinal cord stimulation: stage I trial stimulation. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:1091–1094.

Waldman SD. Cervical radiculopathy. In: *Pain review*. 2nd ed. Philadelphia: Saunders; 2017:236–237.

Waldman SD. Functional anatomy of the cervical spine. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2020.

Fibromyalgia of the Cervical Musculature

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

Fibromyalgia is a chronic pain syndrome that affects a focal or regional portion of the body. Fibromyalgia of the cervical spine is one of the most common painful conditions encountered in clinical practice. The sine qua non for diagnosis is the finding of myofascial trigger points on physical examination. These trigger points are thought to be the result of microtrauma to the affected muscles. Stimulation of the myofascial trigger points reproduces or exacerbates the patient's pain. Although these trigger points are generally localized to the cervical paraspinous musculature, the trapezius, and other muscles of the neck, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment.

The pathophysiology of the myofascial trigger points of fibromyalgia of the cervical spine remains unclear, but tissue trauma seems to be the common denominator. Acute trauma to muscle caused by overstretching commonly results in fibromyalgia. More subtle muscle injury in the form of repetitive microtrauma, damage to muscle fibers from exposure to extreme heat or cold, overuse, chronic deconditioning of the agonist and antagonist muscle unit, or other coexistent disease processes such as radiculopathy may also produce fibromyalgia of the cervical spine.

Various other factors seem to predispose patients to the development of fibromyalgia of the cervical spine. For example, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop fibromyalgia. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. In addition, previous injuries may result in abnormal muscle function and increase the risk of developing fibromyalgia. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological abnormalities including depression and alexithymia, a personality disorder of altered emotional self-awareness.

Often, stiffness and fatigue accompany the pain of fibromyalgia of the cervical spine. These symptoms increase the functional disability associated with this disease and complicate its treatment. Fibromyalgia may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression and alexithymia frequently coexist with the muscle abnormalities, and the management of these concurrent conditions must be an integral part of any successful treatment plan. Studies have suggested that an abnormality in the serotonin transport gene may predispose patients to the development of fibromyalgia as a result of abnormal pain processing.

SIGNS AND SYMPTOMS

As noted earlier, the sine qua non of fibromyalgia of the cervical spine is the myofascial trigger point. This trigger point represents the pathologic lesion and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, is often seen (Fig. 17.1). A positive jump sign is characteristic of fibromyalgia of the cervical spine, as are stiffness of the neck, pain in range of motion, and pain referred to the upper extremities in a nondermatomal pattern. Although this referred pain has been well studied and occurs in a characteristic pattern, it often leads to misdiagnosis.

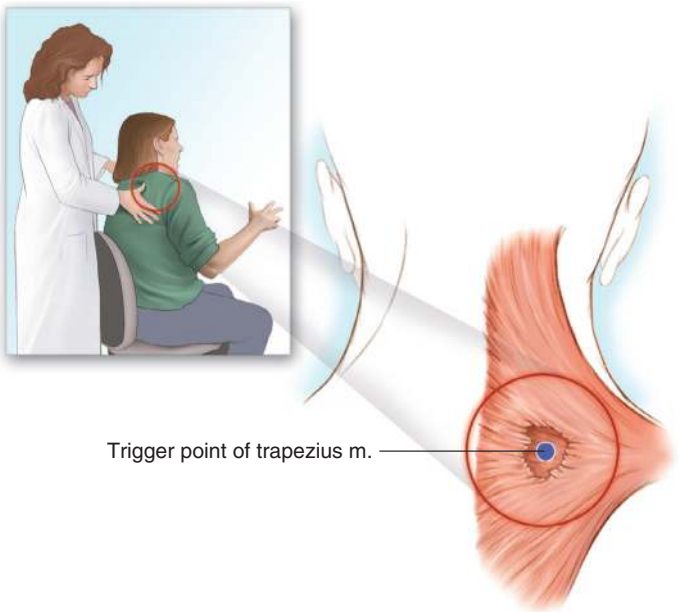

FIG 17.1 Palpation of a trigger point results in a positive jump sign.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with fibromyalgia of the cervical spine, but other investigators have not corroborated this finding. Electrodiagnostic testing has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Recent clinical reports suggest that mean sonographic thickness of the multifidus, splenius capitis, and trapezius is decreased in patients with fibromyalgia of the cervical region. Thus, the diagnosis is based on the clinical findings of trigger points in the cervical paraspinous muscles and an associated jump sign, rather than on specific laboratory, electrodiagnostic, or radiographic testing.

DIFFERENTIAL DIAGNOSIS

The clinician must rule out other disease processes that may mimic fibromyalgia of the cervical spine, including primary inflammatory muscle disease, multiple sclerosis, Lyme disease, hypothyroid disease, and collagen vascular disease (Table 17.1). The judicious use of electrodiagnostic

TABLE 17.1 Medical Disorders That Mimic Symptoms of Fibromyalgia or Are Comorbid With Fibromyalgia

Medical Disorder	Differentiating Signs and Symptoms	Laboratory Tests
Rheumatoid arthritis	Predominant joint pain, joint swelling, and joint line tenderness	Positive rheumatoid factor in 80%–90% of patients, radiographic evidence of joint erosion
Systemic lupus erythematosus	Multisystem involvement, commonly arthritis, arthralgia, rash	Antinuclear antibody test, other autoantibodies
Polyarticular osteoarthritis	Multiple painful joints	Radiographic evidence of joint degeneration
Polymyalgia rheumatica	Proximal shoulder and hip girdle pain, more common in older persons	Elevation of erythrocyte sedimentation rate in ~80% of patients
Polymyositis or other myopathies	Symmetric proximal muscle weakness	Elevated serum muscle enzymes (creatinine kinase, aldolase), abnormal EMG, abnormal muscle biopsy
Spondyloarthropathy	Localization of spinal pain to specific sites in the neck, midthoracic, anterior chest wall, or lumbar regions; objective limitation of spinal mobility resulting from pain and stiffness	Radiographic sacroiliitis, vertebral body radiographic changes
Osteomalacia	Diffuse bone pain, fractures, proximal myopathy, with muscle weakness	Low 25-hydroxyvitamin D levels, low phosphate levels, DEXA scan abnormalities
Lyme disease	Rash, arthritis, or arthralgia; occurs in areas of endemic disease	Positive Lyme serologic test results (ELISA, Western blot)
Hypothyroidism	Cold intolerance, mental slowing, constipation, weight gain, hair loss	Elevated thyroid stimulating level
Sleep apnea	Interrupted breathing during sleep, heavy snoring, excessive sleepiness during the day	Polysomnography abnormalities
Hepatitis C	Right upper quadrant pain, nausea, decreased appetite	Elevated liver enzymes (alanine aminotransferase), hepatitis C antibody, hepatitis C RNA
Hyperparathyroidism	Increased thirst and urination, kidney stones, nausea or vomiting, decreased appetite, thinning bones, constipation	Elevated serum calcium and parathyroid levels
Cushing's syndrome	Hypertension, diabetes, hirsutism, moon facies, weight gain	Elevated 24-hour urinary free cortisol level
Addison's disease	Postural hypotension, nausea, vomiting, skin pigmentation, weight loss	Blunted ACTH stimulation test
Multiple sclerosis	Visual changes (unilateral partial or complete loss, double vision), ascending numbness in a leg or bandlike truncal numbness, slurred speech (dysarthria)	Magnetic resonance imaging of brain or spinal cord, cerebrospinal fluid analysis for immunoglobulins, visual evoked potentials
Neuropathy	Shooting or burning pain, tingling, numbness	Tests to identify underlying cause (e.g., diabetes, herniated disk), EMG, nerve conduction study, nerve biopsy

ACTH, Adrenocorticotropic hormone; *DEXA*, dual-energy x-ray absorptiometry; *ELISA*, enzyme-linked immunosorbent assay; *EMG*, electromyography.

Modified from Arnold LM. The pathophysiology, diagnosis and treatment of fibromyalgia. *Psychiatr Clin North Am*. 2010;33(2):375–408.

testing and radiography can identify coexisting disorders such as a herniated nucleus pulposus or rotator cuff tear. The clinician must also identify any psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with fibromyalgia or other pathologic processes.

TREATMENT

Treatment is focused on blocking the myofascial trigger and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor, has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique is required to prevent infection, as are universal precautions to minimize any risk to the operator. Most side effects of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid pneumothorax when injecting trigger points in proximity to the underlying pleural space.

CLINICAL PEARLS

Fibromyalgia of the cervical spine is a common disorder that often coexists with various somatic and psychological disorders, yet it is often misdiagnosed. In patients suspected of suffering from fibromyalgia of the cervical spine, a careful evaluation is mandatory to identify any underlying disease processes. Treatment is focused on blocking the myofascial trigger to achieve pain relief. This is accomplished with trigger point injections with local anesthetic or saline solution, along with antidepressants to treat underlying depression. Physical therapy, therapeutic heat and cold, transcutaneous nerve stimulation, and electrical stimulation may be helpful in some cases. For patients who do not respond to traditional measures, consideration should be given to the use of botulinum toxin type A injection.

SUGGESTED READINGS

Berger AA, Keefe J, Winnick A, et al. Cannabis and cannabidiol (CBD) for the treatment of fibromyalgia. *Best Pract Res Clin Anaesthesiol*. 2020;34(3):617–631.

Gold K, Burton C, Garaycochea G, et al. Cervical sprain with residual C1–C2 laxity as source of fibromyalgia and multiple sclerosis symptoms? *Arch Phys Med Rehabil*. 2022;103(3): e26–e27. ISSN 0003-9993.

Goldenberg DL, Clauw DJ, Palmer RE, et al. Opioid use in fibromyalgia: a cautionary tale. *Mayo Clin Proc*. 2016;91(5): 640–648.

Gota CE. Fibromyalgia: recognition and management in the primary care office. *Rheum Dis Clin North Am*. 2022;48(2):467–478.

Kueny A, Montesó-Curto P, Lunn S, et al. Fibromyalgia pain and fatigue symptoms in Spanish and United States men. *Pain Manag Nurs*. 2021;22(3):423–428.

Kuzu O, Aras B. Sonographic measurement of the neck extensor muscle thickness in patients with fibromyalgia. *Musculoskelet Sci Pract*. 2022;59: 102541.

Kuzu D, Valentine TR, Kratz AL. Temporal associations between substance use and somatic symptoms in the daily lives of people with fibromyalgia. *J Pain*. 2022;23(suppl 5):55.

Lattanzio SM, Imbesi F. Fibromyalgia associated with repeated gadolinium contrast-enhanced MRI examinations. *Radiol Case Rep*. 2020;15(5):534–541.

Sluka KA, Clauw DJ. Neurobiology of fibromyalgia and chronic widespread pain. *Neuroscience*. 2016;338(3):114–129.

Wolfe F, Clauw DJ, Fitzcharles MA, et al. 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. *Semin Arthritis Rheum*. 2016;46(3):319–329.

18

Cervical Strain

ICD-10 CODE S13.4xxA

THE CLINICAL SYNDROME

Acute cervical strain is a constellation of symptoms consisting of nonradicular neck pain that radiates in a nondermatomal pattern into the shoulders and interscapular region; headache often accompanies these symptoms. The trapezius is commonly affected, with resultant spasm and limited range of motion of the cervical spine (Fig. 18.1). Cervical strain is usually the result of trauma to the cervical spine and associated soft tissues (Fig. 18.2), but it may occur without an obvious inciting incident. Given that over 93% of the world population uses a smartphone, it is not surprising that there has been an increased incidence of cervical strain resulting from poor posture while looking down at the smartphone screen (Fig. 18.3). The relationship of the angle of the cervical spine and the device screen can be quantified using a cumulative average of tilt angles of the neck over time. If the observed tilt angle is excessive, then significant strain is being placed on the cervical spine and soft tissues. The pathologic lesions responsible for this clinical syndrome may emanate from the soft tissues, facet joints, or intervertebral disks.

SIGNS AND SYMPTOMS

Neck pain is the hallmark of cervical strain. It may begin in the occipital region and radiate in a nondermatomal pattern into the shoulders and interscapular region. The pain of cervical strain is often exacerbated by movement of the cervical spine and shoulders. Headaches often occur and may worsen with emotional stress. Sleep disturbance is common, as is difficulty concentrating on simple tasks. Depression may occur with prolonged symptoms.

On physical examination, tenderness is elicited on palpation; spasm of the paraspinous musculature and trapezius is

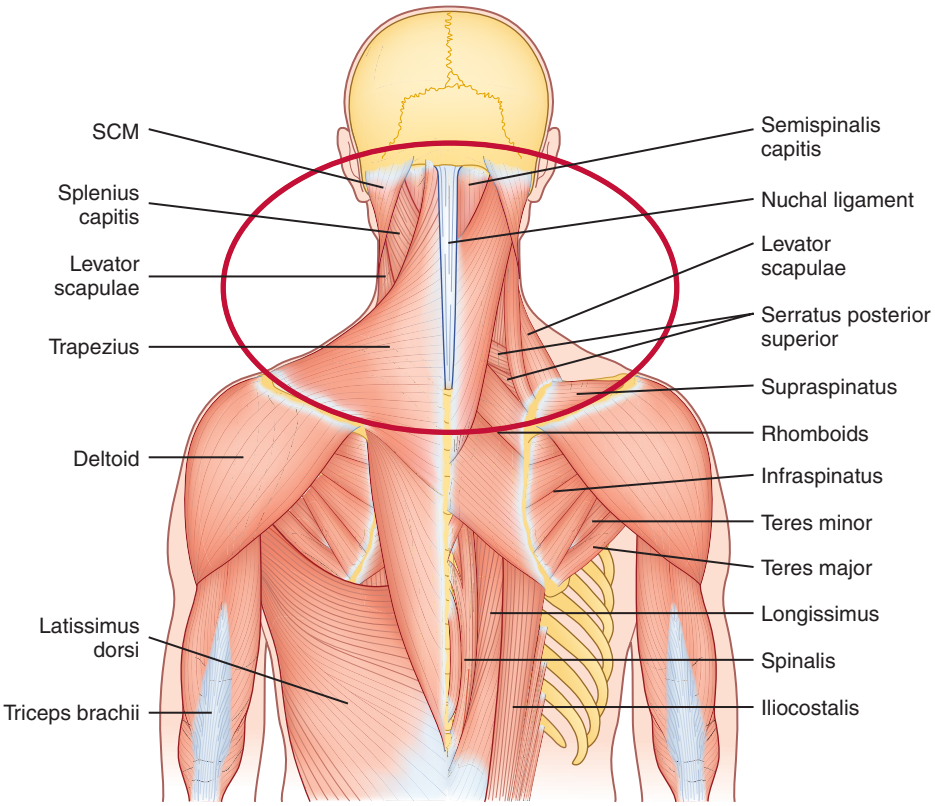

FIG 18.1 The muscles and ligaments of the neck are subject to injury from acute trauma and repetitive stress and overuse injuries. (From Waldman SD. *The spine: pain medicine: a case-based learning series*. Philadelphia: Elsevier, 2022:22–30.)

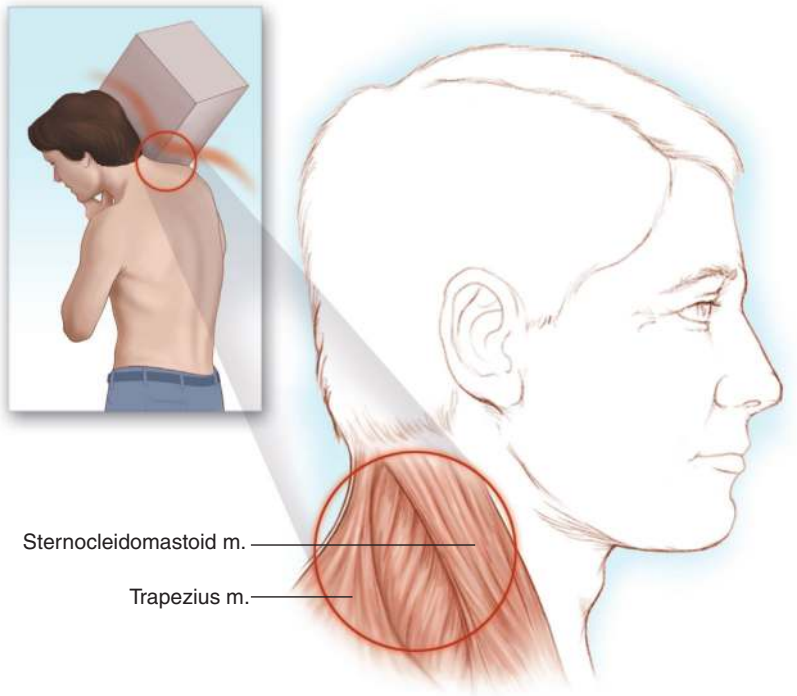

FIG 18.2 Cervical strain is often caused by trauma to the cervical spine and adjacent soft tissues.

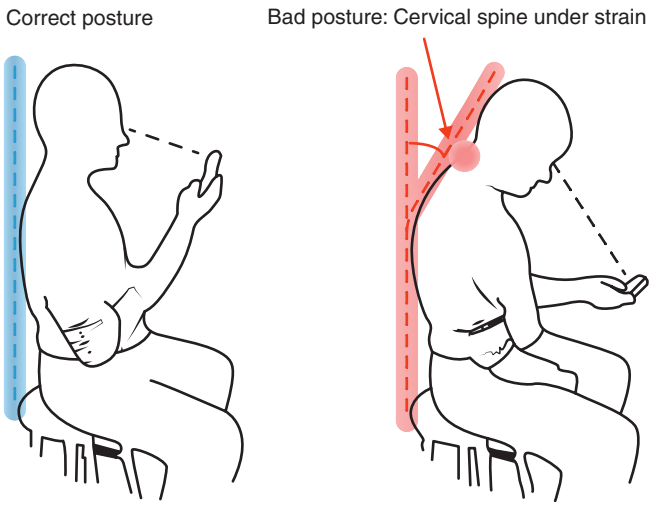

FIG 18.3 Poor posture and an excessive tilt angle of the neck when using a smartphone may result in cervical strain. (From Lamonaca F, Polimeni G, Barbé K, et al. Health parameters monitoring by smartphone for quality of life improvement. *Measurement.* 2015;73:82–94, Fig. 7.)

often present. Decreased range of motion is invariably present, and pain is increased when this maneuver is attempted. The neurologic examination of the upper extremities is within normal limits, despite the frequent complaint of upper extremity pain.

TESTING

No specific test exists for cervical strain. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic cervical strain (see "Differential Diagnosis"). Plain radiographs can delineate any bony abnormality of the cervical spine, including arthritis, fracture, congenital abnormality (e.g., Arnold–Chiari malformation), and tumor (Fig. 18.4). Straightening of the lordotic curve is frequently noted. All patients with the recent onset of cervical strain should undergo magnetic resonance imaging (MRI) of the cervical spine and, if significant occipital or headache symptoms are present, of the brain (Fig. 18.5). Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, human leukocyte antigen-B27 antigen screening, and automated blood chemistry should be performed to rule out occult inflammatory arthritis, infection, and tumor.

DIFFERENTIAL DIAGNOSIS

Cervical strain is a clinical diagnosis supported by a combination of clinical history, physical examination, radiography, and MRI. Pain syndromes that may mimic cervical strain include cervical bursitis, cervical fibromyositis, inflammatory arthritis, and disorders of the cervical spinal cord, roots, plexus, and nerves (Box 18.1).

TREATMENT

Cervical strain is best treated with a multimodality approach. Physical therapy, including heat modalities and deep sedative massage, combined with nonsteroidal antiinflammatory drugs and skeletal muscle relaxants, is a reasonable starting point. For symptomatic relief, cervical epidural block, blockade of the medial branch of the dorsal ramus, or intraarticular injection of the facet joint with local anesthetic and steroid is extremely effective. Underlying sleep disturbance and depression are best treated with a tricyclic antidepressant such as nortriptyline, which can be started at a single bedtime dose of 25 mg.

Cervical facet block is often combined with atlantooccipital block when treating pain in this area. Although the

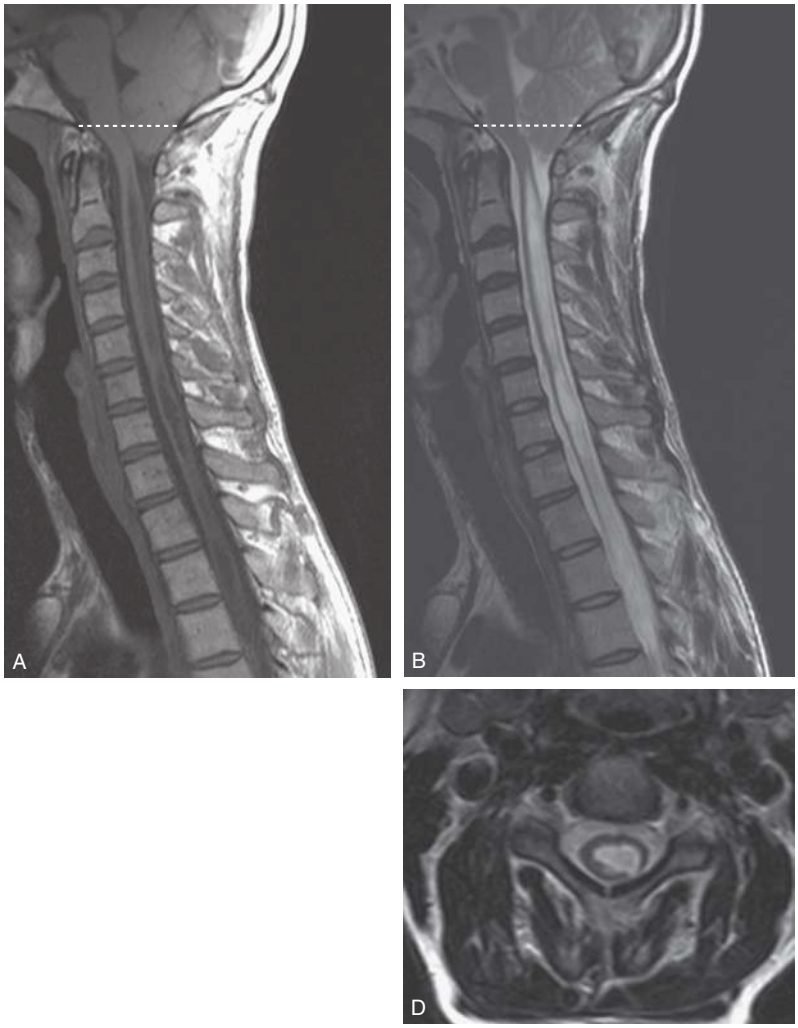

FIG 18.4 Sagittal T1W **(A)** and T2W **(B)** magnetic resonance (MR) images of a patient with Arnold–Chiari malformation type I. The cerebellar tonsils protrude through the foramen magnum (*broken line*), and an associated syrinx is present. The fourth ventricle is normal, and there is no meningocele or other structural defect. **C,** The herniation of the cerebellar tonsils (*white arrows*) is seen on the axial T2W MR image taken through the level of C1. **D,** The syrinx is also well demonstrated on the axial T2W MR image taken through the midcervical spine. (From Waldman SD, Campbell R. *Imaging of pain.* Philadelphia: Saunders; 2011:Fig. 8.1.)

atlanto-occipital joint is not a true facet joint in the anatomic sense, the technique is analogous to the facet joint block commonly used by pain practitioners and may be viewed as such.

COMPLICATIONS AND PITFALLS

The proximity to the spinal cord and exiting nerve roots makes it imperative that cervical epidural block and cervical facet block be carried out only by those familiar with the regional anatomy and experienced in interventional pain management techniques. The proximity to the vertebral artery, combined with the vascular nature of this region, makes the potential for intravascular injection high, and the injection of even a small amount of local anesthetic into the vertebral artery can result in seizures. Given the proximity of the brain and brainstem, ataxia resulting from vascular uptake of local anesthetic is not uncommon after cervical facet block. Many

BOX 18.1 Treatment Modalities for Cervical Strain

- Physical modalities
  - Physical therapy
  - Local heat
  - Deep sedative massage
  - Ice rubs
- Medication management
  - Simple analgesics
  - Nonsteroidal antiinflammatory agents
  - Skeletal muscle relaxants
- Acupuncture
- Manipulative therapy
- Interventional pain management
  - Trigger point injections
  - Medial branch block
  - Cervical epidural block

FIG 18.5 Syringohydromyelia. **A,** T1-weighted sagittal magnetic resonance imaging (MRI) of the cervical spine demonstrating a Chiari type I malformation with low-lying cerebellar tonsils (*straight arrow*) and a tight foramen magnum. A syrinx cavity is noted in the cervical spinal cord (*curved arrow*). **B,** T1-weighted axial MRI demonstrating the eccentric nature of the syrinx cavity, with internal septa or haustrations (*arrows*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al. eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2304.)

patients also complain of a transient increase in headache and cervicalgia after injection of the cervical facet joints.

CLINICAL PEARLS

Cervical strain is a common cause of neck, occipital, shoulder, and upper extremity pain. It is often confused with cervical radiculopathy and cervical fibromyositis. The clinician must rule out diseases of the cervical spinal cord, such as syringomyelia, that may initially manifest in a manner similar to cervical strain. Ankylosing spondylitis may also manifest as cervical strain and must be correctly identified to avoid ongoing joint damage and functional disability.

Many pain specialists believe that cervical facet block and atlanto-occipital block are underused in the treatment of "post-whiplash" cervicalgia and cervicogenic headaches and that they should be considered whenever cervical epidural or occipital nerve blocks fail to provide palliation.

SUGGESTED READINGS

Candido KD, England B. Cervical spine pain related to the facet joints. *Tech Reg Anesth Pain Manag*. 2015;19(3–4):109–118.

Groeneweg R, Haanstra T, Bolman CAW, et al. Treatment success in neck pain: the added predictive value of psychosocial variables in addition to clinical variables. *Scand J Pain*. 2017;14:44–52.

Gustafsson E, Thomée S, Grimby-Ekman A, et al. Texting on mobile phones and musculoskeletal disorders in young adults: a five-year cohort study. *Appl Ergon*. 2017;58:208–214.

Han H, Shin G. Head flexion angle when web-browsing and texting using a smartphone while walking. *Appl Ergon*. 2019;81:284–287.

Hudgins TH, Origenes AK, Pleuhs B, et al. *Essentials of physical medicine and rehabilitation*. 4th ed. Philadelphia: Elsevier; 2020:29–32.

Ris I, Juul-Kristensen B, Boyle E, et al. Chronic neck pain patients with traumatic or non-traumatic onset: differences in characteristics. A cross-sectional study. *Scand J Pain*. 2017;14:1–8.

Vasavada AN, Nevins DD, Monda SM, et al. Gravitational demand on the neck musculature during tablet computer use. *Ergonomics*. 2015;58(6):990–1004.

Verwoerd M, Wittink H, Maissan F, de Raai Ej, Smeets RJEM. Prognostic factors for persistent pain after a first episode of nonspecific idiopathic, non-traumatic neck pain: a systematic review. *Musculoskelet Sci Pract*. 2019;42:13–37.

19

Longus Colli Tendinitis

ICD-10 CODE M65.20

THE CLINICAL SYNDROME

The tendons of the longus colli muscle are prone to the development of tendinitis. Longus colli tendinitis is usually caused either by repetitive trauma to the musculotendinous apparatus or by the deposition of calcium hydroxyapatite crystals. This crystal deposition usually occurs in the superior fibers of the musculotendinous apparatus and is easily identified on a lateral plain radiograph of the neck. The onset of longus colli tendinitis is generally acute, and it is often misdiagnosed as acute pharyngitis or retropharyngeal abscess because the acute onset of retropharyngeal pain is frequently accompanied by a mild elevation in temperature and leukocytosis. Longus colli tendinitis is most often seen in the third to sixth decades of life.

SIGNS AND SYMPTOMS

The pain of longus colli tendinitis is constant and severe and is localized to the retropharyngeal area. It is made worse by swallowing (Fig. 19.1). The patient may complain of acute anterior neck pain in addition to the pain on swallowing. Referred pain from the inflamed longus colli muscle to the anterior and posterior neck often occurs (Fig. 19.2). A mild fever is often present, as is mild leukocytosis. Intraoral palpation of the superior attachment of the muscle usually reproduces the symptoms.

TESTING

Plain radiographs are indicated for all patients who present with retropharyngeal pain. Characteristic amorphous

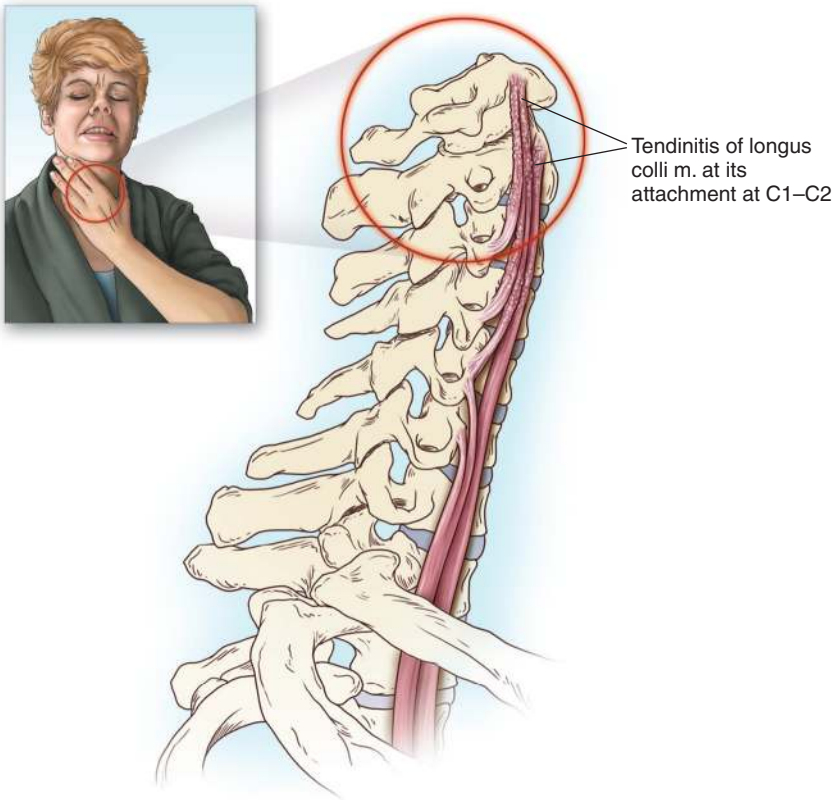

FIG 19.1 The pain of longus colli tendinitis is constant, severe, made worse by swallowing, and localized to the retropharyngeal area.

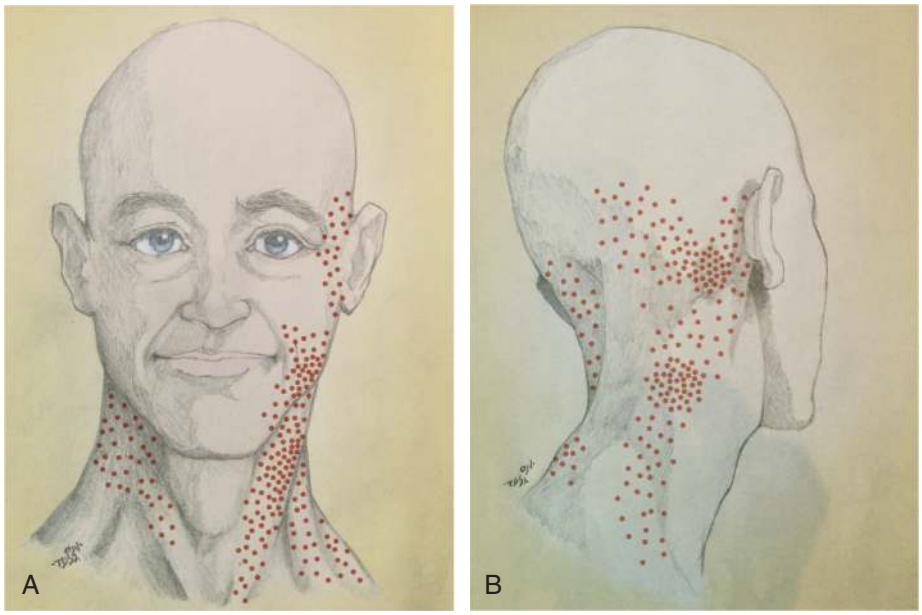

FIG 19.2 A schematic representation of the pattern of pain referral from the longus colli muscle, anterior **(A)**, and posterior **(B)** views. Dot density represents the likelihood of pain being reported at each site. (From Minerbi A, Ratmansky M, Finestone A, et al. The local and referred pain patterns of the longus colli muscle. *J Bodyw Mov Ther*. 2017;21(2):267–273, Fig. 6.)

FIG 19.3 Lateral cervical spine x-ray study showing faint calcification anterior to the junction of C1–C2 (*arrowheads*). (From Guss DA, Jacoby IJ. Longus colli tendinitis causing acute neck pain. *J Emerg Med*. 2002;22(2):211–212.)

calcification of the superior attachment of the musculotendinous unit just below the anterior arch of atlas is highly suggestive of longus colli tendinitis (Fig. 19.3). Computed tomographic scanning may further delineate the problem (Fig. 19.4). The finding of a smooth, linear prevertebral fluid collection is considered pathognomonic for this disease (Figs. 19.5 and 19.6). Unlike in a retropharyngeal or prevertebral abscess, the wall of any fluid-containing structure with air-fluid levels does not enhance (Fig. 19.7). Additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and complete blood chemistry tests, in patients suspected of suffering from longus colli tendinitis.

FIG 19.4 Computed tomography scan showing calcification of the superior aspect of the musculotendinous unit of the longus colli muscle (*arrow*). Note the relationship with the anterior arch of the atlas. (From Omezzine SJ, Hafsa C, Lahmar I, et al. Calcific tendinitis of the longus colli: diagnosis by CT. *Joint Bone Spine*. 2008;75(1):90–91.)

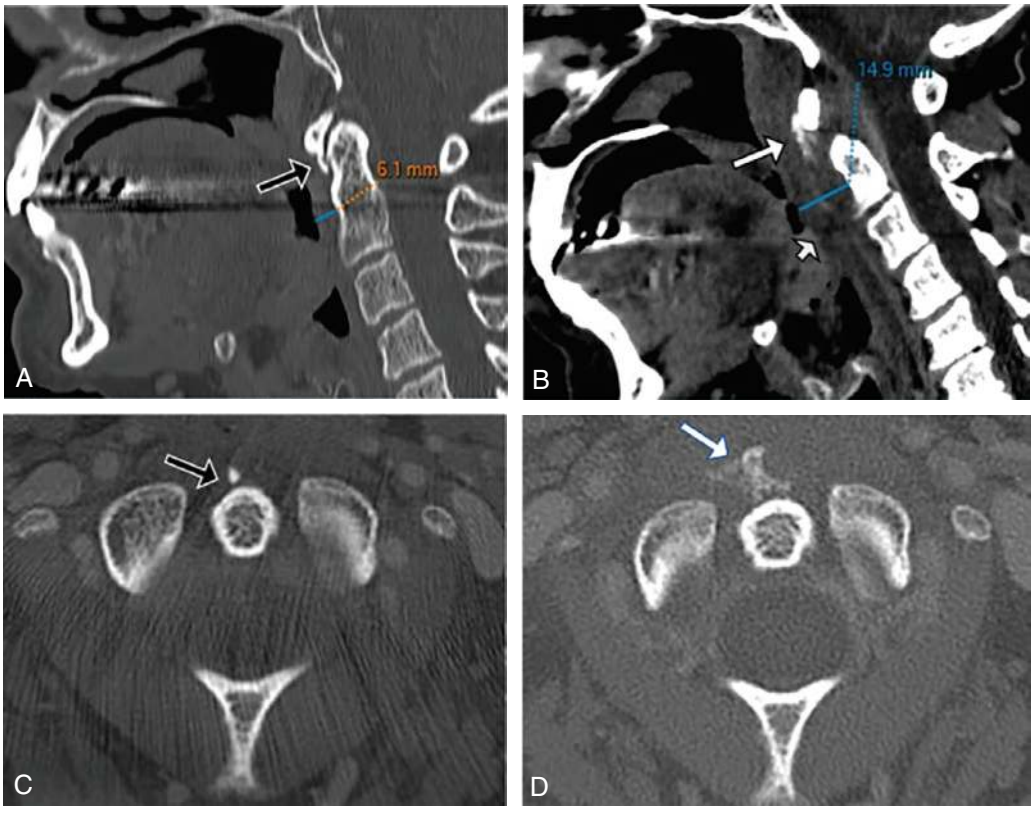

FIG 19.5 Axial and saggital computed tomography (CT) of the neck. Sagittal **(B)** and axial **(D)** CT shows calcifications (*white arrow*) in the longus colli muscle (LCM) in front of the C1–C2 level with poorly defined margins, a thickening (15 mm) (→)of the prevertebral soft tissue. Sagittal **(A)** and axial **(C)** of former CT scan performed 3 years ago showed that the calcification (*black arrow*) in the LCM was smaller with well-defined margins, and no thickening of the prevertebral soft tissue. (From Guerroum H, Koubaa I, Benissad A. Calcific tendinitis of the longus colli: an uncommon cause of neck pain. *Radiol Case Rep*. 2022;17(4):1228–1230.)

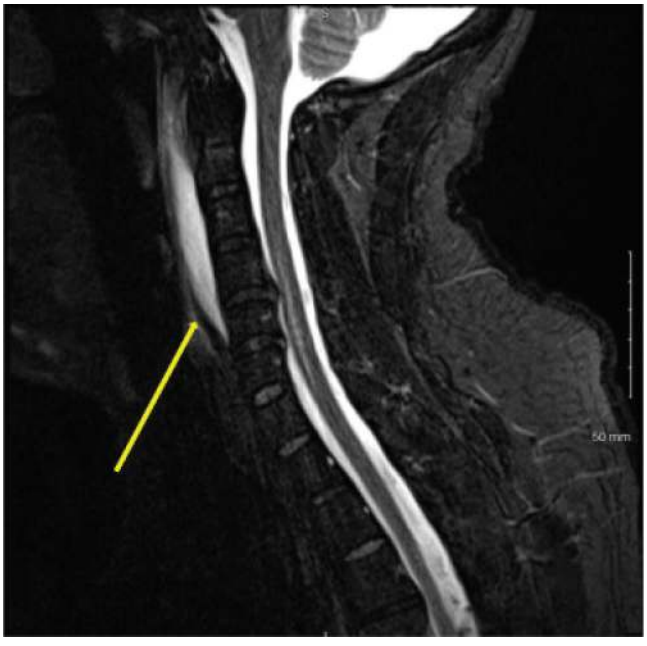

FIG 19.6 Sagittal magnetic resonance imaging with short tau inversion recovery (MRI STIR) demonstrates a retropharyngeal and prevertebral fluid collection that extends from the C1–C2 level inferiorly to the C5 vertebral body. (From Kessler B, Leyva JD, Schmitt C, Murtagh RD. Acute longus colli tendinitis: case report of a rare cause of neck pain. *Radiol Case Rep*. 2020;15(9):1433–1436.)

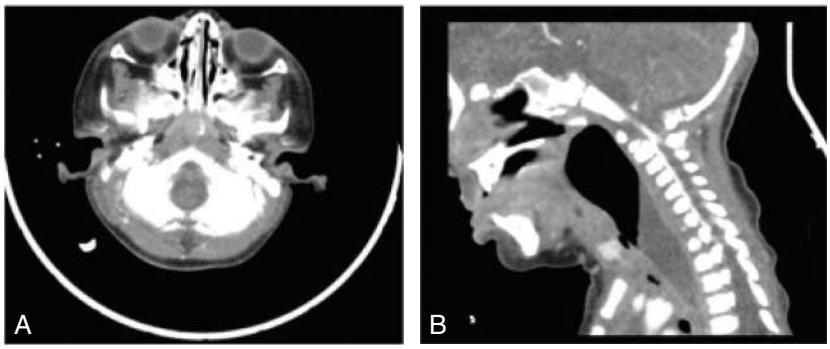

FIG 19.7 Axial (A) and sagittal (B) contrasted computed tomography (CT) scan of head and neck, demonstrating retropharyngeal abscess with large cavity measuring 6 × 9 × 12 cm has wide air-fluid level extending until the level of T4. (From Alkhodair AA, Alkusayer MM, Albadah AA. Retropharyngeal abscess: a rare complication of nasogastric tube insertion. *J Pediatr Surg Case Rep*. 2022; 78:102197.)

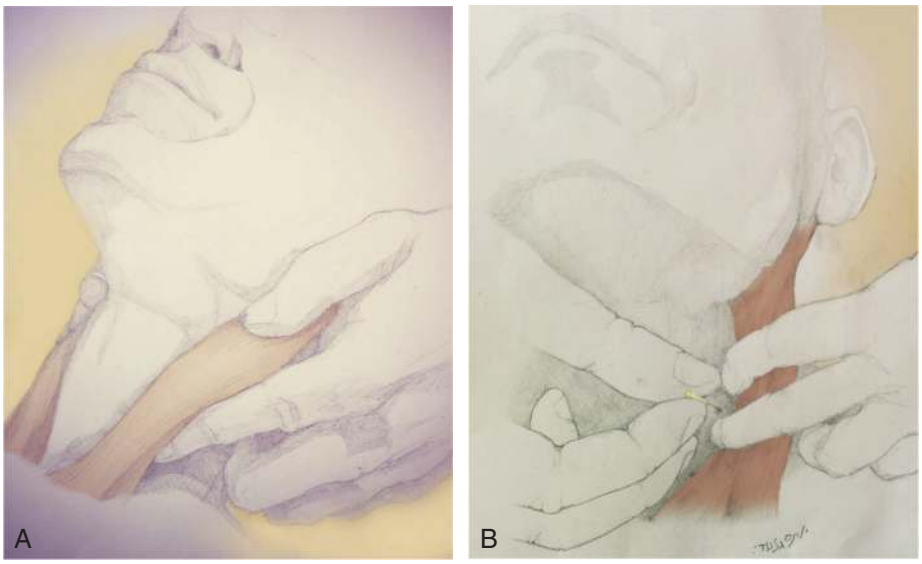

FIG 19.8 A demonstration of the techniques of massaging (A) and dry-needling (B) of the longus colli muscle. (From Minerbi A, Ratmansky M, Finestone A, et al. The local and referred pain patterns of the longus colli muscle. *J Bodyw Mov Ther*. Epub 5 Apr 2017, ISSN 1360–8592, Fig. 2.)

DIFFERENTIAL DIAGNOSIS

Longus colli tendinitis is often misdiagnosed as acute pharyngitis or retropharyngeal abscess. Occasionally, the patient is diagnosed with an early peritonsillar abscess. This delay in diagnosis can often subject the patient to unnecessary antibiotic therapy and occasionally surgical drainage of the suspected "abscess." In some clinical situations, consideration should be given to primary or secondary tumors involving this anatomic region.

TREATMENT

Initial treatment of the pain and functional disability associated with longus colli tendinitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors. Local application of heat and cold, dry needling, and deep sedative massage may also be beneficial (Fig. 19.8). For patients who do not respond to these treatment modalities, injection of the superior portion of the tendon with local anesthetic and steroid is a reasonable next step. Such injection should be considered only if the clinician is certain that no occult infection in this anatomic region exists. Ultrasound needle guidance may simplify needle placement and avoid injury to surrounding structures including the thyroid gland, carotid artery, jugular vein, and exiting cervical nerve roots (Fig. 19.9).

COMPLICATIONS AND PITFALLS

The main pitfalls in the treatment of longus colli tendinitis are failure to diagnose this painful condition in a timely manner

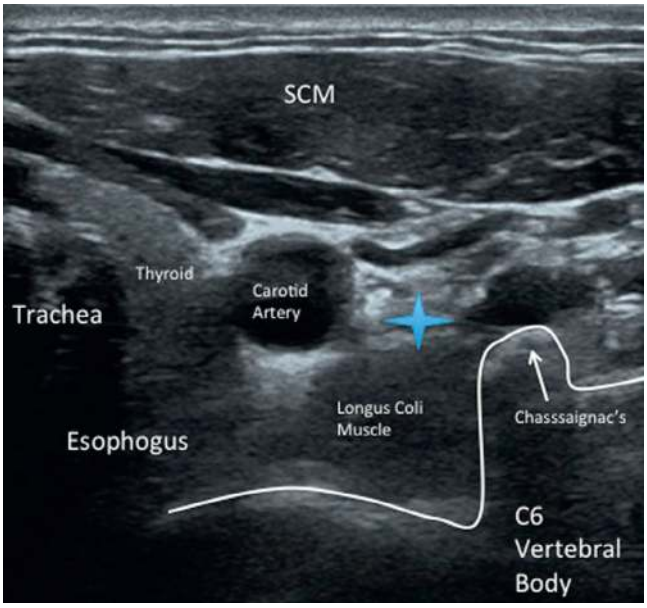

FIG 19.9 Ultrasound needle guidance may simplify needle placement and avoid injury to surrounding structures, including the thyroid gland, carotid artery, jugular vein, and exiting cervical nerve roots.

and mistaking it for a disease requiring more intensive treatment (e.g., retropharyngeal abscess or peritonsillar abscess). Rapid institution of treatment with NSAIDs and reassurance is often all that is required. For more recalcitrant cases, injection with local anesthetic and steroid almost always results in prompt resolution of symptoms. Use of this injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Trauma to the tendon from the injection itself is also a possibility. Tendons that are highly inflamed or previously damaged are subject to rupture if they are injected directly. This complication can often be avoided if the clinician uses a gentle technique and stops injecting immediately on encountering significant resistance. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The musculotendinous unit of the longus colli muscle is susceptible to the development of tendinitis. Calcium hydroxyapatite deposition around the tendon may occur, thus making subsequent treatment more difficult. NSAIDs usually provide excellent palliation of the patient's pain. If they do not, properly performed injection of the inflamed musculotendinous unit with local anesthetic and steroid is a reasonable next step.

SUGGESTED READINGS

Alkhodair AA, Alkusayer MM, Albadah AA. Retropharyngeal abscess: a rare complication of nasogastric tube insertion. *J Pediatr Surg Case Rep*. 2022;78:102197.

Boardman J, Kanal E, Aldred P, et al. Frequency of acute longus colli tendinitis on CT examinations. *Emerg Radiol*. 2017;24(6):645–651.

Guerroum H, Koubaa I, Benissad A. Calcific tendinitis of the longus colli: an uncommon cause of neck pain. *Radiol Case Rep*. 2022;17(4):1228–1230.

Kang T, Park SY, Lee SH, Park JH. Acute calcific tendinitis of the longus colli. *Pain Med*. 2020;21:1706–1708.

Kessler B, Leyva JD, Schmitt C, Murtagh R. Acute longus colli tendinitis: case report of a rare cause of neck pain. *Radiol Case Rep*. 2020;15(9):1433–1436.

Siag K, Mazzawi S, Paker M, et al. Acute longus colli tendinitis and otolaryngology. *Braz J Otorhinolaryngol*. 2022;88(3):351–357

Suh B, Eoh J, Shin J. Clinical and imaging features of longus colli calcific tendinitis: an analysis of ten cases. *Clin Orthop Surg*. 2018;10(2):204–209.

Waldman SD. Longus colli tendinitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Kluwer Wolters; 2016:215–221.

Retropharyngeal Abscess

ICD-10 CODE J39.0

THE CLINICAL SYNDROME

Once a disease almost exclusively seen in children, retropharyngeal abscess is occurring more commonly in the adult population. It may occur as a sequela of upper respiratory tract infection, trauma to the posterior pharynx (e.g., difficult endotracheal intubation), or perforation from a foreign body, among other causes (Fig. 20.1). Often misdiagnosed, retropharyngeal abscess can result in life-threatening complications and, if untreated, death. The mortality and morbidity associated with retropharyngeal abscess are primarily the result of airway obstruction, mediastinitis, spread of infection to the epidural space, necrotizing fasciitis, erosion into the carotid artery, and, in immunocompromised patients, overwhelming sepsis. Lying posterior to the pharynx, the retropharyngeal space is bound by the prevertebral fascia posteriorly, the buccopharyngeal fascia anteriorly, and the carotid sheaths laterally (Fig. 20.2). Extending from the base of the skull inferiorly to the mediastinum, the retropharyngeal space is susceptible to infection by aerobic organisms such as *Streptococcus*, *Staphylococcus*, and *Haemophilus* and anaerobic organisms such as *Bacteroides*. Rarely, fungal and mycobacterial infections of the retropharyngeal space have been reported in immunocompromised patients.

The patient suffering from retropharyngeal abscess initially presents with sore throat, neck pain, and painful and difficult swallowing (Fig. 20.3). This pain becomes more

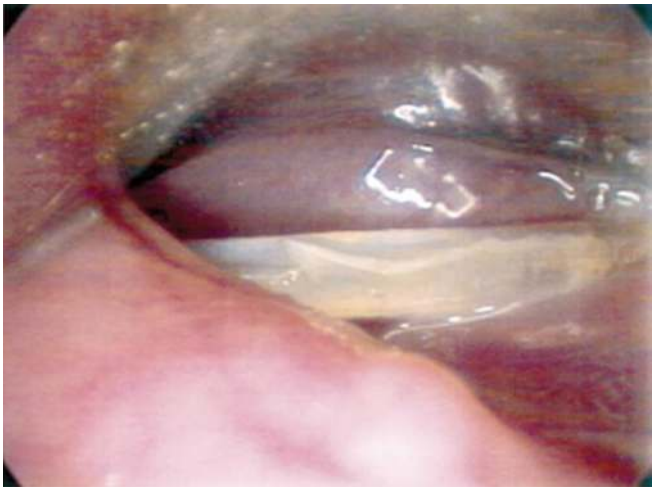

FIG 20.1 Endoscopy showing a foreign body (bone) perforating the cervical esophagus in a patient with a retropharyngeal abscess. (From Poluri A, Singh B, Sperling N, et al. Retropharyngeal abscess secondary to penetrating foreign bodies. *J Craniomaxillofac Surg*. 2000;24(4):243–246.)

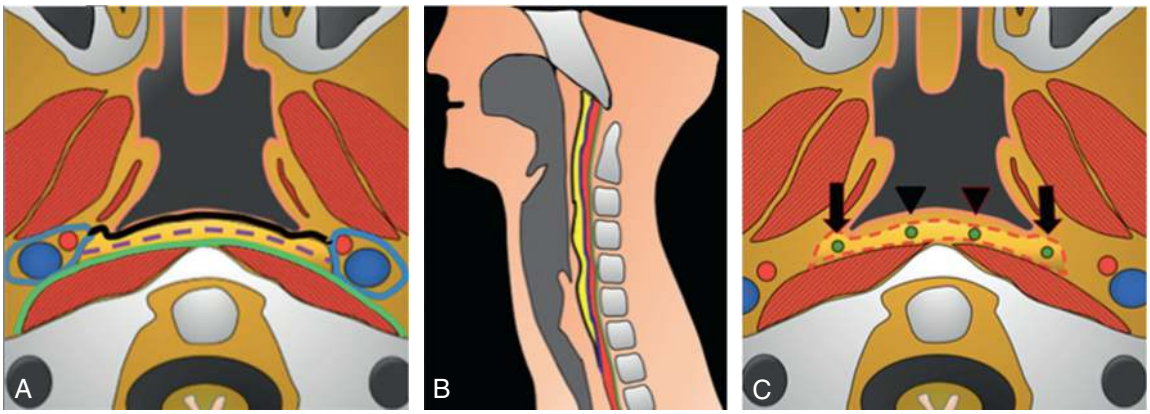

FIG 20.2 Normal anatomy of the retropharyngeal space (RPS). Axial images show visceral fascia (*black line*), prevertebral fascia (*green line*), the carotid sheath (*blue line*), and the alar fascia (*purple dashed line*) **(A)**. A sagittal image shows RPS (*yellow area*), and the danger space (*red area*) **(B)**. Medial retropharyngeal nodes (*arrowhead*) and lateral retropharyngeal nodes (*arrow*) are shown in the RPS **(C)**. (From Tomita H, Yamashiro T, Ikeda H, et al. Fluid collection in the retropharyngeal space: a wide spectrum of various emergency diseases. *Eur J Radiol*. 2016;85(7):1247–1256, Fig. 1.)

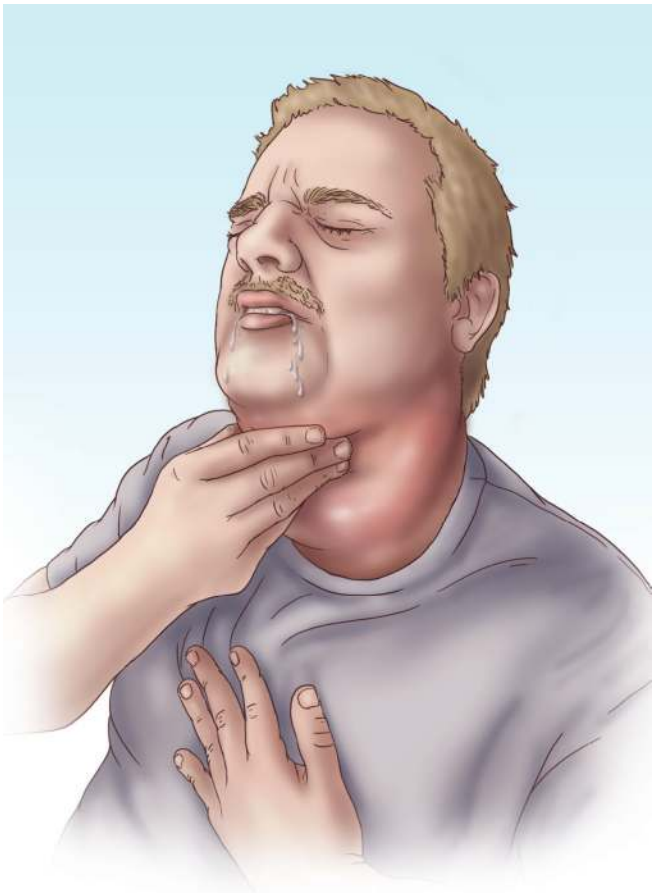

FIG 20.3 The patient suffering from retropharyngeal abscess appears acutely ill and exhibits drooling as swallowing becomes increasingly difficult.

intense and localized as the abscess increases in size and compresses adjacent structures. Low-grade fever and vague constitutional symptoms, including malaise and anorexia, progress to frank sepsis with high-grade fever, rigors, and chills. At this point, the mortality rate associated with retropharyngeal abscess rises dramatically, despite treatment with appropriate antibiotics and surgical drainage of the abscess.

SIGNS AND SYMPTOMS

The patient with retropharyngeal abscess initially presents with ill-defined pain in the general area of the infection. At this point, the patient may have mild pain on swallowing and range of motion of the cervical spine. The physical examination at this point may reveal posterior pharyngeal swelling. Low-grade fever or night sweats may be present. Theoretically, if the patient has received steroids, these constitutional symptoms may be attenuated or their onset may be delayed. As the abscess increases in size, the patient appears acutely ill with fever, rigors, and chills. Drooling may be present as the patient finds it increasingly difficult to swallow. Nuchal rigidity and respiratory stridor may also be

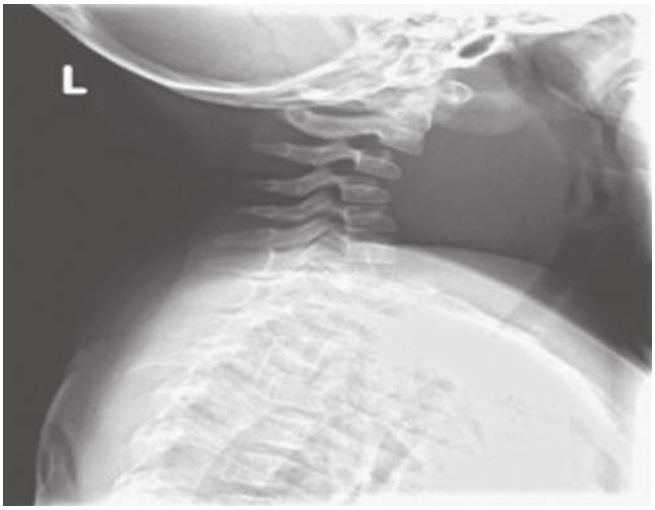

FIG 20.4 Lateral neck radiograph revealing a massive prevertebral swelling that caused cervical spine kyphosis. (From Strovski E, Mickelson J-I, Ludemann JP. Minimally invasive drainage of a giant retropharyngeal abscess. *Int J Pediatr Otorhinolaryngol Extra.* 2009;4(2):92–95.)

evident. Spread to the mediastinum and central nervous system is associated with a high mortality rate in spite of aggressive medical and surgical treatment.

TESTING

Lateral radiography of the neck reveals widening of the retropharyngeal soft tissues in more than 80% of patients suffering from retropharyngeal abscess; clearly defined soft tissue masses with air-fluid levels suggestive of abscess are seen in less than 10% of patients (Fig. 20.4). In this era of readily available magnetic resonance imaging (MRI) and high-speed computed tomography (CT) scanning, it may be more prudent to obtain this noninvasive testing first, given the highly specific diagnostic information obtained (Figs. 20.5 and 20.6). Both MRI and CT are highly accurate in the diagnosis of retropharyngeal abscess and should be obtained on an urgent basis in all patients suspected of suffering from this condition (Fig. 20.7). Ultrasonography may also be useful in identifying retropharyngeal abscess (Fig. 20.8).

All patients suspected of suffering from retropharyngeal abscess should undergo laboratory testing consisting of complete blood cell count, sedimentation rate, and automated blood chemistries. Studies suggest that C-reactive protein testing may also be beneficial because patients with markedly elevated levels have higher morbidity and mortality rates. Blood and urine cultures should be immediately obtained in all patients thought to be suffering from retropharyngeal abscess to allow immediate implementation of antibiotic therapy while the workup is in progress. Gram stains and cultures of the abscess material should also be obtained, but antibiotic treatment should not be delayed while waiting for this information.

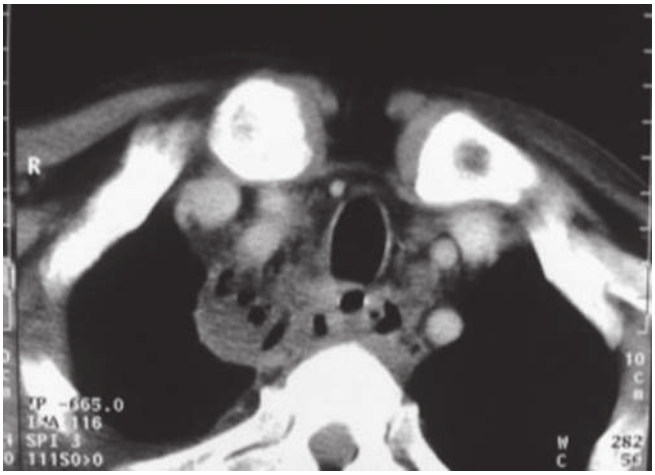

FIG 20.5 Computed tomography scan demonstrating multiple air-fluid collections in the upper mediastinum and the retropharyngeal and parapharyngeal spaces that represented the presence of abscess. (From Abu Abeeleh M, Al Smady M, Qasem H, et al. Descending necrotising mediastinitis: a fatal disease to keep in mind. *Heart Lung Circ*. 2010;19(4):254–256.)

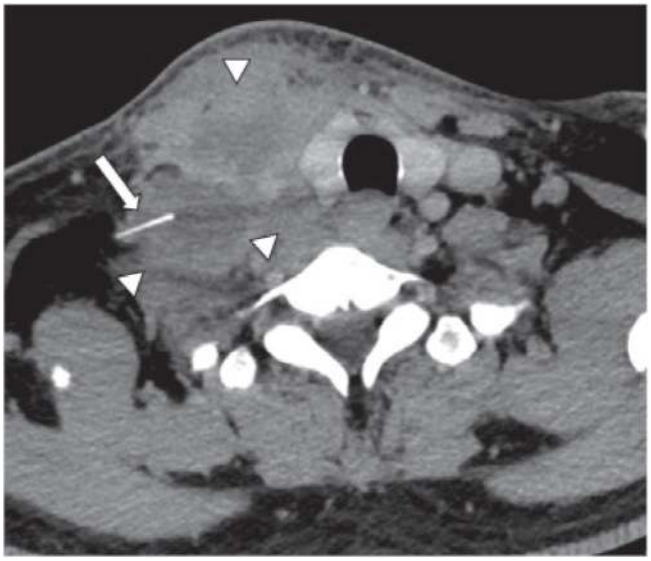

FIG 20.6 Axial contrast-enhanced computed tomography image of the neck of a patient with fever and neck swelling. There is a migrated fish bone in the right supraclavicular region (*arrow*) and formation of a large multiloculated abscess (*arrowheads*). The fish bone was subsequently removed during incision and drainage of the abscess. (From Khoo HW, Ong CYG, Chinchure D. Teach a man to fillet: gastrointestinal and extra-gastrointestinal complications related to fish bone ingestion. *Clin Imaging*. 2021;69:150–157.)

FIG 20.7 Preoperative sagittal contrast-enhanced T1-weighted magnetic resonance imaging showing a prevertebral abscess (*arrow*) and an epidural abscess causing spinal cord compression from C3 to C7 with diskitis at C5–C6. (From Yanni DS, LaBagnara M, Saravanan R, et al. Transcervical drainage of epidural and retropharyngeal abscess. *J Clin Neurosci*. 2010;17(5):636–638.)

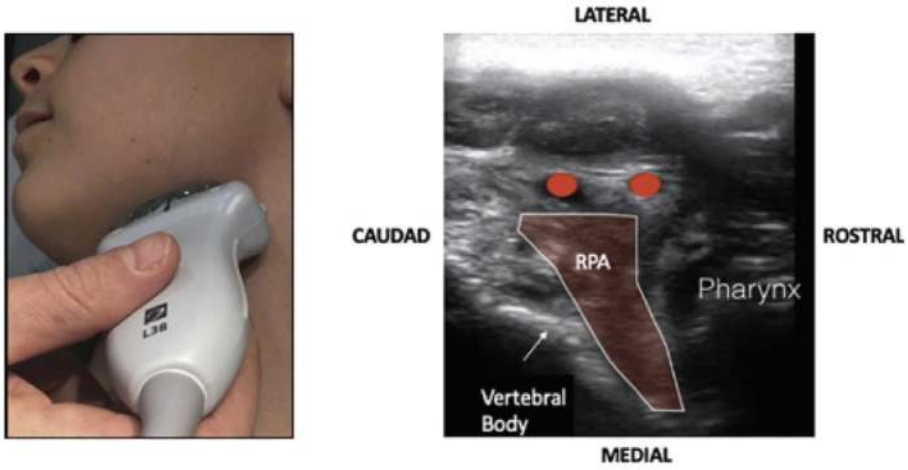

FIG 20.8 Transducer placement and ultrasound imaging for evaluation of retropharyngeal abscess with corresponding abnormal anatomy. (From Malia L, Sivitz A, Chicaiza H. A novel approach: point-of-care ultrasound for the diagnosis of retropharyngeal abscess. *Am J Emerg Med.* 2021;46:271–275.)

DIFFERENTIAL DIAGNOSIS

The diagnosis of retropharyngeal abscess should be strongly considered in any patient with sore throat, fever, neck pain, painful and difficult swallowing, and posterior pharyngeal swelling, especially if the patient has a history of trauma to the retropharyngeal space. Diseases commonly mistaken for retropharyngeal abscess are listed in Box 20.1. Constitutional symptoms associated with serious infection may be attenuated in patients who have been receiving steroids or who are immunocompromised (e.g., acquired immunodeficiency syndrome, malignant disease).

BOX 20.1 Differential Diagnosis of Retropharyngeal Abscess

- Angioedema
- Caustic ingestions
- Cervical epidural abscess
- Cervical subdural abscess
- Epiglottitis
- Esophagitis
- Foreign body of esophagus
- Foreign body of pharynx
- Foreign body of trachea
- Longus colli tendinitis
- Kawasaki's disease
- Mediastinitis
- Meningitis
- Mononucleosis
- Odontogenic infections
- Pediatric fever
- Peritonsillar abscess
- Pharyngitis
- Severe thrush
- Sinusitis

TREATMENT

The rapid initiation of the treatment of retropharyngeal abscess is mandatory if the patient is to avoid significant morbidity and mortality. The treatment of retropharyngeal abscess is aimed at two goals: (1) treatment of the infection with antibiotics and (2) drainage of the abscess to relieve compression on adjacent structures including the airway. Because many retropharyngeal abscesses are caused by *Staphylococcus aureus*, the initial antibiotic regimen should include vancomycin to treat staphylococcal infection. Gram-negative and anaerobic antibiotic coverage should also be started empirically immediately after blood and urine culture samples are taken. Antibiotic therapy can be tailored to the culture and sensitivity reports as they become available. As mentioned, antibiotic therapy should not be delayed while waiting for definitive diagnosis if retropharyngeal abscess is being considered as part of the differential diagnosis.

Antibiotics alone are rarely successful in the treatment of retropharyngeal abscess unless the diagnosis is made very early in the course of the disease; surgical drainage of the abscess is required to effect full recovery. Careful attention to airway management is mandatory in patients suspected of suffering from retropharyngeal abscess, and early endotracheal intubation in a controlled setting is preferred to waiting until respiratory compromise is already present.

Serial CT or MRI scans are useful in following the resolution of retropharyngeal abscess. These imaging tests should be repeated immediately at the first sign of negative change in the patient's clinical status.

COMPLICATIONS AND PITFALLS

Failure to diagnose and treat retropharyngeal abscess rapidly and accurately can result only in disaster for the clinician and patient alike. The insidious onset of airway compromise associated with retropharyngeal abscess can lull the clinician into a sense of false security, and failure to recognize the spread of infection into the central nervous system can result in permanent neurologic damage. If retropharyngeal abscess is suspected, the algorithm listed in Box 20.2 should be followed.

BOX 20.2 Algorithm for Spinal Cord Compression Resulting From Retropharyngeal Abscess

- Immediately obtain blood and urine cultures.
- Immediately start high-dose antibiotics that cover *Staphylococcus aureus*.
- Immediately obtain the most readily available spinal imaging technique that can confirm the presence of spinal cord compression, such as abscess, tumor, and others.
  - Computed tomography
  - Magnetic resonance imaging
  - Myelography
- Simultaneously obtain emergency consultation from a spinal surgeon.
- Continuously and carefully monitor the patient's neurologic status.
- If any of the above is unavailable, arrange emergency transfer of the patient to a tertiary care center by the most rapidly available transportation.
- Repeat imaging and obtain a repeat surgical consultation if any deterioration in the patient's neurologic status occurs.

CLINICAL PEARLS

Delay in diagnosis puts the patient and clinician at tremendous risk for a poor outcome. The clinician should assume that all patients who present with sore throat, fever, neck pain, painful and difficult swallowing, and posterior pharyngeal swelling are suffering from retropharyngeal abscess until proved otherwise and should treat these patients accordingly. Overreliance on a single negative or equivocal imaging test result is a mistake. Serial CT or MRI testing is indicated should any deterioration in the patient's clinical status occur.

SUGGESTED READINGS

Akhavan M. Ear, nose, throat: beyond pharyngitis: retropharyngeal abscess, peritonsillar abscess, epiglottitis, bacterial tracheitis, and postoperative tonsillectomy. *Emerg Med Clin North Am*. 2021;39(3):661–675.

Alkhodair AA, Alkusayer MM, Albadah AA. Retropharyngeal abscess: a rare complication of nasogastric tube insertion. *J Pediatric Surg Case Rep*. 2022;78:102197.

Awobajo MD, Agarwal AN, Hackman SD. Retropharyngeal abscess—an unusual complication in a COVID-19 patient. *Otolaryngol Case Rep*. 2021;20:100312.

Khoo HW, Ong CYG, Chinchure D. Teach a man to fillet: gastrointestinal and extra-gastrointestinal complications related to fish bone ingestion. *Clin Imaging*. 2021;69:150–157.

Malia L, Sivitz A, Chicaiza H. A novel approach: point-of-care ultrasound for the diagnosis of retropharyngeal abscess. *Am J Emerg Med*. 2021;46:271–275.

Sánchez CIS, Angulo CM. Retropharyngeal abscess. Clinical review of twenty-five years. *Acta Otorrinolaringol (Engl Ed.)*. 2021;72(2):71–79.

Tiresse N, Oucharqui S, Benaissa E, et al. The occurrence of a multidrug-resistant tuberculous retropharyngeal abscess in an immunocompetent patient: a case report. *IDCases*. 2021;26:e01282.

21

Cervicothoracic Interspinous Bursitis

ICD-10 CODE M71.50

THE CLINICAL SYNDROME

The interspinous ligaments of the lower cervical and upper thoracic spine and their associated muscles are susceptible to the development of acute and chronic pain symptoms following overuse. Bursitis is thought to be responsible for this pain. Frequently, the patient presents with midline pain after prolonged activity requiring hyperextension of the neck, such as painting a ceiling, or following prolonged use of a computer monitor with too high a focal point.

SIGNS AND SYMPTOMS

The pain is localized to the interspinous region between C7 and T1 and does not radiate. It is constant, dull, and aching. The patient may attempt to relieve the constant ache by assuming a posture of dorsal kyphosis with a thrusting forward of the neck (Fig. 21.1). In contrast to the pain of cervical strain, the pain of cervicothoracic interspinous bursitis often lessens with activity and worsens with rest. On physical examination, tenderness is elicited on deep palpation of the C7–T1 region, often with reflex spasm of the associated paraspinous musculature. Decreased range of motion is invariably present, and pain increases with extension of the lower cervical and upper thoracic spine.

TESTING

No specific test exists for cervicothoracic bursitis, although magnetic resonance imaging (MRI) may reveal inflammation of interspinous bursae (Fig. 21.2). Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic cervicothoracic bursitis (see "Differential Diagnosis"). Plain radiographs can delineate any bony abnormality of the cervical spine, including arthritis, fracture, congenital abnormality (e.g., Arnold–Chiari malformation), and tumor. All patients with the recent onset of cervicothoracic bursitis should undergo MRI of the cervical spine and, if significant occipital or headache symptoms are present, of the brain (Fig. 21.3). Ultrasound imaging may also be useful in further distinguishing solid

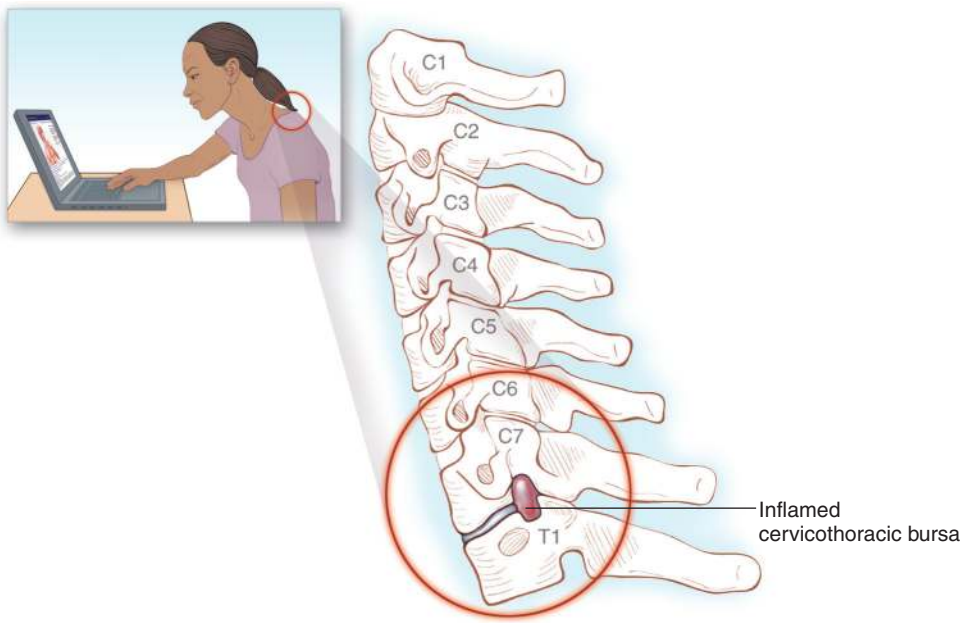

FIG 21.1 A patient with cervicothoracic interspinous bursitis may attempt to relieve the constant ache by assuming a posture of dorsal kyphosis with a thrusting forward of the neck.

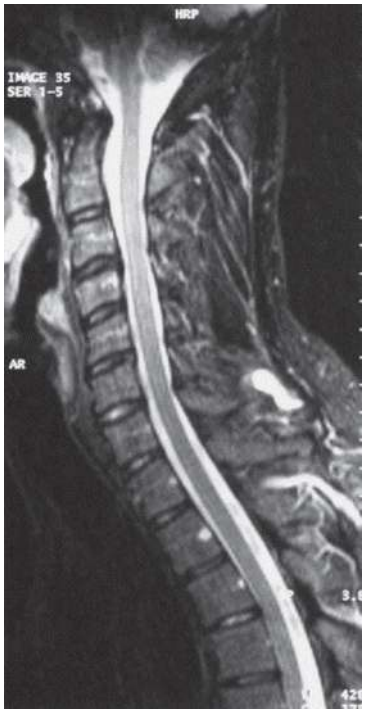

FIG 21.2 Magnetic resonance imaging (T2) of an interspinous bursa measuring 2 × 2 × 2.5 cm between C6 and C7. (From Perka C, Schneider SV, Buttgereit F, Matziolis G. Development of cervical interspinous bursitis after prolonged sports trauma: a case report. *Jt Bone Spine*. 2006;73(1):118–120.)

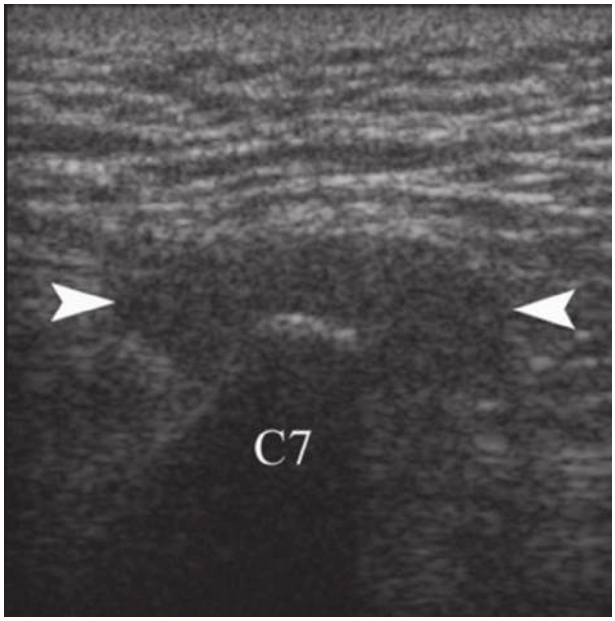

FIG 21.4 Posterior longitudinal gray-scale ultrasound (US) scan in the midline of the cervical spine. Onset of disease in a 78-year-old man with inflammatory pain and stiffness in neck and both shoulders. US demonstrated a hypoechoic swelling (between *arrowheads*) of the soft tissues surrounding spinous process of the seventh cervical vertebra (C7). (From Falsetti P, Acciai C. Ultrasound in assessment of cervical interspinous bursitis in polymyalgia rheumatica. *Jt Bone Spine*. 2013;80(3):342–343, Fig. 1.)

FIG 21.3 Klippel–Feil anomaly. T1-weighted (A) and T2-weighted (B) sagittal magnetic resonance imaging of the cervical spine that demonstrates lack of segmentation of the C4 and C5 vertebrae (*arrows*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al. eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2306.)

from cystic interspinous masses (Fig. 21.4). Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry should be performed to rule out occult inflammatory arthritis, infection, and tumor.

DIFFERENTIAL DIAGNOSIS

Cervicothoracic bursitis is a clinical diagnosis of exclusion supported by a combination of clinical history, physical examination, radiography, and MRI. Pain syndromes that

may mimic cervicothoracic bursitis include cervical strain, cervical fibromyositis, inflammatory arthritis, and disorders of the cervical spinal cord, roots, plexus, and nerves. Congenital abnormalities such as Arnold–Chiari malformation and Klippel–Feil syndrome may also manifest similarly to cervicothoracic bursitis.

TREATMENT

Cervicothoracic bursitis is best treated with a multimodality approach. Physical therapy consisting of the correction of functional abnormalities (e.g., poor posture, improper chair, or computer height), heat modalities, and deep sedative massage, combined with nonsteroidal antiinflammatory drugs (NSAIDs) and skeletal muscle relaxants, is a reasonable starting point. If these treatments fail to provide rapid relief, injection of local anesthetic and steroid into the area between the interspinous ligament and the ligamentum flavum is a reasonable next step (Fig. 21.5). For symptomatic relief, cervical epidural block, blockade of the medial branch of the dorsal ramus, or intraarticular injection of the facet joint with local anesthetic and steroid may also be considered. Antimyotonic agents such as tizanidine may be used if symptoms persist. Underlying sleep disturbance and depression are best treated with a tricyclic antidepressant such as nortriptyline, which can be started at a single bedtime dose of 25 mg.

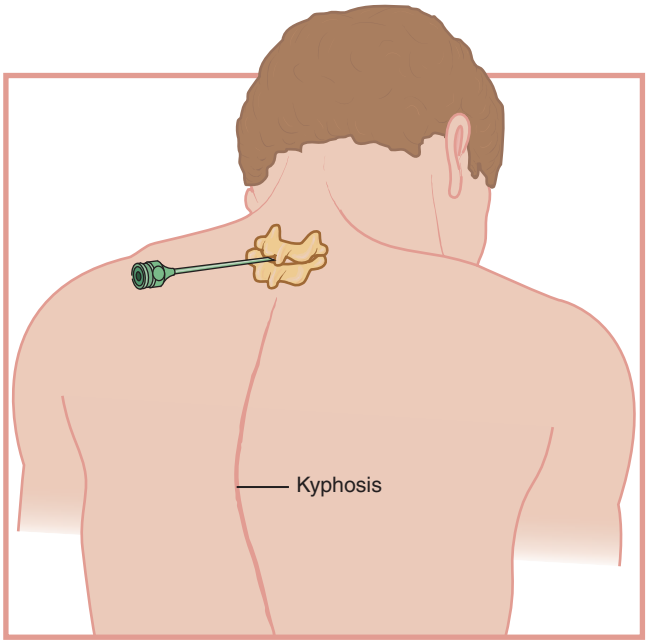

FIG 21.5 Proper needle placement for injection of a cervicothoracic bursa. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Elsevier; 2007.)

COMPLICATIONS AND PITFALLS

The proximity to the spinal cord and exiting nerve roots makes it imperative that injections be performed only by those familiar with the regional anatomy and experienced in interventional pain management techniques. The proximity to the vertebral artery, combined with the vascular nature of this region, renders the potential for intravascular injection high, and the injection of even a small amount of local anesthetic into the vertebral artery can result in seizures. Given the proximity of the brain and brainstem, ataxia resulting from vascular uptake of local anesthetic is not uncommon after injection in this region. Many patients also complain of a transient increase in headache and cervicalgia after injection of the cervical facet joints.

CLINICAL PEARLS

Correction of the functional abnormalities responsible for the development of cervicothoracic bursitis is mandatory if long-lasting relief is to be achieved. Physical modalities, including local heat, gentle stretching exercises, and deep sedative massage, are beneficial and may be started concurrently with a trial of NSAIDs. Injection of local anesthetic and steroid is extremely effective in the treatment of cervicothoracic bursitis pain that fails to respond to more conservative measures. Vigorous exercise should be avoided, because it will exacerbate the patient's symptoms.

SUGGESTED READINGS

Blecher R, Yilmaz E, Tawfik T, et al. Cervico-thoracic interspinous bursitis associated with bilateral upper-extremity numbness: a case report. *Cureus*. 2017;9(11):e1897.

Falsetti P, Acciai C. Ultrasound in assessment of cervical interspinous bursitis in polymyalgia rheumatica. *Jt Bone Spine*. 2013;80:342–343.

Perka C, Schneider SV, Buttgereit F, et al. Development of cervical interspinous bursitis after prolonged sports trauma: a case report. *Jt Bone Spine*. 2006;73(1):118–120.

Waldman SD. Cervicothoracic interspinous bursitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:238–239.

Waldman SD. Injection technique for cervicothoracic bursitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:84–85.

Brachial Plexopathy

ICD-10 CODE G54.0

THE CLINICAL SYNDROME

Brachial plexopathy is a constellation of symptoms consisting of neurogenic pain and associated weakness that radiates from the shoulder into the supraclavicular region and upper extremity (Fig. 22.1). There are many causes of brachial plexopathy, but some of the more common ones include compression of the plexus by cervical ribs or abnormal muscles (e.g., thoracic outlet syndrome), invasion of the plexus by tumor (e.g., Pancoast's tumor syndrome), direct trauma to the plexus (e.g., stretch injuries and avulsions), inflammatory causes (e.g., Parsonage–Turner syndrome, herpes zoster), and postradiation plexopathy (Fig. 22.2).

SIGNS AND SYMPTOMS

Patients suffering from brachial plexopathy complain of pain radiating to the supraclavicular region and upper extremity. The pain is neuritic in character and may take on a deep, boring quality as the plexus is invaded by tumor. Movement of the neck and shoulder exacerbates the pain, so patients often try to avoid such movement. Frozen shoulder often results and may confuse the diagnosis. If thoracic outlet syndrome is suspected, the Adson test may be performed (Fig. 22.3). The test is positive if the radial pulse disappears with the neck extended and the head turned toward the affected side. Because the Adson test is nonspecific, treatment decisions should not be based on this finding alone (see "Testing"). If the patient presents with severe pain that is followed shortly by profound weakness, brachial plexitis should be considered; this can be confirmed with electromyography.

TESTING

All patients presenting with brachial plexopathy, especially those without a clear history of antecedent trauma, must undergo magnetic resonance imaging (MRI) of the cervical spine and the brachial plexus. Computed tomography (CT) scanning and ultrasound imaging are reasonable alternatives if MRI is contraindicated. Electromyography and nerve conduction velocity testing are extremely sensitive, and a skilled electromyographer can delineate which portion of the plexus is abnormal. Ultrasound imaging of the individual nerve roots and surrounding tissues may help identify the

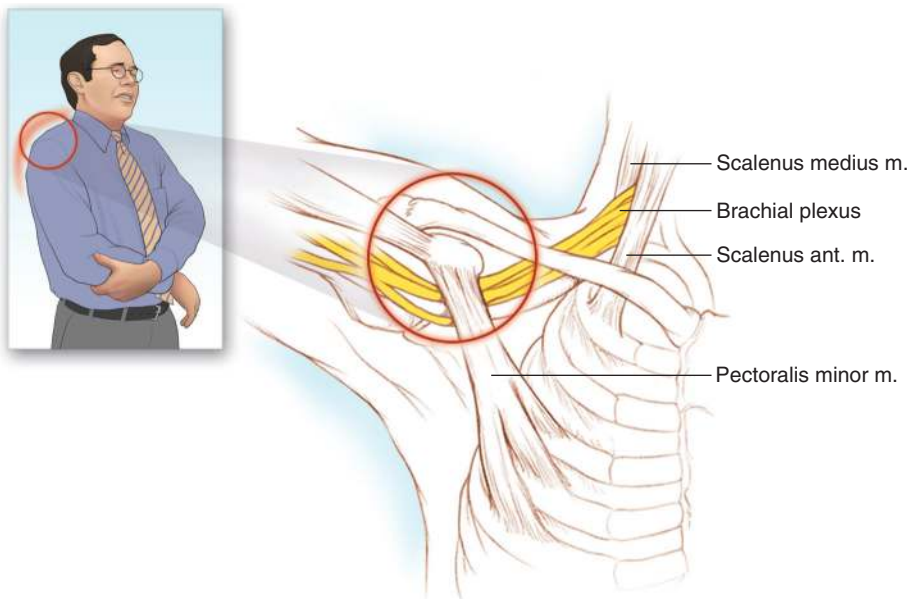

FIG 22.1 The pain of brachial plexopathy radiates from the shoulder and supraclavicular region into the upper extremity.

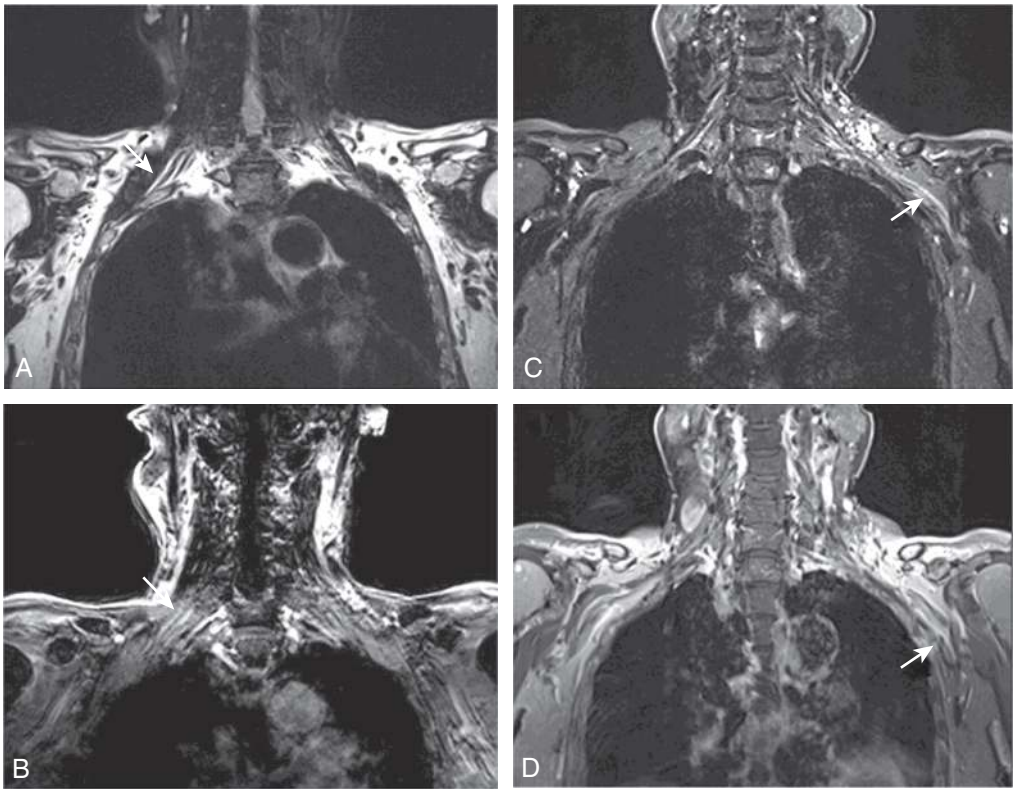

FIG 22.2 Coronal magnetic resonance (MR) scan of the brachial plexus. Case 1. MR images taken 10 days after the onset of motor symptoms show mild swelling of the brachial plexus with T2 hyperintensity **(A)** and corresponding contrast enhancement **(B)**, marked in the upper and middle trunks (*arrows*). Case 2. MR image taken 8 weeks after the onset of motor weakness. T2 STIR (Short Tau Inversion Recovery) coronal **(C)** and gadolinium-enhanced TSE (Turbo Spine Echo) coronal **(D)** images demonstrated increased signal and intense enhancement in the left brachial plexus at the cord level, marked in the medial cord (*arrows*). (From Choi J-Y, Kang CH, Kim B-J, et al. Brachial plexopathy following herpes zoster infection: two cases with MRI findings. *J Neurol Sci*. 2009;285(1–2):224–226.)

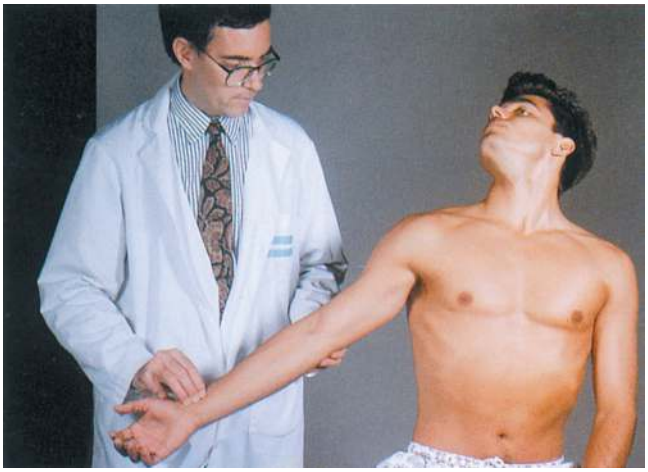

FIG 22.3 Adson test. The patient inhales deeply, extends the neck fully, and turns the head to the affected side. This maneuver tests for compression in the scalene triangle; it is positive if there is a diminution in the radial pulse and reproduction of the patient's symptoms. (From Klippel JH, Dieppe PA. *Rheumatology*. 2nd ed. London: Mosby; 1998.)

neuroanatomic basis of abnormalities identifyed on electrodiagnostic testing. If an inflammatory basis for the plexopathy is suspected, serial electromyography is indicated and MRI of the shoulder muscles often reveals muscle edema and denervation-induced atrophy (Fig. 22.4). If Pancoast's tumor or some other tumor of the brachial plexus is suspected, chest radiographs with apical lordotic views may be helpful. If the diagnosis is in question, screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry should be performed to rule out other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Diseases of the cervical spinal cord, bony cervical spine, and disk can mimic brachial plexopathy. Appropriate testing, including MRI, CT, ultrasonography, and electromyography, can help sort out the myriad possibilities, but the clinician should be aware that more than one pathologic process may be contributing to the patient's symptoms. Syringomyelia,

FIG 22.4 Parsonage–Turner syndrome. Axial short tau inversion recovery **(A)** and sagittal oblique T2-weighted **(B)** images. Increased signal intensity consistent with interstitial muscle edema associated with denervation is seen in the supraspinatus and infraspinatus muscles (*arrows*). (From Edelman RR, Hessellink JR, Zlatkin MB, et al. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3272.)

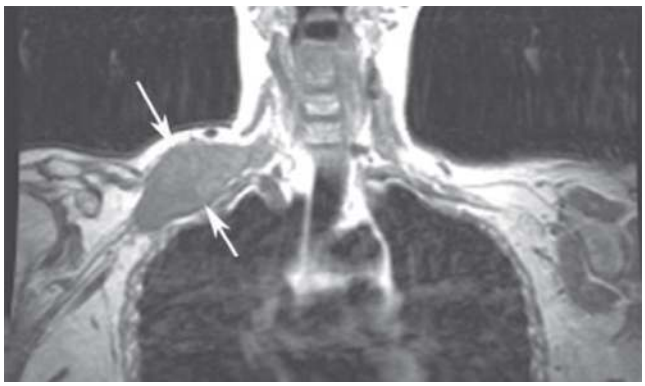

FIG 22.5 Brachial plexus schwannoma, depicted on coronal T1-weighted magnetic resonance (MR) image. Fusiform mass is oriented along the longitudinal axis of the plexus. (From Schweitzer AD, Krol G. Imaging of plexopathy in oncologic patients. In: Newton HB, ed. *Handbook of neuro-oncology neuroimaging*. 2nd ed. San Diego: Academic Press; 2016:763–775.)

tumor of the cervical spinal cord, and tumor of the cervical nerve root as it exits the spinal cord (e.g., schwannoma) can have an insidious onset and be quite difficult to diagnosis (Fig. 22.5). Pancoast's tumor should be high on the list of diagnostic possibilities in all patients presenting with brachial plexopathy in the absence of clear antecedent trauma, especially if there is a history of tobacco use (Figs. 22.6 and 22.7). Lateral herniated cervical disk, metastatic tumor, or cervical spondylosis resulting in significant nerve root compression may also present as brachial plexopathy. Rarely, infection involving the apex of the lung may compress and irritate the plexus.

FIG 22.6 Plain radiograph demonstrating Pancoast tumor on the left. (From Tashi E, Kapisyzi P, Xhemalaj D, Andoni A, Peposhi I. Pancoast tumor approach through oesophagus. *Respir Med Case Rep*. 2017;22:218–219.)

TREATMENT

Drug Therapy

Gabapentin

Gabapentin is the first-line treatment for the neuritic pain of brachial plexopathy. Start with 300 mg gabapentin at bedtime for two nights, and caution the patient about potential side effects, including dizziness, sedation, confusion, and rash. The drug is then increased in 300-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 2400 mg/day is reached. At this point, if the patient has experienced partial

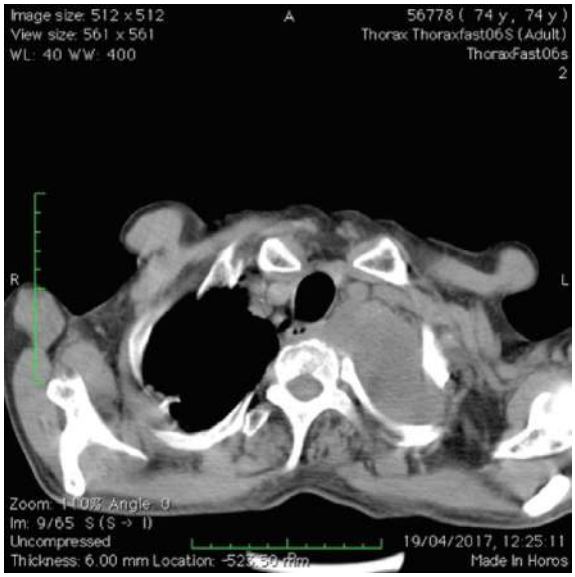

FIG 22.7 Left upper lobe mass. (From Tashi E, Kapisyzi P, Xhemalaj D, Andoni A, Peposhi I. Pancoast tumor approach through oesophagus. *Respir Med Case Rep.* 2017;22:218–219.)

pain relief, blood values are measured and the drug is carefully titrated upward using 100-mg tablets. Rarely is a dose greater than 3600 mg/day required.

Pregabalin

Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine

Carbamazepine is useful in patients who do not obtain pain relief with gabapentin. Despite the safety and efficacy of carbamazepine, confusion and anxiety have surrounded its use. It is sometimes discontinued owing to laboratory abnormalities erroneously attributed to it. Therefore baseline laboratory values consisting of a complete blood count, urinalysis, and automated chemistry profile should be obtained before starting the drug.

Carbamazepine should be initiated slowly if the pain is not out of control, at a starting dose of 100–200 mg at bedtime for two nights. The patient should be cautioned about side effects, including dizziness, sedation, confusion, and rash. The drug is increased in 100- to 200-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 1200 mg/day is reached. Careful monitoring of laboratory parameters is mandatory to avoid the rare possibility of a life-threatening blood dyscrasia; at the first sign of blood count abnormality or rash, the drug should be discontinued. Failure to monitor patients started on carbamazepine can be disastrous, because aplastic anemia can occur. When pain relief is obtained, the patient should be kept at that dosage of carbamazepine for at least 6 months before tapering of the medication is considered. The patient should be instructed that under no circumstances should the drug dosage be changed or the drug refilled or discontinued without the physician's knowledge.

Baclofen

Baclofen may be of value in some patients who fail to obtain relief from gabapentin or carbamazepine. Baseline laboratory tests should be obtained before starting baclofen, and the patient should be cautioned about potential adverse effects, which are the same as those associated with carbamazepine and gabapentin. Baclofen is started with a 10-mg dose at bedtime for two nights; the dosage is then increased in 10-mg increments given in equally divided doses over 7 days, as side effects allow, until pain relief is obtained or a total dose of 80 mg/day is reached. This drug has significant hepatic and central nervous system side effects, including weakness and sedation. As with carbamazepine, careful monitoring of laboratory values is indicated.

When treating individuals with any of these drugs, the physician should make sure that the patient knows that premature tapering or discontinuation of the medication may lead to the recurrence of pain, which will be more difficult to control.

Invasive Therapy

Brachial Plexus Block

Brachial plexus block with local anesthetic and steroid is an excellent adjunct to drug treatment. This technique rapidly relieves pain while medications are being titrated to effective levels. The initial block is carried out with preservative-free bupivacaine combined with methylprednisolone. Subsequent daily nerve blocks are carried out in a similar manner, substituting a lower dose of methylprednisolone. This approach can also be used to control breakthrough pain.

Radiofrequency Destruction of the Brachial Plexus

The brachial plexus can be destroyed by creating a radiofrequency lesion under biplanar fluoroscopic guidance. This procedure is reserved for patients who have failed to respond to all aforementioned treatments and whose pain is secondary to tumor or avulsion of the brachial plexus.

Dorsal Root Entry Zone Lesioning

Dorsal root entry zone lesioning is the neurosurgical procedure of choice for intractable brachial plexopathy in patients who have failed to respond to all aforementioned treatments and whose pain is secondary to tumor or avulsion of the brachial plexus. This is a major neurosurgical procedure and carries significant risks.

Physical Modalities

Physical and occupational therapy to maintain function and palliate pain is a crucial part of the treatment plan for patients suffering from brachial plexopathy. Shoulder abnormalities,

including subluxation and adhesive capsulitis, must be treated aggressively. Occupational therapy to assist in activities of daily living is important to avoid further deterioration of function.

COMPLICATIONS AND PITFALLS

The pain of brachial plexopathy is difficult to treat. It responds poorly to opioid analgesics and may respond poorly to the medications discussed. The uncontrolled pain of brachial plexopathy can lead to suicide, and strong consideration should be given to hospitalizing such patients. Correct diagnosis of the underlying cause is crucial to the successful treatment of the pain and dysfunction associated with brachial plexopathy, because stretch injuries and contusions of the plexus may respond with time, but plexopathy secondary to tumor or avulsion of the cervical roots requires aggressive treatment.

CLINICAL PEARLS

Brachial plexus block with local anesthetic and steroid represents an excellent stopgap measure for patients suffering from the uncontrolled pain of brachial plexopathy while waiting for drug treatments to take effect. Correct diagnosis is paramount to allow the clinician to design a logical treatment plan.

SUGGESTED READING

Abadía LAR, Galvez LA, Zapata SA. Brachial plexopathy due to varicella-zoster virus as the initial presentation of HIV infection: importance of rehabilitation and pain management. *Neurol Perspect*. 2021;1(4):233–235.

Jakobsen RL, Fuglsang-Frederiksen A, Hellfritzsch MB, Qerama E. A prospective study of high resolution ultrasound in brachial plexopathies—correlation with electrophysiological measurements. *Clin Neurophysiol*. 2019;130(7):1144–1150.

Maheshwari S, Panda A, Kushwaha S, et al. Malignant synovial sarcoma: an unpredictable cause of brachial plexopathy. *Clin Neurol Neurosurg*. 2020;193:105767.

Morris BA, Burr AR, Anderson BM, Howard SP. Late radiation related brachial plexopathy after pulsed reduced dose rate reirradiation of an axillary breast cancer recurrence. *Practical Radiat Oncol*. 2021;11(5):319–322.

Waldman SD. Brachial plexopathy. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:260–261.

Waldman SD. Brachial plexus block: interscalene approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:242–248.

Waldman SD. Painful conditions of the brachial plexus. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Kluwer Wolters; 2016:301–312.

23

Pancoast's Tumor Syndrome

ICD-10 CODE C34.10

THE CLINICAL SYNDROME

Pancoast's tumor syndrome is the result of local growth of tumor from the apex of the lung directly into the brachial plexus. Such tumors usually involve the first and second thoracic nerves, as well as the eighth cervical nerve, and produce a classic clinical syndrome consisting of severe arm pain and, in some patients, Horner's syndrome (Fig. 23.1). Destruction of the first and second ribs is also common. Diagnosis is usually delayed, and patients are often erroneously treated for cervical radiculopathy or primary shoulder disease until the diagnosis becomes clear.

SIGNS AND SYMPTOMS

Patients suffering from Pancoast's tumor syndrome complain of pain radiating to the supraclavicular region and upper extremity (Fig. 23.2). Initially, the lower portion of the brachial plexus is involved because the tumor growth is from below, causing pain in the upper thoracic and lower cervical dermatomes. The pain is neuritic and may take on a deep, boring quality as the tumor invades the brachial plexus. Movement of the neck and shoulder exacerbates the pain, so patients often try to avoid such movement. Frozen shoulder often results and may confuse the diagnosis. As the disease progresses, Horner's syndrome may occur (Fig. 23.3).

TESTING

All patients presenting with brachial plexopathy, especially those without a clear history of antecedent trauma, must undergo magnetic resonance imaging (MRI) of the cervical spine and the brachial plexus (Figs. 23.4 and 23.5). Computed tomography (CT) and/or ultrasound imaging is a reasonable alternative if MRI is contraindicated (Fig. 23.6). Positron emission tomography may help clarify the nature of suspicious masses in this regions (Fig. 23.7). Electromyography (EMG) and nerve conduction velocity testing are extremely sensitive, and a skilled electromyographer can determine which portion of the plexus is abnormal. All patients with a significant smoking history and suspected Pancoast's tumor or other tumor of the brachial plexus should undergo chest radiography with apical lordotic views or CT scanning through the apex of the lung. If the diagnosis is in question, screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry should be performed to rule out other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Diseases of the cervical spinal cord, bony cervical spine, and disk can mimic the brachial plexopathy associated with Pancoast's tumor syndrome. Appropriate testing, including MRI and EMG, can help sort out the myriad possibilities, but the clinician should be aware that more than one pathologic process may be contributing to the patient's symptoms. Syringomyelia, tumor of the cervical spinal cord, and tumor of the cervical nerve root as it exits the spinal cord (e.g., schwannoma) can have an insidious onset and be quite difficult to diagnose. Pancoast's tumor should be high on the list of diagnostic possibilities in all patients presenting with brachial plexopathy in the absence of clear antecedent trauma, especially if they have a history of tobacco use. Lateral herniated cervical disk, metastatic tumor, or cervical spondylosis that results in significant nerve root compression may also manifest as brachial plexopathy. Rarely, infection involving the apex of the lung may compress and irritate the plexus.

TREATMENT

The primary treatment of Pancoast's tumor syndrome is aimed at the tumor itself. Based on the cell type and extent of involvement, chemotherapy and radiation therapy may be indicated. Primary surgical treatment of tumors involving the brachial plexus is difficult, and the results are often disappointing.

Drug Therapy

Opioid Analgesics

Opioid analgesics are the mainstay of treatment for the pain associated with Pancoast's tumor syndrome. Although neuropathic pain generally responds poorly to opioid analgesics, given the severity of the pain and the lack of other options, a trial of opioid analgesics is warranted. Administration of a short-acting, potent opioid such as oxycodone is a reasonable starting point. Immediate-release morphine or methadone can also be considered. These drugs can be used in combination with nonsteroidal antiinflammatory drugs and the adjuvant analgesics described here.

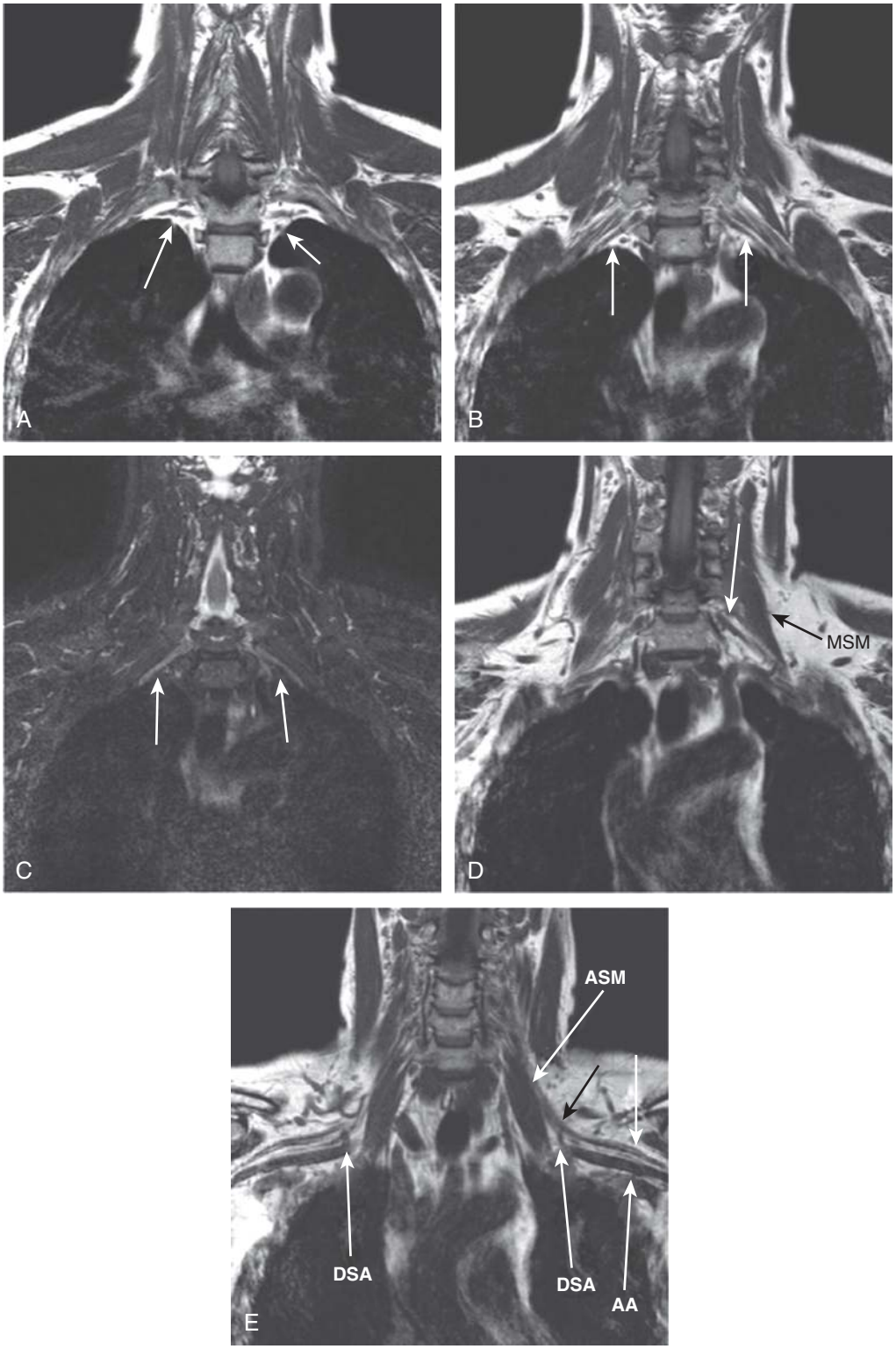

FIG 23.1 Magnetic resonance imaging of normal coronal anatomy. **A,** Most posterior image with the horizontal course of the T1 nerve root (*long arrow*), very close to the lung apex. The *short arrow* points to the stellate ganglion. **B,** Image just anterior to **A** with the C8 nerve roots (*arrows*). **C,** T2-weighted short tau inversion recovery image at the same level as **B** shows the slightly increased signal intensity of the normal C8 nerve roots (*arrows*). **D,** *Arrow* points to the C7 nerve root. *MSM*, Middle scalene muscle. **E,** The cords (*white arrow*) are seen as linear structures above the axillary artery (AA). The dorsal scapular artery (DSA) courses between the trunks of the brachial plexus; the *black arrow* points to the superior trunk. *ASM*, Anterior scalene muscle. (From Van Es HW, Bollen TL, van Heesewijk HP. MRI of the brachial plexus: a pictorial review. *Eur J Radiol*. 2010;74(2):391–402.)

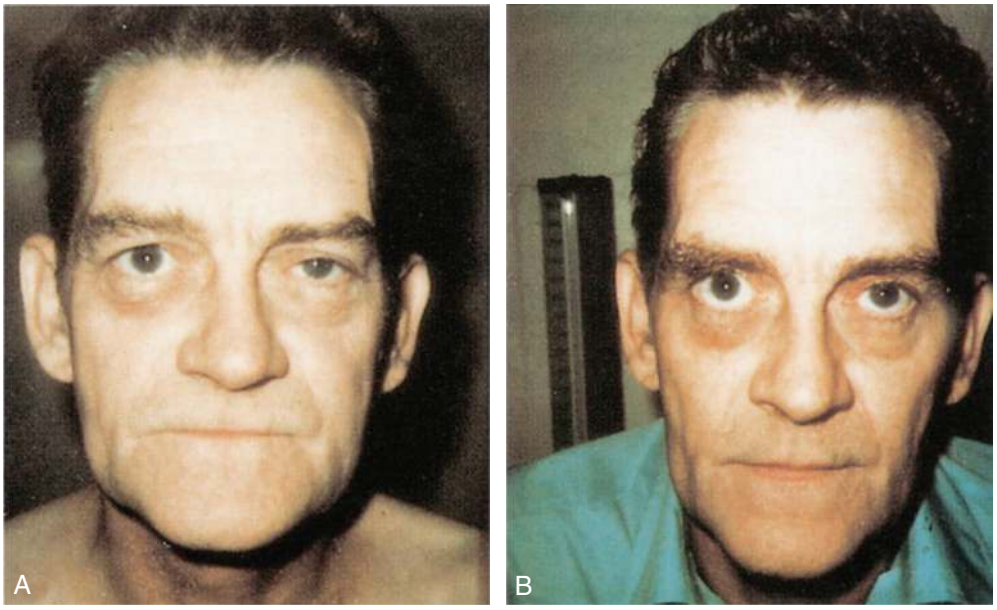

FIG 23.2 Horner's syndrome in a patient suffering from Pancoast's tumor syndrome. **A,** This 58-year-old man presented with chronic left arm and shoulder pain along with progressive weakness of his lower arm and hand. Physical examination showed clinical findings of a superior sulcus (Pancoast's) tumor, ptosis of the left eyelid, miosis of the pupil, decreased sweating of the left face, arm, and upper chest (Horner's syndrome), and a tumor mass in the lung apex that involved the brachial plexus and adjacent rib. **B,** After radiation therapy the manifestations of Horner's syndrome have resolved. Also his pain and neurologic symptoms were reduced. Survival is poor with Pancoast's tumors (under 30% at 5 years) as a result of progressive regional disease but also distant metastases. (From Salgia R, Blanco R, Skarin AT. Lung cancer and tumors of the heart and mediastinum. In: *Atlas of diagnostic oncology*. 4th ed. Philadelphia; 2010:98–159.)

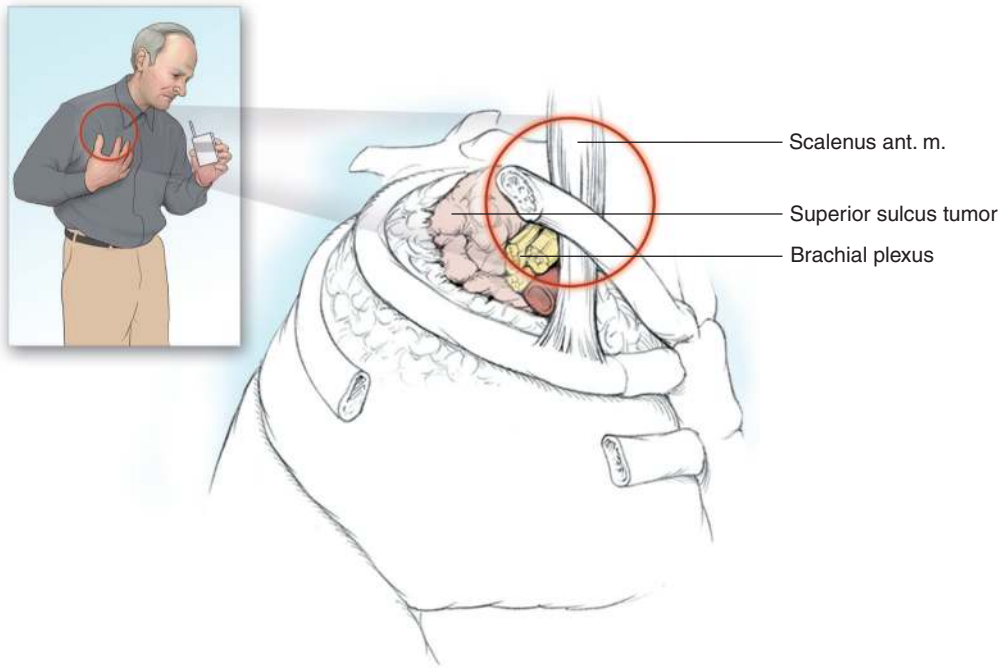

FIG 23.3 Pancoast's tumor should be suspected in patients suffering from shoulder and upper extremity pain who have a history of smoking.

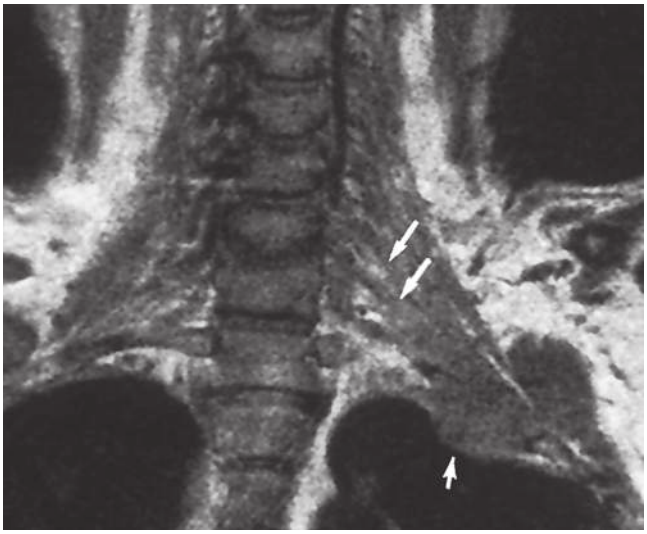

FIG 23.4 Pancoast's tumor (adenocarcinoma) with infiltration of the brachial plexus. A 65-year-old man complained of severe pain in the shoulder radiating to the elbow, the medial side of the forearm, and the fourth and fifth fingers in an ulnar nerve distribution. Screening coronal T1-weighted magnetic resonance imaging shows the brachial plexus from the region of the roots (*long arrows*) to the region of the trunks and divisions, where tumor invasion (*short arrow*) and loss of fat planes on the left are seen. (From Stark DD, Bradley WG Jr., eds. *Magnetic resonance imaging*. 3rd ed. St Louis: Mosby; 1999:2399.)

Gabapentin

Gabapentin is used to treat the neuritic pain of Pancoast's tumor syndrome. The initial dose is 300 mg gabapentin at bedtime for two nights, and the patient should be cautioned about potential side effects, including dizziness, sedation, confusion, and rash. The drug is then increased in 300-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 2400 mg/day is reached. At this point, if the patient has experienced partial pain relief, blood values are measured and the drug is carefully titrated upward using 100-mg tablets. Rarely is a dose greater than 3600 mg/day required.

Pregabalin

Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine

Carbamazepine is useful in patients who do not obtain pain relief with gabapentin. Despite the safety and efficacy of carbamazepine, confusion and anxiety have surrounded its use. The drug is sometimes discontinued owing to laboratory abnormalities erroneously attributed to it. Therefore baseline laboratory values consisting of a complete blood count, urinalysis, and automated chemistry profile should be obtained before starting the drug.

Carbamazepine should be initiated slowly if the pain is not out of control at a starting dose of 100–200 mg at bedtime for two nights. The patient should be cautioned about side effects, including dizziness, sedation, confusion, and rash. The drug is increased in 100- to 200-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 1200 mg/day is reached. Careful monitoring of laboratory parameters is mandatory to avoid the rare possibility of a life-threatening blood dyscrasia; at the first sign of blood count abnormality or rash, the drug should be discontinued. Failure to monitor patients started on carbamazepine can be disastrous, because aplastic anemia can occur. When pain relief is obtained, the patient should be kept at that dosage of carbamazepine for at least 6 months before tapering of the medication is considered. The patient should be instructed that under no circumstances should the drug dosage be changed or the drug refilled or discontinued without the physician's knowledge.

Baclofen

Baclofen may be of value in some patients who fail to obtain relief from the previously mentioned medications. Baseline laboratory tests should be obtained before starting baclofen, and the patient should be cautioned about potential adverse effects, which are the same as those associated with carbamazepine and gabapentin. Baclofen is started with a 10-mg dose at bedtime for two nights; the drug is then increased in 10-mg increments given in equally divided doses over 7 days, as side effects allow, until pain relief is obtained or a total dose of 80 mg/day is reached. This drug has significant hepatic and central nervous system side effects, including weakness and sedation. As with carbamazepine, careful monitoring of laboratory values is indicated.

Invasive Therapy

Brachial Plexus Block

Brachial plexus block with local anesthetic and steroid is an excellent adjunct to drug treatment of Pancoast's tumor syndrome. This technique rapidly relieves pain while medications are being titrated to effective levels. The initial block is carried out with preservative-free bupivacaine combined with methylprednisolone. Subsequent daily nerve blocks are performed in a similar manner, by substituting a lower dose of methylprednisolone. This approach can also be used to control breakthrough pain.

Radiofrequency Destruction of the Brachial Plexus

The brachial plexus can be destroyed by creating a radiofrequency lesion under biplanar fluoroscopic guidance. This procedure is reserved for patients for whom all aforementioned treatments have failed.

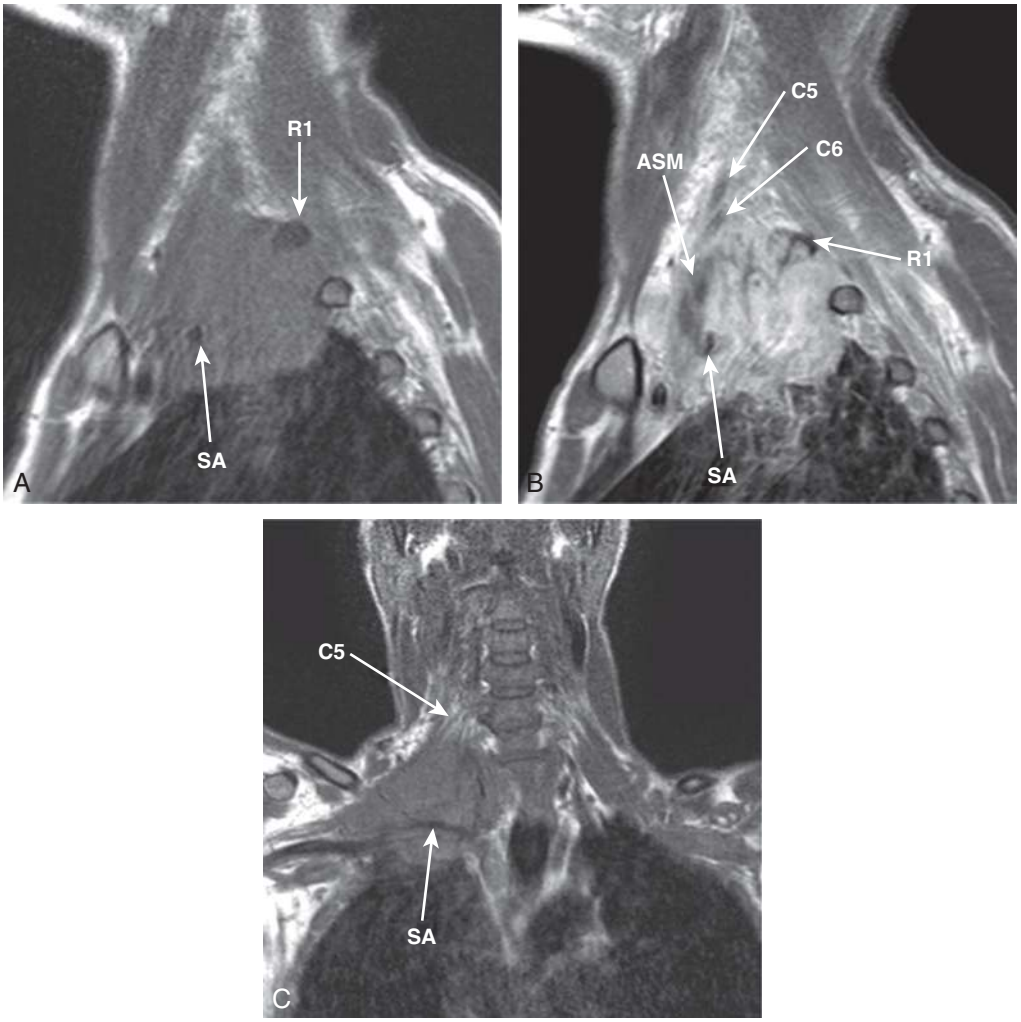

FIG 23.5 Magnetic resonance imaging of an inoperable superior sulcus tumor. A and B, Sagittal T1-weighted image without (A) and with (B) intravenous gadolinium show the extension of the nonsmall-cell lung tumor into the interscalene triangle. With intravenous gadolinium, the nonenhancing nerve roots can be discerned from the enhancing tumor, and tumor is visible up to the C5 nerve root. The subclavian artery (SA) is encased, the tumor surrounds the anterior scalene muscle (ASM), and involvement of the first rib (R1) is evident. C, Coronal T1-weighted image demonstrates the involvement of the C5 nerve root. (From Van Es HW, Bollen TL, van Heesewijk HP. MRI of the brachial plexus: a pictorial review. *Eur J Radiol*. 2010;74(2):391–402.)

Dorsal Root Entry Zone Lesioning

Dorsal root entry zone lesioning is the neurosurgical procedure of choice for intractable brachial plexopathy associated with Pancoast's tumor in patients who have failed to respond to all aforementioned treatment options. This is a major neurosurgical procedure and carries significant risks.

Other Neurosurgical Options

Cordotomy, deep brain stimulation, and thalamotomy have all been tried, with varying degrees of success.

Physical Modalities

Physical and occupational therapy to maintain function and palliate pain is a crucial part of the treatment plan for patients suffering from Pancoast's tumor syndrome. Shoulder abnormalities, including subluxation and adhesive capsulitis, must be aggressively treated. Occupational therapy to assist in activities of daily living is important to avoid further deterioration of function.

COMPLICATIONS AND PITFALLS

The pain of Pancoast's tumor syndrome is difficult to treat. It may respond poorly to any of or all the recommended medications. The uncontrolled pain of Pancoast's tumor syndrome can lead to suicide, and strong consideration should be given to hospitalizing such patients. Correct diagnosis of the underlying cause is crucial, because the pain and dysfunction associated with brachial plexopathy secondary to Pancoast's tumor require aggressive treatment.

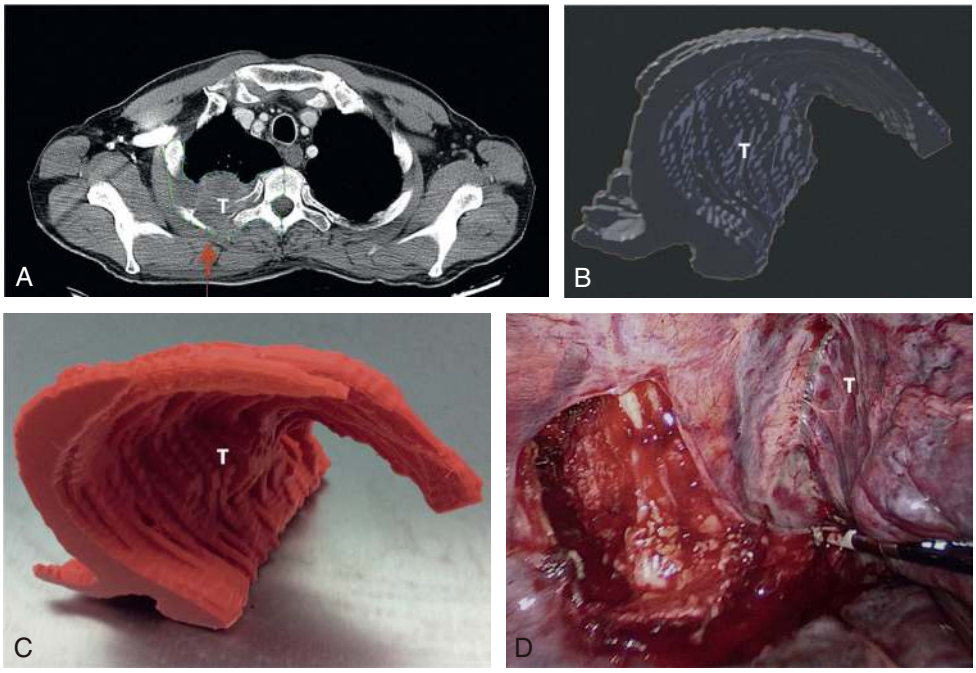

FIG 23.6 Right upper lobe Pancoast's tumor. **A,** Computed tomography of the tumor (T) with region of interest (*green line*) around the tumor, ipsilateral side of spine, and the chest wall (*red arrow*). **B,** Three-dimensional (3D) rendering of extracted topology showing tumor (T), spine, and chest wall. **C,** 3D printed plastic model of the tumor (T), spine, and chest wall. **D,** Thoracoscopic image of the resection of the chest wall and laminectomy with tumor (T) invading the third rib. (From Kim MP, Ta AH, Ellsworth WA IV, et al. Three dimensional model for surgical planning in resection of thoracic tumors. *Int J Surg Case Rep*. 2015;16:127–129.)

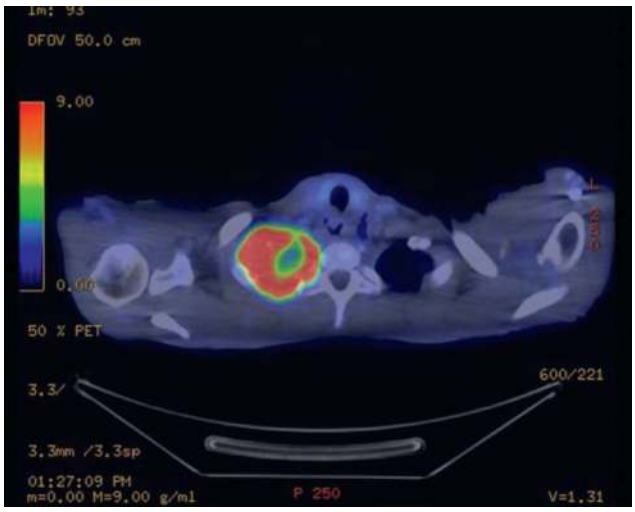

FIG 23.7 Positron emission tomography–computed tomography scan showing Pancoast's tumor. (From Ng DWJ, Tan GHC, Teo MCC. Malignancy arising in a 41-year-old colonic interposition graft. *Asian J Surg*. 2016;39(1):45–47.)

CLINICAL PEARLS

Brachial plexus block with local anesthetic and steroid is an excellent stopgap measure for patients suffering from the uncontrolled pain of brachial plexopathy while waiting for drug treatments to take effect. Correct diagnosis is paramount to allow the clinician to design a logical treatment plan.

SUGGESTED READINGS

Bois MC, Yi JE, Erickson LA. Pancoast tumor of the lung. *Mayo Clin Proc*. 2016;91(5):e69–e70.

Deslauriers J, Tronc F, Fortin D. Management of tumors involving the chest wall including pancoast tumors and tumors invading the spine. *Thorac Surg Clin*. 2013;23(3):313–325.

Ferrante MA, Kissel JT. Neoplastic plexopathies. In: Newton HB, ed. *Handbook of neuro-oncology neuroimaging*. 2nd ed. San Diego: Academic Press; 2016: chap 14, 111–123.

Jakobsen RL, Fuglsang-Frederiksen A, Hellfritzsch MB, Qerama E. A prospective study of high resolution ultrasound in brachial plexopathies—correlation with electrophysiological measurements. *Clin Neurophysiol*. 2019;130(7):1144–1150.

Maheshwari S, Panda A, Kushwaha S, et al. Malignant synovial sarcoma: an unpredictable cause of brachial plexopathy. *Clin Neurol Neurosurg*. 2020;193:105767.

Morris BA, Burr AR, Anderson BM, Howard SP. Late radiation related brachial plexopathy after pulsed reduced dose rate reirradiation of an axillary breast cancer recurrence. *Pract Radiat Oncol*. 2021;11(5):319–322.

Ramírez Abadía LA, Arce Galvez L, Ayala Zapata S. Brachial plexopathy due to varicella-zoster virus as the initial presentation of HIV infection: importance of rehabilitation and pain management. *Neurol Perspect*. 2021;1(4):233–235.

Rao SJ, Sagheer U. Pancoast tumor with cardiac metastasis and intracardiac thrombus. *J Am Coll Cardiol*. 2022;79 (9_suppl):3120.

Tamura M, Hoda MA, Klepetko W. Current treatment paradigms of superior sulcus tumours. *Eur J Cardiothorac Surg*. 2009;36 (4):747–753.

Waldman SD. Painful conditions of the brachial plexus. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Kluwer Wolters; 2016:301–312.

Waldman SD. Brachial plexus block: interscalene approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:242–248.

Waldman SD. Brachial plexopathy. In: *Pain review*. 3rd ed. Philadelphia: Elsevier; 2017:260–261.

Thoracic Outlet Syndrome

ICD-10 CODE G54.0

THE CLINICAL SYNDROME

Thoracic outlet syndrome consists of a constellation of signs and symptoms, including paresthesias and aching pain of the neck, shoulder, and arm. The cause is thought to be compression of the brachial plexus and subclavian artery and vein as they traverse the interscalene triangle, the costoclavicular space, and the subpectoral (subcoracoid) tunnel (Fig. 24.1). Compression or entrapment of the neurovascular structures may be caused by congenitally abnormal structures such as cervical ribs, aberrant scalene muscles, fibrous bands, and/or abnormal pectoralis minor and subscapularis muscles as the plexus cords, subclavian/axillary artery, and the terminal branches of the brachial plexus traverse the subpectoral space (Fig. 24.2). Cervicothoracic tumors and aneurysms must also be considered when evaluating a patient with thoracic outlet syndrome. One or all the structures may be compressed, thus giving the syndrome a varied clinical expression. Thoracic outlet syndrome is seen most commonly in women between 25 and 50 years of age. It has been the subject of significant debate, and the diagnosis and treatment of thoracic outlet syndrome remain controversial.

SIGNS AND SYMPTOMS

Although the symptoms of thoracic outlet syndrome vary, compression of neural structures accounts for most of them. Paresthesias of the upper extremity radiating into the distribution of the ulnar nerve may be misdiagnosed as tardy ulnar palsy. Aching and incoordination of the affected extremity are also common findings. If the pain persists, abnormal position of the shoulder girdle to relieve compression or entrapment of the neurovascular structures may be observed. If vascular compression exists, edema or discoloration of the arm may be noted; in rare instances, venous or arterial thrombosis may occur. Rarely, the symptoms of thoracic outlet syndrome are caused by arterial aneurysm, and auscultation of the supraclavicular region reveals a bruit.

The symptoms of thoracic outlet syndrome may be elicited by various maneuvers, including the Adson test, the costoclavicular test, and the Roo elevated arm stress test, which is also known as the hyperabduction test. The Adson test is carried out by palpating the radial pulse on the affected side with the patient's neck extended and the head turned toward the affected side (Fig. 24.3). A diminished pulse suggests

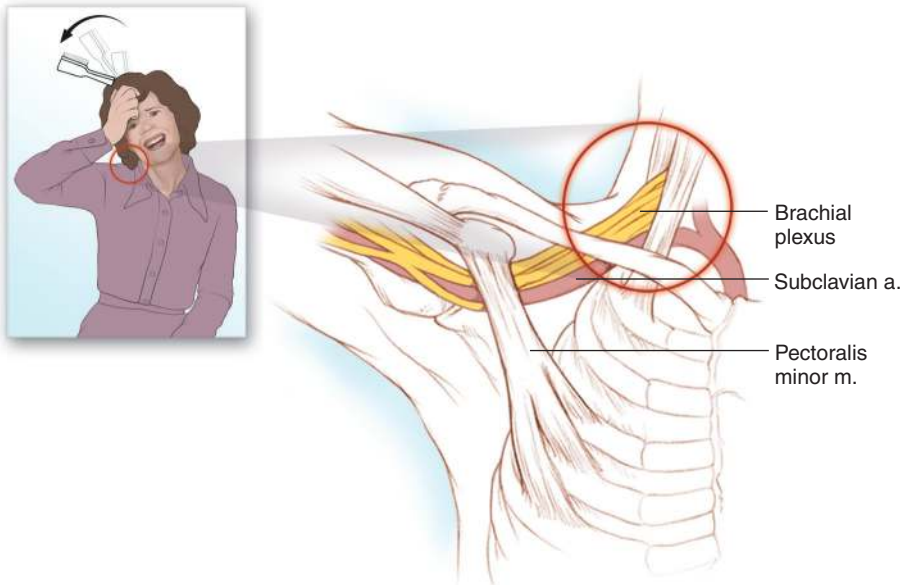

FIG 24.1 Compression of the brachial plexus results in pain and weakness in the affected upper extremity.

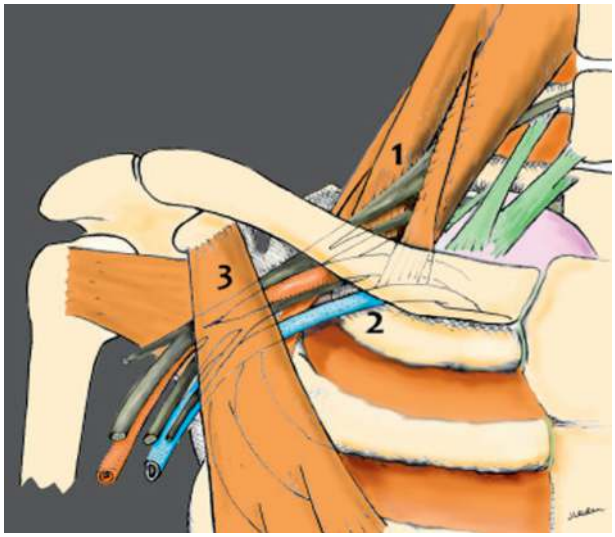

FIG 24.2 The three main sites implicated in neurovascular compression in thoracic outlet syndrome include **(1)** interscalene triangle, **(2)** costoclavicular space, and **(3)** subpectoral tunnel. (From Laulan J. Thoracic outlet syndromes. The so-called "neurogenic types." *Hand Surg Rehabil*. 2016;3(3): 155–164.)

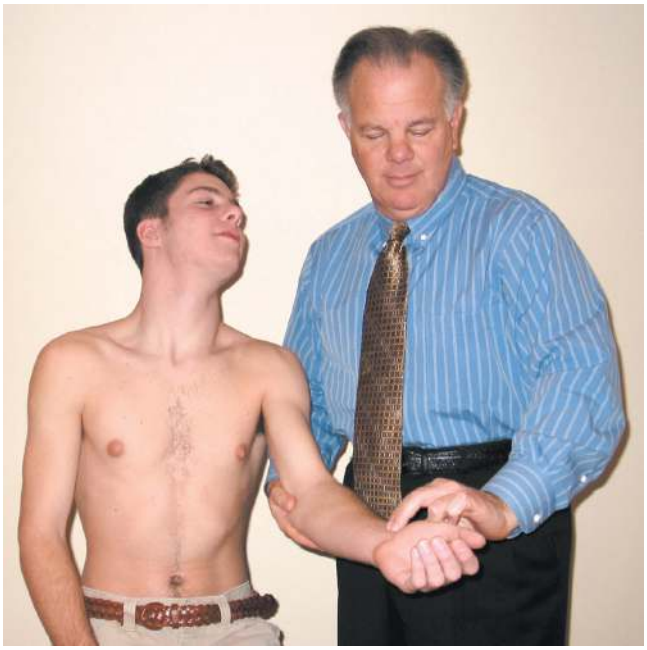

FIG 24.3 The Adson maneuver for thoracic outlet syndrome. (From Waldman D. *Physical diagnosis of pain*. 3rd ed. Philadelphia: Elsevier; 2015.)

thoracic outlet syndrome. The elevated arm stress test is performed by having the patient hold his or her arms over the head and open and close the hands. Normally, patients without thoracic outlet syndrome can perform this maneuver for approximately 3 minutes, whereas those suffering from thoracic outlet syndrome experience the onset of symptoms within 30 seconds.

TESTING

Plain radiographs of the cervical spine should be obtained in all patients suspected of having thoracic outlet syndrome. These films should be carefully reviewed for congenital abnormalities such as cervical ribs or overly elongated transverse processes. Patients should also undergo chest radiography with apical lordotic views to rule out Pancoast's tumor. Magnetic resonance imaging (MRI) of the cervical spine is indicated to identify lesions of the cervical spinal cord and exiting nerve roots, as well as cervical ribs, tumors, and fibrous adhesions (Figs. 24.4 and 24.5). If the diagnosis is still in doubt, MRI of the brachial plexus is indicated to search for an occult pathologic process, including primary tumors of the plexus and aberrant scalene muscles which may cause compression or entrapment (Fig. 24.6). Ultrasonography may also help clarify the diagnosis (Fig. 24.7). Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry may be performed to exclude other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Diseases of the cervical spinal cord, bony cervical spine, and disk can mimic thoracic outlet syndrome. Appropriate testing, including MRI and electromyography, can help sort out the myriad possibilities, but the clinician should be aware that more than one pathologic process may be contributing to the patient's symptoms. Syringomyelia, tumor of the cervical spinal cord, and tumor of the cervical nerve root as it exits the spinal cord (e.g., schwannoma) can have an insidious onset and may be quite difficult to diagnose. Pancoast's tumor should be high on the list of diagnostic possibilities in the absence of clear antecedent trauma, especially if the patient has a history of tobacco use. Lateral herniated cervical disk, metastatic tumor, or cervical spondylosis that results in significant nerve root compression should also be considered. Rarely, infection involving the apex of the lung may compress and irritate the plexus.

TREATMENT

Physical Modalities

The primary treatment for patients suffering from thoracic outlet syndrome is the rational use of physical therapy to maintain function and palliate pain. Shoulder abnormalities, including subluxation and adhesive capsulitis, must be aggressively treated. Occupational therapy to assist in activities of daily living is important to avoid further deterioration of function.

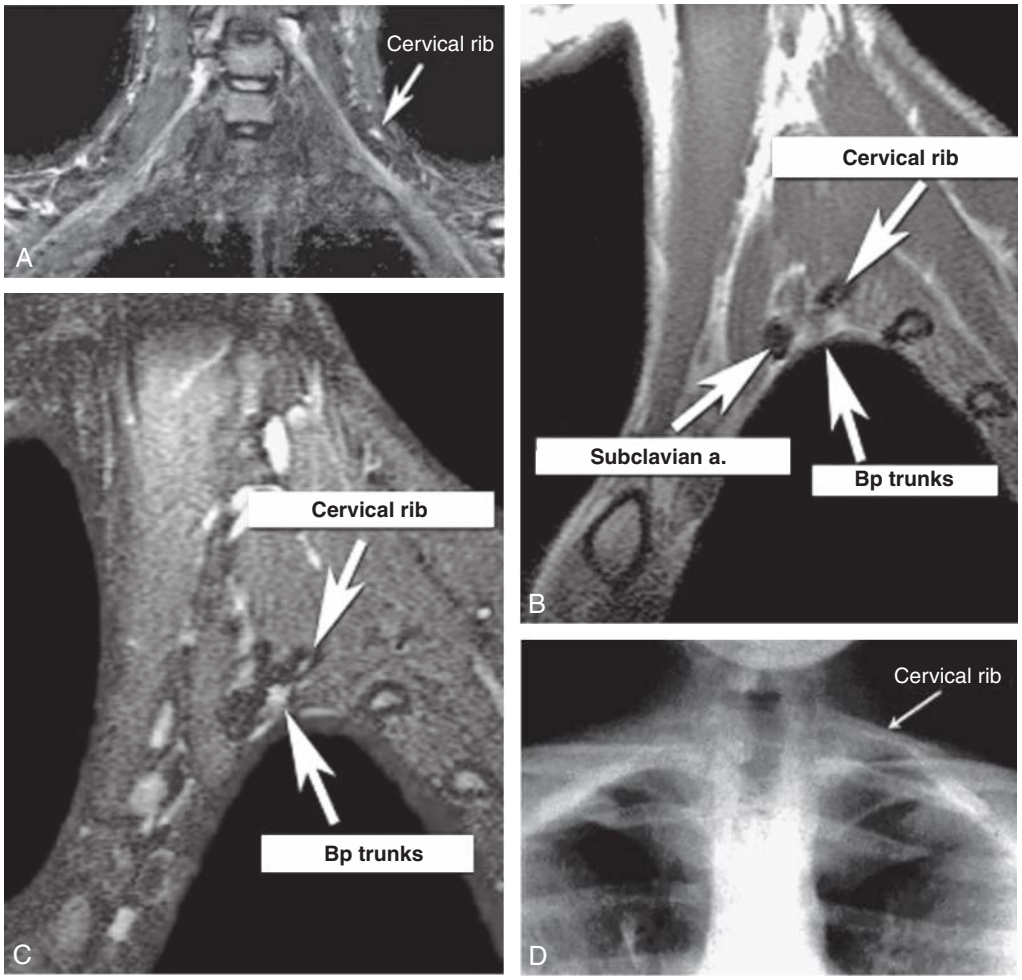

FIG 24.4 A patient with thoracic outlet syndrome on the left side secondary to a cervical rib. **A,** Coronal T2-weighted magnetic resonance imaging (MRI) shows the subtle deviation of the brachial plexus as it crosses the area of the cervical rib. **B** and **C,** Sagittal T1- and T2-weighted MRI at the level of the interscalene triangle shows the close proximity of the cervical rib to the lower trunk of the brachial plexus (Bp). **D,** Plain chest film shows the left cervical rib. The patient underwent surgical removal of the cervical rib, with resulting relief of symptoms. (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2382.)

Drug Therapy

Gabapentin

Gabapentin is the first-line pharmacologic treatment for the neuritic pain of thoracic outlet syndrome. The initial dose is 300 mg gabapentin at bedtime for two nights, and the patient should be cautioned about potential side effects, including dizziness, sedation, confusion, and rash. The drug is then increased in 300-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dosage of 2400 mg/day is reached. At this point, if the patient has experienced partial pain relief, blood values are measured and the drug is carefully titrated upward using 100-mg tablets. Rarely is a dosage greater than 3600 mg/day required.

Carbamazepine

Carbamazepine is useful in patients who do not obtain pain relief with gabapentin. Despite the safety and efficacy of carbamazepine, confusion and anxiety have surrounded its use. The drug is sometimes discontinued owing to laboratory abnormalities erroneously attributed to it. Therefore baseline laboratory values consisting of a complete blood count, urinalysis, and automated chemistry profile should be obtained before starting the drug.

Carbamazepine should be initiated slowly if the pain is not out of control at a starting dose of 100–240 mg at bedtime for two nights. The patient should be cautioned about side effects, including dizziness, sedation, confusion, and rash. The drug is increased in 100- to 200-mg increments given in equally

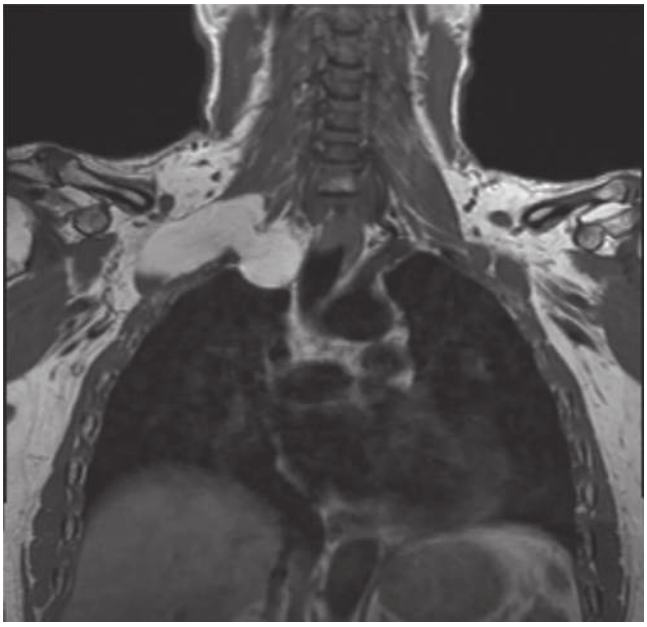

FIG 24.5 Coronal magnetic resonance imaging (MRI) of lipoma. Extrapleural lipoma with both an intrathoracic and extrathoracic component. T1-weighted image. (From Lombardi ME, Fromke E, Cuevas N, Pascarella L. Intrathoracic spindle cell lipoma causing thoracic outlet syndrome. *Ann Vasc Surg Brief Rep Innov*. 2022;2(2):100091.)

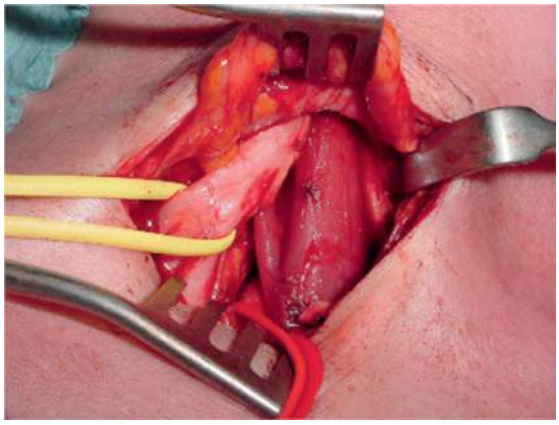

FIG 24.6 Congenital abnormality of scalenus medius muscle penetrated by brachial plexus. (From Thompson JF. Thoracic outlet syndrome. *Surgery (Oxford)*. 2016;34(4):198–202.)

divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dosage of 1240 mg/day is reached. Careful monitoring of laboratory parameters is mandatory to avoid the rare possibility of a life-threatening blood dyscrasia, and at the first sign of blood count abnormality or rash, the drug should be discontinued. Failure to monitor patients started on carbamazepine can be disastrous, because aplastic anemia can occur. When pain relief is obtained, the patient should be kept at that dosage of carbamazepine for at least 6 months before considering tapering of the medication. The patient should be instructed that under no circumstances should the drug dosage be changed or the drug refilled or discontinued without the physician's knowledge.

Pregabalin

Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Baclofen

Baclofen may be of value in some patients who fail to obtain relief with gabapentin or carbamazepine. Baseline laboratory tests should be obtained before starting baclofen, and the patient should be cautioned about potential adverse effects, which are the same as those associated with carbamazepine and gabapentin. Baclofen is started with a 10-mg dose at bedtime for two nights; the drug is then increased in 10-mg increments given in equally divided doses over 7 days, as side effects allow, until pain relief is obtained or a total dosage of 80 mg/day is reached. This drug has significant hepatic and central nervous system side effects, including weakness and sedation. As with carbamazepine, careful monitoring of laboratory values is indicated.

When treating individuals with any of these drugs, the physician should make sure that the patient knows that premature tapering or discontinuation of the medication may lead to the recurrence of pain, which will be more difficult to control.

Invasive Therapy

Brachial Plexus Block

Brachial plexus block with local anesthetic and steroid is an excellent adjunct to drug treatment of thoracic outlet syndrome. This technique rapidly relieves pain while medications are being titrated to effective levels. The initial block is carried out with preservative-free bupivacaine combined with methylprednisolone. Subsequent daily nerve blocks are carried out in a similar manner, by substituting a lower dose of methylprednisolone. This approach can also be used to control breakthrough pain.

Surgery

In the absence of demonstrable disease (e.g., a cervical rib), the outcome of surgical treatment for thoracic outlet syndrome is dismal, regardless of the technique chosen. In patients with a clear cause of their symptoms who have failed to achieve relief from more conservative therapies, however, the judicious use of surgical treatment may be a reasonable last step.

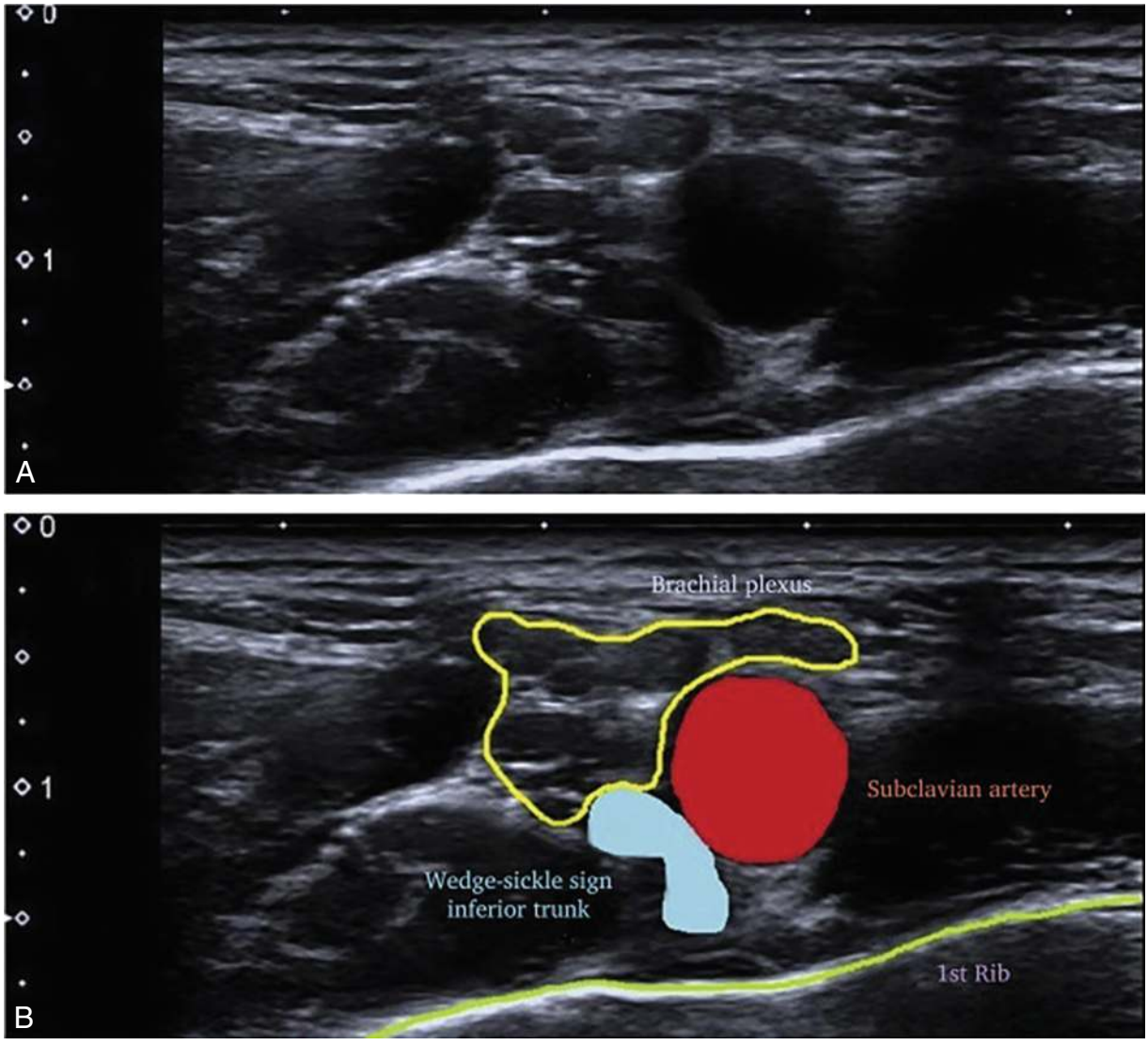

FIG 24.7 Axial image of high resolution ultrasound (HRUS) in a neurogenic thoracic outlet syndrome (NTOS) patient with a wedge-sickle sign **(A)**. The subclavian artery is marked red **(B),** the indented lower trunk of the brachial plexus (wedge-sickle sign) is marked by the gray, the brachial plexus is outlined by the yellow line, and the first rib is marked by the green line. The scale is displayed on the left border in centimetres. (From Pessser N, Teijink JAW, Vervaart K, et al. Value of ultrasound in the diagnosis of neurogenic thoracic outlet syndrome. *Eur J Vasc Endovasc Surg*. 2020;59(5):852–853.)

COMPLICATIONS AND PITFALLS

The pain and dysfunction of thoracic outlet syndrome are difficult to treat. Physical therapy should be the primary modality in any well thought out treatment plan. In general, the pain of thoracic outlet syndrome responds poorly to opioid analgesics, and these drugs should be avoided. The careful use of adjuvant analgesics may help palliate the pain and allow the patient to participate in physical therapy. Correct diagnosis is crucial, because stretch injuries and contusions of the plexus may respond with time, but plexopathy secondary to tumor or avulsion of the cervical roots requires aggressive treatment.

CLINICAL PEARLS

Brachial plexus block with local anesthetic and steroid represents an excellent stopgap measure to palliate the pain associated with thoracic outlet syndrome while waiting for drug treatments to take effect. Correct diagnosis is paramount to allow the clinician to design a logical treatment plan.

SUGGESTED READINGS

Aghamiri SH, Assarzadegan F, Ghaffari M, et al. Recurrent middle cerebral artery stroke caused by arterial thoracic outlet syndrome and coagulopathy. *Radiol Case Rep*. 2022;17(5):1665–1669.

Connolly MR, Auchincloss HG. Anatomy and embryology of the thoracic outlet. *Thorac Surg Clin*. 2021;31(1):1–10.
Illig KA, Rodriguez-Zoppi E. How common is thoracic outlet syndrome? *Thorac Surg Clin*. 2021;31(1):11–17.
Khalilzadeh O, Glover M, Torriani M, Gupta R. Imaging assessment of thoracic outlet syndrome. *Thorac Surg Clin*. 2021;31(1):19–25.
Lombardi ME, Fromke E, Cuevas N, Pascarella L. Intrathoracic spindle cell lipoma causing thoracic outlet syndrome. *Ann Vasc Surg Brief Rep Innov*. 2022;2(2):100091.
Pesser N, Teijink JAW, Vervaart K, et al. Value of ultrasound in the diagnosis of neurogenic thoracic outlet syndrome. *Eur J Vasc Endovasc Surg*. 2020;59(5):852–853.
Suzuki T, Kimura H, Matsumura N, Iwamoto T. Surgical approaches for thoracic outlet syndrome: a review of the literature. *J Hand Surg Glob Online*. 2022.

25

Arthritis Pain of the Shoulder

ICD-10 CODE M19.90

THE CLINICAL SYNDROME

The shoulder joint is susceptible to the development of arthritis from various conditions that cause damage to the joint cartilage. Osteoarthritis is the most common cause of shoulder pain and functional disability (Fig. 25.1). It may occur after seemingly minor trauma or may be the result of repeated microtrauma. Pain around the shoulder and the upper arm that is worse with activity is present in most patients suffering from osteoarthritis of the shoulder. Difficulty sleeping is also common, as is progressive loss of motion.

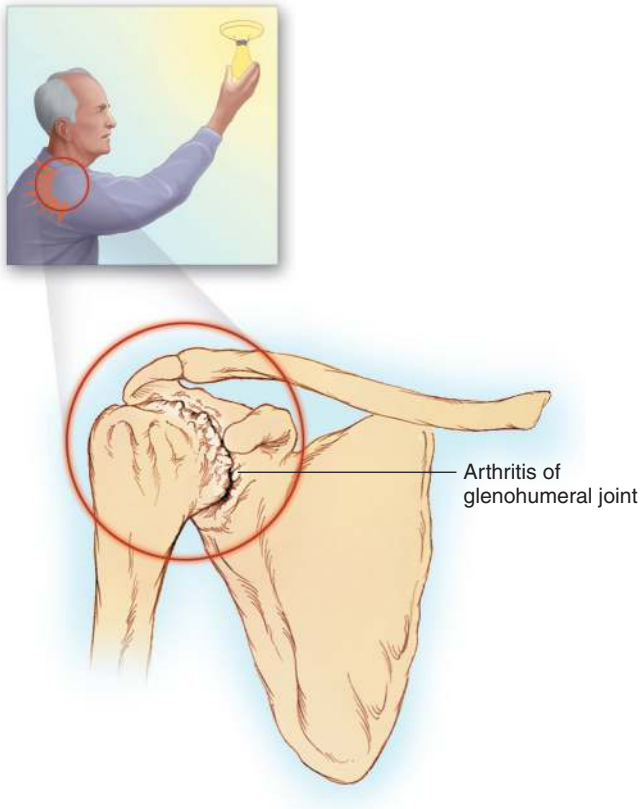

FIG 25.1 Range of motion of the shoulder can precipitate the pain of osteoarthritis.

SIGNS AND SYMPTOMS

Most patients presenting with shoulder pain secondary to osteoarthritis, rotator cuff arthropathy, or posttraumatic arthritis complain of pain that is localized around the shoulder and upper arm. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with the use of the joint, and crepitus may be present on physical examination.

In addition to pain, patients suffering from arthritis of the shoulder joint often experience a gradual reduction in functional ability because of decreasing shoulder range of motion. This change makes simple everyday tasks such as combing one's hair, fastening a brassiere, or reaching overhead quite difficult. With continued disuse, muscle wasting may occur and a frozen shoulder may develop.

TESTING

Plain radiographs are indicated in all patients who present with shoulder pain (Fig. 25.2). Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Computerized tomography may help identify bony abnormalities. Magnetic resonance and ultrasound imaging of the shoulder are indicated if a rotator cuff tear or other soft tissue pathology is suspected (Figs. 25.3 and 25.4). Radionuclide bone scanning is indicated if metastatic disease or primary tumor involving the shoulder is a possibility.

DIFFERENTIAL DIAGNOSIS

Osteoarthritis of the joint is the most common form of arthritis that results in shoulder pain; however, rheumatoid arthritis, posttraumatic arthritis, and rotator cuff arthropathy are also common causes of shoulder pain (Fig. 25.5). Less common causes of arthritis-induced shoulder pain include collagen vascular diseases, infection, villonodular

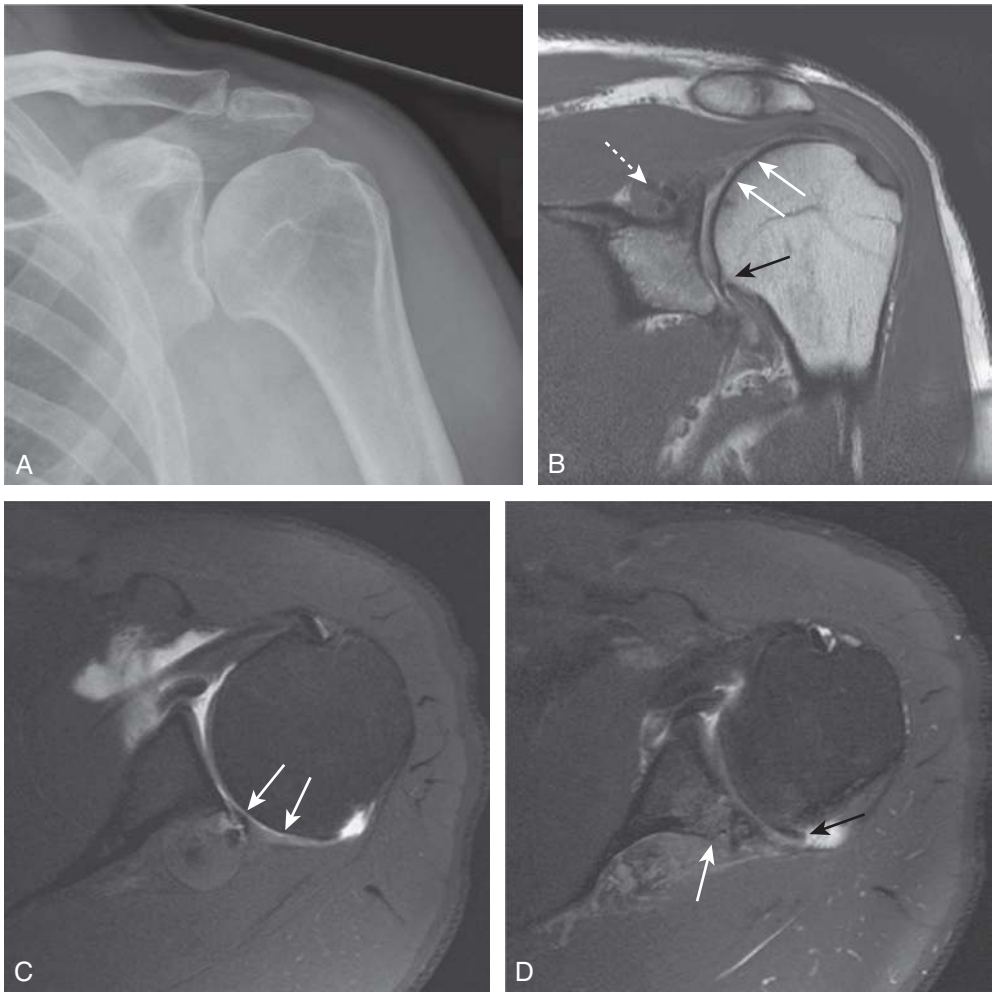

FIG 25.2 **A,** Anteroposterior (AP) radiograph of a patient with early osteoarthritis (OA) of the glenohumeral joint. There is asymmetric joint space narrowing and minor inferior osteophyte formation. The acromioclavicular (AC) joint is normal, and the subacromial space is preserved. **B,** The coronal T1-weighted (T1W) magnetic resonance (MR) arthrogram image demonstrates chondral thinning (*white arrows*), the inferior osteophyte (*black arrow*), and low-signal intensity (SI) loose bodies within the spinoglenoid notch (*broken arrow*). **C,** The chondral thinning is also seen on an axial T1W with fat suppression (FST1W) MR image (*white arrows*). **D,** On a more inferior axial FST1W MR image, the osteophytes (*black arrow*) are visualized in association with bony eburnation of the posterior glenoid (*thick white arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain*. WB Saunders; 2011.)

synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is diagnosed with culture and treated with antibiotics rather than injection therapy. Collagen vascular diseases generally manifest as a polyarthropathy rather than a monoarthropathy limited to the shoulder joint; however, shoulder pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described here.

TREATMENT

Initial treatment of the pain and functional disability associated with osteoarthritis of the shoulder includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

Intraarticular injection of the shoulder is performed by placing the patient in the supine position and preparing the skin overlying the shoulder, subacromial region, and joint space with antiseptic solution. Using strict aseptic technique, the practitioner attaches a sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone to a 1½-inch, 25-gauge needle. The midpoint of the acromion is identified, and at a point approximately 1 inch below the midpoint, the shoulder joint space is identified.

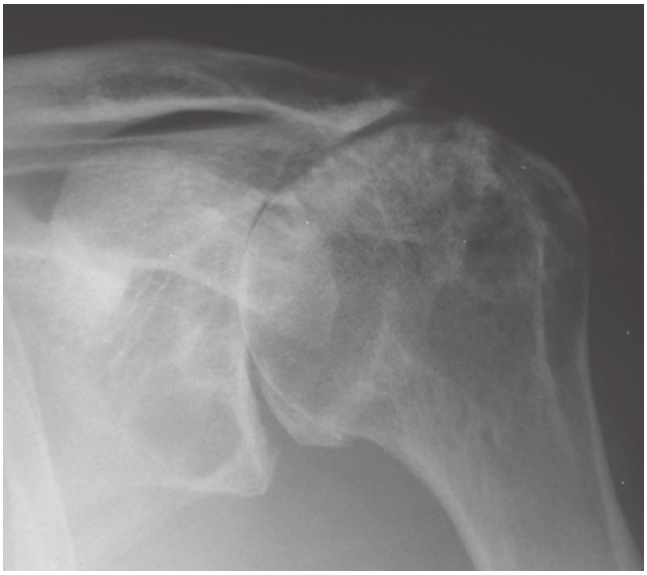

FIG 25.3 Anteroposterior (AP) radiograph of a patient with severe glenohumeral joint osteoarthritis (OA) secondary to rotator cuff failure (cuff arthropathy). Note the superior migration of the humeral head with complete loss of the subacromial space and bony eburnation of the acromion. (From Waldman SD, Campbell RSD. *Imaging of pain*. WB Saunders; 2011.)

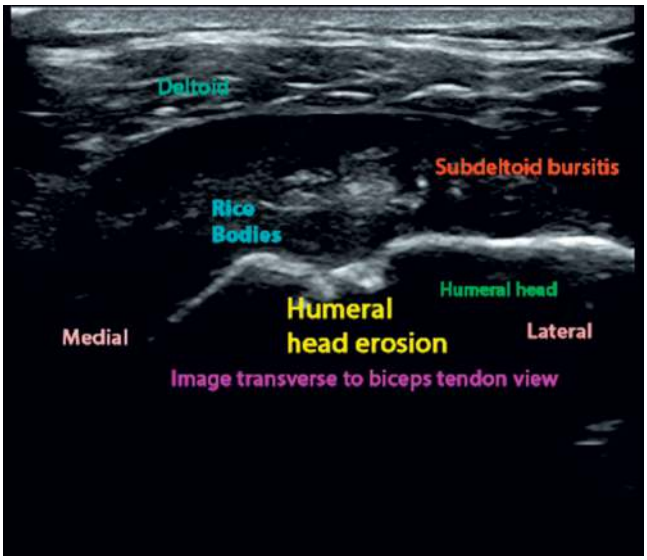

FIG 25.4 Transverse ultrasound image demonstrating significant osteoarthritis of the head of the humerus as evidenced by the defects in the cortical contour.

The needle is carefully advanced through the skin and subcutaneous tissues, through the joint capsule, and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly and slightly more medially. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection is felt; if resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed,

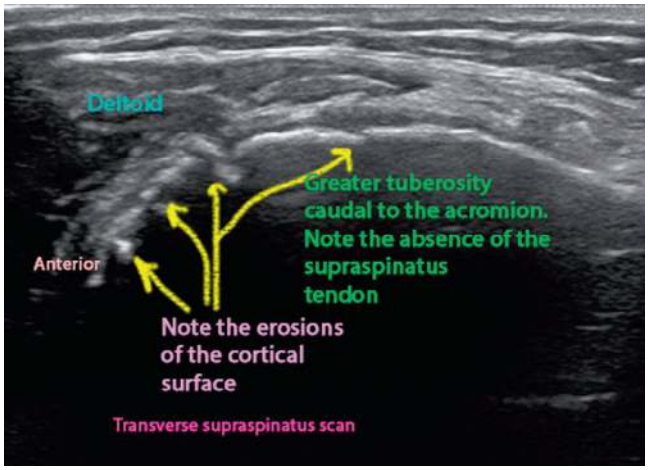

FIG 25.5 Transverse ultrasound image demonstrating significant cortical erosions in a patient with complete rupture of the supraspinatus tendon.

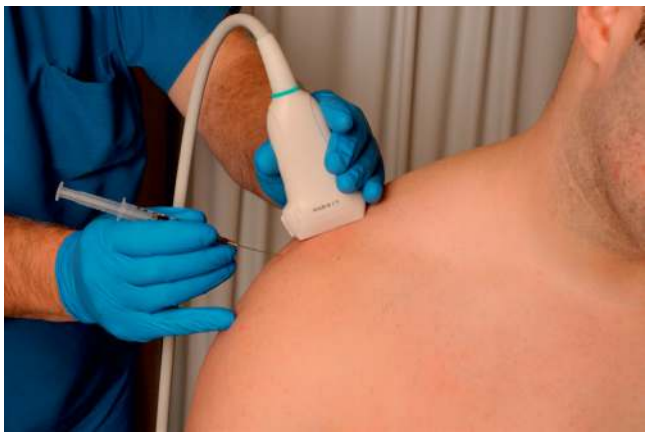

FIG 25.6 Ultrasound needle guidance may aid in the intraarticular placement of the needle in patients in whom anatomic landmarks are difficult to identify.

and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience has suggested that the injection of platelet-rich plasma into the glenohumeral joint may reduce the pain and functional disability associated with osteoarthritis of the shoulder. Ultrasound needle guidance may aid in the intraarticular placement of the needle in patients in whom anatomic landmarks are difficult to identify (Fig. 25.6).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for shoulder pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to

minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of intraarticular injection of the shoulder is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the shoulder joint, and they should be warned of this possibility.

CLINICAL PEARLS

Osteoarthritis of the shoulder is a common complaint encountered in clinical practice. It must be distinguished from other causes of shoulder pain, including rotator cuff tears. Intraarticular injection is extremely effective in the treatment of pain secondary to arthritis of the shoulder joint. Coexistent bursitis and tendinitis may contribute to shoulder pain and necessitate additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with this injection technique.

SUGGESTED READINGS

Adams JE, Steinmann SP. Surgical management of osteoarthritis in the shoulder and elbow. *J Hand Ther*. 2022;35(3):413–417.

Hodgetts C, Bruce W. Epidemiology, common diagnoses, treatments and prognosis of shoulder pain: a narrative review. *Int J Osteopath Med*. 2021;42:11–19.

Mengi A, Guler MA. Nocturnal pain in patients with rotator cuff related shoulder pain: a prospective study. *Musculoskelet Sci Pract*. 2022;59:102536.

Peebles LA, Arner JW, Haber DB, Provencher MT. Glenohumeral resurfacing in young, active patients with end-stage osteoarthritis of the shoulder. *Arthrosc Tech*. 2020;9(9):e1315–e1322.

Waldman SD. Injection of the glenohumeral joint. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:87–90.

Waldman SD. The glenohumeral joint. In: Waldman SD, ed. *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Kluwer Wolters; 2016: 338–343.

Wynell-Mayow W, Chong CC, Musbahi O, Ibrahim E. A lower critical coracoid process angle is associated with type-B osteoarthritis: a radiological study of normal and diseased shoulders. *JSES Int*. 2022;6(3):447–453.

Acromioclavicular Joint Pain

ICD-10 CODE M25.519

THE CLINICAL SYNDROME

The acromioclavicular joint is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are frequently the result of falling directly onto the shoulder when playing sports or riding a bicycle. Repeated strain from throwing or working with the arm raised across the body may also result in trauma to the joint. After trauma, the joint may become acutely inflamed; if the condition becomes chronic, arthritis of the acromioclavicular joint may develop. Cysts of the acromioclavicular joint can become quite large and contribute to functional disability and pain (Fig. 26.1). Rarely, infection of the acromioclavicular joint may occur.

SIGNS AND SYMPTOMS

Patients suffering from acromioclavicular joint dysfunction frequently complain of pain when reaching across the chest (Fig. 26.2). Often, patients are unable to sleep on the affected shoulder and may complain of a grinding sensation in the joint, especially on first awakening. Physical examination may reveal enlargement or swelling of the joint, with tenderness to palpation. Downward traction or passive adduction of the affected shoulder may cause increased pain. Physical examination of the abnormal acromioclavicular joint will reveal positive provocative tests including the acromioclavicular adduction stress test, the chin adduction test, and the Paxino test (Fig. 26.3). If the ligaments of the acromioclavicular joint are disrupted, these maneuvers may reveal actual joint instability (Box 26.1).

TESTING

Plain radiographs of the joint may reveal narrowing or sclerosis, consistent with osteoarthritis or actual separation or dislocation of the joint (Fig. 26.4). Magnetic resonance imaging (MRI) is indicated if disruption of the ligaments is suspected and to clarify the extent of ligamentous injury or to help rule out infection (Fig. 26.5). Ultrasound evaluation of the joint is useful to further delineate acromioclavicular joint pathology (Figs. 26.6 and 26.7). The injection technique described later serves as both a diagnostic and a therapeutic maneuver. In selected patients, arthroscopy of the joint may provide additional diagnostic information. If polyarthritis is

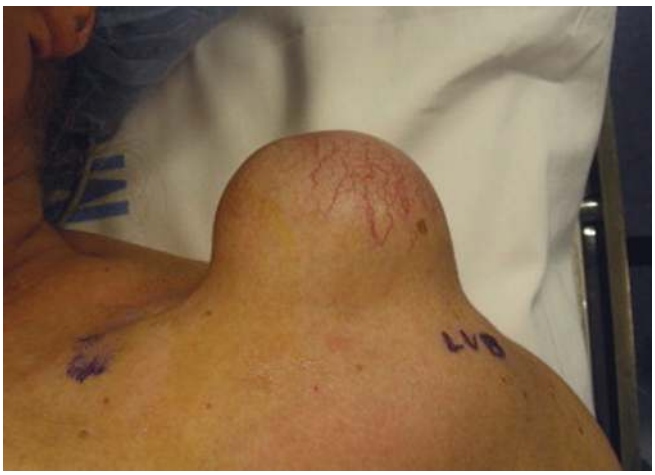

FIG 26.1 Preoperative photograph displaying a large acromioclavicular joint cyst with telangiectatic vessels. (From Nowak DD, Covey AS, Grant RT, Bigliani LU. Massive acromioclavicular joint cyst. *J Shoulder Elbow Surg.* 2009;18(5):e12–e14.)

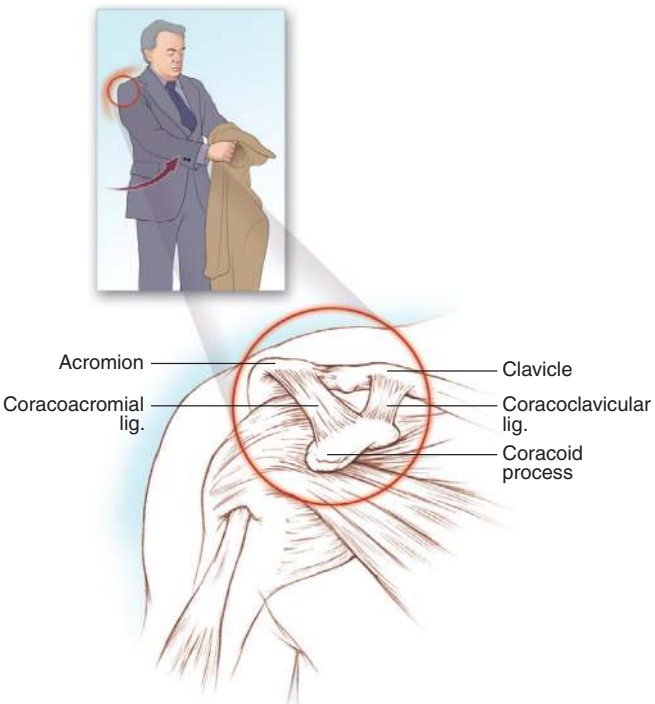

FIG 26.2 Acromioclavicular joint pain is made worse by reaching across the chest.

FIG 26.3 To perform the adduction stress test for acromioclavicular joint dysfunction, the examiner has the patient maximally extend the affected shoulder and arm behind him or her while the examiner exerts forward pressure on the scapula. (From Waldman SD. *Physical diagnosis of pain*. 4th ed. Philadelphia: Elsevier, Fig. 61.5.)

BOX 26.1 Radiographic Classification of Injuries of the Acromioclavicular Joint

- Type I: Normal
- Type II: Subluxation of the acromioclavicular joint space is less than 1 cm; normal coracoclavicular space
- Type III: Subluxation of the acromioclavicular joint space is greater than 1 cm; widening of the coracoclavicular space is more than 50%
- Types IV–VI: Subluxation of the acromioclavicular joint space is more than 1 cm, and widening of the coracoclavicular space is more than 50%; there is associated displacement of the clavicle

present, screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing should be performed.

DIFFERENTIAL DIAGNOSIS

Osteoarthritis of the acromioclavicular joint is a frequent cause of shoulder pain, and it is usually the result of trauma (Box 26.2). However, rheumatoid arthritis and rotator cuff arthropathy are also common causes of shoulder pain that may mimic acromioclavicular joint pain and confuse the diagnosis. Less common causes of arthritis-induced shoulder pain include the collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture, antibiotics, and surgical drainage, rather than injection therapy. Collagen vascular diseases generally manifest as polyarthropathy rather than monarthropathy limited to the shoulder joint; however, shoulder pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described here.

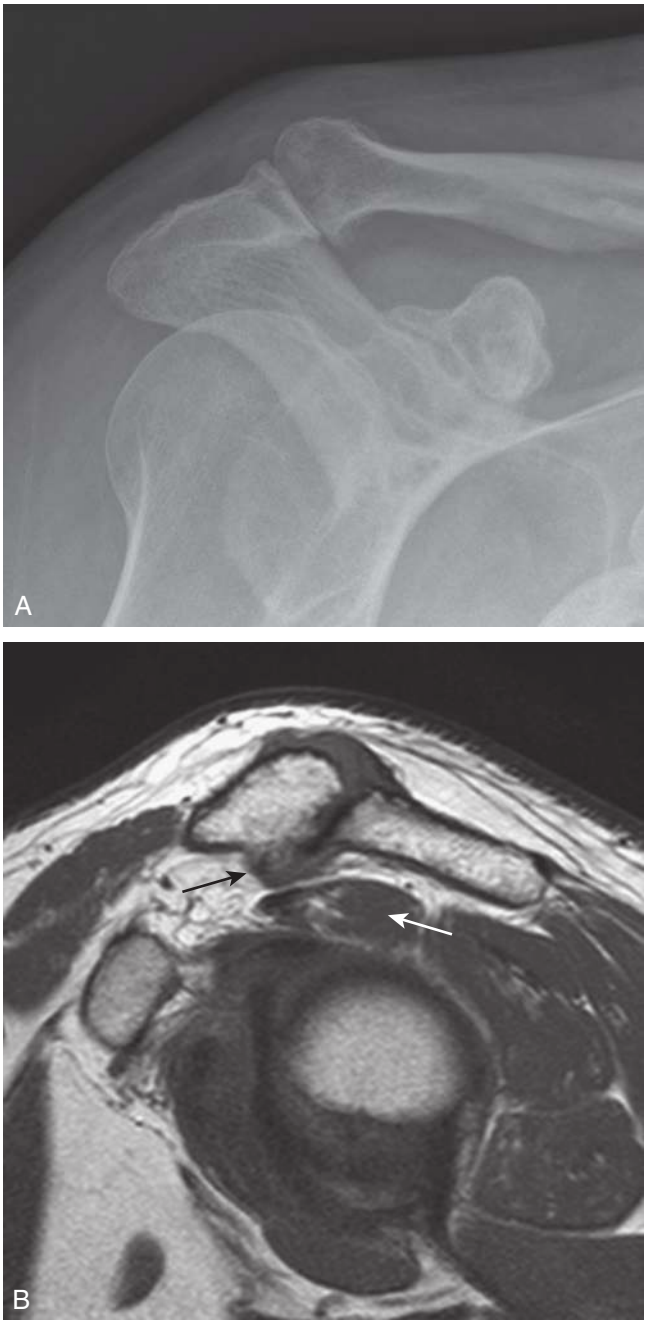

FIG 26.4 A, Oblique radiograph demonstrating typical features of osteoarthritis (OA) changes with osteophytes and sclerosis. B, The sagittal oblique T1-weighted (T1W) magnetic resonance (MR) image shows the inferior osteophyte formation (*black arrow*), which does not impinge on the supraspinatus tendon (*white arrow*). (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Elsevier; 2011: Fig. 89.1.)

TREATMENT

Initial treatment of the pain and functional disability associated with acromioclavicular joint pain includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For

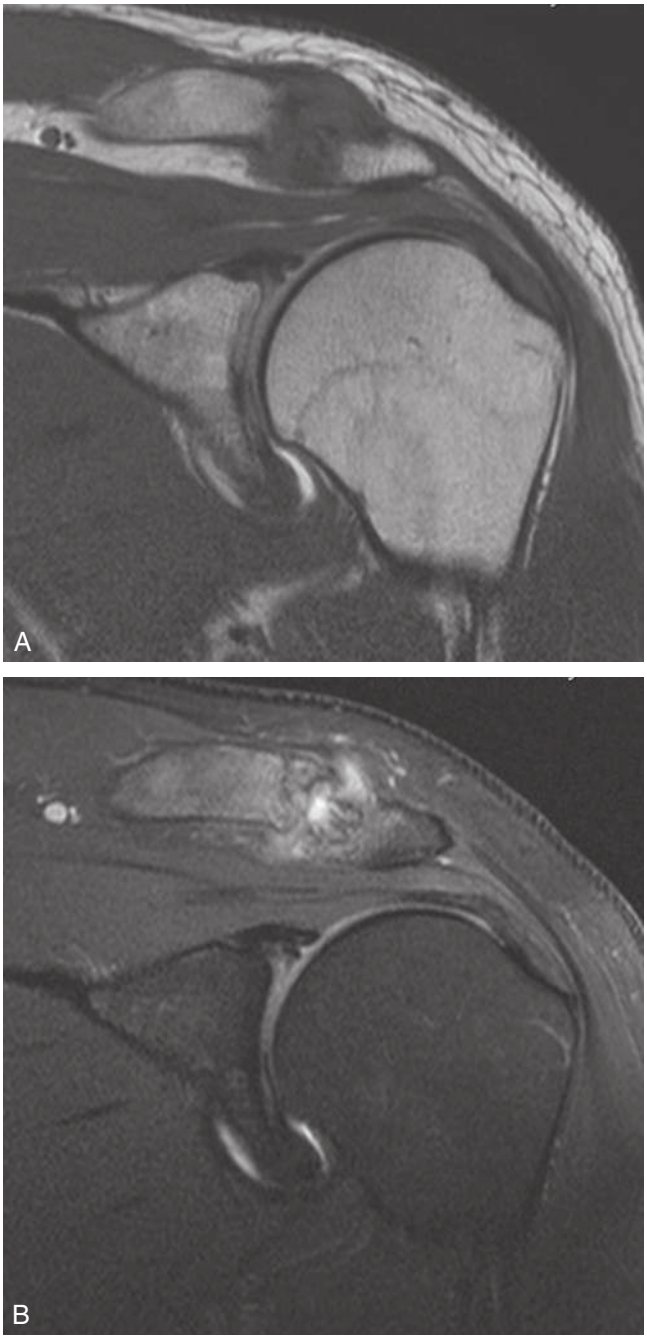

FIG 26.5 Coronal oblique T1-weighted (T1W) **(A)** and fast spin T2 weighted (FST2W) **(B)** magnetic resonance (MR) images of a patient with osteoarthritis (OA) of the acromioclavicular (AC) joint with marrow edema and subchondral cyst formation. In addition, there is subluxation of the joint, indicating joint instability, which may contribute to subacromial impingement. (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Elsevier; 2011: Fig. 89.3.)

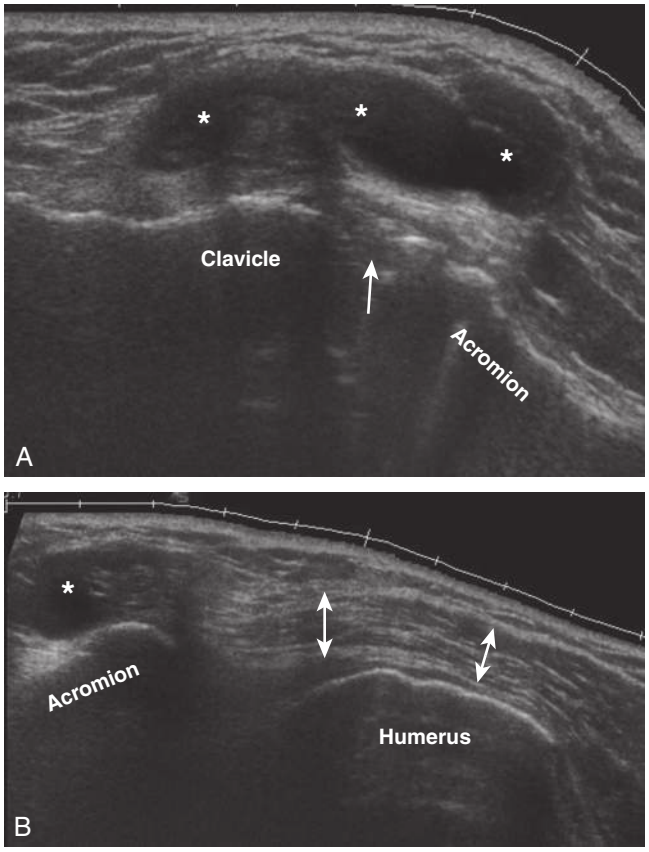

FIG 26.6 **A,** Coronal ultrasound (US) image of the acromioclavicular (AC) joint (*white arrow*) with an overlying loculated anechoic cystic structure (*asterisks*). **B,** The coronal image of the rotator cuff shows the deltoid muscle (*double-headed arrows*) lying directly on the humeral head with absence of the supraspinatus as a result of a massive cuff tear. The cyst occurs as a result of glenohumeral joint fluid communicating through the AC joint in association with the cuff tear, a finding referred to as the "geyser" phenomenon. A portion of the cyst is also visible on this image (*asterisk*). (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Elsevier; 2011: Fig. 89.4.)

patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

Intraarticular injection of the acromioclavicular joint is performed by placing the patient in the supine position and preparing the skin overlying the superior shoulder and distal clavicle with antiseptic solution. A sterile syringe containing 1 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The top of the acromion is identified, and, at a point approximately 1 inch medially, the acromioclavicular joint space is identified. The needle is carefully advanced through the skin and subcutaneous tissues, through the joint capsule, and into the joint (Fig. 26.8). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected slightly more medially. After the joint space is entered, the contents of the syringe are gently injected. Some resistance to injection should be felt, because the joint space is small and the joint capsule is dense. If significant resistance is encountered, however, the needle is probably in a ligament and should be advanced slightly into the joint space until the injection can proceed with only limited resistance. If no resistance is encountered on injection,

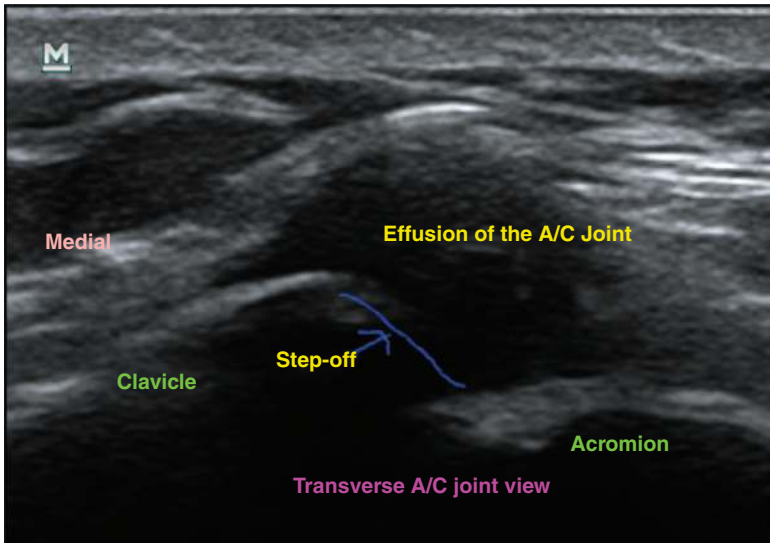

FIG 26.7 Transverse ultrasound view of the acromioclavicular joint showing effusion and a step-off indicating ligamentous injury. (Courtesy Steven Waldman, MD.)

BOX 26.2 Differential Diagnosis of Acromioclavicular Joint Pain

- Clavicle fractures
- Coracoid fractures
- Rotator cuff injury
- Shoulder dislocation
- Shoulder impingement syndrome
- Osteoarthritis
- Inflammatory arthritis
- Glenoid labrum tear
- Septic arthritis
- Brachial plexus injury
- Parsonage–Turner syndrome
- Os acromiale
- Distal clavicle osteolysis

the joint space is probably not intact, and MRI is recommended. After injection, the needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that the intraarticular injection of platelet-rich plasma may provide improved healing of acromioclavicular joint pathology. Ultrasound guidance may improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify (Fig. 26.9).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for shoulder pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to

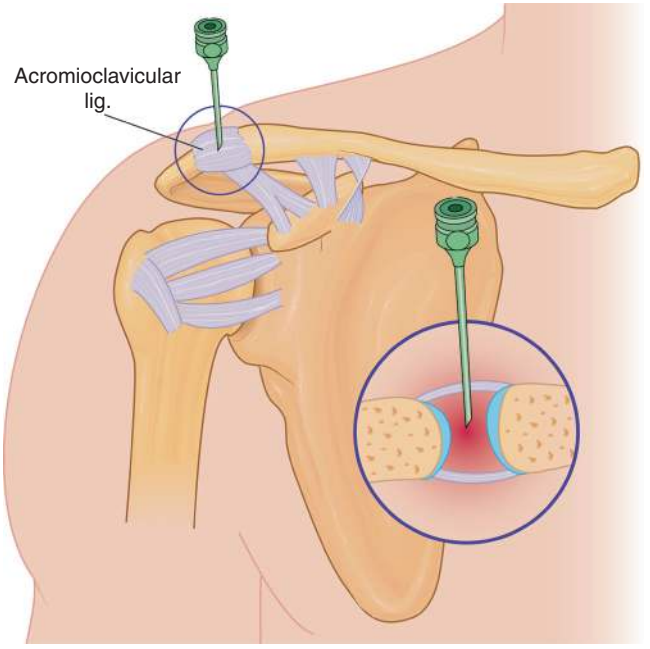

FIG 26.8 Proper needle placement for acromioclavicular joint injection. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:41.)

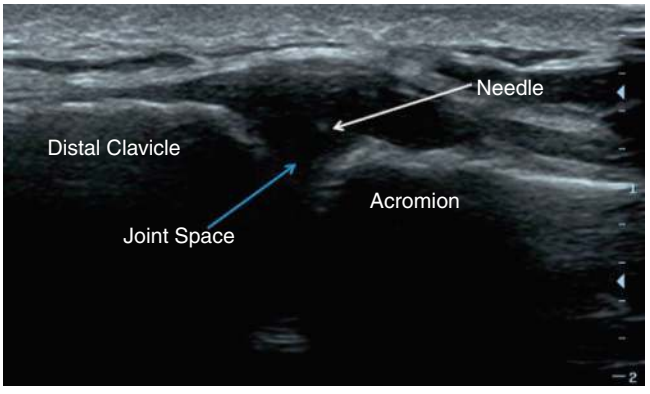

FIG 26.9 Transverse ultrasound image demonstrating the needle tip within the acromioclavicular joint using an out-of-plane approach.

minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of intraarticular injection of the acromioclavicular joint is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the acromioclavicular joint, and patients should be warned of this possibility.

CLINICAL PEARLS

Intraarticular injection is extremely effective in the treatment of pain secondary to arthritis of the acromioclavicular joint. Coexistent bursitis and tendinitis may contribute to shoulder pain and necessitate additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with this injection technique.

SUGGESTED READINGS

Allen H, Chan BY, Davis KW, Blankenbaker DG. Overuse injuries of the shoulder. *Radiol Clin North Am*. 2019;57(5):897–909.

Armstrong A. Evaluation and management of adult shoulder pain: a focus on rotator cuff disorders, acromioclavicular joint arthritis, and glenohumeral arthritis. *Med Clin North Am*. 2014;98(4):755–775.

Cibulas A, Leyva A, Cibulas G, et al. Acute shoulder injury. *Radiol Clin North Am*. 2019;57(5):883–896.

Denker J, McCarty LP III. Acromioclavicular joint injuries overhead athletes. *Oper Tech Sports Med*. 2016;24(3):213–222.

Favorito PJ, Herbst KA. Acromioclavicular joint injuries. In: Greiwe MR, ed. *Shoulder and elbow trauma and its complications: Volume 1: The shoulder*. Cambridge: Woodhead Publishing; 2015:215–231. *Woodhead Publishing Series in Biomaterials*; Vol. 102.

Netter FH. Shoulder (glenohumeral joint). In: *Atlas of human anatomy*. 4th ed. Philadelphia, PA: Saunders; 2006.

Reschke D, Dagrosa R, Matteson DT. An unusual cause of shoulder pain and weakness. *Am J Emerg Med*. 2018;36(12):2339.e5–2339.e6.

Waldman SD. Clinical correlates: functional anatomy of the shoulder. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2016.

Waldman SD. Acromioclavicular joint. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:90–91.

Waldman SD. Acromioclavicular joint injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:90–93.

Waldman SD. Disorders of the acromioclavicular joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:143–151.

27

Subdeltoid Bursitis

ICD-10 CODE M75.50

THE CLINICAL SYNDROME

The subdeltoid bursa lies primarily under the acromion and extends laterally between the deltoid muscle and the joint capsule under the deltoid muscle (Fig. 27.1). It may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The subdeltoid bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries frequently take the form of direct trauma to the shoulder when playing sports or falling off a bicycle. Repeated strain from throwing, bowling, carrying a heavy briefcase, working with the arm raised across the body, rotator cuff injuries, or repetitive motion associated with assembly-line work may result in inflammation of the subdeltoid bursa. If the inflammation becomes chronic, calcification of the bursa may occur.

Patients suffering from subdeltoid bursitis frequently complain of pain with any movement of the shoulder, but especially with abduction (Fig. 27.2). The pain is localized to the subdeltoid area, with referred pain often noted at the insertion of the deltoid at the deltoid tuberosity on the upper third of the humerus. Patients are often unable to sleep on the affected shoulder and may complain of a sharp, catching sensation when abducting the shoulder, especially on first awakening.

SIGNS AND SYMPTOMS

Physical examination may reveal point tenderness over the acromion; occasionally, swelling of the bursa gives the affected deltoid muscle an edematous feel. Passive elevation and medial rotation of the affected shoulder reproduce the pain, as do resisted abduction and lateral rotation. Sudden release of resistance during this maneuver markedly increases the pain. Rotator cuff tear may mimic or coexist with subdeltoid bursitis and may confuse the diagnosis (see "Differential Diagnosis").

TESTING

Plain radiographs of the shoulder may reveal calcification of the bursa and associated structures, consistent with chronic inflammation. Magnetic resonance imaging is indicated to confirm the diagnosis of subdeltoid bursitis or if a diagnosis of tendinitis, partial disruption of the ligaments, or rotator cuff tear is being considered (Figs. 27.3 and 27.4). Ultrasound imaging may further delineate the cause of the patient's pain (Figs. 27.5 and 27.6). Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Radionuclide bone scanning is indicated if metastatic disease or primary tumor involving the shoulder is a possibility. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Subdeltoid bursitis is one of the most common causes of shoulder joint pain. Osteoarthritis, rheumatoid arthritis, posttraumatic arthritis, and rotator cuff arthropathy are also common causes of shoulder pain that may coexist with subdeltoid bursitis (Box 27.1). Less common causes of arthritis-induced shoulder pain include collagen vascular diseases, infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics, rather than with injection therapy. Collagen vascular diseases generally manifest as polyarthropathy rather than monarthropathy limited to the shoulder joint; however, shoulder pain secondary to collagen vascular disease responds exceedingly well to the injection technique described here.

TREATMENT

Initial treatment of the pain and functional disability associated with subdeltoid bursitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection of local anesthetic and steroid into the subdeltoid bursa is a reasonable next step.

Injection into the subdeltoid bursa is performed by placing the patient in the supine position and preparing the skin overlying the superior shoulder, acromion, and distal clavicle with antiseptic solution. A sterile syringe containing

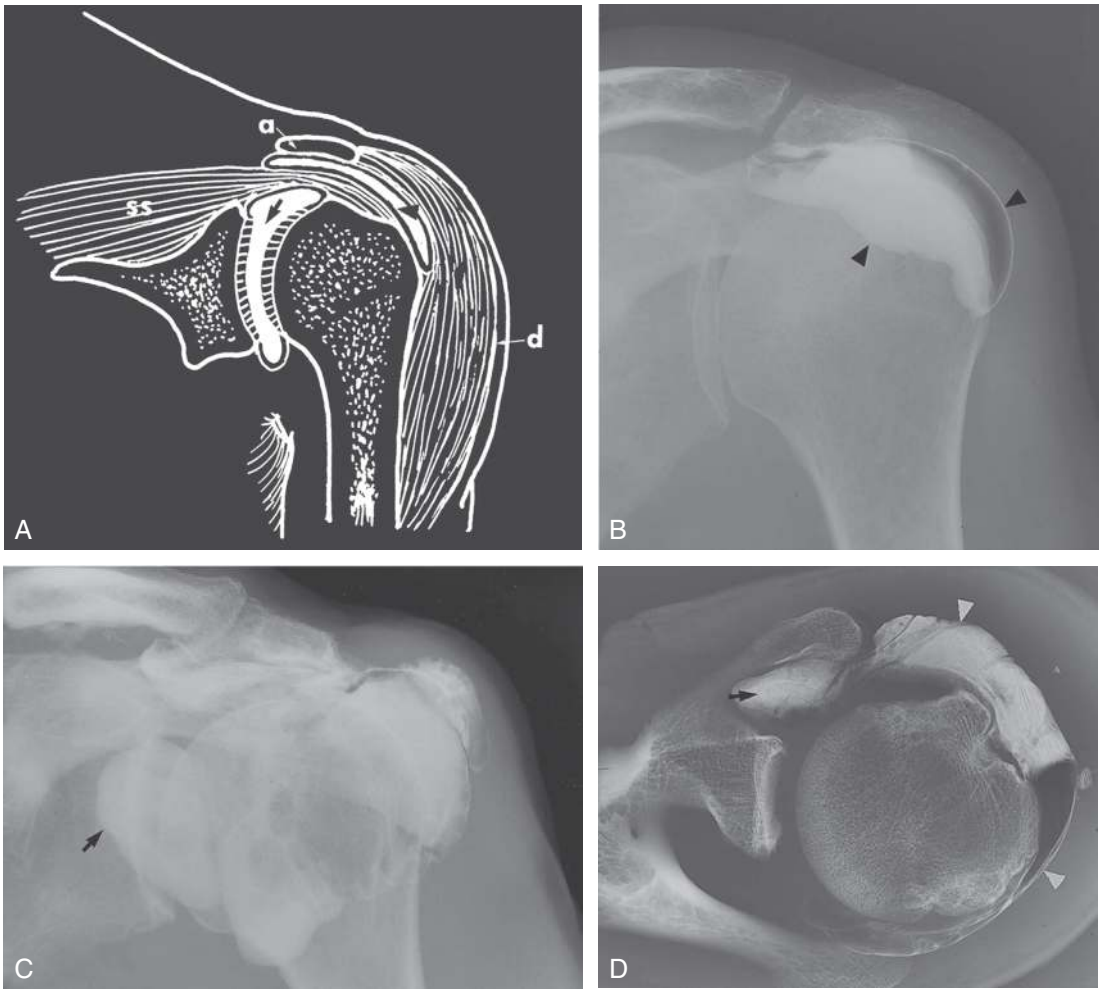

FIG 27.1 Normal anatomy of the subacromial (subdeltoid) bursa. **A,** Diagram of a coronal section of the shoulder shows the glenohumeral joint (*arrow*) and subacromial (subdeltoid) bursa (*arrowhead*), separated by a portion of the rotator cuff (i.e., supraspinatus tendon). The supraspinatus (*ss*) and deltoid (*d*) muscles and the acromion (*a*) are indicated. **B,** Subdeltoid-subacromial bursogram, accomplished with the injection of both radiopaque contrast material and air, shows the bursa (*arrowheads*) sitting like a cap on the humeral head and greater tuberosity of the humerus. Note that the joint is not opacified, indicative of an intact rotator cuff. **C,** In a different cadaver, a subacromial-subdeltoid bursogram shows much more extensive structure as a result of opacification of the subacromial, subdeltoid, and subcoracoid (*arrow*) portions of the bursa. **D,** Radiograph of a transverse section of the specimen illustrated in **(C)** shows both the subdeltoid (*arrowheads*) and subcoracoid (*arrow*) portions of the bursa. The glenohumeral joint is not opacified. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3072.)

4 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The lateral edge of the acromion is identified, and at the midpoint of the lateral edge, the injection site is identified. At this point, the needle is carefully advanced with a slightly cephalad trajectory through the skin and subcutaneous tissues beneath the acromion capsule and into the bursa (Fig. 27.7). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected slightly more inferiorly. After the bursa is entered, the contents of the syringe are gently injected while the needle is slowly withdrawn. Resistance to injection should be minimal unless calcification of the bursal sac is present, in which case resistance to needle advancement is associated with a gritty feel. Significant calcific bursitis may ultimately require surgical excision to achieve complete relief of symptoms. After injection, the needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Clinical case reports suggest that the injection of rilonacept, an interleukin-1 trap, may provide an alternative to the use of steroids in the treatment of subdeltoid bursitis. Ultrasound guidance may

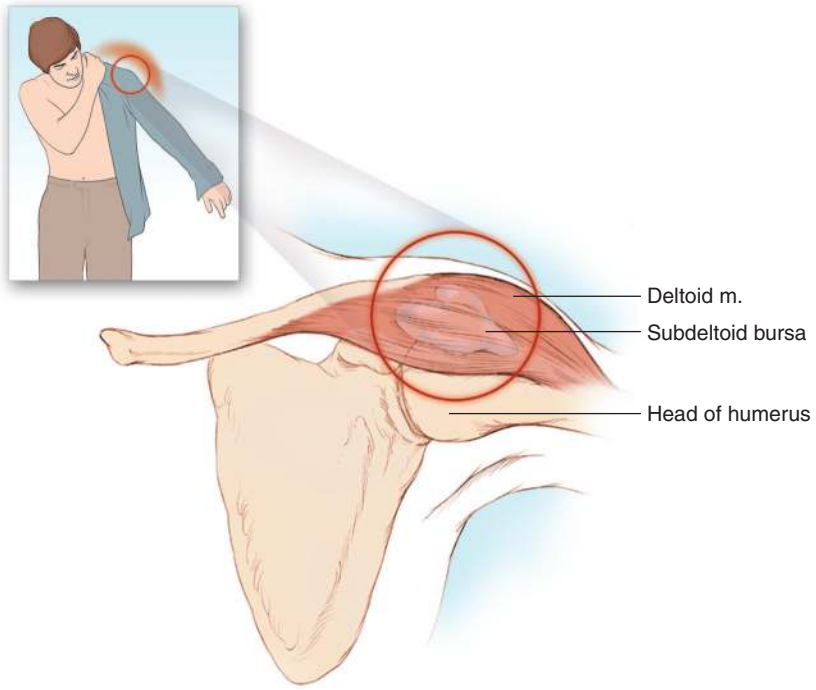

FIG 27.2 Abduction of the shoulder exacerbates the pain of subdeltoid bursitis.

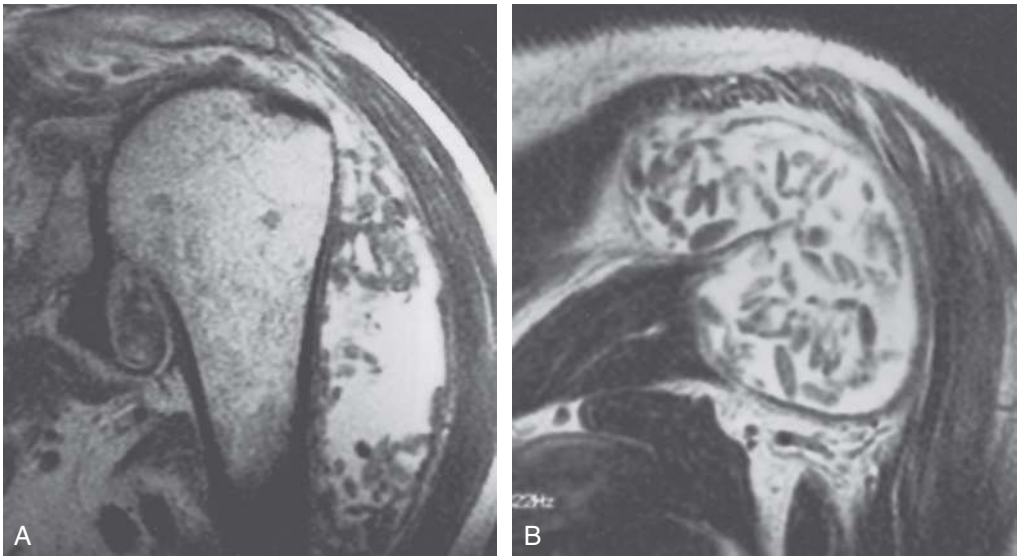

FIG 27.3 Subacromial-subdeltoid bursitis. **A, B,** Oblique, coronal, fast spin-echo (TR 300, TE 99) magnetic resonance images (**B** is posterior to **A**) show massive distention of the bursa with fluid of high signal intensity, and synovial proliferative tissue and rice bodies of low signal intensity, in a patient with probable rheumatoid arthritis. The rotator cuff is torn and retracted, and the glenohumeral joint is also involved. (From Resnick D. *Diagnosis of bone and joint disorders.* 4th ed. Philadelphia: Saunders; 2002:4256.)

improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for shoulder pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied

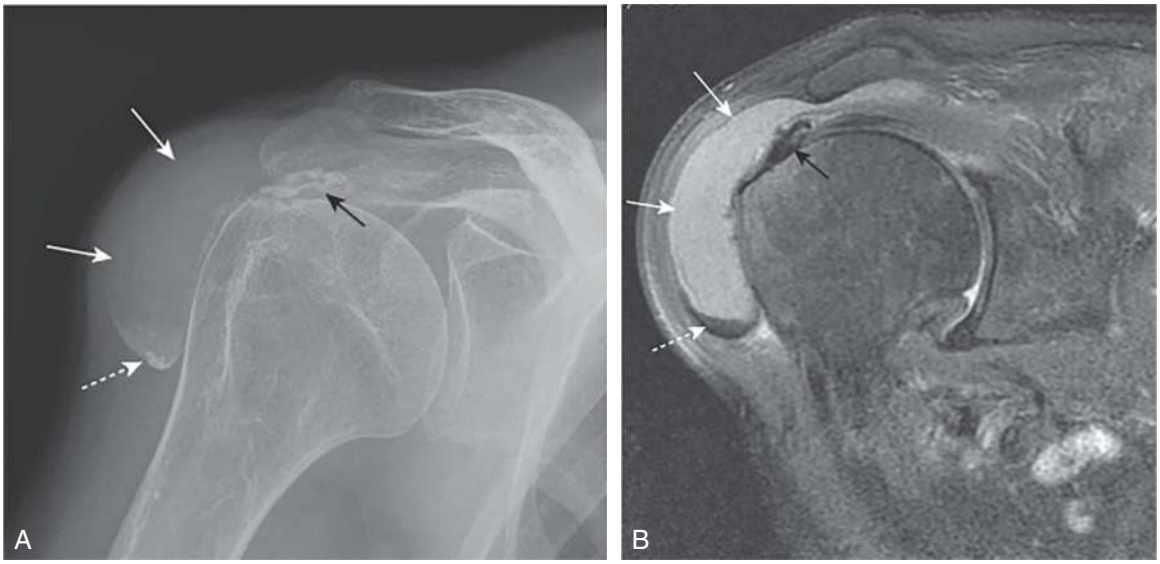

FIG 27.4 **A,** Anterior posterior (AP) radiograph of a patient with an acute inflammatory subdeltoid bursitis. There is soft tissue thickening (*white arrows*) due to bursal distention, with crystals collecting in the dependent portion of the bursa (*broken white arrow*), originating from a focus of calcific tendinitis within the rotator cuff (*black arrow*). **B,** The corresponding coronal FST2W MR image shows the same features with a high-SI fluid-filled bursa (*white arrows*), low-SI crystals within the bursa (*broken white arrow*), and the supraspinatus tendon (*black arrow*). (From Subdeltoid bursitis. Waldman SD, Campbell RSD, eds. *Imaging of pain*. Philadelphia: W.B. Saunders; 2011: chap 99, 253–254.)

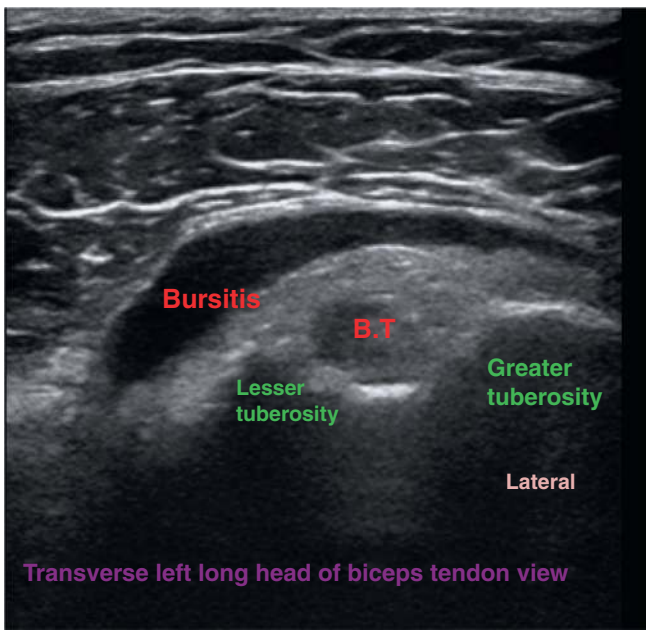

FIG 27.5 Longitudinal ultrasound image of subdeltoid bursitis. Note relationship of the biceps tendon (*B.T.*), the bursa, and the humeral head.

to the injection site immediately after injection. The major complication of injection of the subdeltoid bursa is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection of the subdeltoid bursa, and patients should be warned of this possibility.

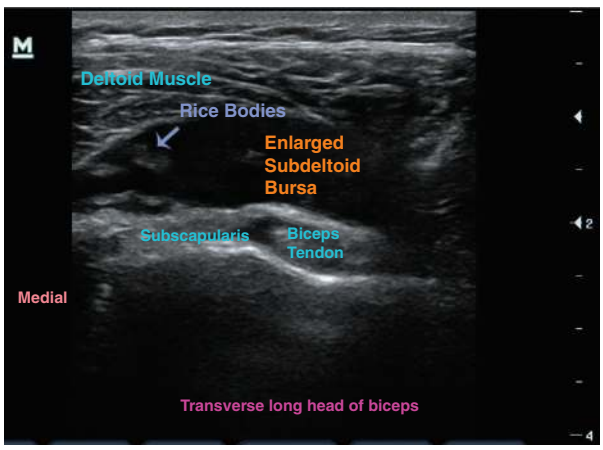

FIG 27.6 Sonographic finding of subdeltoid bursitis in patient with mixed connective tissue disease. Note the rice bodies within the enlarged, fluid-filled subdeltoid bursa.

BOX 27.1 Differential Diagnosis of Subdeltoid Bursitis

- Osteoarthritis
- Rheumatoid arthritis
- Posttraumatic arthritis
- Rotator cuff arthropathy
- Impingement syndromes
- Collagen vascular diseases
- Septic arthritis
- Villonodular synovitis
- Lyme disease

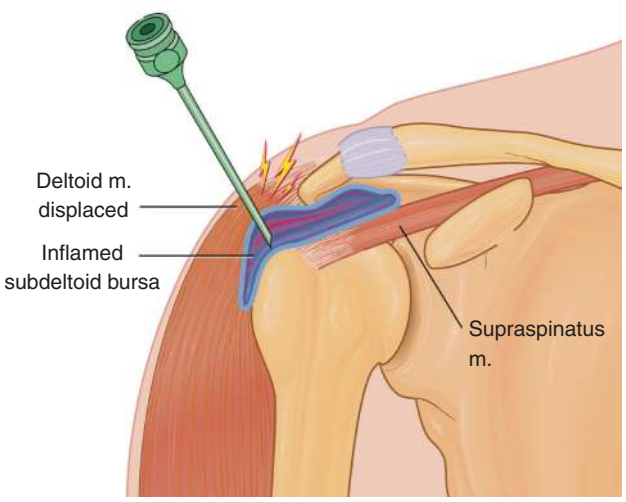

FIG 27.7 Proper needle placement for injection of the subdeltoid bursa. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

CLINICAL PEARLS

This injection technique is extremely effective in the treatment of pain secondary to subdeltoid bursitis. Coexistent arthritis and tendinitis may contribute to shoulder pain, necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

SUGGESTED READINGS

Kang BS, Lee SH, Cho Y, et al. Acute calcific bursitis after ultrasound-guided percutaneous barbotage of rotator cuff calcific tendinopathy: a case report. *PM&R*. 2016;8(8):808–812.

Waldman SD. Ultrasound-guided injection technique for subdeltoid. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:327–334.

Waldman SD. Subdeltoid bursitis and other disorders of the subdeltoid bursa. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:206–213.

Waldman SD. Subdeltoid bursitis injection In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:147–150.

Waldman SD. The subdeltoid bursa. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:91.

Wu T, Song HX, Dong Y, et al. Ultrasound-guided versus blind subacromial—subdeltoid bursa injection in adults with shoulder pain: a systematic review and meta-analysis. *Semin Arthritis Rheum*. 2015;45(3):374–378.

Bicipital Tendinitis

ICD-10 CODE M75.20

THE CLINICAL SYNDROME

The tendons of the long and short heads of the biceps are particularly susceptible to the development of tendinitis. Bicipital tendinitis is usually caused at least partially by impingement on the tendons of the biceps at the coracoacromial arch. The onset of bicipital tendinitis is generally acute, occurring after overuse or misuse of the shoulder joint, such as trying to start a recalcitrant lawn mower, practicing an overhead tennis serve, or performing an overaggressive follow-through when driving golf balls. The biceps muscle and tendons are susceptible to trauma and to wear and tear. If the damage is severe enough, the tendon of the long head of the biceps can rupture, leaving the patient with a telltale "Popeye" biceps (named after the cartoon character). This deformity can be accentuated by having the patient perform Ludington's maneuver: placing his or her hands behind the head and flexing the biceps muscle (see Chapter 31).

SIGNS AND SYMPTOMS

The pain of bicipital tendinitis is constant and severe and is localized in the anterior shoulder over the bicipital groove (Fig. 28.1). A catching sensation may accompany the pain. Significant sleep disturbance is often reported. The patient may attempt to splint the inflamed tendons by internal rotation of the humerus, which moves the biceps tendon from beneath the coracoacromial arch. Patients with bicipital tendinitis have a positive Yergason's test result—that is, production of pain on active supination of the forearm against resistance with the elbow flexed at a right angle (Fig. 28.2). Bursitis often accompanies bicipital tendinitis.

In addition to pain, patients suffering from bicipital tendinitis often experience a gradual reduction in functional ability because of decreasing shoulder range of motion that makes simple everyday tasks such as combing one's hair, fastening a brassiere, and reaching overhead quite difficult. With continued disuse, muscle wasting may occur, and a frozen shoulder may develop.

TESTING

Plain radiographs are indicated for all patients who present with shoulder pain. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the shoulder is indicated if rotator cuff tear is suspected and to further delineate shoulder pathology (Figs. 28.3–28.5). Arthroscopy can aid in the diagnosis and treatment of bicipital tendinitis in selected patients (Fig. 28.6). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

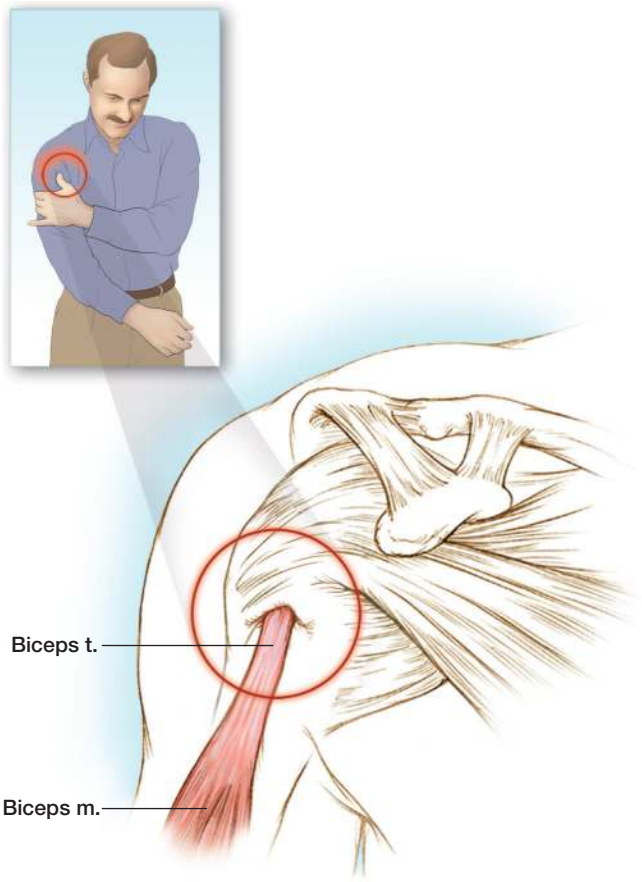

FIG 28.1 Palpation of the bicipital groove exacerbates the pain of bicipital tendinitis.

DIFFERENTIAL DIAGNOSIS

Bicipital tendinitis is usually a straightforward clinical diagnosis. However, coexisting bursitis or tendinitis of the shoulder from overuse or misuse may confuse the diagnosis.

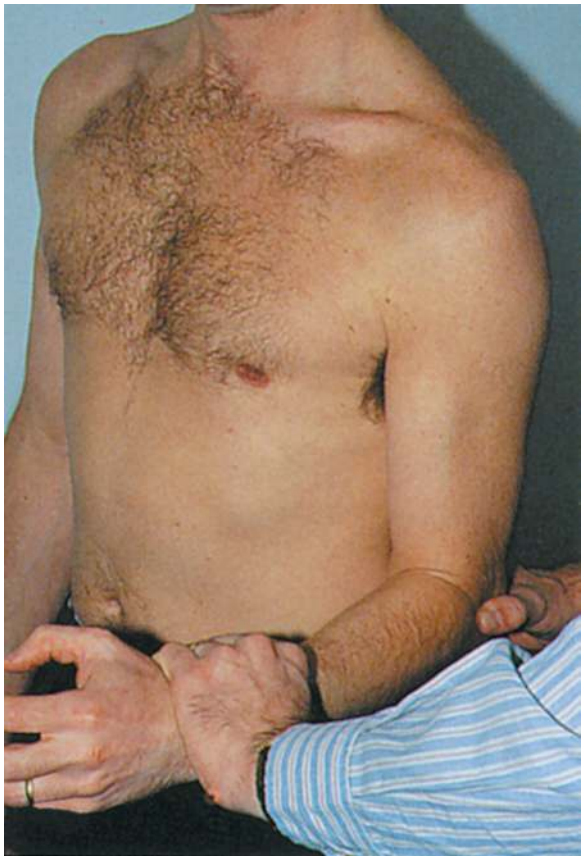

FIG 28.2 Yergason's test for bicipital tendinitis. (From Klippel JH, Dieppe PA. *Rheumatology*. 2nd ed. London: Mosby; 1998.)

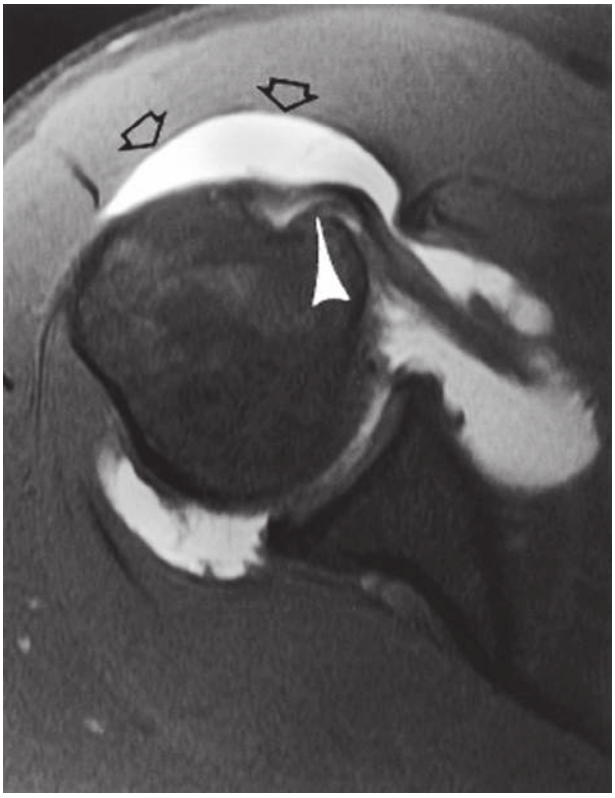

FIG 28.3 "Perched" biceps tendon. Fat-suppressed, T1-weighted, axial magnetic resonance arthrogram reveals a flattened biceps tendon (*large arrowhead*) draped over the lesser tuberosity in the presence of a normally configured bicipital groove. The presence of contrast material in the subacromial-subdeltoid bursa (*open arrowheads*) indicates a coexisting full-thickness tear of the rotator cuff. (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2005:3161.)

Occasionally, partial rotator cuff tear can be mistaken for bicipital tendinitis. In some clinical situations, consideration should be given to primary or secondary tumors involving the shoulder, superior sulcus of the lung, or proximal humerus. The pain of acute herpes zoster, which occurs before eruption of a vesicular rash, can also mimic bicipital tendinitis.

TREATMENT

Initial treatment of the pain and functional disability associated with bicipital tendinitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

Injection for bicipital tendinitis is carried out by placing the patient in the supine position with the arm externally rotated approximately 45 degrees. The coracoid process is identified anteriorly. Just lateral to the coracoid process is the lesser tuberosity, which can be more easily palpated as the arm is passively rotated. The point overlying the tuberosity is marked with a sterile marker. The skin overlying the anterior shoulder is prepared with antiseptic solution. A sterile syringe containing 1 mL of 0.25% preservative-free bupivacaine and

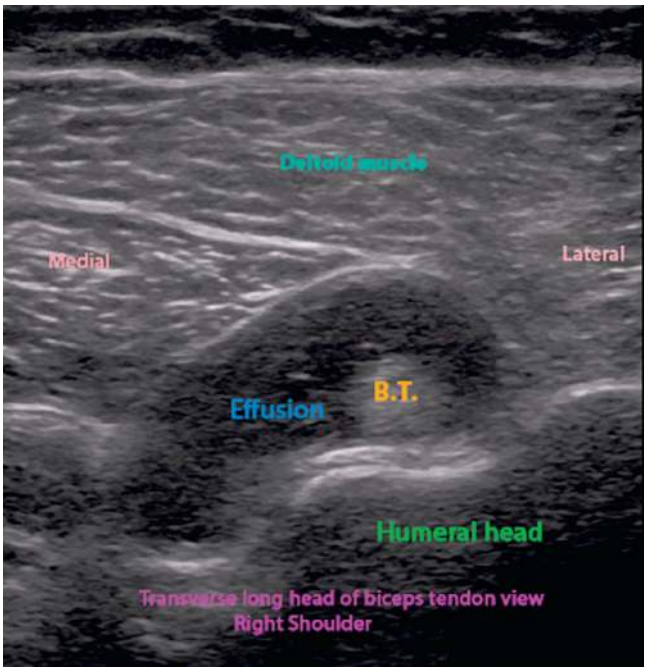

FIG 28.4 Transverse ultrasound image demonstrating significant effusion surrounding the right biceps tendon (*B.T.*)

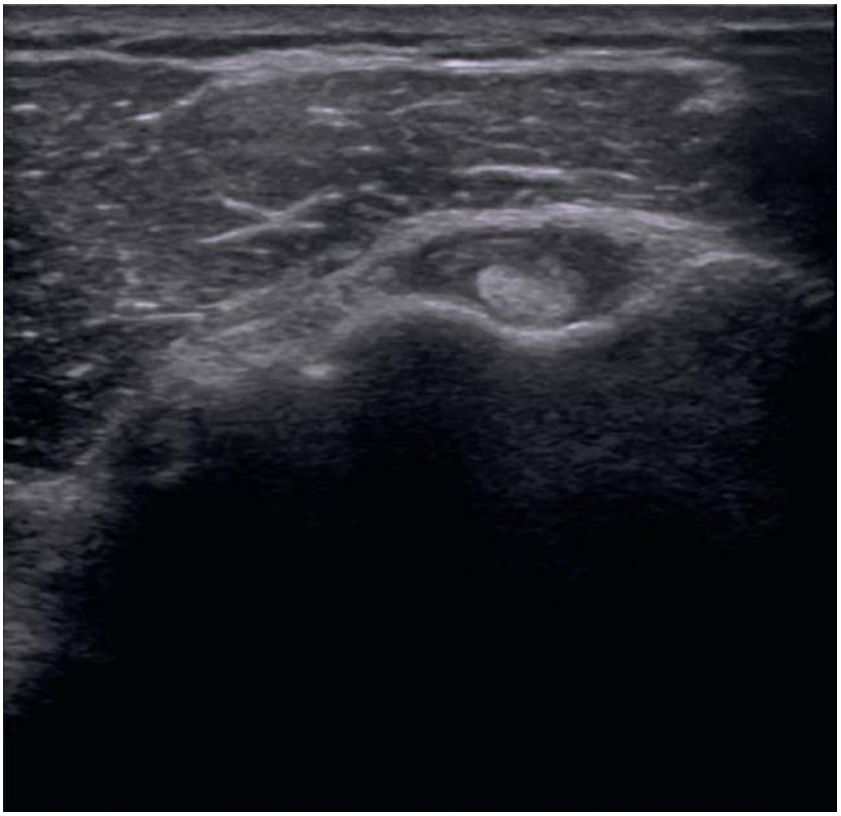

FIG 28.5 Longitudinal ultrasound image demonstrating an effusion along a portion of the biceps tendon. (Courtesy Dr. Steven Waldman.)

40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The previously marked point is palpated, and the insertion of the biceps tendon is reidentified with the gloved finger. The needle is carefully advanced at this point through the skin, subcutaneous tissues, and underlying tendon until it impinges on bone. The needle is then withdrawn 1–2 mm out of the periosteum of the humerus, and the contents of the syringe are gently injected. The clinician should feel slight resistance to injection. If no resistance is encountered, either the needle tip is in the joint space itself or the tendon is ruptured. If resistance is significant, the needle tip is probably in the substance of a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that injection of platelet-rich plasma around the inflamed tendon may provide improved healing of tendinopathy. Ultrasound guidance may improve the accuracy of needle placement is patients in whom anatomic landmarks are hard to identify (Fig. 28.7).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of this injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Trauma to the biceps tendon from the injection itself is also a possibility. Tendons that are highly inflamed or previously damaged are subject to rupture if they are injected directly. This complication can often be avoided if the clinician uses a gentle technique and stops injecting immediately if significant resistance is encountered. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

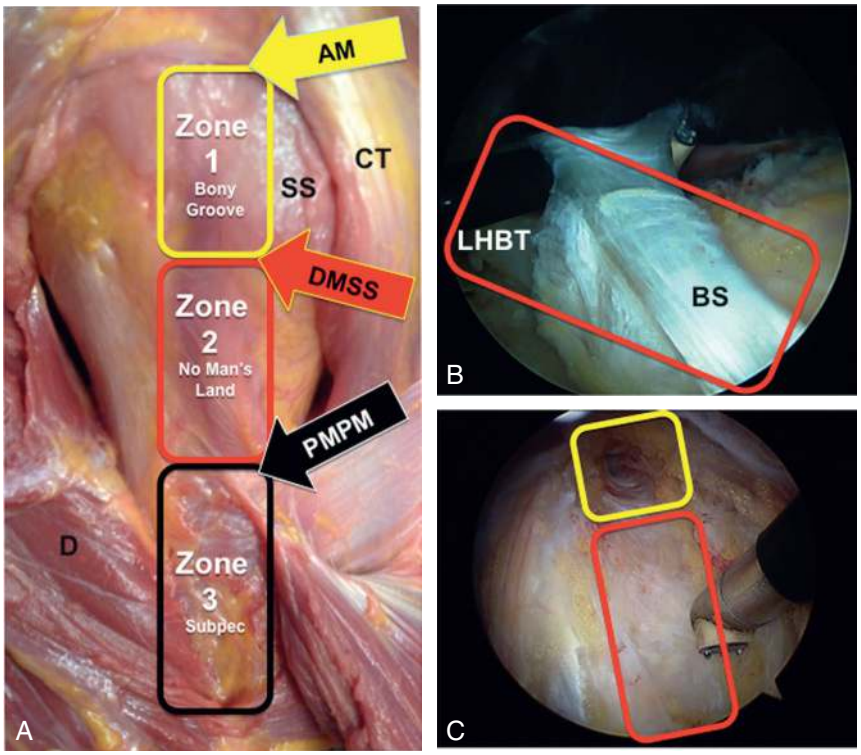

FIG 28.6 Anatomy of the biceps tendon. A soft tissue sheath **(A, B)** consistently covers the long head of the biceps tendon to the level of the proximal margin of the pectoralis major tendon (PMPM) and contributes to the roof of the bicipital tunnel. The sheath is clearly visible during open procedures **(A)** and extraarticular arthroscopic procedures within the subdeltoid space **(B, C)**. The fibroosseous bicipital tunnel consists of three distinct anatomic zones **(A)**. Zone 1 represents the traditional bony bicipital groove (*yellow box*) beginning at the articular margin (*AM*) and ending at the distal margin of the subscapularis tendon (*DMSS*). Zone 2 (*red box*) extends from the DMSS to the PMPM and represents a "no man's land" because it is not viewable from arthroscopy above or from subpectoral exposure below. Zone 3 is distal to the PMPM and represents the subpectoral region. The sheath overlying zone 2 can be robust **(B)**. *BS*, Bicipital sheath; *CT*, conjoint tendon; *D*, deltoid; *SS*, subscapularis. (From Taylor SA, Fabricant PD, Bansal M, et al. The anatomy and histology of the bicipital tunnel of the shoulder. *J Shoulder Elb Surg*. 2015;24(4):511–519.)

FIG 28.7 Ultrasound-guided injection of the bicipital groove and its contents.

CLINICAL PEARLS

The musculotendinous unit of the shoulder joint is susceptible to the development of tendinitis for several reasons. First, the joint is subjected to many different repetitive motions. Second, the space in which the musculotendinous unit functions is restricted by the coracoacromial arch, a configuration that makes impingement likely with extreme movements of the joint. Third, the blood supply to the musculotendinous unit is poor, and this complicates the healing of microtrauma. All these factors can contribute to tendinitis. If the inflammation continues, calcium deposition around the tendon may occur and make subsequent treatment more difficult.

The injection technique described is extremely effective in the treatment of pain secondary to bicipital tendinitis. Coexistent bursitis and arthritis may contribute to shoulder pain, thus necessitating additional treatment with more localized injection of anesthetic and methylprednisolone. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with this injection technique.

SUGGESTED READINGS

Taylor SA, Fabricant PD, Bansal M, et al. The anatomy and histology of the bicipital tunnel of the shoulder. *J Shoulder Elb Surg*. 2015;24(4):511–519.

Waldman SD. Bicipital tendinitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2017:114–117.

Waldman SD. The biceps tendon. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:91–92.

Waldman SD, Campbell RSD. Biceps tendinopathy. In: *Imaging of pain*. Philadelphia: Saunders; 2011:245–246.

Waldman SD. Bicipital tendinitis and other disorders of the biceps tendon. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:196–205.

Waldman SD. Ultrasound-guided injection technique for bicipital tendinitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:311–319.

Wu T, Song HX, Dong Y, et al. Ultrasound-guided versus blind subacromial—subdeltoid bursa injection in adults with shoulder pain: a systematic review and meta-analysis. *Semin Arthritis Rheum*. 2015;45(3):374–378.

29

Avascular Necrosis of the Glenohumeral Joint

ICD-10 CODE M87.029

THE CLINICAL SYNDROME

Avascular necrosis, which is also known as osteonecrosis, of the glenohumeral joint is an often missed diagnosis. Like the scaphoid, the glenohumeral joint is extremely susceptible to this disease because of the tenuous blood supply of the articular cartilage, which is only 1.0–1.2 mm thick at the center of the humeral head. This blood supply is easily disrupted, often leaving the proximal portion of the bone without nutrition leading to osteonecrosis (Fig. 29.1). Avascular necrosis of the glenohumeral joint is a disease of the fourth and fifth decades, except when it is secondary to collagen vascular disease. Avascular necrosis of the glenohumeral joint is more common in men. The disease is bilateral in 50%–55% of cases.

Factors predisposing to avascular necrosis of the glenohumeral joint are listed in Box 29.1. They include trauma to the joint; corticosteroid use; Cushing's disease; alcohol abuse; and connective tissue diseases, especially systemic lupus erythematosus, osteomyelitis, human immunodeficiency virus infection, organ transplantation, hemoglobinopathies including sickle cell disease, hyperlipidemia, gout, renal failure, pregnancy, and radiation therapy involving the femoral head.

The patient with avascular necrosis of the glenohumeral joint complains of pain over the affected glenohumeral joint or glenohumeral joints that may radiate into the proximal upper extremity and shoulder. The pain is deep and aching, and patients often complain of a catching sensation with range of motion of the affected glenohumeral joint or glenohumeral joints. Range of motion decreases as the disease progresses.

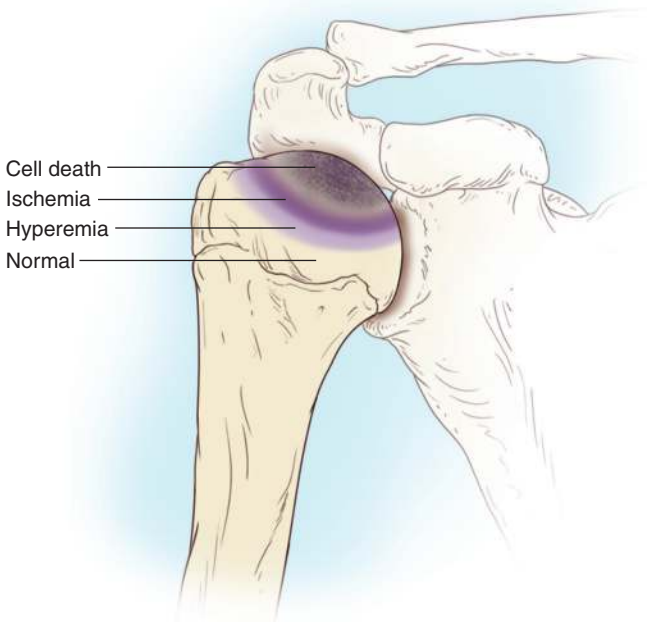

FIG 29.1 The pain of avascular necrosis of the glenohumeral joint is worsened by passive and active range of motion.

SIGNS AND SYMPTOMS

Physical examination of patients suffering from avascular necrosis of the glenohumeral joint reveals pain to deep palpation of the glenohumeral joint. The pain can be worsened by passive and active range of motion. A click or crepitus may also be appreciated by the examiner during range of motion of the glenohumeral joint. Range of motion is invariably decreased.

TESTING

Plain radiographs are indicated in all patients who present with avascular necrosis of the glenohumeral joint, to rule out underlying occult bony disease and to identify sclerosis and

BOX 29.1 Predisposing Factors for Avascular Necrosis of the Glenohumeral Joint

- Trauma to the glenohumeral joint
- Steroids
- Cushing's disease
- Alcohol abuse
- Connective tissue diseases, especially systemic lupus erythematosus
- Osteomyelitis
- Human immunodeficiency virus infection
- Organ transplantation
- Hemoglobinopathies, including sickle cell disease
- Hyperlipidemia
- Gout
- Renal failure
- Pregnancy
- Radiation therapy

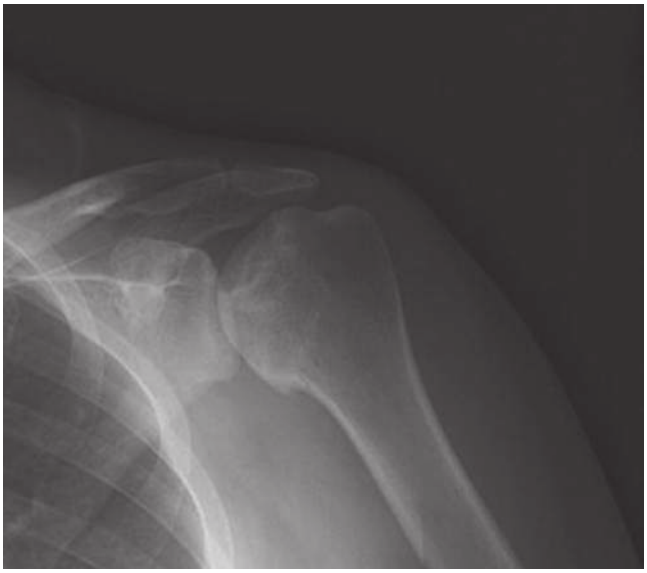

FIG 29.2 Preoperative radiographs from a 60-year-old patient with avascular necrosis. (From Hollis R, Yamaguchi K. Avascular necrosis of the shoulder. *Semin Arthroplasty*. 2008; 19(1):19–22.)

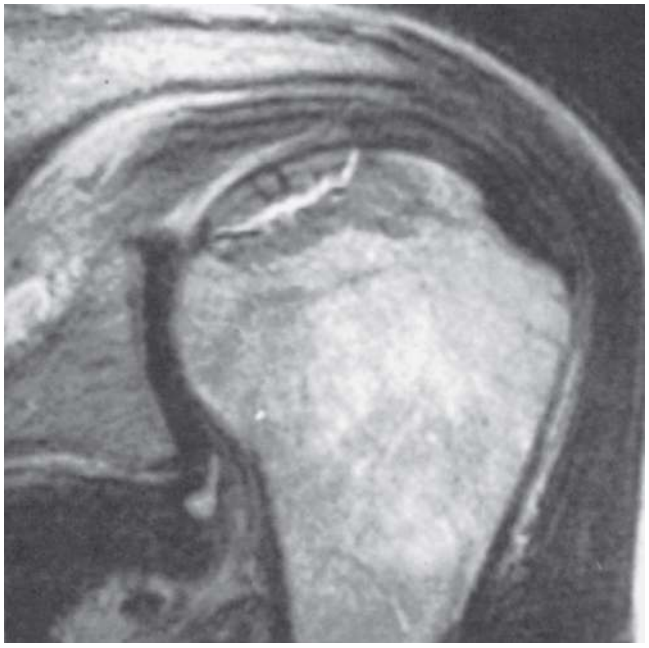

FIG 29.3 Osteonecrosis: humeral head. A coronal oblique T2-weighted (TR/TE, 3600/96) fast spin-echo magnetic resonance image shows the classic features of osteonecrosis, including a crescent sign. (Courtesy Eilenberg S. From Resnick D, Kang HS, Pretterklieber ML, eds. *Internal derangement of joints*. 2nd ed. Philadelphia: Saunders; 2007:404.)

fragmentation of the humoral head, although plain radiographs can be notoriously unreliable early in the course of the disease (Fig. 29.2). Based on the patient's clinical presentation, additional testing including complete blood cell count, uric acid, sedimentation rate, and antinuclear antibody testing may also be indicated. Magnetic resonance imaging of the glenohumeral joint is indicated in all patients suspected of suffering from avascular necrosis of the glenohumeral joint and when other causes of joint instability, infection, or tumor are suspected (Fig. 29.3). Administration of gadolinium followed by postcontrast imaging may help delineate the adequacy of blood supply; contrast enhancement of the glenohumeral joint is a good prognostic sign. Electromyography is indicated if coexistent cervical radiculopathy or brachial plexopathy is suspected. A very gentle intraarticular injection of the glenohumeral joint with small volumes of local anesthetic provides immediate reduction of the pain and helps demonstrate that the nidus of the patient's pain is in fact the glenohumeral joint. Ultimately, total joint replacement will be required in most patients suffering from avascular necrosis of the glenohumeral joint, although newer joint preservation techniques are becoming more popular in younger, more active patients, given the short life expectancy of total shoulder prostheses.

DIFFERENTIAL DIAGNOSIS

Coexistent arthritis and gout of the glenohumeral joint, joint bursitis, infection, and tendinitis may also coexist with avascular necrosis of the glenohumeral joints and exacerbate the pain and disability of the patient (Fig. 29.4).

FIG 29.4 Axial T1 sequence showing collections (peripherally enhancing) in subscapularis and biceps muscle, consistent with septic arthritis. (From Maranya SO, Mutiso VM. Bilateral glenohumeral septic arthritis secondary to mastitis with subsequent avascular necrosis: a case report. *Int J Surg Case Rep* 2021;88:106502.)

Labrum tears, ligament tears, bone cysts, bone contusions, and fractures may also mimic the pain of avascular necrosis of the glenohumeral joint, as can occult metastatic disease.

TREATMENT

Initial treatment of the pain and functional disability associated with avascular necrosis of the glenohumeral joint should include a combination of the nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and decreased weight bearing of the affected glenohumeral joint. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, an injection of a local anesthetic into the glenohumeral joint may be a reasonable next step to provide palliation of acute pain. Clinical reports suggest that the intraarticular injection of platelet-rich plasma and/or stem cells may provide some resolution of the osteonecrosis of the femoral head, so this new treatment may be worthy of consideration in patients suffering from avascular necrosis of the glenohumeral joint. Vigorous exercises should be avoided because they will exacerbate the patient's symptoms. Ultimately, surgical repair in the form of total joint arthroplasty is the treatment of choice.

COMPLICATIONS AND PITFALLS

Failure to treat significant avascular necrosis of the glenohumeral joint surgically usually results in continued pain and disability and, in most patients, leads to ongoing damage to the glenohumeral joint (see Fig. 29.2). Injection of the joint with local anesthetic is a relatively safe technique if the clinician is attentive to detail, uses small amounts of local anesthetic, and avoids high injection pressures that may further damage the joint. Another complication of this injection technique is infection. This complication should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after this injection technique, and patients should be warned of this possibility.

CLINICAL PEARLS

Avascular necrosis of the glenohumeral joint is a diagnosis that is often missed, leading to much unnecessary pain and disability. The clinician should include avascular necrosis of the glenohumeral joint in his or her differential diagnosis for all patients complaining of shoulder joint pain, especially if any of the predisposing factors listed in Box 29.1 are present. Coexistent arthritis, tendinitis, and gout may also contribute to the pain and may require additional treatment. The use of physical modalities, including local heat and cold, as well as decreased weight bearing, may provide symptomatic relief. Vigorous exercises should be avoided because they will exacerbate the patient's symptoms and may cause further damage to the shoulder. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

SUGGESTED READINGS

Alkhateeb JM, Arafah MA, Tashkandi M, Al Qahtani SM. Surgical treatment of humeral head avascular necrosis in patients with sickle cell disease: a systematic review. *JSES Int*. 2021;5(3):391–397. https://doi.org/10.1016/j.jseint.2021.01.011.

Hernigou J, Bastard C, Dubory A, Zilber S, Flouzat Lachaniette CH, Rouard H, Hernigou P. Cell therapy for posttraumatic shoulder osteonecrosis. *Morphologie*. 2021;105(349):162–169. https://doi.org/10.1016/j.morpho.2020.11.006.

Lancigu R, Rony L. Aseptic osteonecrosis of the shoulder: etiologies, diagnosis and medical management. *Morphologie*. 2021;105(349):148–154. https://doi.org/10.1016/j.morpho.2020.12.010.

Le Coz P, Herve A, Thomazeau H. Surgical treatments of atraumatic avascular necrosis of the shoulder. *Morphologie*. 2021;105(349):155–161. https://doi.org/10.1016/j.morpho.2021.02.007.

Maranya SO, Mutiso VM. Bilateral glenohumeral septic arthritis secondary to mastitis with subsequent avascular necrosis: a case report. *Int J Surg Case Rep*. 2021;88:106502. https://doi.org/10.1016/j.ijscr.2021.106502.

Ristow JJ, Ellison CM, Mickschl DJ, Kenneth CB, Haidet KC, Gray JR, Grindel SI. Outcomes of shoulder replacement in humeral head avascular necrosis. *J Shoulder Elb Surg*. 2019;28(1):9–14. https://doi.org/10.1016/j.jse.2018.06.031.

Adhesive Capsulitis of the Shoulder

ICD-10 CODE M75.00

THE CLINICAL SYNDROME

The shoulder joint is susceptible to the development of various conditions that cause damage or inflammation to the joint cartilage, ligaments, tendons, and soft tissues. Although most of these conditions can cause pain and functional disability, a favorable outcome is expected when they are properly managed. In some patients, however, increasing pain and inflammation lead to the development of edema and stiffness of the soft and connective tissues of the shoulder and result in the formation of fibrous adhesions that severely restrict the range of motion of the joint. If this condition is untreated, significant pain and functional disability, and ultimately a frozen shoulder can result. This condition tends to occur more commonly in females and in patients over the age of 40 unless there is a history of antecedent trauma.

Diseases that predispose the patient to the development of adhesive capsulitis can be divided into two general categories: (1) those within the shoulder and proximal upper extremity (e.g., rotator cuff tendinopathy, subdeltoid bursitis, and biceps tendon tendinopathy) and (2) diseases outside the shoulder region (e.g., stroke, diabetes, myocardial infarction, tuberculosis, Parkinson's disease, hypothyroidism [Box 30.1], and reflex sympathetic dystrophy).

Regardless of the underlying cause of adhesive capsulitis, failure of prompt diagnosis and treatment of this condition uniformly results in a poor clinical outcome.

BOX 30.1 Signs and Symptoms of Hypothyroidism

- Fatigue
- Increased sensitivity to cold
- Dry skin
- Thinning hair
- Weight gain
- Puffy face
- Hoarseness
- Constipation
- Bradycardia
- Depression
- Goiter
- Muscle weakness
- Myxedema

SIGNS AND SYMPTOMS

Most patients presenting with shoulder pain secondary to adhesive capsulitis complain of pain that is localized around the shoulder and upper arm. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical examination.

In addition to pain, patients suffering from adhesive capsulitis of the shoulder joint often experience a gradual reduction in functional ability because of decreasing shoulder range of motion that makes simple everyday tasks, such as combing one's hair, fastening a brassiere, or reaching overhead, quite difficult (Fig. 30.1). With continued disuse, muscle wasting may occur, and a frozen shoulder may develop (Fig. 30.2; Box 30.2). Sleep disturbance is quite common in patients suffering from adhesive capsulitis and may further exacerbate the patient's pain.

TESTING

Plain radiographs are indicated in all patients who are suspected of suffering from adhesive capsulitis, to rule out other causes of shoulder pain. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging of the shoulder is indicated to identify treatable shoulder abnormalities (e.g., rotator cuff tears), as well as to define the extent of adhesive capsulitis (Figs. 30.2–30.4). Radionuclide bone scanning is indicated if metastatic disease or primary tumor involving the shoulder is a possibility. Diseases outside the shoulder region may cause shoulder pain (e.g., pericarditis, hypothyroidism, and reflex sympathetic dystrophy), and specific testing to rule out these disorders is mandatory if successful diagnosis and treatment are to be expected.

DIFFERENTIAL DIAGNOSIS

Osteoarthritis of the joint is the most common form of arthritis that results in shoulder pain; however, rheumatoid arthritis, posttraumatic arthritis, and rotator cuff arthropathy are also common causes of shoulder pain. Less common causes of arthritis-induced shoulder pain include collagen

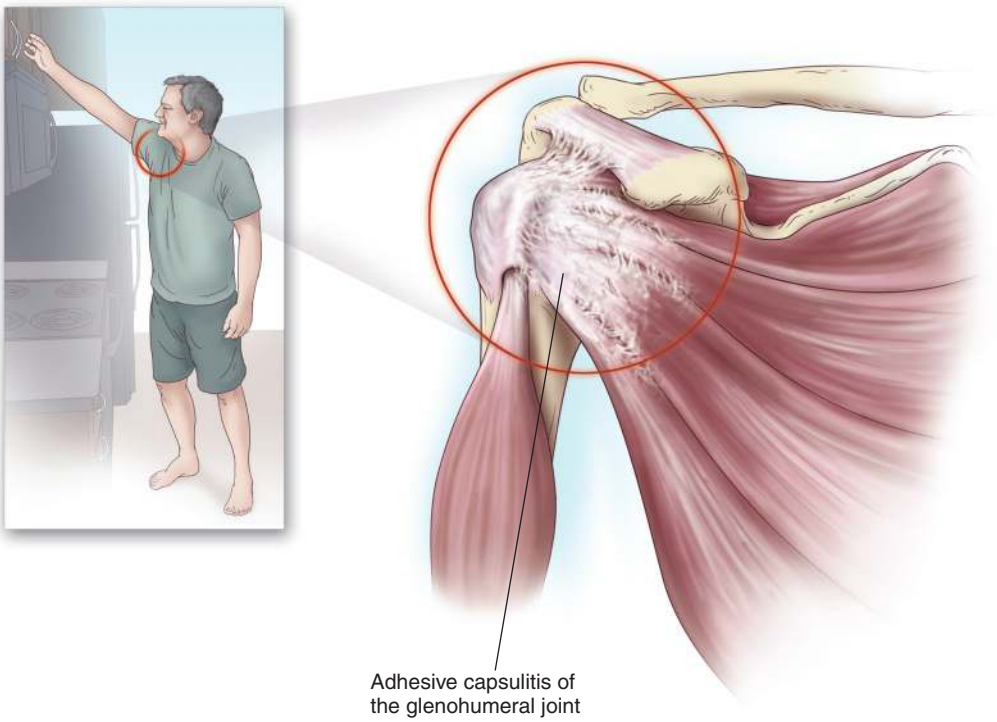

FIG 30.1 Patients suffering from adhesive capsulitis of the shoulder joint often experience a gradual reduction in functional ability because of decreasing shoulder range of motion that makes simple everyday tasks quite difficult.

FIG 30.2 Superior labral tear from anterior to posterior lesion demonstrated on magnetic resonance imaging of the shoulder. Coronal oblique fat-suppressed T1-weighted fast spin-echo direct magnetic resonance arthrogram image demonstrating a detached tear of the superior glenoid labrum (*arrow*) extending into the long head of biceps tendon (*arrowhead*). (From Lee JC, Guy S, Connell D, et al. MRI of the rotator interval of the shoulder. *Clin Radiol*. 2007;62(5):416–423.)

FIG 30.3 Adhesive capsulitis demonstrated on magnetic resonance imaging (MRI). Sagittal oblique T1-weighted fast spin-echo MRI demonstrating enhancing soft tissue (*arrowheads*) surrounding the coracohumeral ligament (*straight arrow*) and extending toward the intraarticular portion of the long head of the biceps tendon (*curved arrow*). (From Lee JC, Guy S, Connell D, et al. MRI of the rotator interval of the shoulder. *Clin Radiol*. 2007;62(5):416–423.)

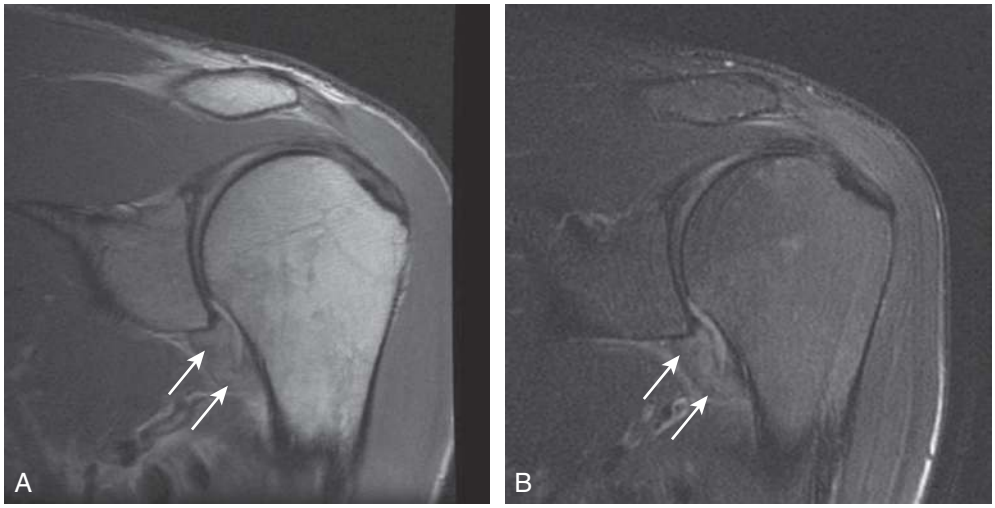

FIG 30.4 A, Coronal proton density and T2-weighted with fat suppression (FST2W) image. B, Magnetic resonance (MR) image in a different patient with adhesive capsulitis. There are thickening and high signal intensity (SI) within the inferior glenohumeral ligament and the capsule of the axillary pouch (*white arrows*). Only a small joint effusion is present. (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Elsevier; 2011: Fig. 94.2.)

BOX 30.2 Stages of Adhesive Capsulitis

- Stage 1: Aching stage
- Stage 2: Freezing stage
- Stage 3: Frozen stage
- Stage 4: Thawing stage

vascular diseases, infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is diagnosed with culture and treated with antibiotics, rather than with injection therapy. Collagen vascular diseases generally manifest as polyarthropathy rather than as monarthropathy limited to the shoulder joint; however, shoulder pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described here.

TREATMENT

Initial treatment of the pain and functional disability associated with adhesive capsulitis of the shoulder includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial as may be the use of ultrasound therapy. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step (Fig. 30.5).

Intraarticular injection of the shoulder is performed by placing the patient in the supine position and preparing the skin overlying the shoulder, subacromial region, and joint space with antiseptic solution. A sterile syringe containing 2 mL of 0.30% preservative-free bupivacaine and 40 mg

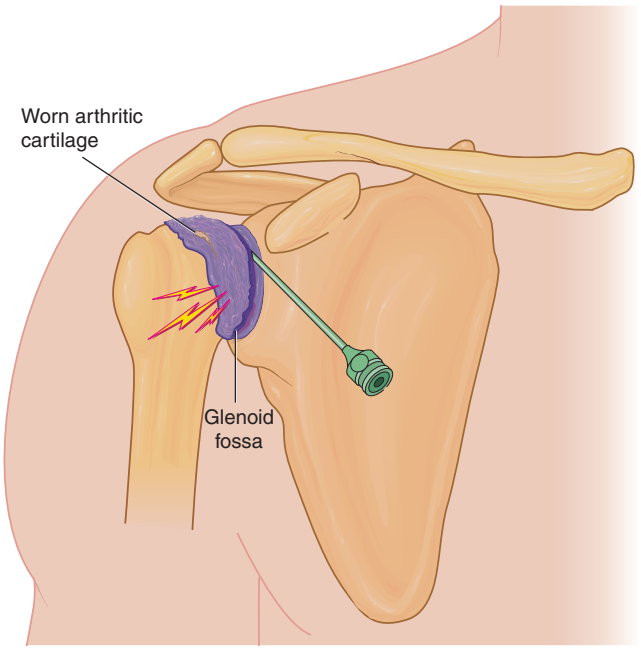

FIG 30.5 Injection technique for adhesive capsulitis of the shoulder. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:58.)

methylprednisolone is attached to a 1½-inch, 30-gauge needle using strict aseptic technique. The midpoint of the acromion is identified; at a point approximately 1 inch below the midpoint, the shoulder joint space is identified. The needle is carefully advanced through the skin and subcutaneous tissues, through the joint capsule, and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly and slightly more medially. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection

should be felt; if resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Studies suggest that the use of ultrasound guidance may improve the accuracy of needle placement for those less familiar with this technique (Fig. 30.6). This technique can be used for lysis of adhesions via distention arthrography, manipulation of the shoulder under local anesthesia, and a two-needle technique can be used to barbotage and remove calcifications (Figs. 30.7 and 30.8). Rarely, surgery will be required to remove adhesions.

Physical modalities, including local heat, ultrasound therapy, and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for shoulder pain. Transcutaneous nerve stimulation and acupuncture may also provide alternative treatment options. The use of extracorporeal shock wave therapy may be useful in recalcitrant cases. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Treatment of any underlying reflex sympathetic dystrophy of the affected extremity with stellate ganglion blocks should be introduced early in course of the disease.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of intraarticular injection of the shoulder is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 30% of patients complain of a transient increase in pain after intraarticular injection of the shoulder joint, and patients should be warned of this possibility.

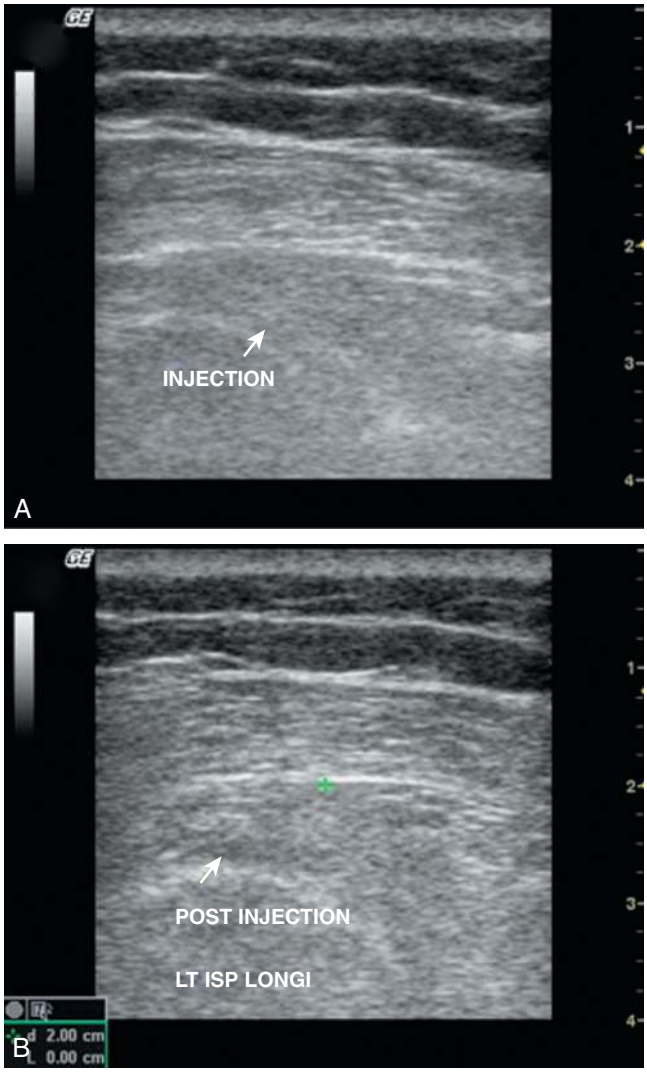

FIG 30.6 Ultrasound study showing the needle tip in the articular capsule **(A)** and the capsular distention **(B)**. (From Lee H-J, Lim K-B, Kim D-Y, et al. Randomized controlled trial for efficacy of intraarticular injection for adhesive capsulitis: ultrasonography-guided versus blind technique. *Arch Phys Med Rehabil.* 2009;90(12):1997–2002.)

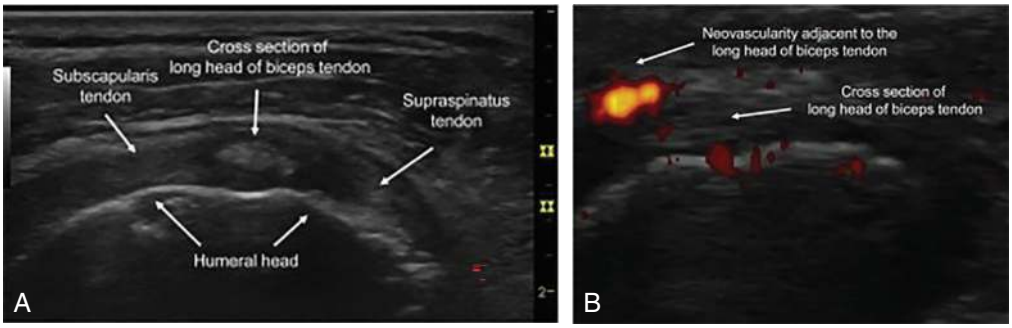

FIG 30.7 Ultrasound of a patient with frozen shoulder. **A,** Gray-scale ultrasound scan of the rotator interval of the left shoulder. **B,** Neovascularity adjacent to the long head of biceps tendon. (From Lewis J, Frozen shoulder contracture syndrome—aetiology, diagnosis and management. *Man Ther.* 2015;20(1):2–9.)

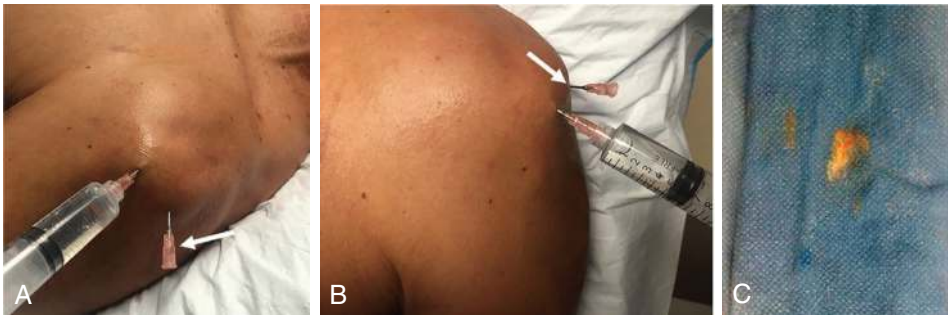

FIG 30.8 Ultrasound (US)-guided two-needle technique for calcific barbotage procedure. **A, B,** Anterior and lateral views of needle positioning for two-needle technique for calcific barbotage procedure. Note the dependent position of the open needle (*arrow*). **C,** Irrigated calcium hydroxyapatite on a sterile blue towel. (From Pourcho AM, Colio SW, Hall MM. Ultrasound-guided interventional procedures about the shoulder: anatomy, indications, and techniques. *Phys Med Rehabil Clin N Am*. 2016;27(3):555–572.)

CLINICAL PEARLS

Adhesive capsulitis of the shoulder is a common complaint encountered in clinical practice. The pathophysiology behind the proliferation of fibroblasts and deposition of dense collagen matrix appears to be in part secondary to the accumulation of advanced glycation end products that result in the cross-linking and stabilization of collage. It must be distinguished from other causes of shoulder pain, including rotator cuff tears. Diseases outside the shoulder region may be responsible for the development of adhesive capsulitis and must be diagnosed and treated if a successful clinical outcome is to be expected. Intraarticular injection is extremely effective in the treatment of pain secondary to arthritis of the shoulder joint. Coexistent bursitis and tendinitis may contribute to shoulder pain and necessitate additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with this injection technique.

SUGGESTED READINGS

Arkkila PE, Kantola IM, Viikari JS, Ronnemaa T. Shoulder capsulitis in type I and II diabetic patients: association with diabetic complications and related diseases. *Ann Rheum Dis*. 1996;55(12):907–914.

Cain EL, Stephen MK, Wilk KE. Adhesive capsulitis of the shoulder. In: Anderws, Wilk and Reynolds *The athlete's shoulder*. 2nd ed. Philadelphia: Elsevier; 2009:293–321.

Dalton SE. Clinical examination of the painful shoulder. *Baillière Clin Rheumatol*. 1989;3(3):453–474.

Davies AM. Imaging the painful shoulder. *Curr Orthop*. 1992;6(1): 32–38.

Lee H-J, Lim K-B, Kim D-Y, Lee K-T. Randomized controlled trial for efficacy of intra-articular injection for adhesive capsulitis: ultrasonography-guided versus blind technique. *Arch Phys Med Rehabil*. 2009;90(12):1997–2002.

Monach PA. Shoulder pain. In: Mushlin and Green *Decision making in medicine*. 3rd ed. Philadelphia: Mosby Elsevier; 2010:532–523.

Reutter TRC. Shoulder pain. In: Mushlin and Green *Decision making in pain management*. 2nd ed. Philadelphia: Mosby Elsevier; 2006:160–162.

Waldman SD. Injection technique for frozen shoulder. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Saunders; 2017:155–159.

Waldman SD, Campbell RSD. Adhesive capsulitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:239–240.

31

Biceps Tendon Tear

ICD-10 CODE M66.829

THE CLINICAL SYNDROME

The tendons of the long and short heads of the biceps are particularly susceptible to the development of tendinitis. Biceps tendon tear is usually caused at least partially by impingement on the tendons of the biceps at the coracoacromial arch. The onset of pain and functional disability associated with biceps tendon tear is generally acute, occurring after overuse or misuse of the shoulder joint, such as trying to start a recalcitrant lawn mower, practicing an overhead tennis serve, or performing an overaggressive follow-through when driving golf balls (Fig. 31.1). More common in men, proximal rupture of the tendon of the long head of the biceps tendon accounts for more than 97% of biceps tendon ruptures; ruptures of the distal portion of the biceps tendon occur less than 3% of the time. Rupture of the long head of the biceps tendon generally occurs in the fourth to sixth decades, but it can occur in younger age groups involved in high-risk activities such as snowboarding.

The biceps muscle and tendons are intimately involved in shoulder and upper extremity function and are susceptible to trauma and to wear and tear (Fig. 31.2). If the damage is severe enough, the tendon of the long head of the biceps can rupture, leaving the patient with a telltale "Popeye" biceps (named after the cartoon character) (Fig. 31.3). This deformity can be accentuated by having the patient perform Ludington's maneuver: placing his or her hands behind the head and flexing the biceps muscle (Fig. 31.4).

SIGNS AND SYMPTOMS

In most patients, the pain of biceps tendon tear occurs acutely and is accompanied by a pop or snapping sound. The pain is constant and severe and is localized in the anterior shoulder

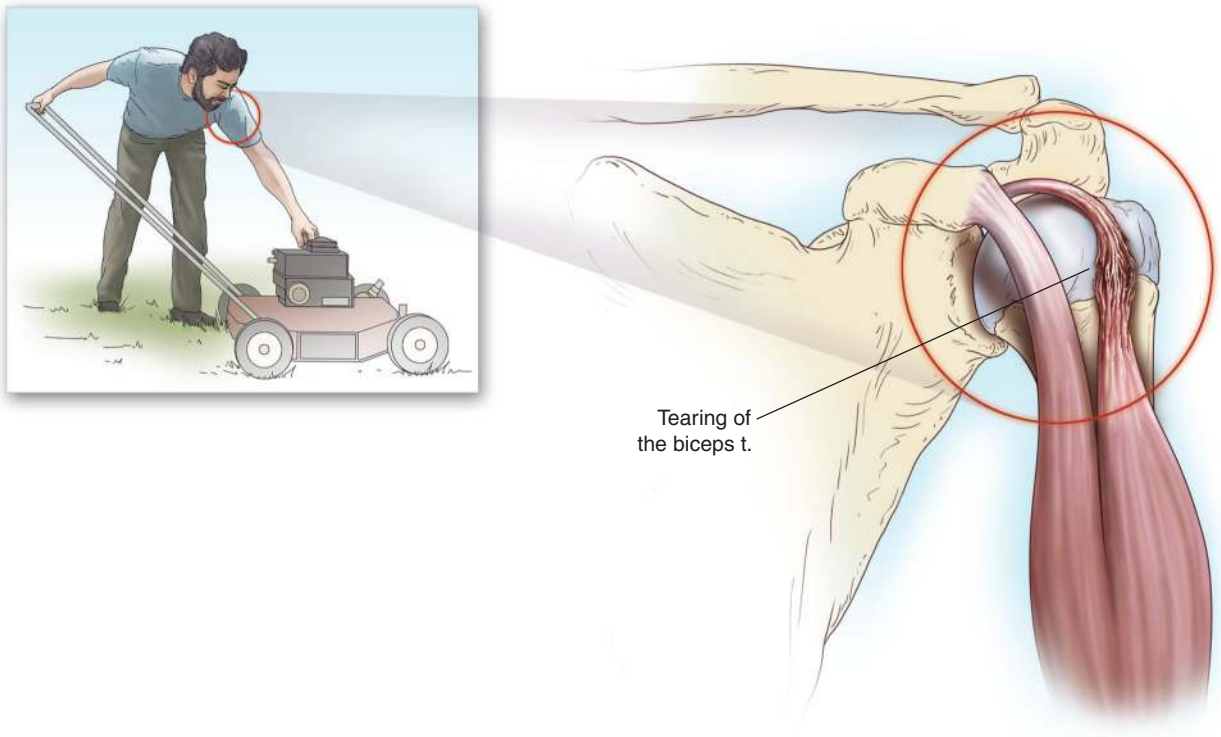

FIG 31.1 The onset of pain and functional disability associated with biceps tendon tear is generally acute, occurring after overuse or misuse of the shoulder joint, such as trying to start a recalcitrant lawn mower.

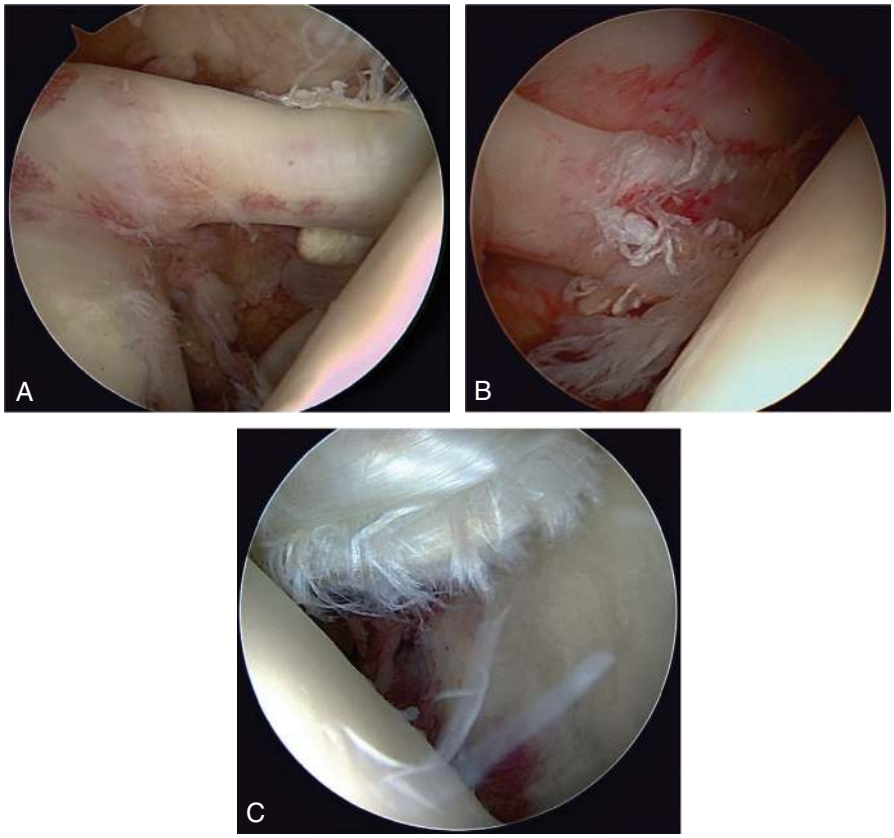

FIG 31.2 Arthroscopic images of the long head of the biceps tendon as seen from the posterior portal showing synovitis **(A),** partial tear **(B),** and delamination **(C)** of the long head of the biceps, concomitant with rotator cuff tear. (From Virk MS, Cole BJ. Proximal biceps tendon and rotator cuff tears. *Clin Sports Med*. 2016;35(1):153–161.)

FIG 31.3 Typical Popeye deformity seen with rupture of the long head of the biceps tendon. (From Virk MS, Cole BJ. Proximal biceps tendon and rotator cuff tears. *Clin Sports Med*. 2016;35(1):153–161.)

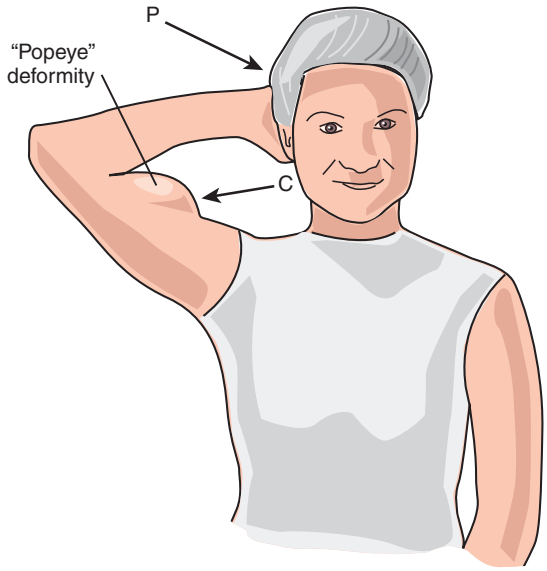

FIG 31.4 The Ludington's maneuver for ruptured long tendon of the biceps. *C*, Contraction of biceps; *P*, pressure applied. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:78.)

over the bicipital groove. Ecchymosis may be present if the trauma is acute and severe. Significant sleep disturbance is often reported. Patients with a partial tendon tear and significant tendinitis may attempt to splint the affected shoulder by internal rotation of the humerus, which moves the biceps tendon from beneath the coracoacromial arch. Patients with biceps tendon tear have a positive Ludington's test result, as described earlier. Bursitis and tendinitis often accompany biceps tendon tear. Occasionally, patients with acute tear of the long tendon of the biceps may experience only vague discomfort and seek medical attention only because of the cosmetic abnormality of retracted biceps tendon and muscle. Occasionally, without treatment, frozen shoulder may develop.

TESTING

Plain radiographs are indicated for all patients who present with shoulder pain. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging of the shoulder is indicated if tendinopathy or tear of the biceps tendon is suspected (Figs. 31.5 and 31.6). Ultrasound imaging may also help further delineate the pathology responsible for the patient's pain and functional disability (Fig. 31.7). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Biceps tendon tear is usually a straightforward clinical diagnosis. However, coexisting bursitis or tendinitis of the shoulder from overuse or misuse may confuse the diagnosis. Occasionally, partial rotator cuff tear can be mistaken for biceps tendon tear. In some clinical situations, consideration

FIG 31.5 Sagittal T2-weighted fast spin-echo magnetic resonance image demonstrates a swollen, hyperintense, but intact long head of biceps tendon within the rotator interval, indicative of biceps tendinopathy *(arrow)*.

FIG 31.6 Ruptured biceps tendon. Axial proton density **(A)** and axial T2-weighted **(B)** magnetic resonance images of the elbow demonstrate torn biceps tendon with tendon swelling, intratendinous abnormal signal, and peritendinous edema *(arrows)*. Sagittal fat-saturated proton density image **(C)** shows retraction of the blunt torn tendon into the soft tissues of the antecubital region with adjacent edema. (From DeLee JC, Drez DD, Miller M, eds. *Orthopaedic sports medicine: principles and practice*. 3rd ed. Philadelphia: Saunders; 2010:570.)

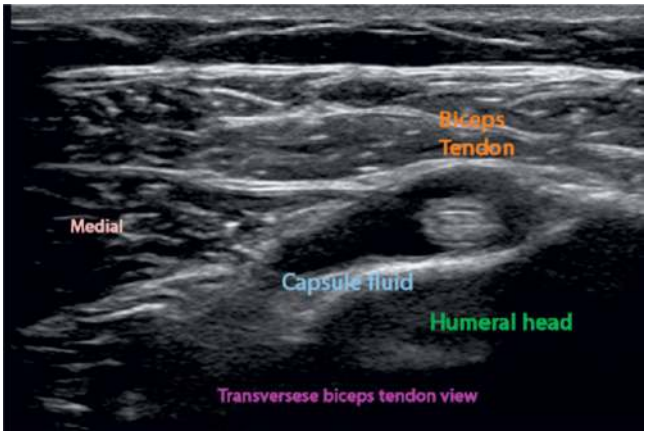

FIG 31.7 Biceps sheath effusion. Transverse ultrasound image demonstrating bicipital tendinitis with effusion.

should be given to primary or secondary tumors involving the shoulder, superior sulcus of the lung, or proximal humerus. The pain of acute herpes zoster, which occurs before eruption of a vesicular rash, can also mimic biceps tendon tear.

TREATMENT

Initial treatment of the pain and functional disability associated with biceps tendon tear includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities and who appear to have significant local pain in the region of the bicipital groove, injection with local anesthetic and steroid is a reasonable next step.

Injection for biceps tendon tear is carried out by placing the patient in the supine position with the arm externally rotated approximately 45 degrees. The coracoid process is identified anteriorly. Just lateral to the coracoid process is the lesser tuberosity, which can be more easily palpated as the arm is passively rotated. The point overlying the tuberosity is marked with a sterile marker. The skin overlying the anterior shoulder is prepared with antiseptic solution. A sterile syringe containing 1 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The previously marked point is palpated, and the insertion of the biceps tendon is reidentified with the gloved finger. The needle is carefully advanced at this point through the skin, subcutaneous tissues, and underlying tendon until it impinges on bone. The needle is then withdrawn 1–2 mm out of the periosteum of the humerus, and the contents of the syringe are gently injected. Slight resistance to injection should be felt. If no resistance is encountered, either the needle tip is in the joint space itself or the tendon is ruptured. If resistance is significant, the needle tip is probably in the substance of a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Occasionally, surgical repair of the tendon is undertaken if the patient is experiencing significant functional disability or is unhappy with the cosmetic defect resulting from the retracted tendon and muscle.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of this injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Trauma to the biceps tendon from the injection itself is also a possibility. Tendons that are highly inflamed or previously damaged are subject to rupture if they are injected directly. This complication can often be avoided if the clinician uses a gentle technique and stops injecting immediately if significant resistance is encountered. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The musculotendinous unit of the shoulder joint is susceptible to the development of tendinitis for several reasons. First, the joint is subjected to many different repetitive motions. Second, the space in which the musculotendinous unit functions is restricted by the coracoacromial arch, thus making impingement likely with extreme movements of the joint. Third, the blood supply to the musculotendinous unit is poor, so the healing of microtrauma is difficult. All these factors can contribute to tendinitis. Calcium deposition around the tendon may occur if the inflammation continues and may complicate subsequent treatment.

The injection technique described is extremely effective in the treatment of pain secondary to biceps tendon tear. Coexistent bursitis and arthritis may contribute to shoulder pain, necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with this injection technique.

SUGGESTED READINGS

Caekebeke P, Schenkels E, Bell SN, van Riet. R. Distal biceps provocation test. *J Hand Surg Am*. 2021;46:710.e1–710.e4. https://doi.org/10.1016/j.jhsa.2020.12.012.

MacInnes SJ, Crawford LA, Shahane SA. Disorders of the biceps and triceps tendons at the elbow. *Orthop Trauma*. 2016;30(4):346–354.

McFarland EG, Borade A. Examination of the biceps tendon. *Clin Sports Med*. 2016;35(1):29–45.

Nicolay RW, Cort DL, Selley RS, et al. Partial rupture of the distal biceps brachii tendon: a magnetic resonance imaging analysis. *J Shoulder Elb Surg*. 2020;29(9):1859–1868. https://doi.org/10.1016/j.jse.2020.04.021.

Pallante GD, O'Driscoll SW. Return of an intact hook test result: clinical assessment of biceps tendon integrity after surgical repair. *Orthop J Sports Med*. 2019;7:1–6. https://doi.org/10.1177/2325967119827311.

Pitsilos C, Gigis I, Chitas K, et al. Systematic review of distal biceps tendon rupture in athletes: treatment and rehabilitation. *J Shoulder Elb Surg*. 2022;31:1763–1772. https://doi.org/10.1016/j.jse.2022.02.027.

Shim SS, Strauch RJ. A novel clinical test for partial tears of the distal biceps brachii tendon: the TILT sign. *Clin Anat*. 2018;31:301–303.

Thomas JR, Lawton JN. Biceps and triceps ruptures in athletes. *Hand Clin*. 2017;33(1):35–46.

Vestermark GL, Bryce AVD, Connor PM, et al. The prevalence of rotator cuff pathology in the setting of acute proximal biceps tendon rupture. *J Shoulder Elb Surg*. 2018;27(7):1258–1262. https://doi.org/10.1016/j.jse.2018.01.006.

Virk MS, Cole BJ. Proximal biceps tendon and rotator cuff tears. *Clin Sports Med*. 2016;35(1):153–161.

Waldman SD. The biceps tendon. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:91–92.

Waldman SD. Bicipital tendinitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:114–117.

Waldman SD, Campbell RSD. Biceps tendinopathy. In: *Imaging of pain*. Philadelphia: Saunders; 2011:245–246.

Supraspinatus Syndrome

ICD-10 CODE M79.74

THE CLINICAL SYNDROME

The supraspinatus muscle is susceptible to the development of myofascial pain syndrome. Flexion-extension and lateral motion stretch injuries to the neck, shoulder, and upper back or repeated microtrauma secondary to activities that require working overhead or repeatedly reaching across one's body, such as painting ceilings, assembly-line work, or even watching television while reclining on a couch, may result in the development of myofascial pain in the supraspinatus muscle.

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the supraspinatus muscle often have referred pain in the shoulder that radiates down into the upper extremity.

The trigger point is pathognomonic of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but referred pain as well. In addition, one often sees an involuntary withdrawal of the stimulated muscle, called "a jump sign," which is characteristic of myofascial pain syndrome. In patients with supraspinatus syndrome, the trigger point overlies the superior border of the scapula (Fig. 32.1).

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. In spite of this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, but trigger points are thought to result from microtrauma to the affected muscle. This trauma may occur from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to the development of myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The supraspinatus muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

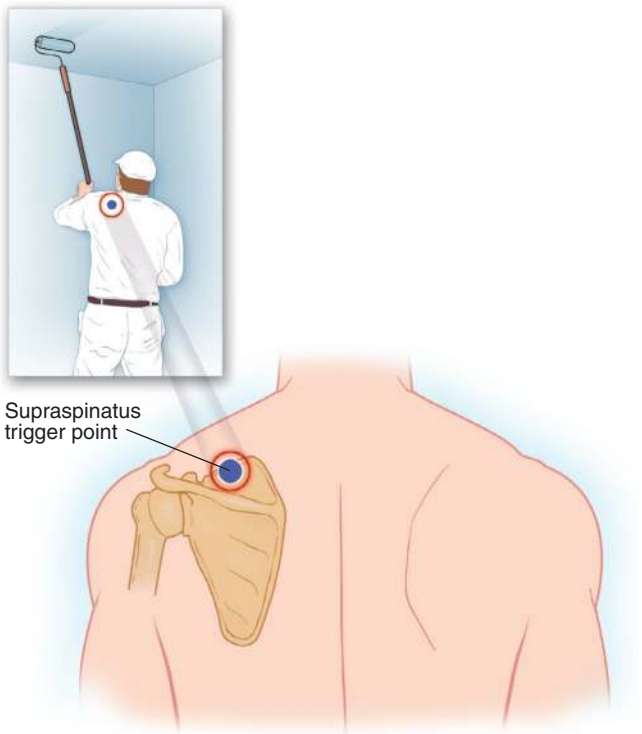

FIG 32.1 In patients with supraspinatus syndrome, the trigger point overlies the superior border of the scapula.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

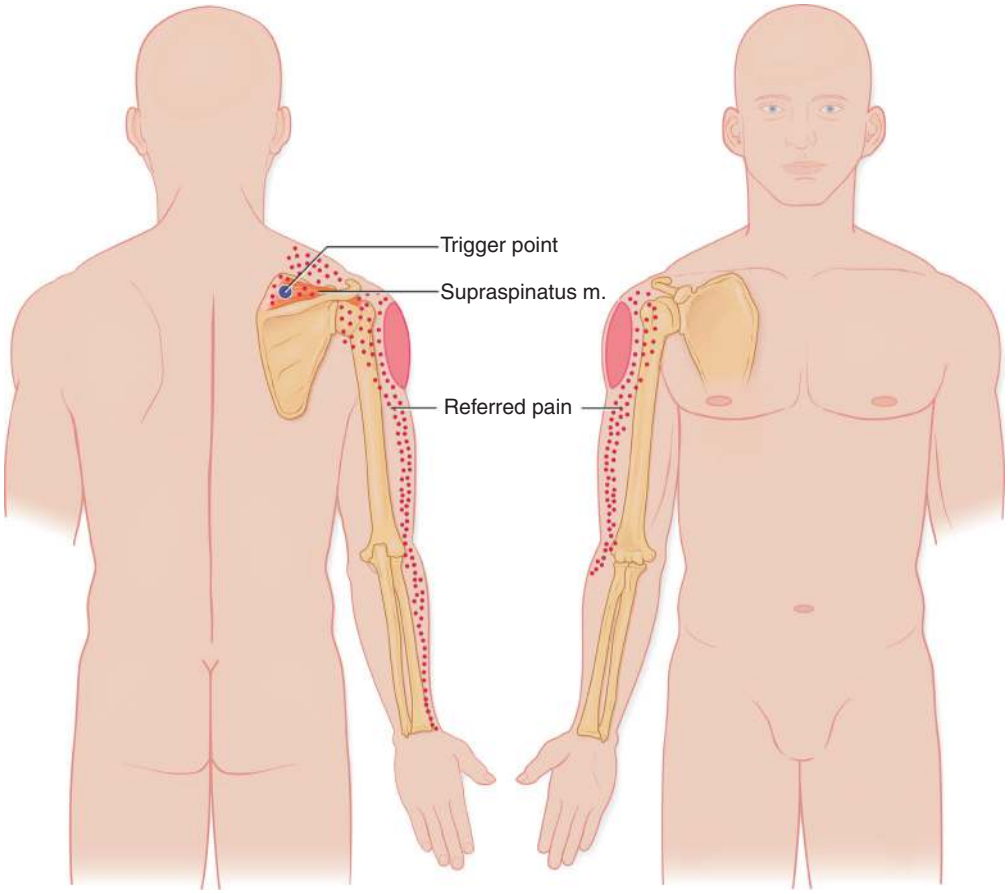

FIG 32.2 Pain on range of motion and pain referred to the shoulder and upper extremities in a nondermatomal pattern are characteristic of supraspinatus syndrome. (From Waldman SD. Deltoid syndrome. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:83.)

SIGNS AND SYMPTOMS

The sine qua non of supraspinatus syndrome is the identification of a myofascial trigger point—a local point of exquisite tenderness—overlying the superior border of the scapula. Mechanical stimulation of the trigger point by palpation or stretching produces intense local pain as well as referred pain, and the jump sign may be present. Other findings characteristic of supraspinatus syndrome are pain on range of motion of the affected scapula and shoulder and pain referred to the shoulder and upper extremities in a nondermatomal pattern (Fig. 32.2).

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with supraspinatus syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing of patients suffering from supraspinatus syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective testing, the clinician must use electrodiagnostic and radiographic means to rule out other disease processes that may mimic supraspinatus syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of supraspinatus syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of having supraspinatus syndrome. The clinician must rule out other coexisting disease processes that may mimic supraspinatus syndrome, including primary inflammatory muscle disease, multiple sclerosis, and collagen vascular disease. Electrodiagnostic and radiographic testing can help identify coexisting disorders such as shoulder bursitis, tendinitis, and rotator cuff tears. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with supraspinatus syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood,

an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injection is extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the trauma to underlying structures. Special care must be taken to avoid pneumothorax when injecting trigger points in proximity to the underlying pleural space.

CLINICAL PEARLS

Although supraspinatus syndrome is a common disorder, it is often misdiagnosed. Therefore, in patients suspected of suffering from supraspinatus syndrome, a careful evaluation is mandatory to identify any underlying disease processes. Supraspinatus syndrome often coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208(15):87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego, CA: Academic Press; 2017:e158–e167.

Gota CE. Fibromyalgia: recognition and management in the primary care office. *Rheum Dis Clin North Am*. 2022;48(2): 467–478. https://doi.org/10.1016/j.rdc.2022.02.006.

Karayanni H, Dror AA, Oren DA, Sela E, Granot I, Srouji S. Exacerbation of chronic myofascial pain during COVID-19. *Adv Oral Maxillofac Surg*. 2021;1:100019. https://doi.org/10.1016/j.adoms.2021.100019.

Monach PA. Shoulder pain. In: Mushlin SB, Greene HL, eds. *Decision making in medicine*. 3rd ed. Philadelphia: Mosby; 2010:522–523.

Ughreja RA, Venkatesan P, Gopalakrishna DB, Preet Singh Y. Effectiveness of myofascial release on pain, sleep, and quality of life in patients with fibromyalgia syndrome: a systematic review. *Complement Ther Clin Pract*. 2021;45:101477. https://doi.org/10.1016/j.ctcp.2021.101477.

Waldman SD. Supraspinatus syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2017:78–83.

33

Rotator Cuff Tear

ICD-10 CODES S43.429A, M75.10

THE CLINICAL SYNDROME

Rotator cuff tears are a common cause of shoulder pain and dysfunction. A rotator cuff tear frequently occurs after seemingly minor trauma to the musculotendinous unit of the shoulder. However, in most cases, the pathologic process responsible for the tear has been a long time in the making and is the result of ongoing tendinitis. The rotator cuff is made up of the subscapularis, supraspinatus, infraspinatus, and teres minor muscles and the associated tendons (Fig. 33.1). The function of the rotator cuff is to rotate the arm and help provide shoulder joint stability along with the other muscles, tendons, and ligaments of the shoulder.

The supraspinatus and infraspinatus muscle tendons are particularly susceptible to the development of tendinitis, for several reasons. First, the joint is subjected to many different repetitive motions. Second, the space in which the musculotendinous unit functions is restricted by the coracoacromial arch, thus making impingement likely with extreme joint movements. Third, the blood supply to the musculotendinous unit is poor, and this makes healing of microtrauma difficult. All these factors can contribute to tendinitis of one or more tendons of the shoulder joint. Calcium deposition around the tendon may occur if the inflammation continues and complicates subsequent treatment. Bursitis often accompanies rotator cuff tears and may require specific treatment.

In addition to pain, patients suffering from rotator cuff tear often experience a gradual reduction in functional ability because of decreasing shoulder range of motion that makes simple everyday tasks such as combing one's hair, fastening a brassiere, or reaching overhead quite difficult. With continued disuse, muscle wasting may occur, and a frozen shoulder may develop.

SIGNS AND SYMPTOMS

Patients presenting with rotator cuff tear frequently complain that they cannot raise the affected arm above the level of the shoulder without using the other arm to lift it (Fig. 33.2). On physical examination, weakness on external rotation is noted if the infraspinatus is involved, and weakness on abduction above the level of the shoulder is evident if the supraspinatus is involved. Tenderness to palpation in the subacromial region is often present. Patients with partial rotator cuff tears lose the ability to reach overhead smoothly. Patients with complete tears exhibit anterior migration of the humeral head, as well as a complete inability to reach above the level of the shoulder. A positive drop arm test—the inability to hold the arm abducted at the level of the shoulder after the supported arm is released—is often seen with complete tears of the rotator cuff (Fig. 33.3). The result of Moseley's test for rotator cuff tear is also positive; this test is performed by having the patient actively abduct the arm to 80 degrees and then adding gentle resistance, which forces the arm to drop if complete rotator cuff tear is present. Passive range of motion of the shoulder is normal, but active range of motion is limited.

The pain of rotator cuff tear is constant and severe and is made worse with abduction and external rotation of the shoulder. Significant sleep disturbance is often reported. Patients may attempt to splint the inflamed subscapularis tendon by limiting medial rotation of the humerus.

TESTING

Plain radiographs are indicated in all patients who present with shoulder pain (Fig. 33.4). Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) and ultrasound imaging of the shoulder are indicated if rotator cuff tendinopathy and or tear is suspected (Figs. 33.5–33.8).

DIFFERENTIAL DIAGNOSIS

Because rotator cuff tears may occur after seemingly minor trauma, the diagnosis is often delayed. The tear may be either partial or complete, further confusing the diagnosis, although a careful physical examination can distinguish between the two. Tendinitis of the musculotendinous unit of the shoulder frequently coexists with bursitis of the associated bursae of the shoulder joint and creates additional pain and functional disability. This pain can cause the patient to splint the shoulder group, resulting in abnormal movement of the shoulder that puts additional stress on the rotator cuff and can lead to further trauma. With rotator cuff tears, passive range of motion is normal, but active range of motion is limited; with frozen shoulder, both passive range of motion and active range of motion are limited. Rotator

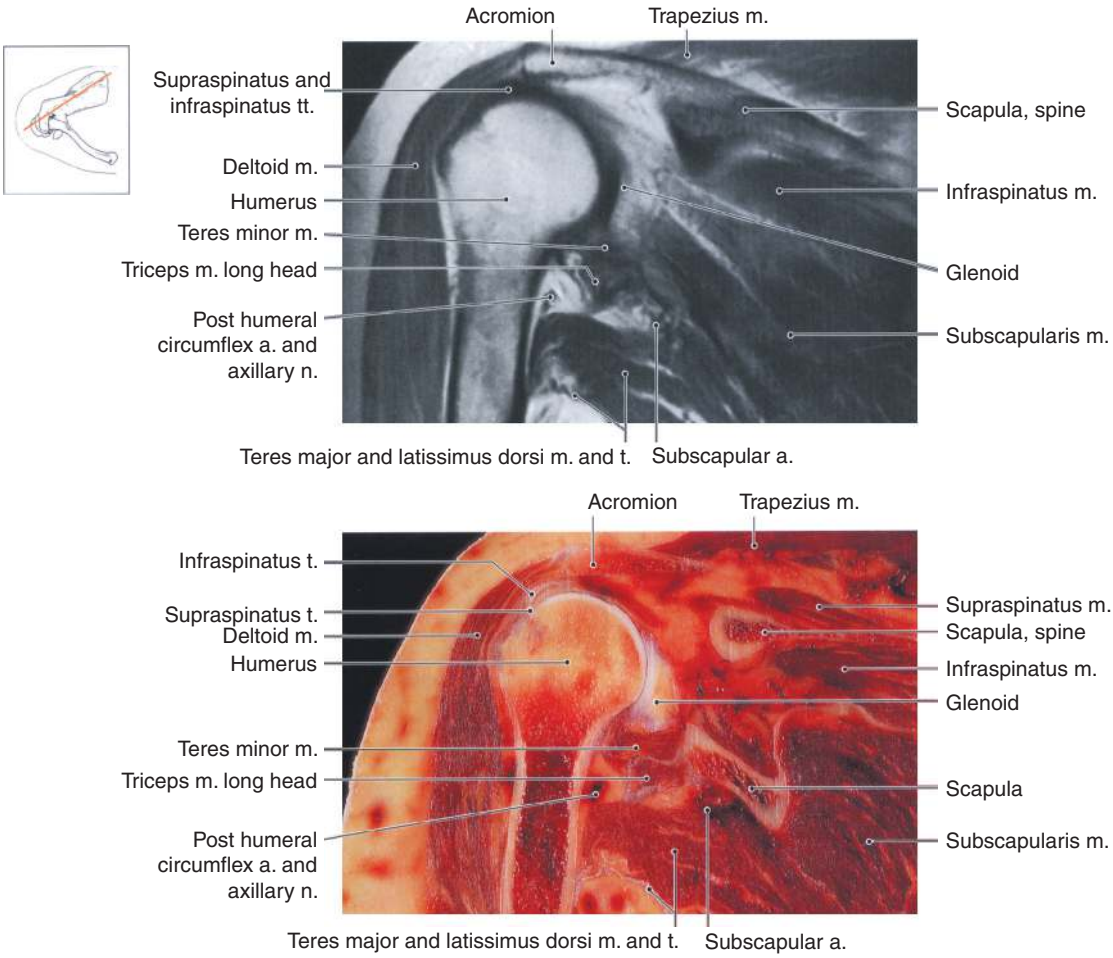

FIG 33.1 Muscles and tendons of the rotator cuff. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas*. 2nd ed. Philadelphia: Saunders; 2002:5.)

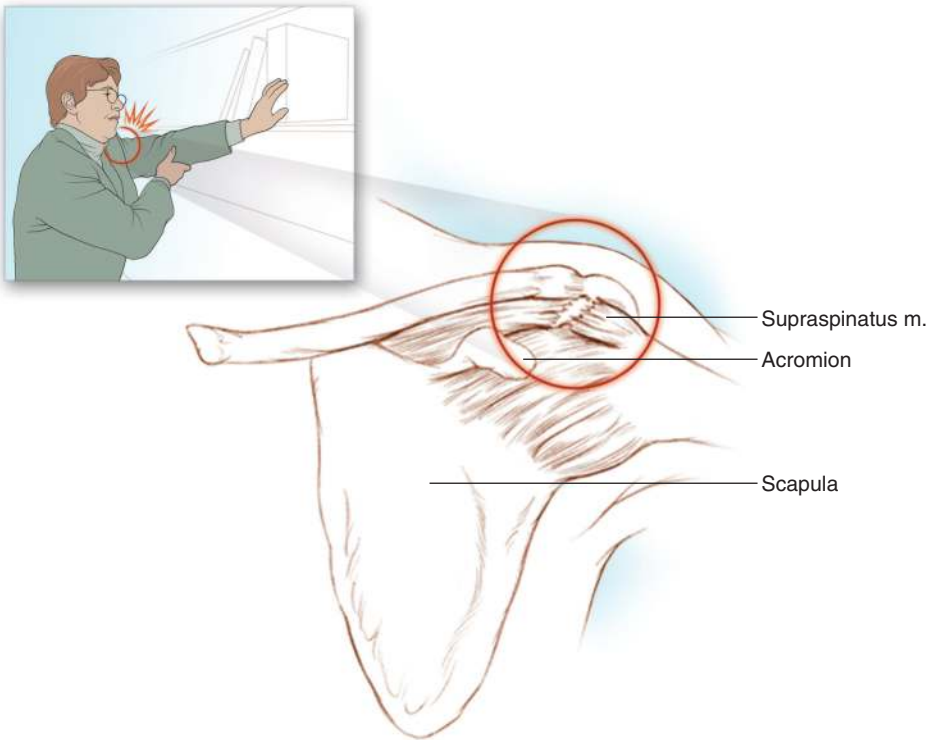

FIG 33.2 Inability to elevate the arm above the level of the shoulder is the hallmark of rotator cuff dysfunction.

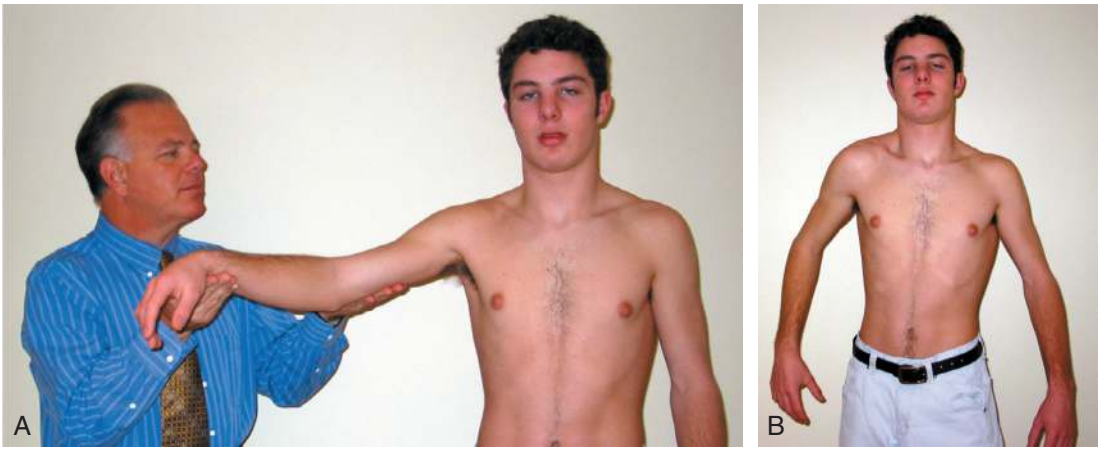

FIG 33.3 A, The drop arm test for complete rotator cuff tear. B, A patient with a complete rotator cuff tear is unable to hold the arm in the abducted position, and it falls to the patient's side. The patient often shrugs or hitches the shoulder forward to use the intact muscles of the rotator cuff and the deltoid to keep the arm in the abducted position. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:91–92.)

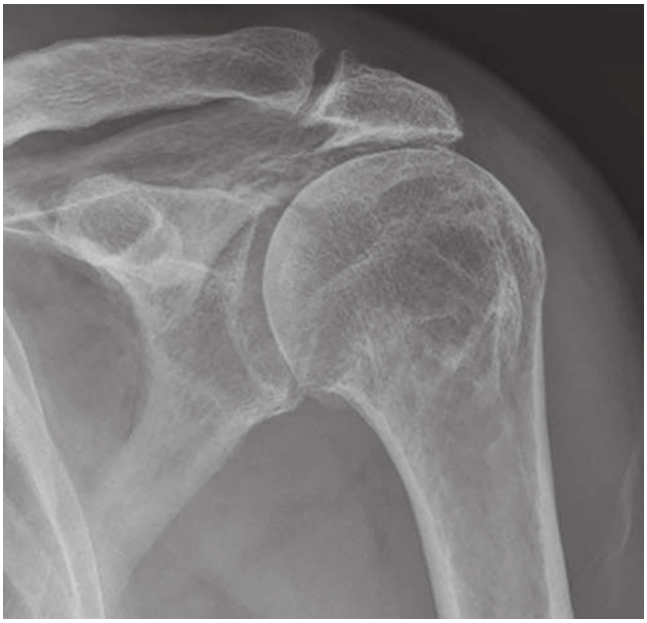

FIG 33.4 Anteroposterior radiograph of the shoulder in a patient with a chronic rotator cuff tear. There is marked narrowing of the subacromial space secondary to proximal humeral head migration. (From Full thickness tear of the rotator cuff. Waldman SD, Robert SD, eds., *Imaging of pain*, Campbell, CA: WB Saunders; 2011: chap 93, 237–238.)

cuff tear rarely occurs before the age 40, except in cases of severe acute trauma to the shoulder.

TREATMENT

Initial treatment of the pain and functional disability associated with rotator cuff tear includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, the injection technique described here is a reasonable next step before surgical intervention.

Injection for rotator cuff tear is carried out by placing the patient in the supine position and preparing the skin overlying the superior shoulder, acromion, and distal clavicle with antiseptic solution. A sterile syringe containing 4 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The lateral edge of the acromion is identified, and at the midpoint of the lateral edge, the injection site is identified. With a slightly cephalad trajectory, the needle is carefully advanced through the skin, subcutaneous tissues, and deltoid muscle beneath the acromion process. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected slightly more inferiorly. After the needle is in place, the contents of the syringe are gently injected. Resistance to injection should be minimal unless calcification of the subacromial bursal sac is present. This calcification can be recognized as resistance to needle advancement, with an associated gritty feel. Significant calcific bursitis may ultimately require irrigation and barbotage or surgical excision to obtain complete relief of symptoms. After injection the needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance may be useful in those patients in whom anatomic landmarks are difficult to identify as well as to improve accuracy of needle placement. Recent clinical reports suggest that the injection of platelet-rich plasma and/or stem cells into the region of tendinopathy may aid in symptom relief and healing.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Transcutaneous nerve

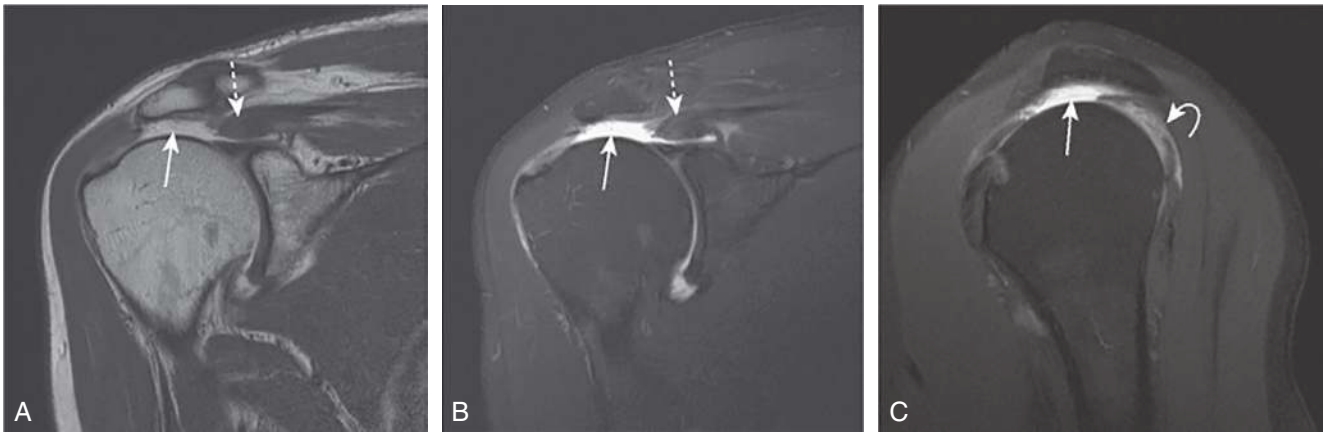

FIG 33.5 Coronal oblique T1-weighted (T1W) **(A)** and T2W with fat suppression (FST2W) **(B)** magnetic resonance (MR) arthrogram images of a patient with a full-thickness tear of the supraspinatus tendon. The tendon defect is outlined by the high-SI contrast medium (*white arrows*), and the torn tendon end is visible medially (*broken white arrows*). **C,** The sagittal oblique FST2W MR image also demonstrates the tendon tear (*white arrow*), and the infraspinatus tendon posteriorly (*curved arrow*) is thickened and has high SI because of associated tendinopathy. (From Full thickness tear of the rotator cuff. Waldman SD, Robert SD, eds., *Imaging of pain*. Campbell, CA: W.B. Saunders; 2011: chap 93, 237–238.)

FIG 33.6 Massive tear of the rotator cuff. **A,** Coronal oblique T2-weighted magnetic resonance imaging (MRI). The supraspinatus tendon is retracted to the medial glenoid margin *(arrow)*. Severe atrophy is evident. **B,** Sagittal oblique T2-weighted MRI. Note the "bald" humeral head. The tear extends from the subscapularis to the infraspinatus tendon *(arrows)*. (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3225.)

stimulation may also reduce pain control. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms and may lead to complete tendon rupture.

COMPLICATIONS AND PITFALLS

One major complication is failure to identify a partial rotator cuff tear correctly and to treat it before it becomes complete. This usually occurs because MRI of the shoulder is not performed and the diagnosis is made on clinical grounds alone.

The injection technique described is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of the injection

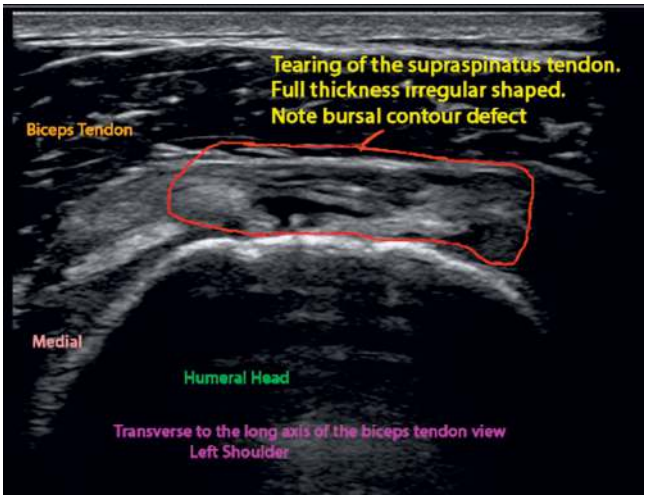

FIG 33.7 Transverse ultrasound scan demonstrating full thickness, irregular tearing of the supraspinatus tendon with loss of the bursal contour.

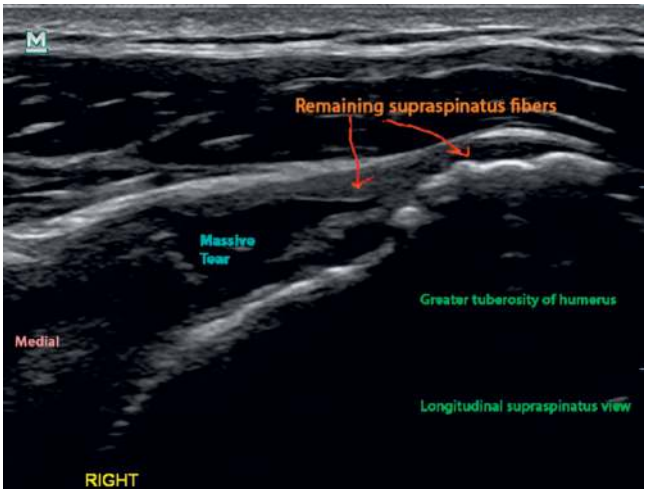

FIG 33.8 Longitudinal ultrasound image demonstrating a massive tear of the supraspinatus tendon with only a few tendon fibers still intact.

technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Trauma to the rotator cuff from the injection itself is also a possibility. Tendons that are highly inflamed or previously damaged are subject to rupture if they are injected directly, which could convert a partial tear into a complete one. This complication can be avoided if the clinician uses gentle technique and stops injecting immediately if significant resistance is encountered. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Injection is extremely effective in the treatment of pain secondary to rotator cuff tear. This technique is not a substitute for surgery, but it can be used to palliate the pain of partial tears or when surgery for complete tears is not being contemplated. Coexistent bursitis and arthritis may contribute to shoulder pain, thus necessitating more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with this injection technique. Partial tears may be amenable to arthroscopic or minimal-incision surgery, and the clinician should not wait until the tear is complete before obtaining orthopedic consultation.

SUGGESTED READINGS

Allen H, Chan BY, Davis KW, Blankenbaker DG. Overuse injuries of the shoulder. *Radiol Clin North Am*. 2019;58(5):898–909.

Cibulas A, Leyva A, Cibulas G, et al. Acute shoulder injury. *Radiol Clin North Am*. 2019;58(5):883–898.

Kang B-S, Lee SH, Cho Y, et al. Acute calcific bursitis after ultrasound-guided percutaneous barbotage of rotator cuff calcific tendinopathy: a case report. *PM&R*. 2016;8(8):808–812.

Netter FH. Shoulder (glenohumeral joint). In: *Atlas of human anatomy*. 4th ed. Philadelphia: Saunders; 2008.

Waldman SD, ed. Atlas of pain management injection techniques. 4th ed. Philadelphia: Saunders; 2018:94–98.

Waldman SD, ed. Clinical correlates: diseases of the rotator cuff. *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:87–92.

Waldman SD. Rotator cuff tear. Waldman SD, eds., Atlas of common pain syndromes. 4th ed. 2019:129–133.

Waldman SD. Injection technique for rotator cuff tear. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:132–135.

Waldman SD. Rotator cuff disease. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:186–195.

Waldman SD. The rotator cuff. In: *Waldman Pain Review*. 2nd ed. Philadelphia: Elsevier; 2017:93–94.

Waldman SD. Clinical correlates: functional anatomy of the shoulder. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2018.

Deltoid Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The deltoid muscle is susceptible to the development of myofascial pain syndrome. Flexion–extension and lateral motion stretch injuries or impact injuries to the deltoid muscle during football or repeated microtrauma secondary to jobs that require prolonged lifting may result in the development of myofascial pain in the deltoid muscle (Fig. 34.1).

Deltoid syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the deltoid muscle often have referred pain in the shoulder that radiates down into the upper extremity.

The trigger point is pathognomonic of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, one often sees an involuntary withdrawal of the stimulated muscle, called a "jump sign," characteristic of myofascial pain syndrome. Patients with deltoid syndrome exhibit trigger points in both the anterior and posterior fibers of the muscle (Fig. 34.2).

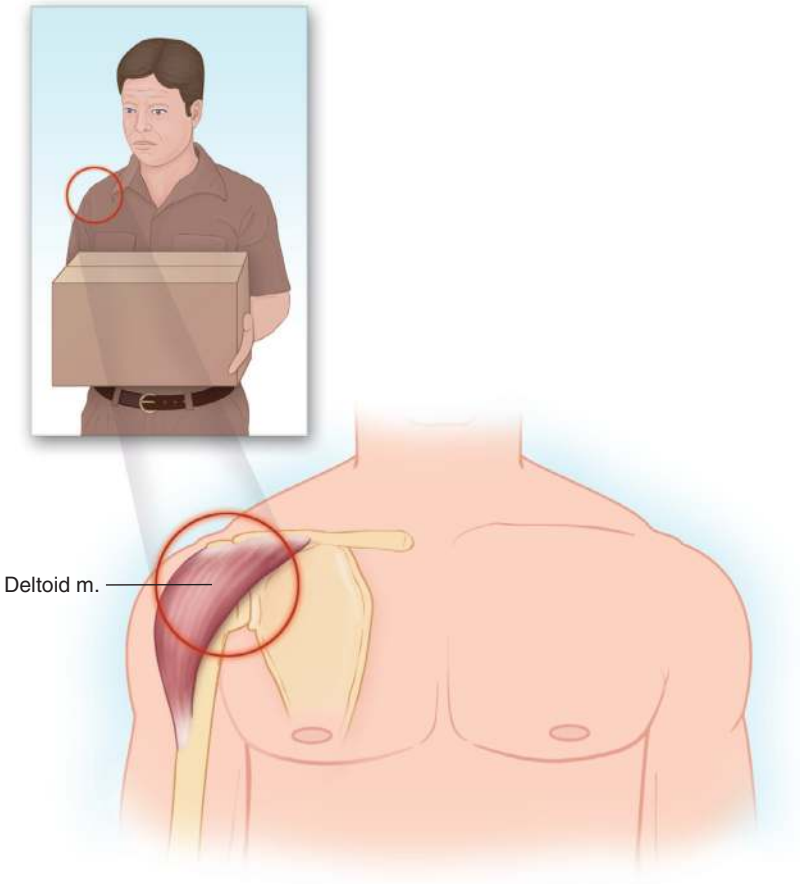

FIG 34.1 Jobs that require prolonged lifting may lead to the development of myofascial pain in the deltoid muscle.

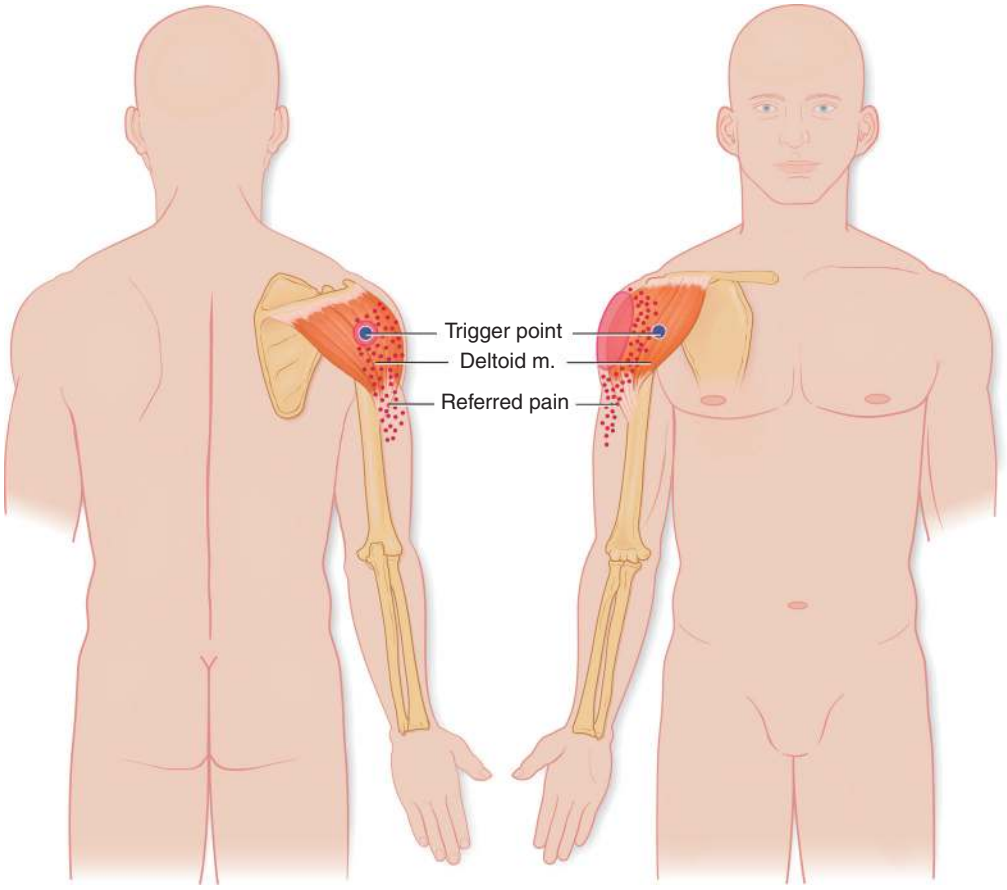

FIG 34.2 Patients with deltoid syndrome have trigger points in both the anterior and posterior fibers of the muscle. (From Waldman SD. Deltoid syndrome. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:83.)

Taunt bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to result from microtrauma to the affected muscle. This trauma may occur from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The deltoid muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The sine qua non of deltoid syndrome is the identification of a myofascial trigger point—a local point of exquisite tenderness—overlying the superior border of the scapula. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. The jump sign is also characteristic of deltoid syndrome, as is pain over the deltoid muscle that is referred into the proximal lateral upper extremity (see Fig. 34.2).

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with deltoid syndrome, but this finding has not been corroborated by

other investigators. Electrodiagnostic testing of patients suffering from deltoid syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic deltoid syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of deltoid syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from deltoid syndrome. The clinician must rule out other coexisting disease processes that may mimic deltoid syndrome, including primary inflammatory muscle disease, multiple sclerosis, and collagen vascular disease. Electrodiagnostic and radiographic testing can help identify coexisting disorders such as bursitis, tendinitis, and rotator cuff tears (Fig. 34.3). The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with deltoid syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

FIG 34.3 Anterior and posterior extension of a large full-thickness tear *(arrows)*. **A,** Coronal oblique T2-weighted magnetic resonance imaging (MRI). **B,** Coronal oblique T2-weighted MRI, more posteriorly. **C,** Axial T2-weighted MRI. (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3237.)

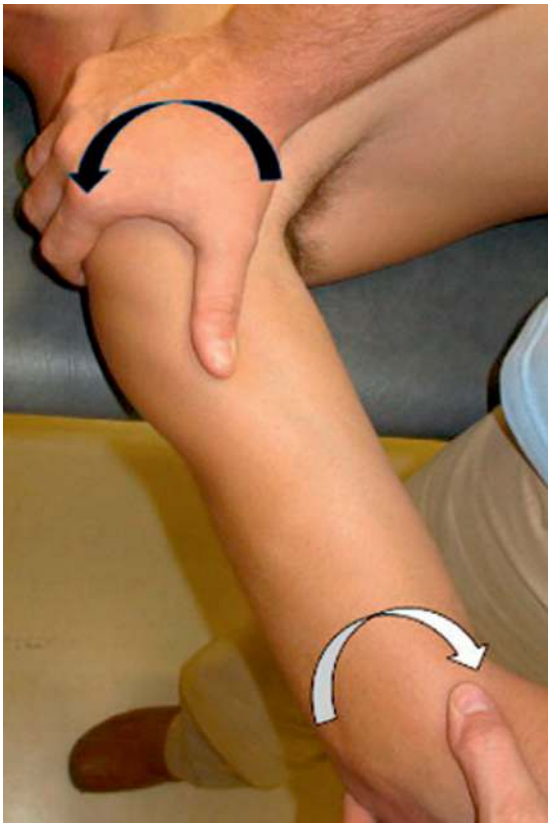

FIG 34.4 Dynamic transverse strokes of anterior deltoid muscle trigger points. The *black arrow* shows the anterior to posterior stroke of the deltoid and the *white arrow* shows the internal rotation movement. (From Fernández de las Peñas C, Cleland J, Huijbregts P, eds. *Neck and arm pain syndromes*. Edinburgh: Churchill Livingstone; 2011.)

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections, antidepressants, and physical therapy to achieve pain relief (Fig. 34.4). Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid pneumothorax when injecting trigger points in proximity to the underlying pleural space.

CLINICAL PEARLS

Although deltoid syndrome is a common disorder, it is often misdiagnosed. Therefore in patients suspected of suffering from deltoid syndrome, a careful evaluation to identify underlying disease processes is mandatory. Deltoid syndrome often coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208(15):87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Fernández de las Peñas C, Ge H-Y, Dommerholt J. Manual treatment of myofascial trigger points. In: Fernández de las Peñas C, Cleland J, Huijbregts P, eds. *Neck and arm pain syndromes*. Edinburgh: Churchill Livingstone; 2011:419–429.

Waldman SD. Deltoid syndrome. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:82–84.

Teres Major Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The teres major muscle is susceptible to the development of myofascial pain syndrome. Stretch or impact injuries to the teres major muscle sustained while playing sports or in motor vehicle accidents, as well as falls onto the lateral scapula, have been implicated in the evolution of teres major syndrome. In addition, repeated microtrauma secondary to reaching up and behind, such as when retrieving a briefcase from the backseat of a car, overhead throwing, and other sports injuries may result in the development of myofascial pain in the teres major muscle (Figs. 35.1 and 35.2).

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the teres major muscle often have referred pain in the shoulder that radiates down into the upper extremity.

The trigger point is pathognomonic of myofascial pain syndrome and is characterized by a local point of exquisite tenderness. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, is often seen and is characteristic of myofascial pain syndrome. Patients with teres major syndrome exhibit trigger points lateral to the scapula in the teres major muscle (Fig. 35.3).

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent

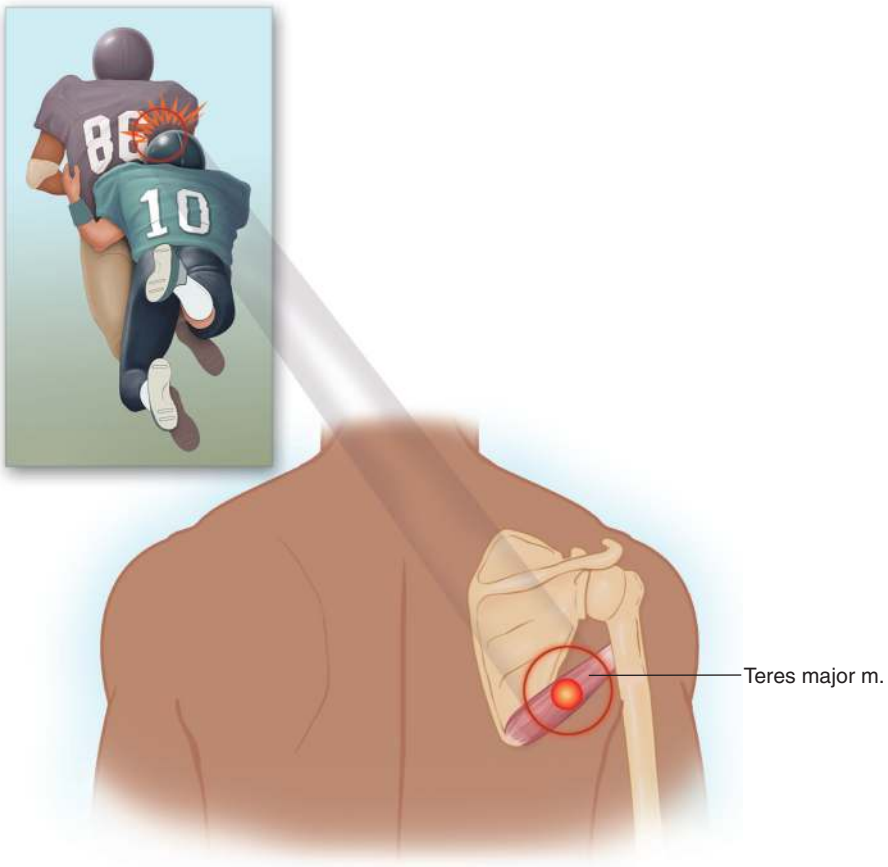

FIG 35.1 Repeated microtrauma secondary to reaching up and behind, as well as playing sports such as football, may result in the development of myofascial pain in the teres major muscle.

FIG 35.2 Teres major injury in a professional baseball player: magnetic resonance imaging (MRI). Coronal oblique **(A)** and axial T1-weighted spin-echo fat-suppressed **(B)** images of the right shoulder acquired using a 1.5-Tesla magnet after the administration of intravenous contrast from patient one demonstrate enhancing fluid *(straight arrows* and *arrowheads)* tracking caudally and posteriorly from the inferior aspect of the glenohumeral joint and indicating an acute, traumatic soft tissue injury of the teres major muscle, consistent with a grade II strain. Also visible is enhancement at the distal supraspinatus footprint *(curved arrow)* that is typical of degenerative tendinosis associated with overhand throwing. **C,** Sagittal oblique T2-weighted fast spin-echo fat-suppressed image shows hyperintense fluid signal outlining the teres major proximal myotendinous junction (TM), as well as intramuscular "feathery" edema *(arrowheads)* typical of an acute myotendinous strain. *LD*, Latissimus dorsi muscle. (From Leland M, Ciccotti MG, Cohen SB, et al. Teres major injuries in two professional baseball pitchers. *J Shoulder Elb Surg*. 2009;18(6):e1–e5.)

physical finding, the pathophysiology of the myofascial trigger point remains elusive, but trigger points are thought to result from microtrauma to the affected muscle. This trauma may occur from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to the development of myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been

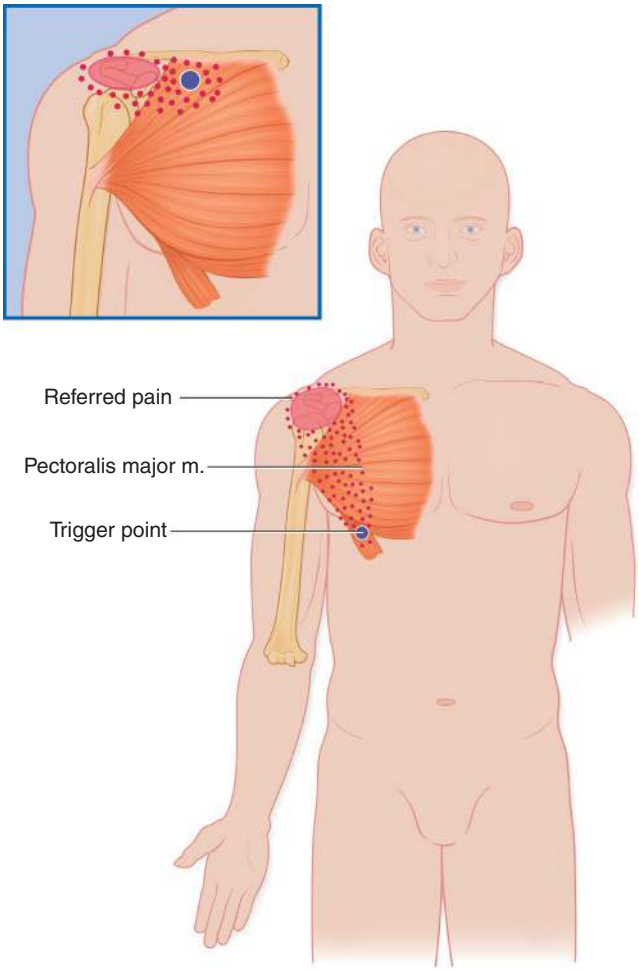

FIG 35.3 Patients with teres major syndrome have a trigger point lateral to the scapula in the teres major muscle. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:86.)

implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The teres major muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point, the pathologic lesion of teres major syndrome, is characterized by a local point of exquisite tenderness in the axillary or posterior portion of the muscle. Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain and referred pain. In addition, the jump sign is characteristic of teres major syndrome, as is pain over the teres major muscle that is referred to the proximal portion of the posterolateral upper extremity.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with teres major syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing of patients suffering from teres major syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic teres major syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of teres major syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from teres major syndrome. The clinician must rule out other coexisting disease processes that may mimic teres major syndrome, including primary inflammatory muscle disease, multiple sclerosis, and collagen vascular disease. Electrodiagnostic and radiographic testing can help identify coexisting disorders such as shoulder bursitis, tendinitis, and rotator cuff tears. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with teres major syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of

the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections, antidepressants, and physical therapy to achieve pain relief (Fig. 35.4). Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

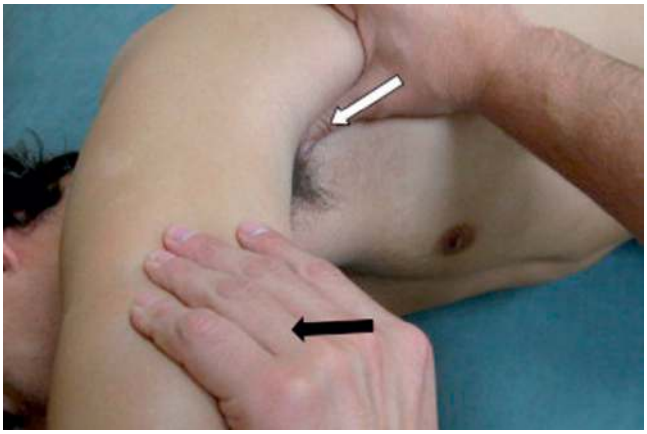

FIG 35.4 Stretching compression of teres major muscle taut band. The *black arrow* shows the shoulder abduction and the *white arrow* shows the stabilization force of the therapist. (From Fernández de las Peñas C, Ge H-Y, Dommerholt J. Manual treatment of myofascial trigger points. In: Fernández de las Peñas C, Cleland J, Huijbregts P, eds. *Neck and arm pain syndromes*. Edinburgh: Churchill Livingstone; 2011:419–429.)

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid pneumothorax when injecting trigger points in proximity to the underlying pleural space.

CLINICAL PEARLS

Although teres major syndrome is a common disorder, it is often misdiagnosed. Therefore in patients suspected of suffering from teres major syndrome, a careful evaluation to identify underlying disease processes is mandatory. Teres major syndrome commonly coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208(15):87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:chap 16(e), e158–e167.

Fernández de las Peñas CF, Ge HY, Dommerholt J. Manual treatment of myofascial trigger points. In: Fernández de las Peñas C, Cleland J, Huijbregts P, eds. *Neck and arm pain syndromes*. Edinburgh: Churchill Livingstone; 2011:419–429.

Waldman SD. Teres major syndrome. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:88–90.

Scapulocostal Syndrome

ICD-10 CODE G56.80

THE CLINICAL SYNDROME

Scapulocostal syndrome consists of a constellation of symptoms including unilateral pain and associated paresthesias at the medial border of the scapula, referred pain radiating from the deltoid region to the dorsum of the hand, and decreased range of motion of the scapula (Fig. 36.1). Scapulocostal syndrome is commonly referred to as traveling salesman's shoulder, because it is frequently seen in individuals who repeatedly reach backward to retrieve something from the back seat of a car (Fig. 36.2). Scapulocostal syndrome is an overuse syndrome caused by repeated improper use of the muscles of scapular stabilization—the levator scapulae, pectoralis minor, serratus anterior, rhomboids, and, to a lesser extent, infraspinatus, and teres minor.

Scapulocostal syndrome is a chronic myofascial pain syndrome, and the sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain and referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, is often seen and is characteristic of myofascial pain syndrome. Almost all patients with scapulocostal syndrome have a prominent infraspinatus trigger point, which is best demonstrated by having the patient place the hand of the affected side over the deltoid of the opposite shoulder (Fig. 36.3). This maneuver laterally rotates the affected scapula and allows palpation and subsequent injection of the infraspinatus trigger point. Other trigger points along the medial border of the scapula may be present and may be amenable to injection therapy.

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to result from microtrauma to the affected muscle. This trauma may occur from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome.

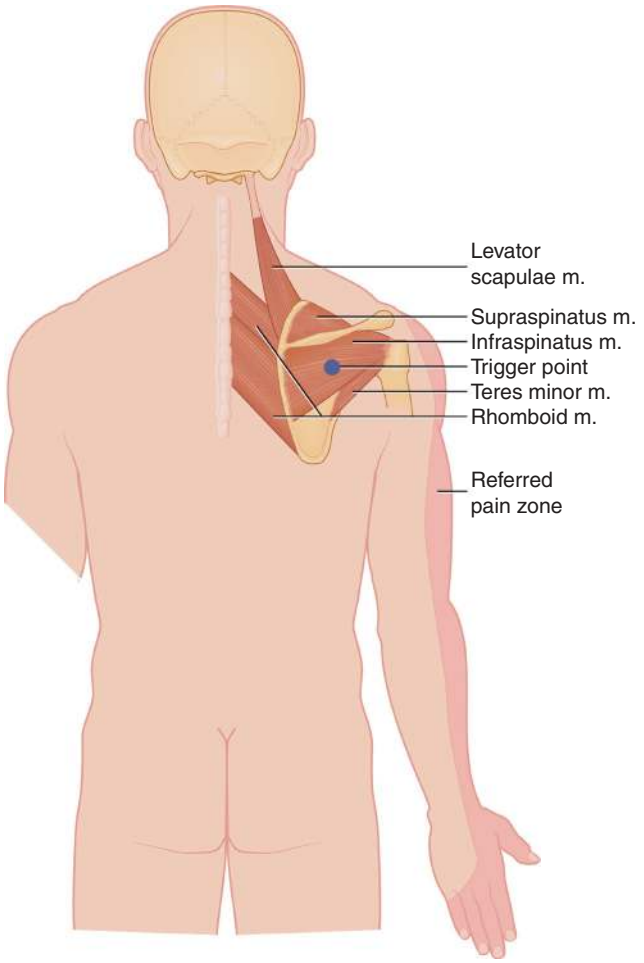

FIG 36.1 Scapulocostal syndrome involves unilateral pain and associated paresthesias at the medial border of the scapula, referred pain radiating from the deltoid region to the dorsum of the hand, and decreased range of motion of the scapula. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007.)

For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these factors may be intensified if the

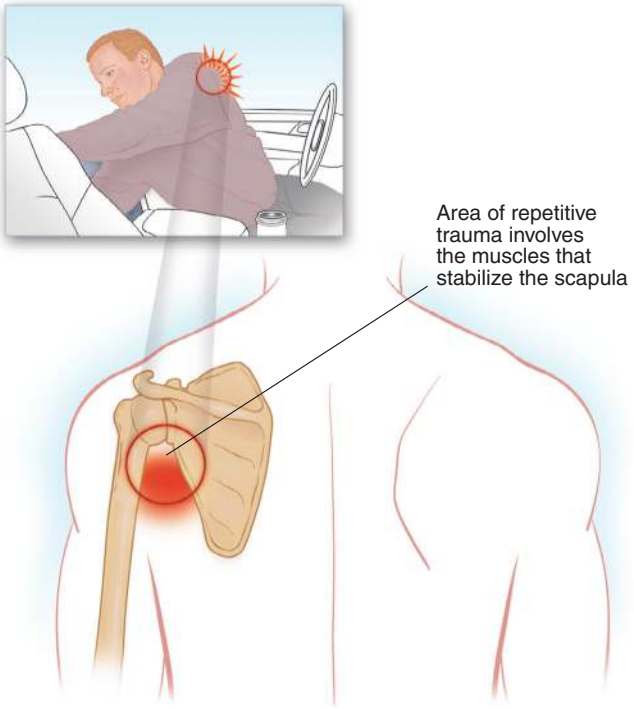

FIG 36.2 Scapulocostal syndrome is also called traveling salesman's shoulder because it is frequently seen in individuals who repeatedly reach backward to retrieve something from the back seat of a car.

patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The muscle groups involved in scapulocostal syndrome seem to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point is the pathologic lesion of scapulocostal syndrome; it is characterized by a local point of exquisite tenderness in the infraspinatus muscle. As noted earlier, this infraspinatus trigger point can best be demonstrated by having the patient place the hand of the affected side over the deltoid of the opposite shoulder. Other trigger points may be present along the medial border of the scapula.

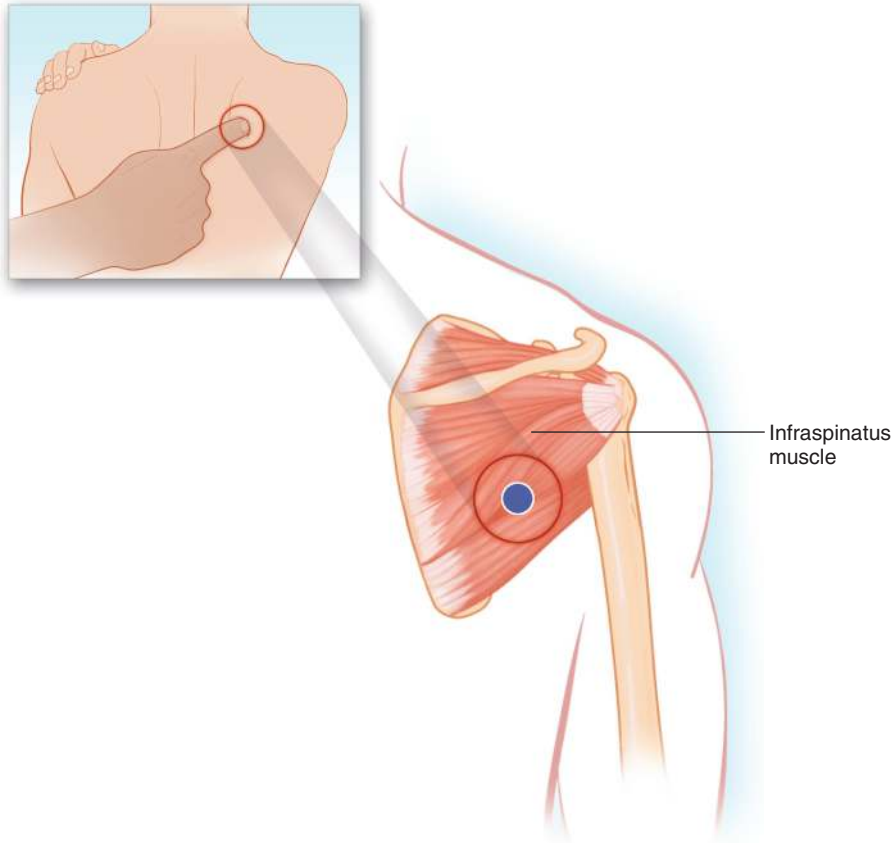

FIG 36.3 The infraspinatus trigger point can be demonstrated by having the patient place the hand of the affected side over the deltoid of the opposite shoulder.

Mechanical stimulation of the trigger point by palpation or stretching produces intense local pain as well as referred pain. The jump sign is characteristic of scapulocostal syndrome, as is pain over the infraspinatus muscle that radiates from the deltoid region to the dorsum of the hand.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with scapulocostal syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing of patients suffering from scapulocostal syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic scapulocostal syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of scapulocostal syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from scapulocostal syndrome. The clinician must rule out other coexisting disease processes that may mimic scapulocostal syndrome, including primary inflammatory muscle disease, isolated tears of the infraspinatus musculotendinous unit, multiple sclerosis, and collagen vascular disease (Fig. 36.4). Electrodiagnostic and radiographic testing can help identify coexisting disorders such as shoulder bursitis, tendinitis, and rotator cuff tears. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with scapulocostal syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger point and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients with scapulocostal syndrome, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin-norepinephrine reuptake inhibitor has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

FIG 36.4 Disruption of the infraspinatus musculotendinous unit. Coronal T2-weighted magnetic resonance imaging scan showing high signal at the site of the disruption *(large white arrow)* and an increased pennate angle *(small white arrow)* resulting from muscular retraction of the infraspinatus. Edema is also seen in the infraspinatus muscle belly. *Infra*, Infraspinatus; *T minor*, teres minor. (From Lunn JV, Castellanos-Rosas J, Tavernier T, et al. A novel lesion of the infraspinatus characterized by musculotendinous disruption, edema, and late fatty infiltration. *J Shoulder Elb Surg*. 2008; 17(4):546–553.)

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections, antidepressants, and physical therapy to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid pneumothorax when injecting trigger points in proximity to the underlying pleural space.

CLINICAL PEARLS

Although scapulocostal syndrome is a common disorder, it is often misdiagnosed. Therefore in patients suspected of suffering from scapulocostal syndrome, a careful evaluation to identify underlying disease processes is mandatory. Scapulocostal syndrome commonly coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Abrams B, Goodman S. Scapulocostal syndrome. In: Waldman SD, ed. *Pain management*. Philadelphia: WB Saunders; 2007: 627–632.

Buttagat V, Eungpinichpong W, Chatchawan U, et al. Therapeutic effects of traditional Thai massage on pain, muscle tension and anxiety in patients with scapulocostal syndrome: a randomized single-blinded pilot study. *J Bodyw Mov Ther*. 2012;16(1):57–63.

Buttagat V, Taepa N, Suwannived N, et al. Effects of scapular stabilization exercise on pain related parameters in patients with scapulocostal syndrome: a randomized controlled trial. *J Bodyw Mov Ther*. 2016;20(1):115–122.

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208(15):87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Fernández de las Peñas C, Ge H-Y, Dommerholt J. Manual treatment of myofascial trigger points. In: Fernández de las Peñas C, Cleland J, Huijbregts P, eds. *Neck and arm pain syndromes*. Edinburgh: Churchill Livingstone; 2011:419–429.

37

Arthritis Pain of the Elbow

ICD-10 CODE M19.90

THE CLINICAL SYNDROME

Elbow pain secondary to degenerative arthritis is frequently encountered in clinical practice. Osteoarthritis is the most common form of arthritis that results in elbow joint pain. Tendinitis and bursitis may coexist with arthritis pain, which makes the correct diagnosis more difficult. The olecranon bursa lies in the posterior aspect of the elbow joint and may become inflamed as a result of direct trauma or overuse of the joint. Bursae susceptible to the development of bursitis also exist between the insertion of the biceps and the head of the radius, as well as in the antecubital and cubital areas.

In addition to pain, patients suffering from arthritis of the elbow joint often experience a gradual reduction in functional ability because of decreasing elbow range of motion that makes simple everyday tasks, such as using a computer keyboard, holding a coffee cup, or turning a doorknob, quite difficult (Fig. 37.1). With continued disuse, muscle wasting may occur, and adhesive capsulitis with subsequent ankylosis may develop.

SIGNS AND SYMPTOMS

Most patients with elbow pain secondary to osteoarthritis or posttraumatic arthritis complain of pain that is localized around the elbow and forearm. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients also complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical examination.

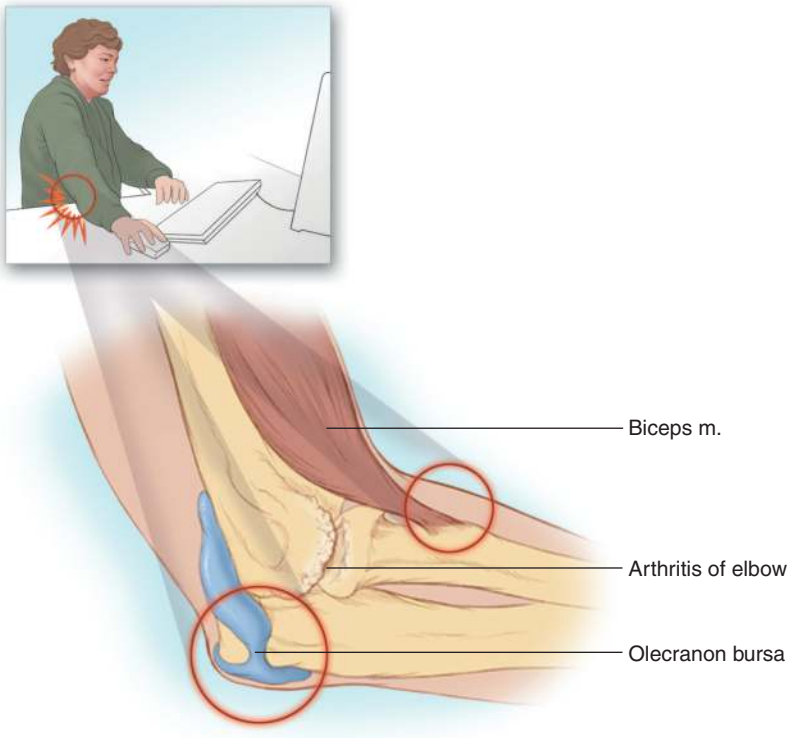

FIG 37.1 Arthritis of the elbow can cause pain and functional disability during common everyday tasks.

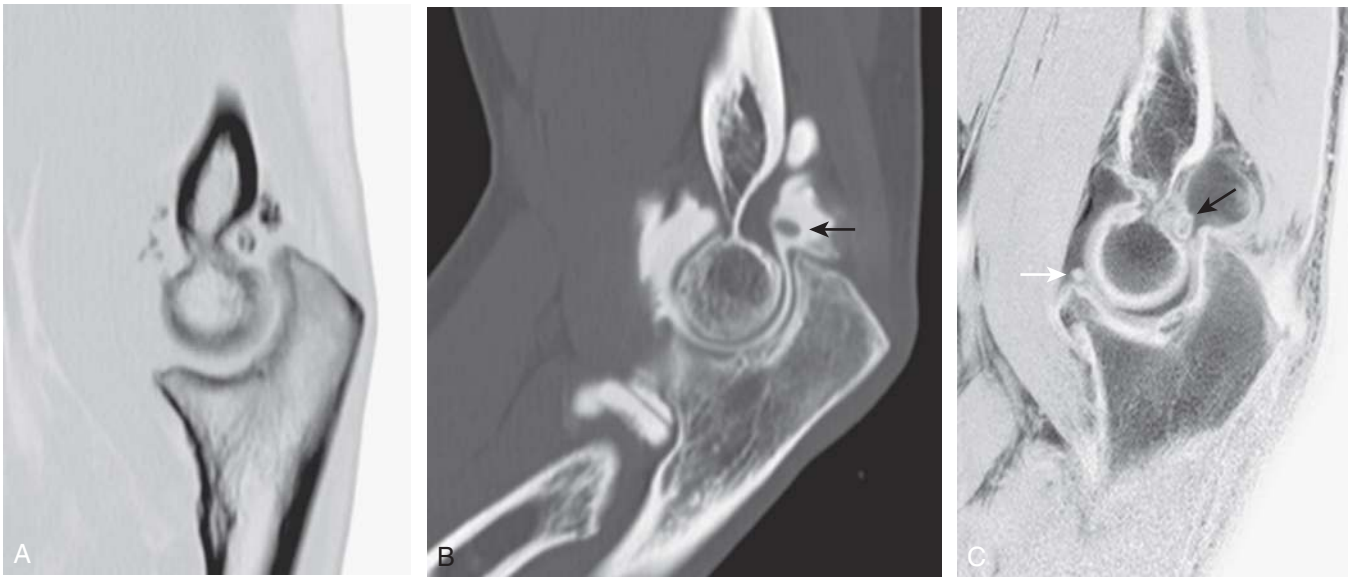

FIG 37.2 A, Sagittal computed tomography (CT) scan demonstrating radiopaque loose bodies in both the posterior olecranon fossa and the anterior coronoid fossa of the distal humerus. B, A sagittal CT arthrogram in a different patient demonstrates a nonradiopaque cartilaginous loose body *(black arrow)* that was not visible on the CT scan without contrast agent. C, A sagittal T1-weighted (T1W) magnetic resonance (MR) arthrogram image from a third patient with an ossific loose body posteriorly *(black arrow)* and a second cartilaginous loose body anteriorly *(white arrow)*. Established osteoarthritis changes are seen. (From Waldman SD, Campbell RSD. *Imaging of pain.* Philadelphia: Elsevier; 2011.)

TESTING

Plain radiographs should be obtained in all patients who present with elbow pain. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Computerized tomography may be useful to identify bony abnormalities (Fig. 37.2). Magnetic resonance and/or ultrasound imaging of the elbow is indicated if joint instability, nerve entrapment, tumor, or other soft tissue abnormality is suspected (Fig. 37.3).

DIFFERENTIAL DIAGNOSIS

Rheumatoid arthritis, posttraumatic arthritis, and psoriatic arthritis are common causes of elbow pain. Less common causes of arthritis-induced elbow pain include collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; treatment is with culture and antibiotics rather than injection therapy. Collagen vascular diseases generally manifest as polyarthropathy rather than as monarthropathy limited to the elbow joint; however, elbow pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described later. The clinician should always be aware of the possibility of uncommon causes of elbow pain including tumor or joint mice (Figs. 37.4 and 37.5).

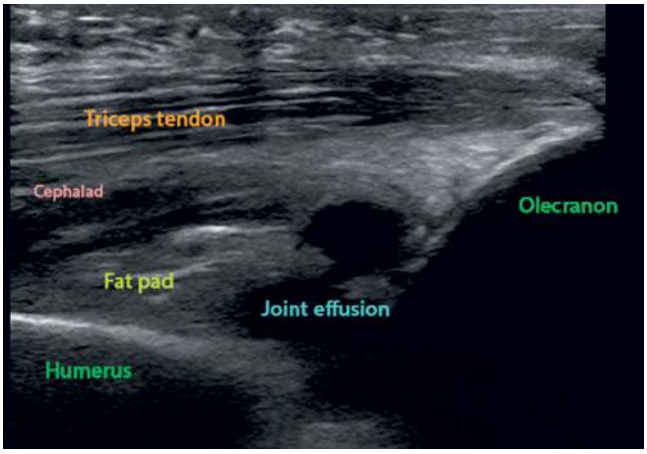

FIG 37.3 Longitudinal ultrasound image of the posterior elbow demonstrating a joint effusion.

TREATMENT

Initial treatment of the pain and functional disability associated with arthritis of the elbow includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

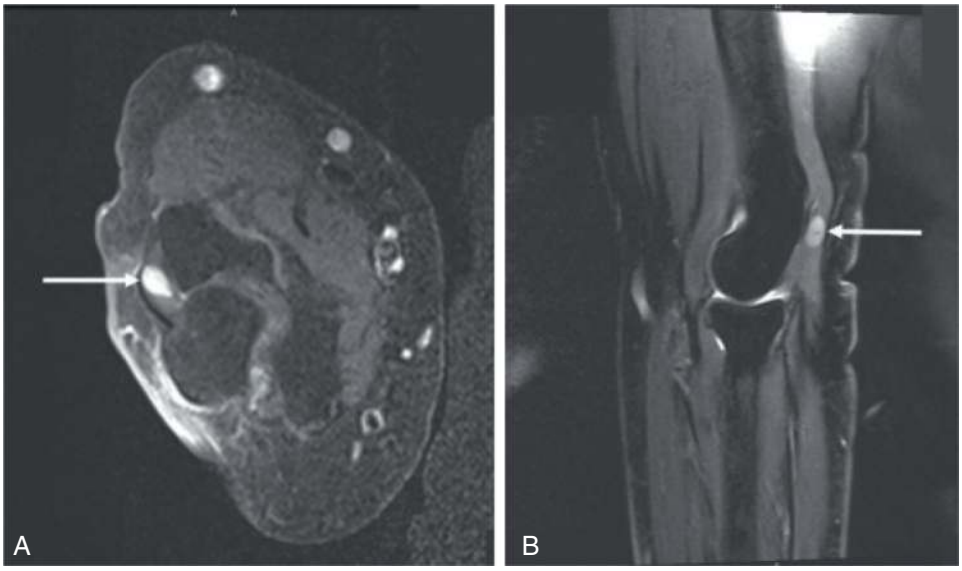

FIG 37.4 A, Magnetic resonance imaging (MRI). Axial T1 TSE with gadolinium injection. *White arrow*: ovoid mass deep at the junction between the anconeus and distal triceps muscle, well defined. The lesion is homogeneously enhanced by gadolinium. B, MRI. Sagittal DP FS. *White arrow* pointing the lesion. (From Maximen J, Vallée N, Ropars M, Kim W. Chronic pain of the elbow due to angioleiomyoma: a case report. *JSES Int.* 2021;5(3):561–563.)

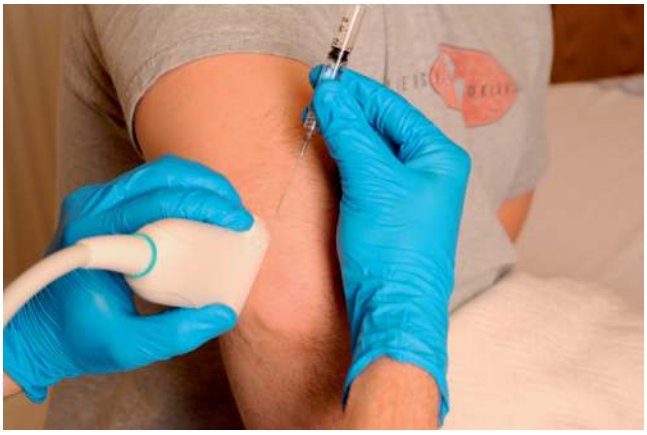

FIG 37.5 Per in plane needle position for ultrasound-guided injection of the elbow joint.

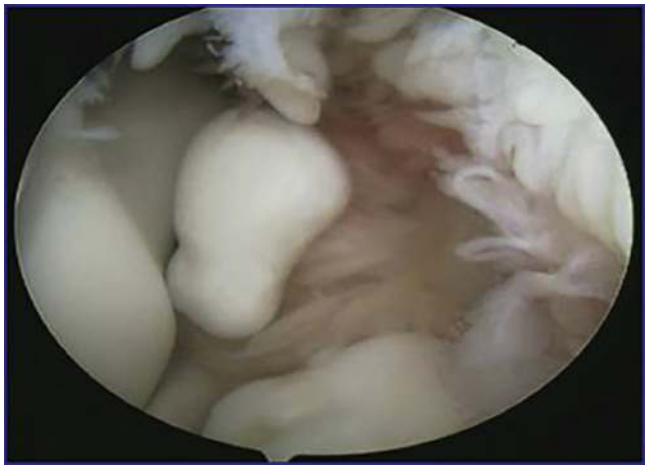

FIG 37.6 Arthroscopic picture of the elbow showing an anterior compartment loose body. (From Alnusif NS, Matache BA, AlQahtani SM, et al. Effectiveness of radiographs and computed tomography in evaluating primary elbow osteoarthritis. *J Shoulder Elb Surg.* 2021;30(suppl 7):S8–S13.)

Intraarticular injection of the elbow is carried out with the patient in the supine position, the arm fully adducted at the patient's side, the elbow flexed, and the dorsum of the hand resting on a folded towel. A total of 5 mL local anesthetic and 40 mg methylprednisolone are drawn up in a 12-mL sterile syringe. After sterile preparation of the skin overlying the posterolateral aspect of the joint, the head of the radius is identified. Just superior to the head of the radius is an indentation that represents the space between the radial head and the humerus. Using strict aseptic technique, a 1-inch, 25-gauge needle is inserted just above the superior aspect of the head of the radius through the skin, subcutaneous tissues, and joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. After the joint space has been entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that the intraarticular injection of platelet-rich plasma may provide improved healing of elbow joint pathology. Ultrasound guidance may improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify (Fig. 37.6).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after

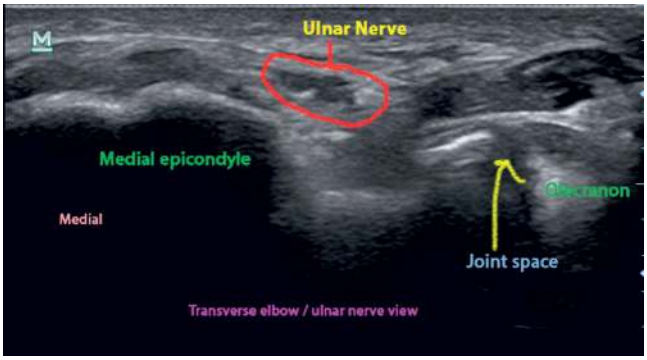

FIG 37.7 Transverse ultrasound image of the posterior elbow demonstrating the relationship of the ulnar nerve to the medial epicondyle, ulnar nerve, joint space, and olecranon process.

the patient undergoes injection for elbow pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The major complication of intraarticular injection of the elbow is infection, although it should be exceedingly rare if strict aseptic technique is followed. The ulnar nerve is especially susceptible to damage at the elbow, and care must be taken to avoid this structure when performing intraarticular injection. Ultrasound imaging may help avoid this complication (Fig. 37.7). Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the elbow joint, and patients should be warned of this possibility.

CLINICAL PEARLS

Pain and functional disability of the elbow are often the result of degenerative arthritis of the joint. Coexistent bursitis and tendinitis may contribute to elbow pain and may confuse the diagnosis. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with the intraarticular injection technique.

SUGGESTED READINGS

Alnusif NS, Matache BA, AlQahtani SM, et al. Effectiveness of radiographs and computed tomography in evaluating primary elbow osteoarthritis. *J Shoulder Elb Surg*. 2021;30(7):S8–S13.

Chung KC, Lark ME. Upper extremity injuries in tennis players: diagnosis, treatment, and management. *Hand Clin*. 2017;33(1):175–186.

Martinez-Catalan N, Sanchez-Sotelo J. Primary elbow osteoarthritis: evaluation and management. *J Clin Orthop Trauma*. 2021;19:67–74. https://doi.org/10.1016/j.jht.2021.05.009.

Maximen J, Vallée N, Ropars M, Kim W. Chronic pain of the elbow due to angioleiomyoma: a case report. *JSES Int*. 2021;5(3):561–563. https://doi.org/10.1016/j.jseint.2021.01.003.

Taylor A, Wolff AL. The forgotten radial nerve: a conceptual framework for treatment of lateral elbow pain. *J Hand Ther*. 2021;34(2):323–329.

Waldman SD. Abnormalities of the posterior elbow joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:259–268.

Waldman SD. Functional anatomy of the elbow joint. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:98–99.

Waldman SD. Intra-articular injection of the elbow joint. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:168–171.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the elbow. In: *Imaging of pain*. Philadelphia: Saunders; 2011:259–260.

Tennis Elbow

ICD-10 CODE M77.10

THE CLINICAL SYNDROME

Tennis elbow (also known as lateral epicondylitis) is caused by repetitive microtrauma to the extensor tendons of the forearm. The pathophysiology of tennis elbow initially involves microtearing at the origin of the extensor carpi radialis and extensor carpi ulnaris. Secondary inflammation may become chronic as a result of continued overuse or misuse of the extensors of the forearm. Coexistent bursitis, arthritis, or gout may perpetuate the pain and disability of tennis elbow.

The most common nidus of pain from tennis elbow is the bony origin of the extensor tendon of the extensor carpi radialis brevis at the anterior facet of the lateral epicondyle. Less commonly, tennis elbow pain originates from the origin of the extensor carpi radialis longus at the supracondylar crest; rarely, it originates more distally, at the point where the extensor carpi radialis brevis overlies the radial head. The olecranon bursa lies in the posterior aspect of the elbow joint and may also become inflamed (bursitis) as a result of direct trauma to the joint or its overuse. Other bursae susceptible to the development of bursitis lie between the insertion of the biceps and the head of the radius, as well as in the antecubital and cubital areas.

Tennis elbow occurs in individuals engaged in repetitive activities such as hand grasping (e.g., shaking hands) or high-torque wrist turning (e.g., scooping ice cream) (Fig. 38.1). Tennis players develop tennis elbow by two different mechanisms: (1) increased pressure grip strain as a result of playing with a too heavy racket, and (2) making backhand shots with a leading shoulder and elbow rather than keeping the shoulder and elbow parallel to the net. Other racket sports players are also susceptible to the development of tennis elbow.

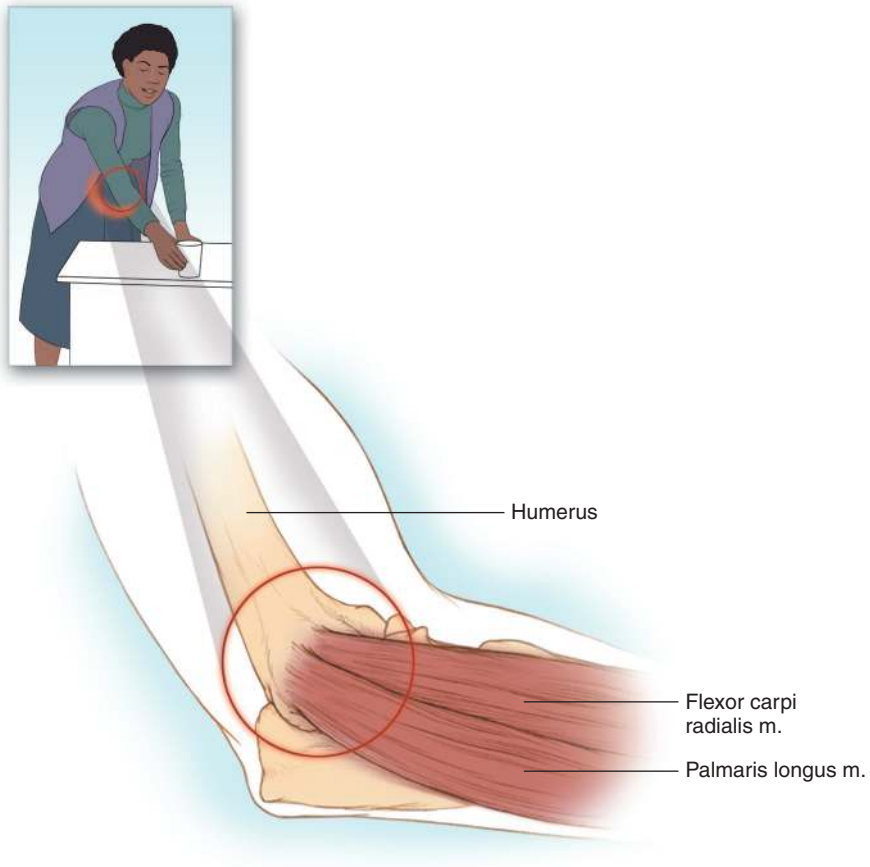

FIG 38.1 The pain of tennis elbow is localized to the lateral epicondyle.

SIGNS AND SYMPTOMS

The pain of tennis elbow is localized to the region of the lateral epicondyle. This pain is constant and is made worse with active contraction of the wrist. Patients note the inability to hold a coffee cup or use a hammer. Sleep disturbance is common. On physical examination, tenderness is elicited along the extensor tendons at or just below the lateral epicondyle. Many patients with tennis elbow exhibit a bandlike thickening within the affected extensor tendons. Elbow range of motion is normal, but grip strength on the affected side is diminished. Patients with tennis elbow have a positive tennis elbow test result. This test is performed by stabilizing the patient's forearm and then having the patient clench his or her fist and actively extend the wrist. The examiner then attempts to force the wrist into flexion (Fig. 38.2). Sudden severe pain is highly suggestive of tennis elbow (Table 38.1).

FIG 38.2 Test for tennis elbow. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms.* Philadelphia: Saunders; 2006:138.)

TESTING

Electromyography can help distinguish cervical radiculopathy and radial tunnel syndrome from tennis elbow. Plain radiographs should be obtained in all patients who present with elbow pain to rule out joint mice and other occult bony diseases. Ultrasound imaging will help quantify the extent of tendinopathy and identify other occult causes of the patient's pain symptomatology (Figs. 38.3 and 38.4). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging of the elbow is indicated if joint instability is suspected or if the symptoms of tennis elbow persist (Fig. 38.5). The injection technique (Fig. 38.6) described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Radial tunnel syndrome and, occasionally, C6–C7 radiculopathy can mimic tennis elbow. Radial tunnel syndrome is caused by entrapment of the radial nerve below the elbow. With radial tunnel syndrome, the maximal tenderness to palpation is distal to the lateral epicondyle over the radial nerve, whereas with tennis elbow, the maximal tenderness to palpation is over the lateral epicondyle (Fig. 38.7).

TREATMENT

Initial treatment of the pain and functional disability associated with tennis elbow includes a combination of nonsteroidal anti-inflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors

TABLE 38.1 Characteristics of Radial Tunnel Syndrome and Lateral Epicondylitis

Characteristic	Radial Tunnel Syndrome	Lateral Epicondylitis (Tennis Elbow)
Frequency	Rare (2% of all peripheral nerve compressions of the upper limb)	Common cause of lateral elbow pain
Cause	Compression of the radial nerve	Caused by overuse of the extensor and supinator muscles
Characteristic patient	Anybody with repetitive, stressful pronation and supination (e.g., tennis players, Frisbee players, swimmers, powerlifters)	Tennis players
Pain location	Pain over the neck of the radius and lateral aspect of the proximal forearm over the extensor muscles themselves (distal to where the pain is located in Lateral Epicondyle (LE))	Pain and tenderness over the lateral epicondyle and immediately distal to it (at the origin of the extensor muscles)
Pain radiation	Pain can radiate proximally and (more commonly) distally	Usually localized without radiation
Provocative tests (much overlap between the two entities)	Pain with resisted extension of the middle finger with the forearm pronated and the elbow extended. Pain with resisted forearm supination with the elbow fully extended	Pain with resisted wrist extension or elbow supination with the elbow extended. Pain with forceful wrist flexion or forearm pronation

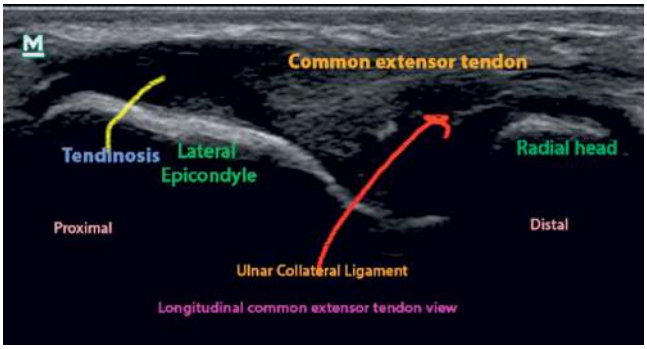

FIG 38.3 Longitudinal ultrasound image demonstrating the classic tendinosis of the common extensor tendon observed in tennis elbow.

and physical therapy. Local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities, injection of local anesthetic and steroid is a reasonable next step.

Injection for tennis elbow is performed by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow flexed, and the dorsum of the hand resting on a folded towel to relax the affected tendons. A total of 1 mL local anesthetic and 40 mg methylprednisolone are drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the posterolateral aspect of the joint, the lateral epicondyle is identified. Using strict aseptic technique,

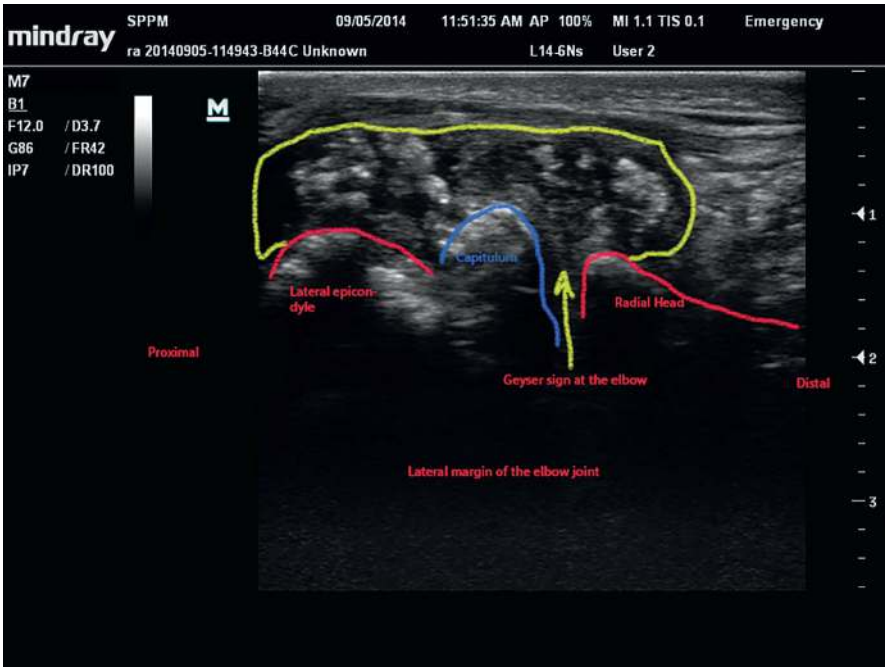

FIG 38.4 As tennis elbow persists, bony abnormalities of the lateral epicondyle are commonly seen as demonstrated in this ultrasound image.

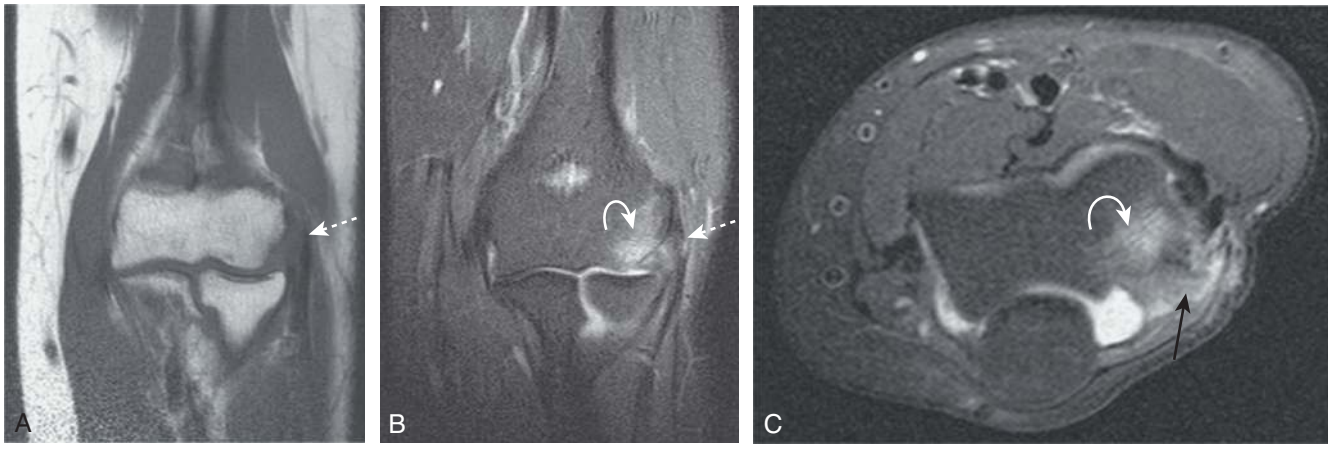

FIG 38.5 Coronal PD **(A)** and FST2W **(B)** magnetic resonance (MR) images of a patient with tennis elbow. There are thickening and increased SI within the common extensor tendon *(broken white arrow)* along with associated underlying bone marrow edema *(curved arrow)*. **C,** The bone marrow is also seen on the axial FST2W MR image *(curved arrow)*, and the soft tissue thickening and increased SI posterior to the extensor tendon probably reflect associated soft tissue impingement *(black arrow)*. *FST2W*, fast spin T2 weighted; *PD* proton density; *SI*, signal intensity. (From Waldman SD, Campbell RSD. *Imaging of pain.* Philadelphia: Elsevier; 2011.)

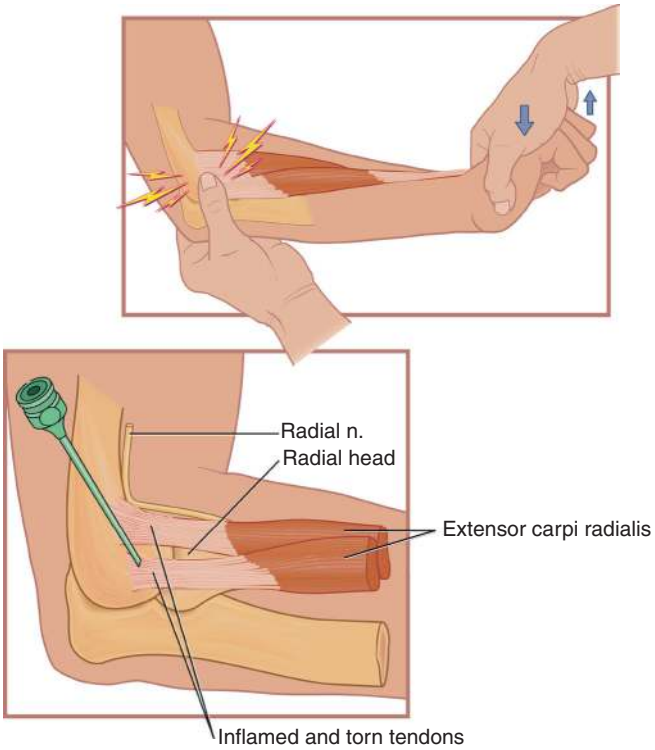

FIG 38.6 Proper needle placement for injection for tennis elbow. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

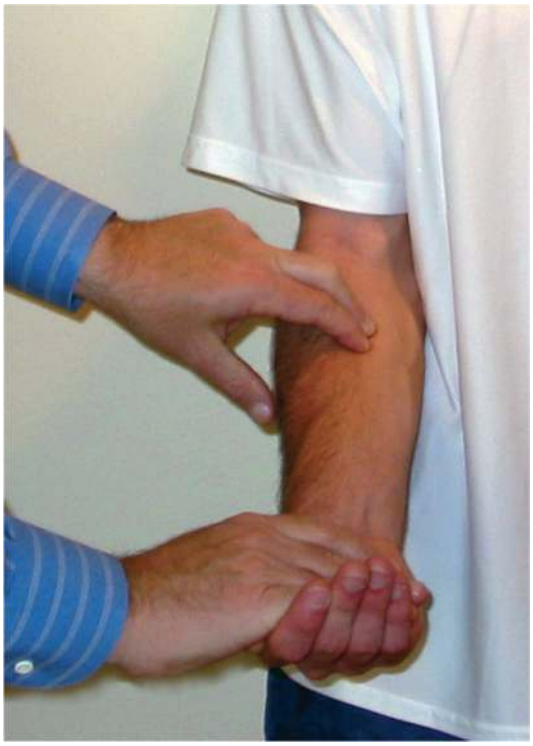

FIG 38.7 Palpation of the radial nerve in the forearm to diagnose radial tunnel syndrome.

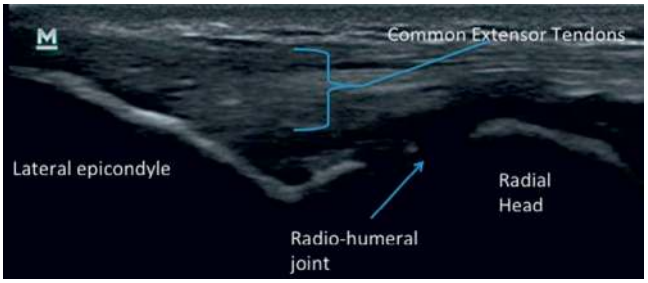

FIG 38.8 Longitudinal ultrasound image demonstrating the gentle slope of the lateral epicondyle, the river-like appearing extensor tendons inserted into the lateral epicondyle, and the hill-shaped radial head.

a 1-inch, 25-gauge needle is inserted perpendicular to the lateral epicondyle through the skin and into the subcutaneous tissue overlying the affected tendon (see Fig. 38.6). If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that the injection of type A botulinum toxin and platelet-rich plasma and/or stem cells may provide improved symptom relief and healing of tennis elbow. Ultrasound guidance may improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify (Fig. 38.8).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for tennis elbow. Low-level laser therapy may also be beneficial. A Velcro counterforce orthotic band placed around the extensor tendons may also help relieve the symptoms. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The major complication associated with tennis elbow is rupture of the inflamed tendon either from repetitive trauma or from injection directly into the tendon. To prevent inflamed and previously damaged tendons from rupturing, the needle position should be confirmed to be outside the tendon before the clinician proceeds with the injection. Another complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. The injection technique is safe if careful attention is paid to the clinically relevant anatomy; in particular, the ulnar nerve is susceptible to damage at the elbow. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to tennis elbow. Coexistent bursitis and tendinitis may contribute to elbow pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with the injection technique. Cervical radiculopathy and radial tunnel syndrome may mimic tennis elbow and must be excluded.

SUGGESTED READING

Chung KC, Lark ME. Upper extremity injuries in tennis players: diagnosis, treatment, and management. *Hand Clin*. 2017;33(1):175–186.

Palacio EP, Schiavetti RR, Kanematsu M, et al. Effects of platelet-rich plasma on lateral epicondylitis of the elbow: prospective randomized controlled trial. *Rev Bras Ortop*. 2016;51(1):90–95.

Waldman SD. Lateral epicondylitis injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:149–152.

Waldman SD. Lateral epicondyle injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:189–193.

Waldman SD. Radial tunnel syndrome. In: *Atlas of uncommon pain syndromes*. 4th ed. Philadelphia: Elsevier; 2019:113–117.

Waldman SD. Tennis elbow. In: *Atlas of common pain syndromes*. 4th ed. Philadelphia: Elsevier; 2019:149–152.

Waldman SD. Tennis elbow. In: *Pain review*. 2nd ed. Philadelphia: Saunders Elsevier; 2017:250–251.

Waldman SD. Tennis elbow. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:250–251.

Waldman SD. Tennis elbow and other abnormalities of the elbow. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:216–225.

Waldman SD. The tennis elbow test. In: *Physical diagnosis of pain. An atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:130–132.

Waldman SD, Campbell RSD. Tennis elbow. In: *Imaging of pain*. Philadelphia: Saunders; 2011:261–262.

Golfer's Elbow

ICD-10 CODE M77.00

THE CLINICAL SYNDROME

Golfer's elbow (also known as medial epicondylitis) is caused by repetitive microtrauma to the flexor tendons of the forearm in a manner analogous to tennis elbow. The pathophysiology of golfer's elbow initially involves microtearing at the origin of the pronator teres, flexor carpi radialis, flexor carpi ulnaris, and palmaris longus (Fig. 39.1). Secondary inflammation may become chronic as a result of continued overuse or misuse of the flexors of the forearm. The most common nidus of pain from golfer's elbow is the bony origin of the flexor tendon of the flexor carpi radialis and the humeral heads of the flexor carpi ulnaris and pronator teres at the medial epicondyle of the humerus. Less commonly, golfer's elbow pain originates from the ulnar head of the flexor carpi ulnaris at the medial aspect of the olecranon process. Coexistent bursitis, arthritis, or gout may perpetuate the pain and disability of golfer's elbow.

Golfer's elbow occurs in individuals engaged in repetitive flexion activities, such as throwing baseballs or footballs, carrying heavy suitcases, and driving golf balls. These activities have in common repetitive flexion of the wrist and strain on the flexor tendons resulting from excessive weight or sudden arrested motion. Many of the activities that cause tennis elbow can also cause golfer's elbow.

SIGNS AND SYMPTOMS

The pain of golfer's elbow is localized to the region of the medial epicondyle (Fig. 39.2). This pain is constant and is made worse with active contraction of the wrist. Patients note the inability to hold a coffee cup or use a hammer. Sleep disturbance is common. On physical examination, tenderness is elicited along the flexor tendons at or just below the medial epicondyle (Fig. 39.3). Many patients with golfer's elbow exhibit a bandlike thickening within the affected flexor

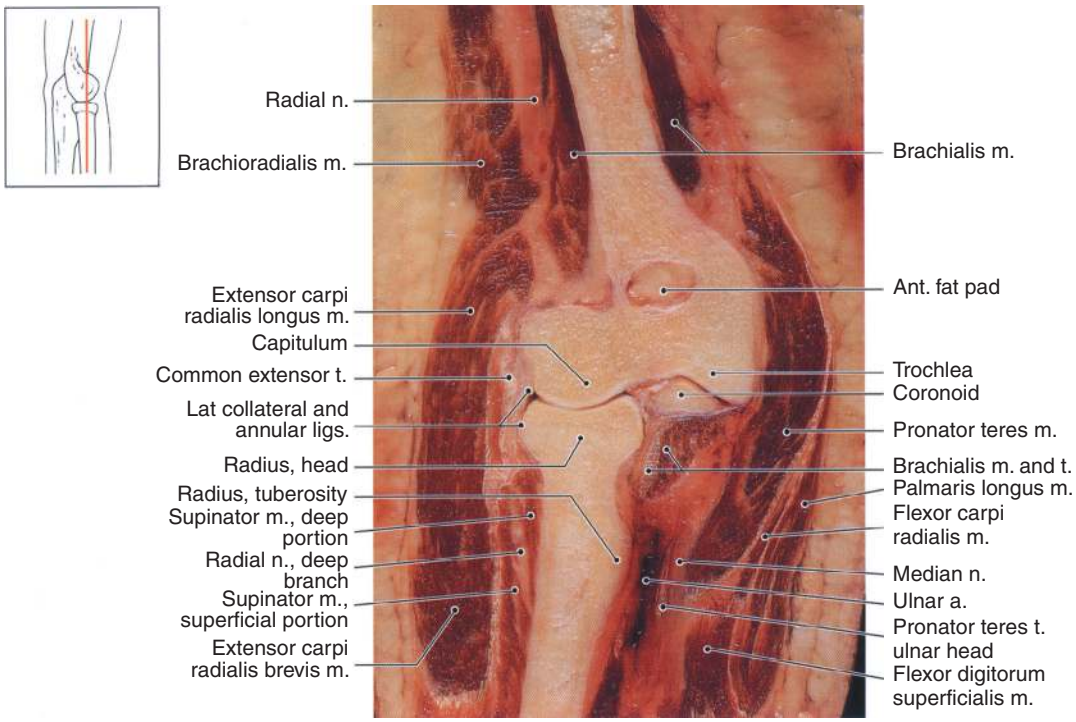

FIG 39.1 Origins of the pronator teres, flexor carpi radialis, flexor carpi ulnaris, palmaris longus, and medial epicondyle. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas.* 2nd ed. Philadelphia: Saunders; 2002:89.)

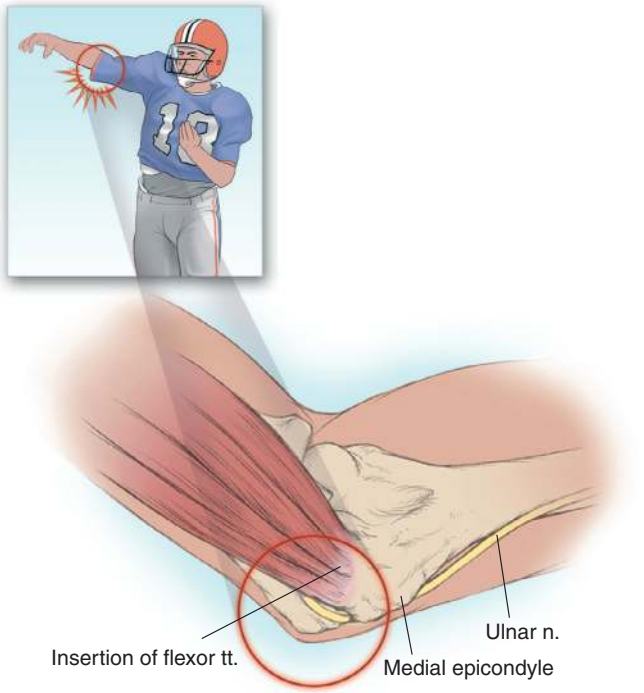

FIG 39.2 The pain of golfer's elbow occurs at the medial epicondyle.

FIG 39.3 Palpation of the medial epicondyle of the elbow will cause pain in patient's suffering from golfer's elbow.

tendons. Elbow range of motion is normal, but grip strength on the affected side is diminished. Patients with golfer's elbow have a positive golfer's elbow test result. This test is performed by stabilizing the patient's forearm and then having the patient actively flex the wrist. The examiner then attempts to force the wrist into extension (Fig. 39.4). Sudden severe pain is highly suggestive of golfer's elbow.

TESTING

Plain radiographs should be obtained in all patients who present with elbow pain to rule out joint mice and other occult bony disease. Based on the patient's clinical presentation,

FIG 39.4 Test for golfer's elbow. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms.* Philadelphia: Saunders; 2006:140.)

additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Ultrasound imaging will help quantify the extent of tendinopathy and identify other occult causes of the patient's pain symptomatology (Figs. 39.5 and 39.6). Magnetic resonance imaging of the elbow is indicated if joint instability is suspected or if the symptoms of golfer's elbow persists (Fig. 39.7). Electromyography (EMG) is indicated to diagnosis entrapment neuropathy at the elbow and to distinguish golfer's elbow from cervical radiculopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Occasionally, C6–C7 radiculopathy mimics golfer's elbow; however, patients suffering from cervical radiculopathy usually have neck pain and proximal upper extremity pain in addition to symptoms below the elbow. As noted earlier, EMG can distinguish radiculopathy from golfer's elbow. Bursitis, arthritis, and gout may also mimic golfer's elbow, thus confusing the diagnosis. The olecranon bursa lies in the posterior aspect of the elbow joint and may become inflamed as a result of direct trauma to the joint or its overuse. Other bursae susceptible to the development of bursitis are located between the insertion of the biceps and the head of the radius, as well as in the antecubital and cubital areas.

TREATMENT

Initial treatment of the pain and functional disability associated with golfer's elbow includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities,

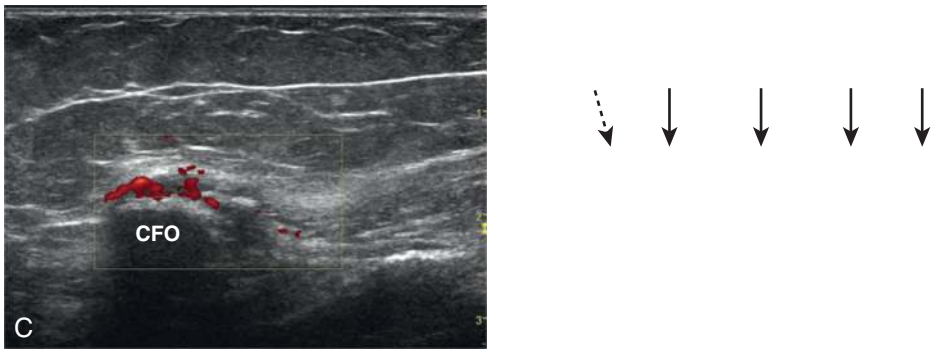

FIG 39.5 A, Radiograph of a middle-aged woman with golfer's elbow demonstrates a few small areas of calcification in the CFO adjacent to the medial epicondyle. B and C, The corresponding ultrasound (US) images show an echo-poor tendon with small echogenic foci of calcification *(white arrows)* and neovascularization, as evident by increased blood flow on Doppler imaging (C) consistent with tendinopathy. D, US-guided injection and dry needling shows the needle *(black arrows)* with the tip adjacent to the areas of calcification *(broken black arrow)*. *CFO*, Common flexor origin. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011.)

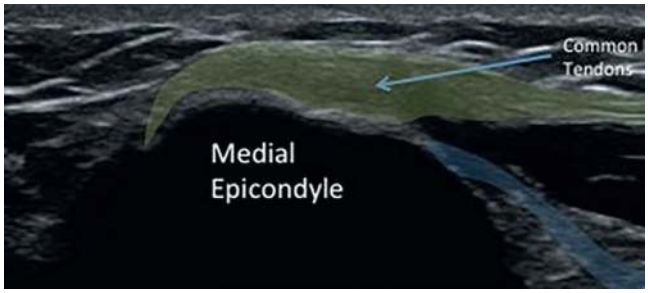

FIG 39.6 Longitudinal ultrasound image of common flexor tendons.

injection with local anesthetic and steroid is a reasonable next step.

Injection for golfer's elbow is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow fully extended, and the dorsum of the hand resting on a folded towel to relax the affected tendons. A total of 1 mL local anesthetic and 40 mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the medial aspect of the joint, the medial epicondyle is identified. Using strict aseptic technique, a 1-inch, 25-gauge needle is inserted perpendicular to the medial epicondyle through the skin and into the

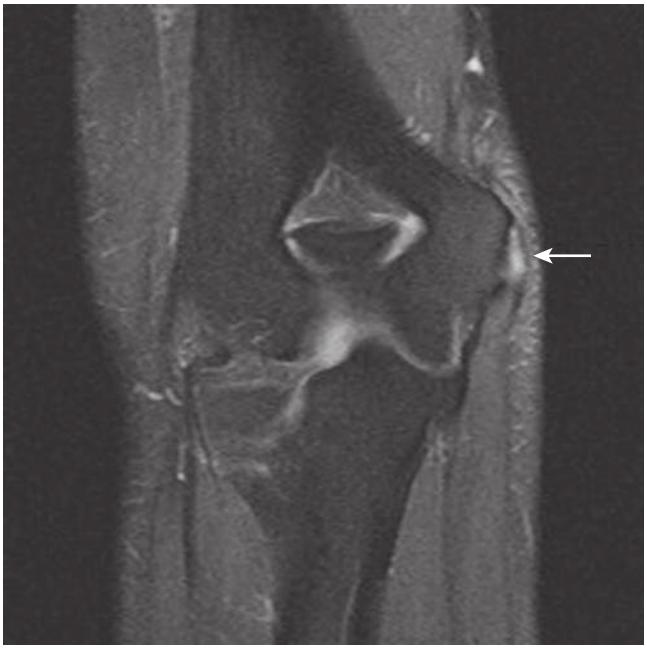

FIG 39.7 T2-coronal magnetic resonance image of medial epicondylitis with a pathologic increase in signal intensity at the origin of the flexor-pronator mass *(arrow)*. (From Van Hofwegen C, Baker CL III, Baker CL Jr. Epicondylitis in the athlete's elbow. *Clin Sports Med*. 2010;29(4):577–597.)

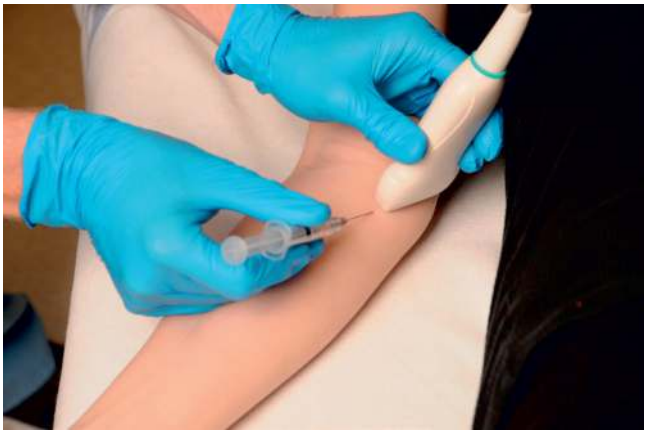

FIG 39.8 Proper needle placement for ultrasound-guided golfer's elbow injection.

subcutaneous tissue overlying the affected tendon. If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. Little resistance to injection should be felt. If significant resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can proceed with less resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that the injection of type A botulinum toxin and platelet-rich plasma and/or stem cells may provide improved symptom relief and healing of golfer's elbow. Ultrasound guidance may improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify (Fig. 39.8).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for elbow pain. A Velcro band placed around the flexor tendons may also help relieve the symptoms. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The major complications associated with this injection technique are related to trauma to the inflamed and previously damaged tendon, which may rupture if injected directly. Therefore, the needle position should be confirmed to be outside the tendon before the clinician proceeds with the injection. Another complication of the injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Injection is safe if careful attention is paid to the clinically relevant anatomy; in particular, the ulnar nerve is susceptible to damage at the elbow. Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the elbow joint, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to golfer's elbow. Coexistent bursitis and tendinitis may contribute to elbow pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with this injection technique. Cervical radiculopathy may mimic golfer's elbow and must be excluded.

SUGGESTED READINGS

McMurtrie A, Watts AC. Tennis elbow and golfer's elbow. *Orthop Trauma*. 2012;26(5):337–344.

Pitzer ME, Seidenberg PH, Bader DA. Elbow tendinopathy. *Med Clin North Am*. 2014;98(4):833–849.

Vinod AV, Ross G. An effective approach to diagnosis and surgical repair of refractory medial epicondylitis. *J Shoulder Elb Surg*. 2015;24(8):1172–1177.

Waldman SD. Golfer's elbow and other abnormalities of the medial elbow. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:295–303.

Waldman SD. Golfer's elbow. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:251–252.

Waldman SD. Medial epicondyle injection for golfer's elbow. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:198–202.

Waldman SD, Campbell RSD. Golfer's elbow. In: *Imaging of pain*. Philadelphia: Saunders; 2011:263–264.

40

Distal Biceps Tendon Tear

ICD-10 CODE S53.499A

THE CLINICAL SYNDROME

Rupture of the distal tendon of the biceps occurs much less frequently than rupture of the long head of the biceps. Proximal rupture of the tendon of the long head of the biceps tendon accounts for more than 97% of biceps tendon ruptures, whereas ruptures of the distal portion of the biceps tendon occur less than 3% of the time. Occurring most commonly in men in the fourth to sixth decades, disruption of the distal biceps tendon is usually the result of an acute traumatic event secondary to a sudden eccentric load on the tendon, such as trying to start a recalcitrant lawn mower, practicing an overhead tennis serve, lifting weights, or performing overaggressive follow-through when driving golf balls (Fig. 40.1). Falls on a flexed and supinated elbow have also been associated with tears and rupture of the distal biceps tendon, as has abuse of anabolic steroids in athletes.

The biceps muscle and proximal and distal tendons are intimately involved in shoulder and elbow function and are susceptible to trauma and to wear and tear. If the damage is severe enough, the distal tendon of the biceps can rupture, leaving the patient with a palpable defect in the antecubital fossa and weakness of upper extremity flexion and supination (Figs. 40.2 and 40.3).

SIGNS AND SYMPTOMS

In most patients, the pain of distal biceps tendon tear occurs acutely, is often quite severe, and is accompanied by a pop or snapping sound. The pain is constant and severe and is localized to the region surrounding the antecubital fossa. Patients with complete distal biceps tendon tear experience weakness of upper extremity flexion and supination. An obvious defect is palpable in the antecubital fossa in patients with complete

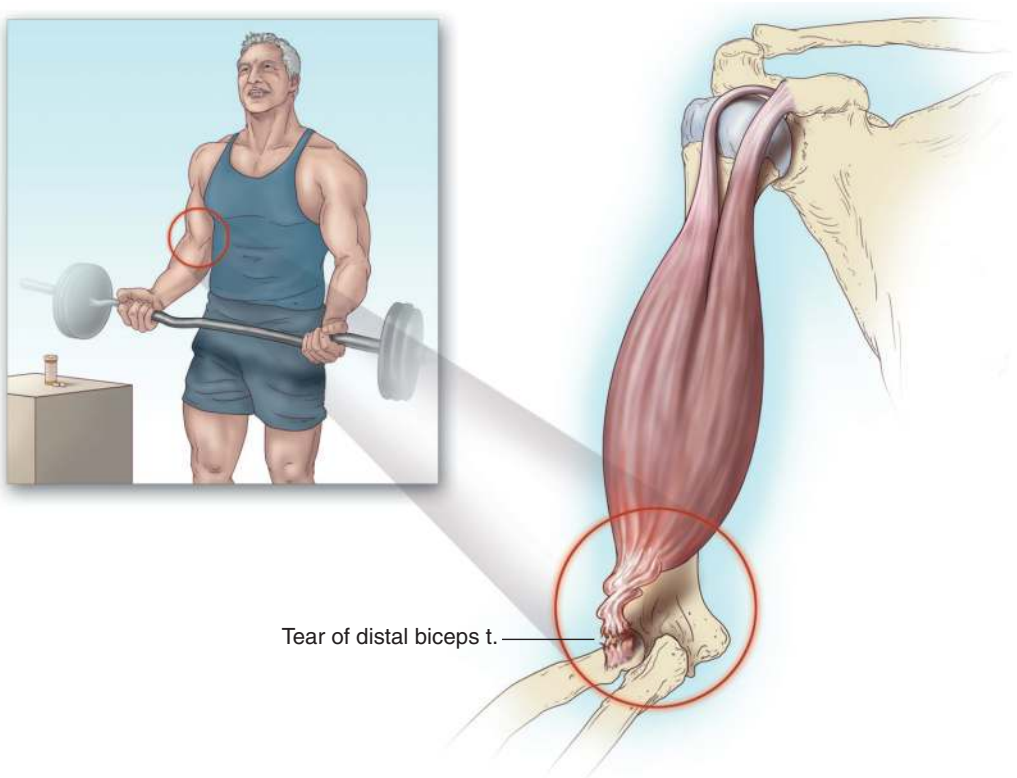

FIG 40.1 Patients with tears of the distal biceps tendon experience weakness of flexion and supination of the affected extremity.

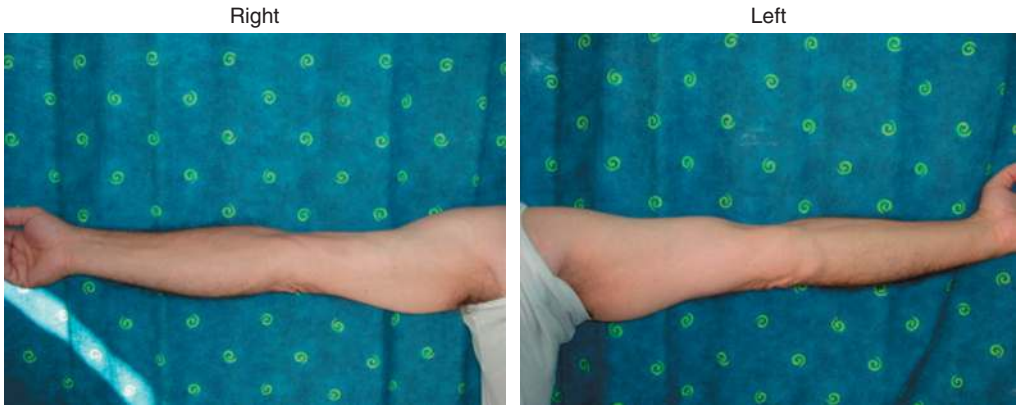

FIG 40.2 A 49-year-old man, 2 years following nonoperative management of a complete avulsion of the right distal biceps tendon. A deficit of distal biceps brachii mass in the right distal arm is clearly seen. (From Hetsroni I, Pilz-Burstein R, Nyska M, et al. Avulsion of the distal biceps brachii tendon in middle-aged population: is surgical repair advisable? A comparative study of 22 patients treated with either nonoperative management or early anatomical repair. *Injury*. 2008;39(7):753–760.)

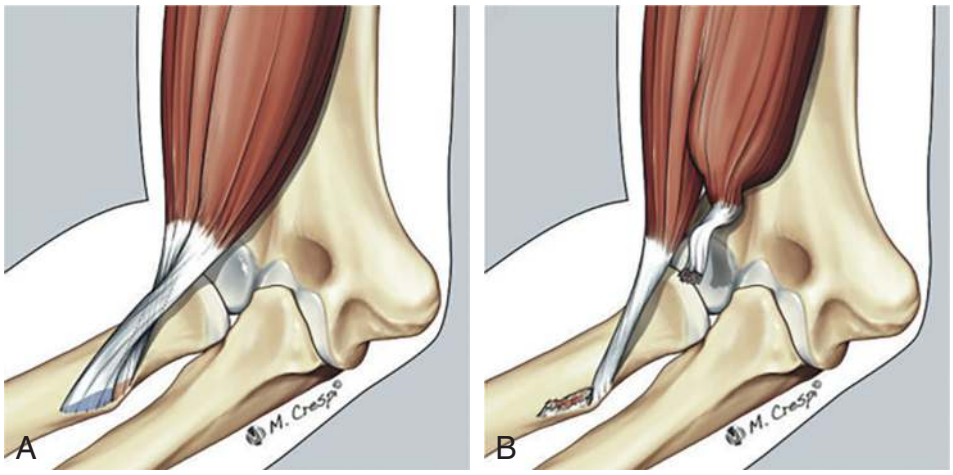

FIG 40.3 A, Illustration demonstrating insertion of a bifid distal biceps tendon on the radial tuberosity. The short head is distal to the long head and occupies a larger footprint area. B, With an eccentric flexion injury, it is postulated that the distal part of the tendon ruptures first and because of the bifid arrangement the force propagates between the long and short head, leaving the long head intact. (From Iqbal K, Leung B, Phadnis J. Distal biceps short head tears: repair, reconstruction, and systematic review. *J Shoulder Elb Surg*. 2020;29(11):2353–2363.)

rupture of the distal biceps tendon (Fig. 40.4). A reverse Popeye sign is usually present (see Fig. 40.2).

TESTING

Plain radiographs are indicated for all patients who present with elbow pain. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Ultrasound imaging may help further delineate the extent of tendinopathy and identify other abnormalities responsible for the patient's pain and functional disability (Fig. 40.5). Magnetic resonance imaging of the elbow is indicated if tendinopathy or if partial tear or complete rupture of the biceps tendon is suspected (Figs. 40.6 and 40.7).

DIFFERENTIAL DIAGNOSIS

Tear of the distal biceps tendon is usually a straightforward clinical diagnosis. However, coexisting bursitis or tendinitis of the elbow from overuse or misuse may confuse the diagnosis. In some clinical situations, consideration should be given to primary or secondary tumors involving the elbow. Nerve entrapments of the elbow and forearm can also complicate the diagnosis.

TREATMENT

Initial treatment of the pain and functional disability associated with distal biceps tendon tear includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy.

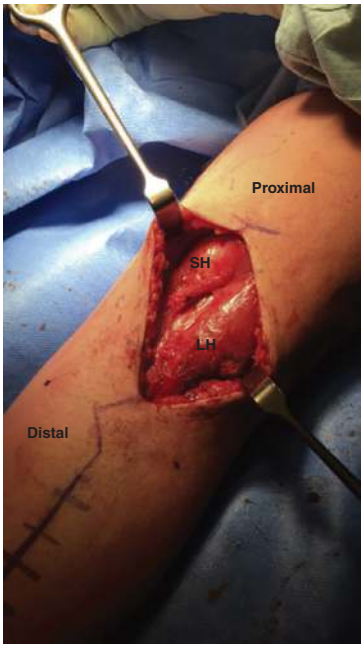

FIG 40.4 Intraoperative image of chronic short head rupture with intact long head (LH) and retraction of short head (SH) and tethering to surrounding scar tissue. On passive forearm rotation, there was normal motion and excursion of the LH muscle belly and tendon with no movement of the SH muscle belly, confirming complete rupture and retraction. (From Iqbal K, Leung B, Phadnis J. Distal biceps short head tears: repair, reconstruction, and systematic review. *J Shoulder Elb Surg*. 2020;29(11):2353–2363.)

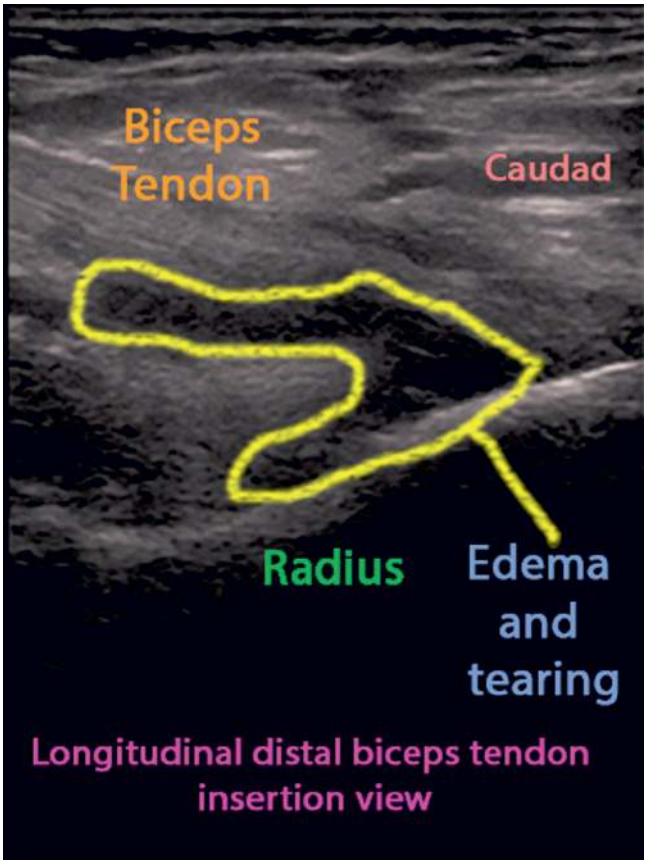

FIG 40.5 Longitudinal ultrasound image demonstrating tearing and edema of the distal biceps tendinous insertion.

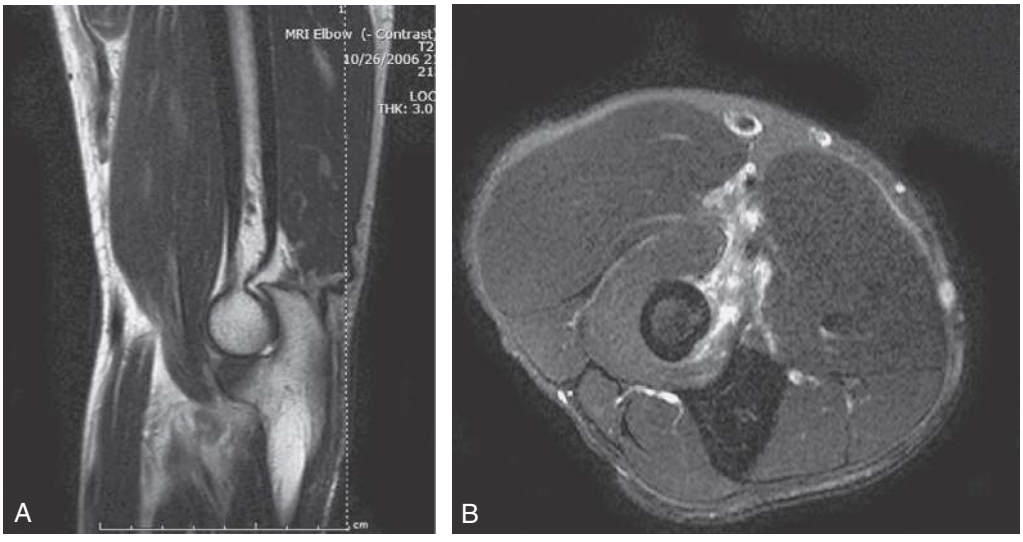

FIG 40.6 T2-weighted magnetic resonance imaging of a distal biceps tendon tear. **A,** Sagittal image with a retracted tendon stump. **B,** Axial image with visible hemorrhage surrounding the insertion site at the bicipital tuberosity of the proximal radius. (Courtesy Garcia GM, Department of Radiology, University of Texas Health Science Center, San Antonio. From Huber FG. Arm. In: DeLee JC, Drez DD, Miller M, eds. *DeLee and Drez's orthopaedic sports medicine: principles and practice*. 3rd ed. Philadelphia: Saunders; 2010:1169.)

Local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities and who appear to have significant local pain in the region of the distal biceps tendon, careful injection with local anesthetic and steroid is a reasonable next step.

Injection for distal biceps tendon tear is carried out by placing the patient in the sitting position with the elbow flexed to approximately 90 degrees. If intact, the distal biceps tendon is easily identified by palpation at the antecubital fossa. If the tendon is absent, the area of defect is identified.

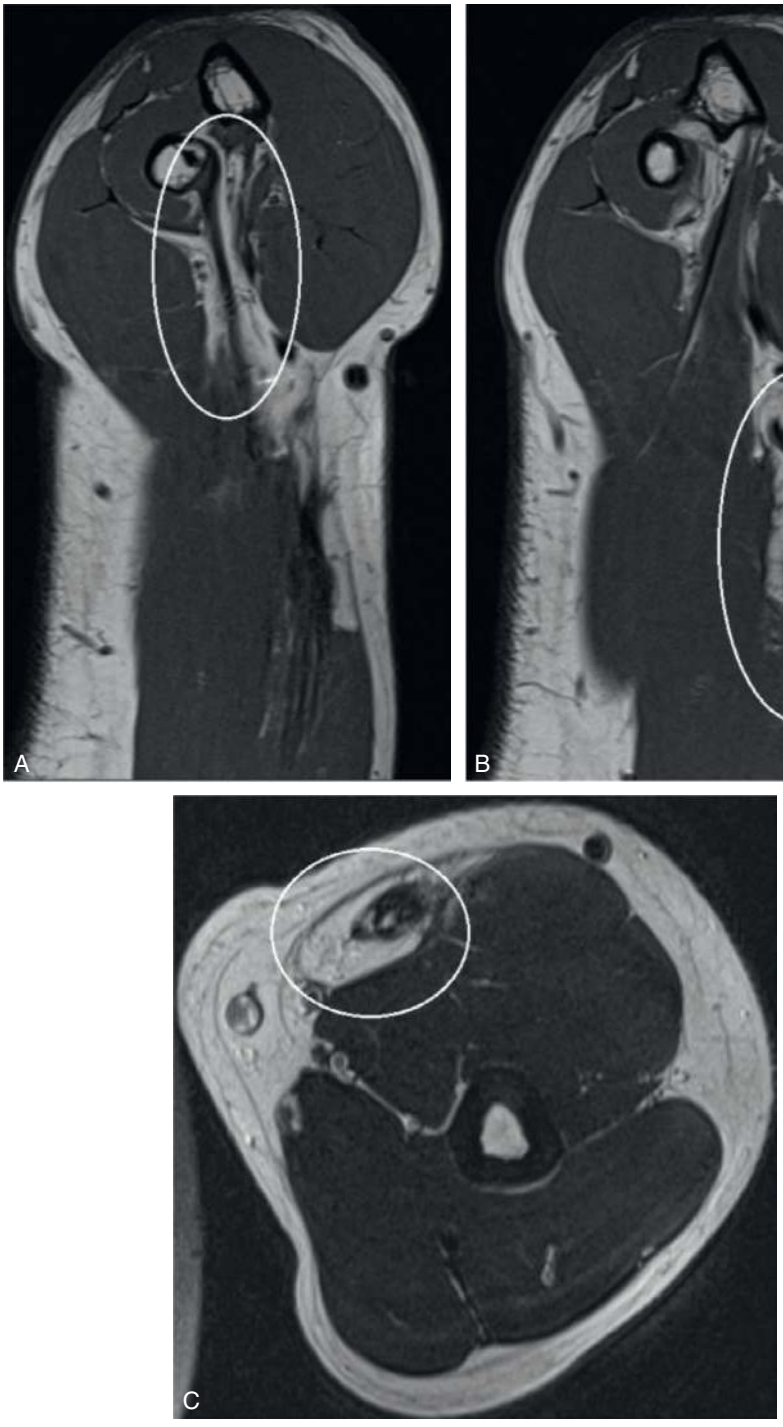

FIG 40.7 MRI FABS view of patient with chronic short head rupture: **A,** intact long head tendon seen inserting into bicipital tuberosity; **B,** significant retraction of short head musculotendinous unit proximally; **C,** axial slices demonstrating two heads visible as separate entities with fat surrounding the medial short head. *MRI*, Magnetic resonance imaging; *FABS*, flexion, abduction external rotation protocol. (From Iqbal K, Leung B, Phadnis J. Distal biceps short head tears: repair, reconstruction, and systematic review. *J Shoulder Elb Surg*. 2020;29(11):2353–2363.)

The point overlying the distal tendon or defect is marked with a sterile marker. The skin-overlying antecubital fossa is then prepared with antiseptic solution. A sterile syringe containing 1 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 11/12-inch, 25-gauge needle using strict aseptic technique. The previously marked point is palpated, and the distal biceps tendon or area of defect is reidentified with the gloved finger. The needle is carefully advanced at this point through the skin and subcutaneous tissues until it impinges on the distal biceps tendon or enters the area of defect. The needle is then withdrawn 1–2 mm out of the substance of the tendon, and the contents

of the syringe are gently injected. Slight resistance to injection should be felt. If no resistance is encountered, the tendon is ruptured. If resistance is significant, the needle tip is probably in the substance of the tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that the injection of type A botulinum toxin and platelet-rich plasma and/or stem cells may provide improved symptom relief and healing of tendinopathy of the distal biceps tendon. Ultrasound guidance may improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Occasionally, surgical repair of the tendon is undertaken if the patient is experiencing significant functional disability or is unhappy with the cosmetic defect caused by the retracted tendon and muscle.

COMPLICATIONS AND PITFALLS

This injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of this injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Trauma to the distal biceps tendon from the injection itself is also a possibility. Tendons that are highly inflamed or previously damaged are subject to rupture if they are injected directly. This complication can often be avoided if the clinician uses a gentle technique and stops injecting immediately if significant resistance is encountered. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The distal biceps tendon ruptures much less commonly than the proximal long head of the biceps tendon, although the forces that can cause either end of the tendon to rupture are similar. The injection technique described is extremely effective in the treatment of pain secondary to biceps tendon tear. Coexistent bursitis and arthritis may contribute to shoulder pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs or COX-2 inhibitors can be used concurrently with this injection technique.

SUGGESTED READINGS

Bhatia DN, Kandhari V, DasGupta B. Cadaveric study of insertional anatomy of distal biceps tendon and its relationship to the dynamic proximal radioulnar space. *J Hand Surg Am*. 2017;42(1):e15–e23.

Caekebeke P, Schenkels E, Bell SN, van Riet R. Distal biceps provocation test. A novel clinical test for partial tears of the distal biceps brachii tendon: the TILT sign. *J Hand Surg Am*. 2021;46:710.e1–710.e4.

Iqbal K, Leung B, Phadnis J. Distal biceps short head tears: repair, reconstruction, and systematic review. *J Shoulder Elb Surg*. 2020;29(11):2353–2363.

MacInnes SJ, Crawford LA, Shahane SA. Disorders of the biceps and triceps tendons at the elbow. *Orthop Trauma*. 2016;30(4):346–354.

McFarland EG, Borade A. Examination of the biceps tendon. *Clin Sports Med*. 2016;35(1):29–45.

Nicolay RW, Lawton CD, Selley RS, Johnson DJ, Vassa RR, Prescott AE, Omar IM, Marra G. Partial rupture of the distal biceps brachii tendon: a magnetic resonance imaging analysis. *J Shoulder Elb Surg*. 2020;29(9):1859–1868. https://doi.org/10.1016/j.jse.2020.04.021.

Pallante GD, O'Driscoll SW. Return of an intact hook test result: clinical assessment of biceps tendon integrity after surgical repair. *Orthop J Sports Med*. 2019;7(2):2325967119827311. https://doi.org/10.1177/2325967119827311.

Pitsilos C, Gigis I, Chitas K, Papadopoulos P, Ditsios K. Systematic review of distal biceps tendon rupture in athletes: treatment and rehabilitation. *J Shoulder Elb Surg*. 2022;31(8):1763–1772. https://doi.org/10.1016/j.jse.2022.02.027.

Stoll LE, Huang JI. Surgical treatment of distal biceps ruptures. *Orthop Clin North Am*. 2016;47(1):189–205.

Thomas JR, Lawton JN. Biceps and triceps ruptures in athletes. *Hand Clin*. 2017;33(1):35–46.

Vestermark GL, Van Doren BA, Connor PM, Fleischli JE, Piasecki DP, Hamid N. The prevalence of rotator cuff pathology in the setting of acute proximal biceps tendon rupture. *J Shoulder Elb Surg*. 2018;27(7):1258–1262.

Virk MS, Cole BJ. Proximal biceps tendon and rotator cuff tears. *Clin Sports Med*. 2016;35(1):153–161. https://doi.org/10.1016/j.jse.2018.01.006.

Waldman SD, Campbell RSD. Anatomy: special considerations of the elbow. In: *Imaging of Pain*. Philadelphia: Saunders; 2011:263–264.

Waldman SD, Campbell RSD. Biceps tendinopathy. In: *Imaging of pain*. Philadelphia: Saunders; 2011:245–246.

Waldman SD. Bicipital tendinitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017: 114–117.

Waldman SD. Disorders of the distal biceps tendon and bicipitoradial bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:304–315.

Waldman SD. The biceps tendon. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:91–92.

Thrower's Elbow

ICD-10 CODE M24.829

THE CLINICAL SYNDROME

Thrower's elbow is a valgus stress overload syndrome caused by continual microtrauma to the medial and lateral elbow from a repetitive throwing motion. Also known as little leaguer's elbow, the pathophysiology of thrower's elbow initially involves damage secondary to significant valgus stress placed in the medial structures and compression of the lateral structures of the elbow during throwing activities. The medial epicondyle, medial collateral ligaments, and the medial epicondylar apophysis are especially susceptible to this repetitive stress, and ongoing tissue damage often exceeds the ability of the athlete's body to repair the damage (see Fig. 39.1). When this occurs, the result is acute, localized, medial elbow pain combined with decreased throwing accuracy and throwing distance.

Thrower's elbow is the name given to the constellation of symptoms, rather than a single pathologic process, that results from this repetitive microtrauma to the elbow. Contributing to this symptom complex are medial epicondylitis (golfer's elbow), growth abnormalities of the medial epicondyle (medial epicondylar apophysitis), medial epicondylar fragmentation, stress fractures involving the medial epicondylar epiphysis, and avulsion fractures of the medial epicondyle. In addition, the findings of osteochondrosis of the humeral capitellum, osteochondritis dissecans of the humeral capitellum, osteochondritis of the radial head, hypertrophy of the ulna, traction apophysitis of the olecranon, triceps tendinitis, and mild instability of the ulnar collateral ligament complex may be observed alone or in combination with the foregoing pathologic processes. Less commonly, nerve entrapment syndromes and subluxation of the ulnar nerve can also occur (Box 41.1).

BOX 41.1 Pathology Contributing to Thrower's Elbow

- Medial epicondylitis (golfer's elbow)
- Medial epicondylar apophysitis
- Medial epicondylar fragmentation
- Stress fractures involving the medial epicondylar epiphysis
- Avulsion fractures of the medial epicondyle
- Osteochondrosis of the humeral capitellum
- Osteochondritis dissecans of the humeral capitellum
- Osteochondritis of the radial head
- Hypertrophy of the ulna
- Traction apophysitis of the olecranon
- Triceps tendinitis
- Mild instability of the ulnar collateral ligament complex

SIGNS AND SYMPTOMS

The pain of thrower's elbow almost always includes pain localized to the region of the medial epicondyle in a manner analogous to golfer's elbow (Fig. 41.1). The patient may note the inability to hold a coffee cup or use a hammer, and the examiner may notice reduced grip strength. Sleep disturbance is common.

Other symptoms are the result of the other specific pathologic processes at play at the time of the examination. Patients suffering from thrower's elbow may exhibit mild instability of the ulnar collateral ligament complex caused by repetitive stretch injuries, as well as a decreased ability to extend the elbow fully. Active compression across the radiocapitellar joint from muscular forces may reproduce the patient's pain, as will an active radiocapitellar compression test, which is performed by having the patient pronate and supinate the forearm in full extension (Fig. 41.2).

Physical examination may also reveal localized tenderness along the flexor tendons at or just below the medial epicondyle. If the patient has an acute injury to the elbow, swelling and ecchymosis may be present. Increased valgus angle greater than 11 degrees in male patients and 13 degrees in female patients may also be noted. Flexion contracture may be present and results in a loss of full elbow extension (Fig. 41.3). In some high-performing athletes, these range-of-motion abnormalities represent adaptive changes and are not often the sole cause of the patient's pain symptoms. Palpation of the ulnar collateral ligament may reveal tenderness to palpation or complete disruption (Fig. 41.4A). Valgus instability in the patient suspected of suffering from thrower's elbow can best be assessed by performing the milking maneuver of Veltri, which is done by grasping the thrower's thumb with the arm in the fully cocked position (90 degrees of shoulder abduction and 90 degrees of elbow flexion) and then applying valgus stress by pulling down on the thumb (see Fig. 41.4B). The test result is considered positive if the patient's pain is reproduced.

The point at which the patient notes the onset of pain during the five-step throwing sequence may give the clinician a clue to the pathologic process primarily responsible

FIG 41.1 Adult male pitcher at the beginning (A) and end (B) of the late cocking phase of the throwing motion. This phase begins as the foot contacts the ground and ends as the arm reaches maximal external rotation. (From DeLee JC, Drez DD, Miller M, eds. *DeLee and Drez's orthopaedic sports medicine: principles and practice*. 3rd ed. Philadelphia: Saunders; 2010:1215.)

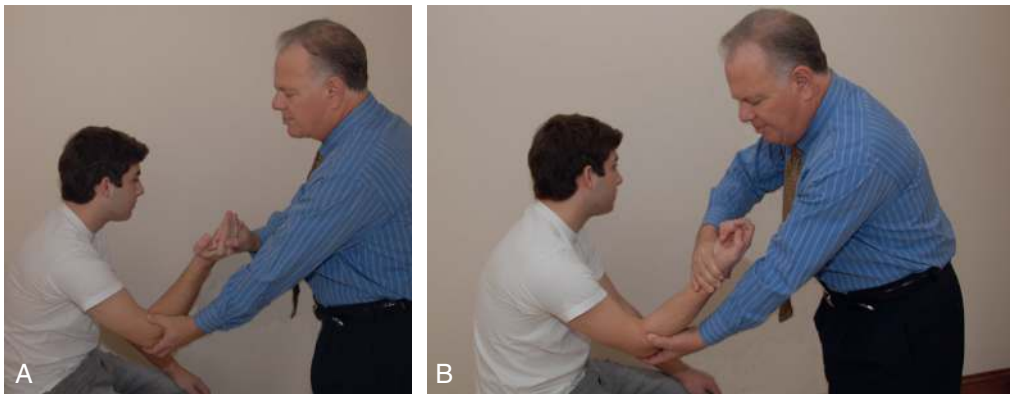

FIG 41.2 The active radiocapitellar compression test is performed by having the patient pronate and supinate the forearm in full extension. **A,** The wrist of the affected extremity is extended and deviated radially. **B,** The radiocapitellar joint is then compressed while the patient's elbow is actively flexed and extended and the forearm is pronated and supinated.

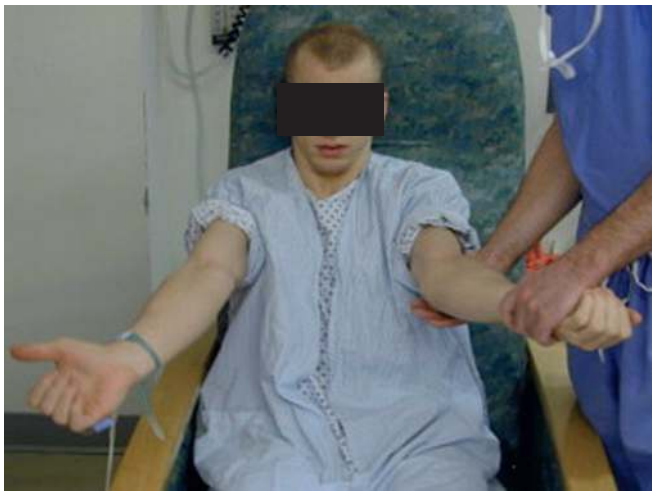

FIG 41.3 The patient is unable to extend his left elbow fully. (From Erne HC, Zouzias IC, Rosenwasser MP. Medial collateral ligament reconstruction in the baseball pitcher's elbow. *Hand Clin*. 2009;25(3):339–346.)

for the pain; this knowledge can be especially useful when many elbow abnormalities are occurring simultaneously (Fig. 41.5). If the patient's pain occurs primarily during the acceleration phase, the clinician should pay special attention to the ulnar collateral ligament complex because the pain is often the result of elbow instability caused by stretching injuries. If the pain occurs primarily during late deceleration, medial epicondylitis and, less commonly, an ulnar nerve disorder are often the cause. Pain during deceleration should alert the clinician to pay special attention to the posterior elements of the elbow because olecranon and triceps tendon abnormalities and joint mice may be the problem.

TESTING

Plain radiographs should be obtained in all patients who present with elbow pain, to rule out joint mice and other occult bony pathologic processes such as avulsion fractures of the

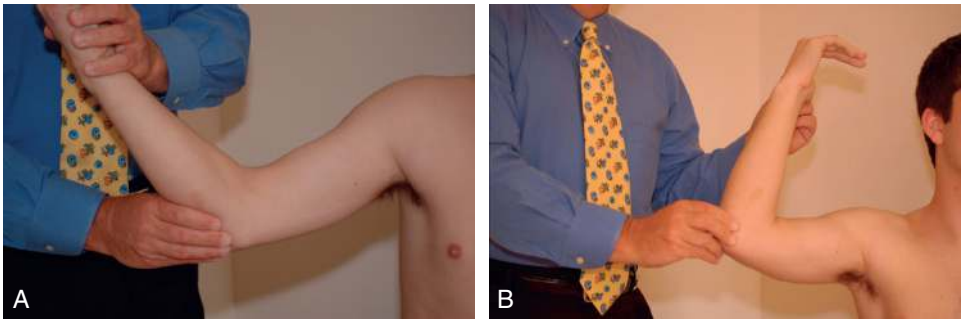

FIG 41.4 A, Palpation of the ulnar collateral ligament. Palpation of the anterior band of the ulnar collateral ligament is performed with the elbow in 70–90 degrees of flexion. B, The milking maneuver of Veltri is performed by grasping the thrower's thumb with the arm in the fully cocked position and then applying valgus stress by pulling down on the thumb in a manner analogous to milking a cow.

Phase 1: Windup	Begins with the pitcher balancing his or her weight over the rear leg, with the elbow flexed and the forward leg flexed at least 90 degrees.	
Phase 2: Early cocking/stride	Starts with the lead leg beginning to descend toward the plate, and the two arms separate. The elbow moves from extension into flexion of 80–100 degrees.	
Phase 3: Late cocking	Begins when the humerus is in extreme abduction and external rotation and the elbow is flexed. The lead foot contacts the ground, the pelvis and trunk rotate, and elbow torque transfers valgus force across the elbow joint. During this phase, medial tension and lateral compression forces are applied to the elbow.	
Phase 4: Acceleration/deceleration	Begins at ball release. Maximal external shoulder rotation occurs to release ball, with trunk rotating and the elbow rapidly extending. Ends when the shoulder has reached full internal rotation with the body decelerating the arm and dissipating the forces in the elbow and shoulder.	
Phase 5: Follow-through	Final phase of the baseball pitch and ends with the pitcher reaching a balanced fielding position with full-trunk rotation and the body weight fully transferred from the rear leg to the forward leg and the elbow flexing into a relaxed position.	

FIG 41.5 The throwing sequence. (Modified from DeLee JC, Drez DD, Miller M, eds. *DeLee and Drez's orthopaedic medicine: principles and practice*. 3rd ed. Philadelphia: Saunders; 2010:1232.)

olecranon (Fig. 41.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the elbow is indicated if joint instability is suspected or if the symptoms of thrower's elbow persist (Figs. 41.7 and 41.8). Electromyography (EMG) is indicated to diagnose entrapment neuropathy at the elbow and to rule out cervical radiculopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

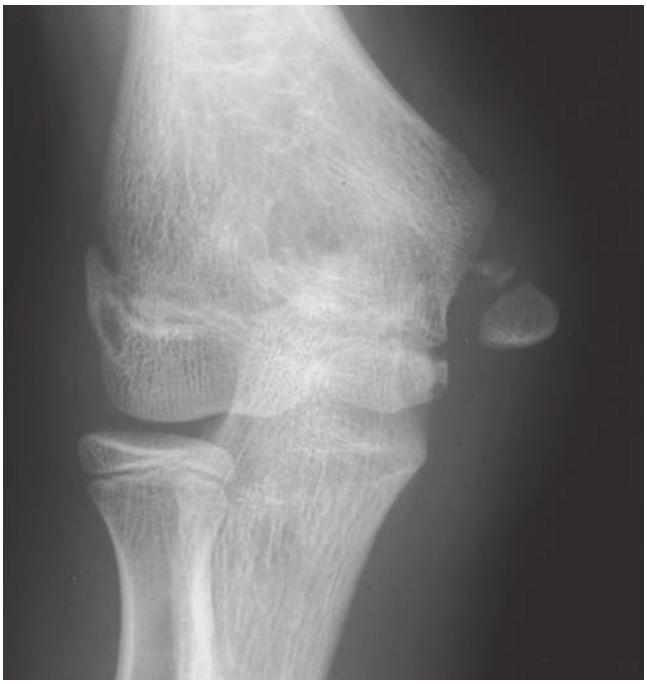

FIG 41.6 Anteroposterior (AP) radiograph demonstrating an acute avulsion injury of the medial epicondylar ossification center that has been displaced and requires surgical reattachment. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011.)

DIFFERENTIAL DIAGNOSIS

Occasionally, cervical radiculopathy mimics thrower's elbow; however, patients suffering from cervical radiculopathy usually have neck pain and proximal upper extremity pain in addition to symptoms below the elbow. As noted earlier, EMG can distinguish radiculopathy from thrower's elbow. Bursitis, arthritis, and gout may also mimic thrower's elbow and confuse the diagnosis. The olecranon bursa lies in the posterior aspect of the elbow joint and may become inflamed as a result of direct trauma to the joint or its overuse. Other bursae susceptible to the development of bursitis are located between the insertion of the biceps and the head of the radius, as well as in the antecubital and cubital areas.

TREATMENT

Initial treatment of the pain and functional disability associated with thrower's elbow includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

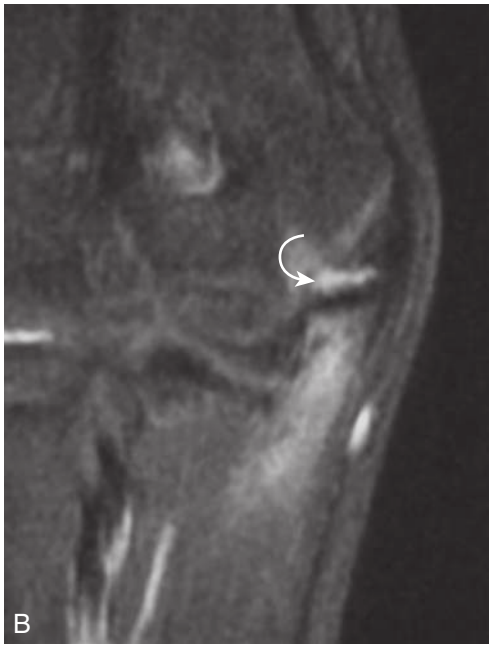

FIG 41.7 A and B, Consecutive coronal T2-weighted with fat suppression (FST2W) magnetic resonance (MR) images of the elbow in a child with recent strain injury of the medial epicondyle. The medial ossification center is not displaced *(white arrow)*, but there are distal soft tissue edema *(broken white arrow)* and a high-signal intensity cleft tear in the proximal flexor digitorum *(curved white arrow)*. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011.)

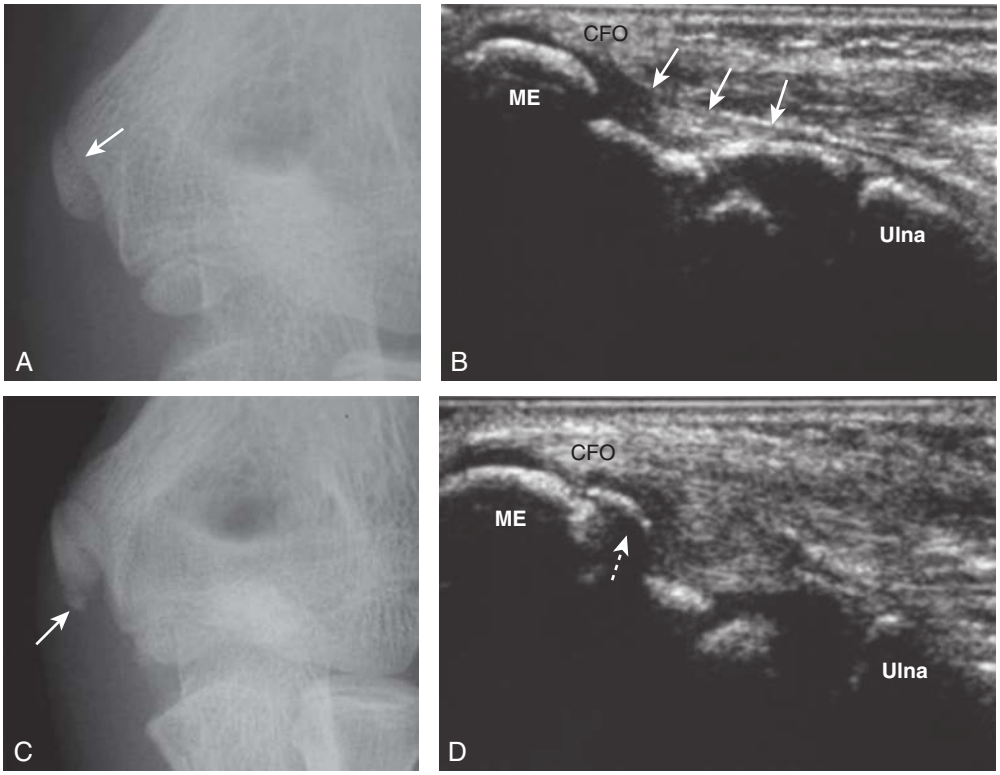

FIG 41.8 **A,** Anteroposterior (AP) radiograph of a normal medial epicondyle *(white arrow)*. **B,** The corresponding ultrasound (US) image shows the medial epicondyle (ME) and the echogenic common flexor origin (CFO). The ulnar collateral ligament (UCL) can be seen as a thin, echo-bright structure *(white arrows)* extending from the distal humerus and attaching to the ulna. **C,** An AP radiograph in a symptomatic child shows fragmentation of the ME *(white arrow)*. **D,** The corresponding longitudinal US image demonstrates the fragmented epicondyle *(broken white arrow)*. The UCL is not visualized because of a chronic tear. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011.)

Injection for thrower's elbow is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow fully extended, and the dorsum of the hand resting on a folded towel to relax the affected tendons. A total of 1 mL local anesthetic and 40 mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the medial aspect of the joint, the medial epicondyle is identified. Using strict aseptic technique, a 1-inch, 25-gauge needle is inserted perpendicular to the medial epicondyle through the skin and into the subcutaneous tissue overlying the affected tendon (Fig. 41.9). If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. Little resistance to injection should be felt. If significant resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can proceed with less resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. The injection of platelet-rich plasma and/or stem cells may improve symptom relief and healing of this pathologic condition.

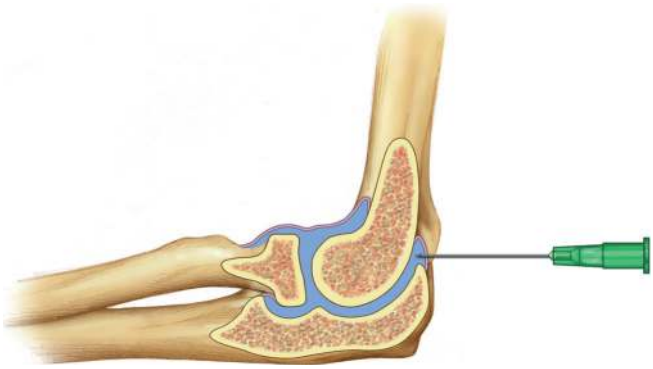

FIG 41.9 Elbow joint injection: posterior approach. A posterior puncture site between the medial and lateral epicondyles just proximal to the olecranon can be used to enter the elbow joint, although this is not recommended because the needle is not seen along its axis and is therefore more difficult to control precisely. (From Peterson JJ, Fenton DS, Czervionke LF. *Image-guided musculoskeletal interventions*. Philadelphia: Saunders; 2008:52.)

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for elbow pain. A Velcro band placed around the flexor tendons may also help relieve the symptoms. Occupational therapy for activities of daily living education may also be beneficial. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms, and return to play should not occur until the patient's symptoms are completely resolved.

COMPLICATIONS AND PITFALLS

The major complications associated with this injection technique are related to trauma to the inflamed and previously damaged tendon, which may rupture if it is injected directly. Therefore, the needle position should be confirmed to be outside the tendon before the clinician proceeds with the injection. Another complication of the injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Injection is safe if careful attention is paid to the clinically relevant anatomy; in particular, the ulnar nerve is susceptible to damage at the elbow. Approximately 25% of patients complain of a transient increase in pain after this injection technique, and patients should be warned of this possibility.

CLINICAL PEARLS

A complete understanding of the biomechanics of overhead throwing is essential if the clinician is to diagnose and treat thrower's elbow successfully. The injection technique described is extremely effective in the treatment of pain secondary to thrower's elbow. Coexistent bursitis and tendinitis may contribute to elbow pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with this injection technique. Cervical radiculopathy may mimic thrower's elbow and must be excluded.

SUGGESTED READINGS

Ciccotti MC, Ciccotti MG. Exam and imaging of the throwing elbow. *Oper Tech Sports Med*. 2020;28(2):150732. https://doi.org/10.1016/j.otsm.2020.150732.

Frantz T, Frangiamore SJ, Schickendantz MS. Anatomical considerations of throwing elbow injuries. *Oper Tech Sports Med*. 2020;28(2):150731. https://doi.org/10.1016/j.otsm.2020.150731.

Gehrman MD, Grandizio LC. Elbow ulnar collateral ligament injuries in throwing athletes: diagnosis and management. *J Hand Surg*. 2022;47(3):266–273. https://doi.org/10.1016/j.jhsa.2021.11.026.

Kurokawa D, Muraki T, Ishikawa H, et al. The Influence of pitch velocity on medial elbow pain and medial epicondyle abnormality among youth baseball players. *Am J Sports Med*. 2020;48:1601–1607.

Read PJ, Morrison WB. Imaging injuries in throwing sports beyond the typical shoulder and elbow pathologies. *Radiol Clin North Am*. 2016;54(5):857–864.

Sakata J, Tamaki T, Kishino A, et al. Risk factors for throwing elbow injuries during pitching analyzed by simulation using human musculoskeletal model in youth baseball pitchers. *J Shoulder Elb Surg*. 2021;30(6):1309–1315. https://doi.org/10.1016/j.jse.2021.01.039.

Sundaram N, Stacy GS. MR imaging findings of the throwing elbow. *Clin Radiol*. 2020;75(2):e18–e19.

Waldman SD. Golfer's elbow and other abnormalities of the medical elbow joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:295–303.

Waldman SD, Campbell RSD. Little leaguer's elbow. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:265–267.

Anconeus Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The anconeus muscle is susceptible to the development of myofascial pain syndrome. Such pain is most often the result of repetitive microtrauma to the muscle caused by such activities as prolonged ironing, handshaking, or digging (Fig. 42.1). Tennis injuries caused by an improper one-handed backhand technique have also been implicated as an inciting factor in myofascial pain syndrome, as has blunt trauma to the muscle.

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the anconeus muscle often have referred pain in the ipsilateral forearm.

The trigger point is pathognomonic of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, is often seen and is characteristic of myofascial pain syndrome. Patients with anconeus syndrome have a trigger point over the superior insertion of the muscle (Fig. 42.2).

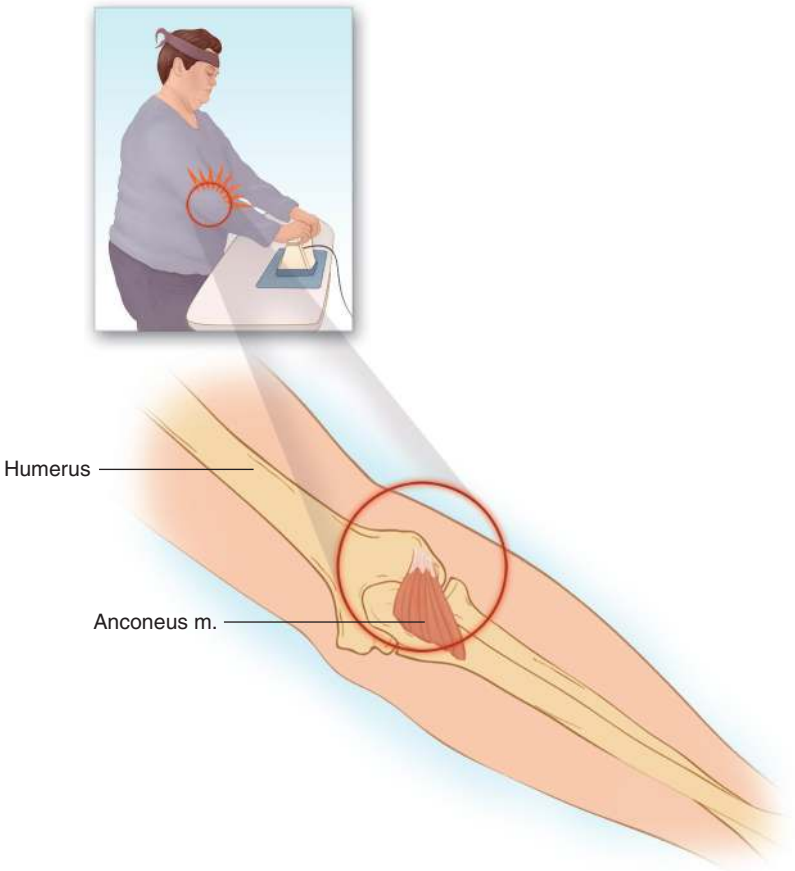

FIG 42.1 Myofascial pain syndrome affecting the anconeus muscle usually results from repetitive microtrauma from activities such as prolonged ironing.

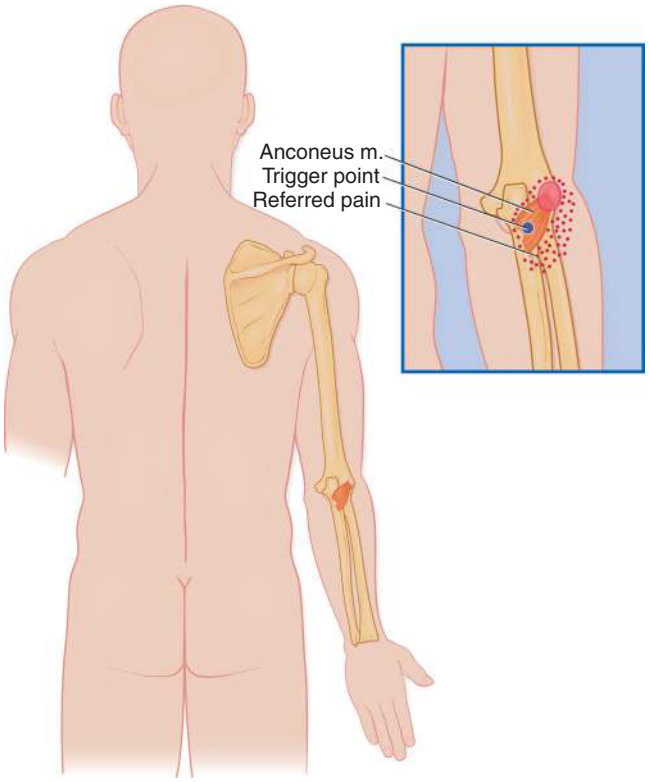

FIG 42.2 Patients with anconeus syndrome have a trigger point over the superior insertion of the muscle. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:134.)

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to result from microtrauma to the affected muscle. This trauma may result from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The anconeus muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point is the pathologic lesion of anconeus syndrome, and it is characterized by a local point of exquisite tenderness over the superior insertion of the muscle (see Fig. 42.2). Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain and referred pain. The jump sign is also characteristic of anconeus syndrome, as is pain over the anconeus muscle that is referred to the ipsilateral forearm.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with anconeus syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing of patients suffering from anconeus syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic anconeus syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of anconeus syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from anconeus syndrome. The anconeus muscle is susceptible to the development of a compartment syndrome, and this possibility should be considered in the differential diagnosis (Fig. 42.3). The clinician must rule out other coexisting disease processes that may mimic anconeus syndrome, including primary inflammatory muscle disease, multiple sclerosis, and collagen vascular disease. Electrodiagnostic and radiographic testing can help identify coexisting disorders such as bursitis, tendinitis, and epicondylitis (Fig. 42.4). The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with anconeus syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger point and achieving prolonged relaxation of the affected muscle.

Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from anconeus syndrome, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor, has also shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

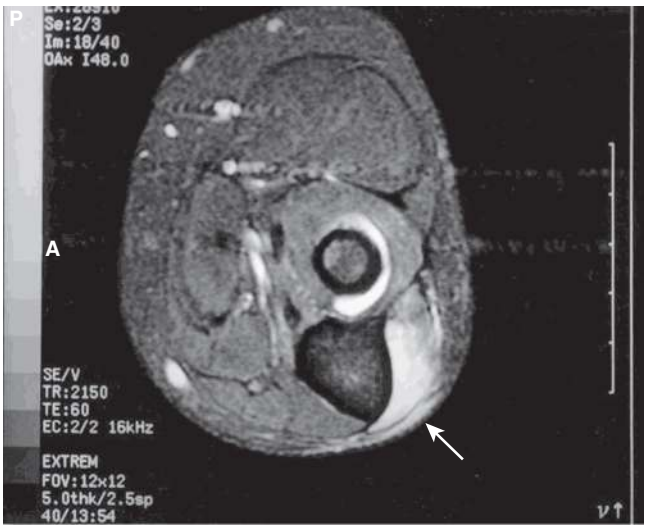

FIG 42.3 Magnetic resonance imaging indicates a signal change at the anconeus muscle consistent with edema (*arrow*). (From Steinmann SP, Bishop AT. Chronic anconeus compartment syndrome: a case report. *J Hand Surg*. 2000;25(5):959–961.)

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the clinician. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures.

FIG 42.4 Anconeus muscle tear with edema in a 30-year-old male bodybuilder who experienced acute pain during weightlifting. Transaxial T1-weighted **(A)** and T2-weighted **(B)** spin-echo magnetic resonance images, the latter obtained with fat suppression, show altered morphology. In **B,** increased signal intensity is evident in the anconeus muscle and surrounding tissues (*arrows*). Inflammatory changes about the biceps tendon are also seen. The radius (r) and ulna (u) are indicated. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3065.)

CLINICAL PEARLS

Although anconeus syndrome is a common disorder, it is often misdiagnosed. Therefore, in patients suspected of suffering from anconeus syndrome, a careful evaluation to identify underlying disease processes is mandatory. Anconeus syndrome commonly coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Badre A, Axford DT, Banayan S, et al. Role of the anconeus in the stability of a lateral ligament and common extensor origin–deficient elbow: an in vitro biomechanical study. *J Shoulder Elb Surg*. 2019;28(5):974–981. https://doi.org/10.1016/j.jse.2018.11.040.

Gangatharam S. Anconeus syndrome: a potential cause for lateral elbow pain and its therapeutic management—a case report. *J Hand Ther*. 2021;34(1):131–134. https://doi.org/10.1016/j.jht.2019.04.002.

Nascimento SRR, Ruiz CR. A study on the prevalence of the anconeus epitrochlearis muscle by magnetic resonance imaging. *Rev Brasileira de Ortopedia (Engl Ed)*. 2018;53(3):373–377. https://doi.org/10.1016/j.rboe.2018.03.015.

Suwannakhan A, Chaiyamoon A, Yammine K, et al. The prevalence of anconeus epitrochlearis muscle and Osborne's ligament in cubital tunnel syndrome patients and healthy individuals: an anatomical study meta-analysis. *Surgeon*. 2021;19(6):e402–e411. https://doi.org/10.1016/j.surge.2020.12.006.

Waldman SD. Injection technique for anconeus syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:172–174.

Supinator Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

As its name implies, the supinator muscle supinates the forearm. Curving around the upper third of the radius, the supinator muscle is composed of a superficial and a deep layer. The superficial layer originates in a tendinous insertion from the lateral epicondyle of the humerus, the radial collateral ligament of the elbow, and the annular ligament of the supinator crest of the ulna.

The supinator muscle is susceptible to the development of myofascial pain syndrome. This pain is most often the result of repetitive microtrauma to the muscle caused by such activities as turning a screwdriver, prolonged ironing, handshaking, or digging with a trowel (Fig. 43.1). Tennis injuries caused by an improper one-handed backhand technique have also been implicated as an inciting factor in myofascial pain syndrome, as has blunt trauma to the muscle.

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the supinator muscle often have referred pain in the ipsilateral forearm.

The trigger point is pathognomonic of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, an involuntary withdrawal of the stimulated muscle, called a jump sign, is often seen and is characteristic of myofascial pain syndrome. Patients with supinator syndrome have a trigger point over the superior portion of the muscle (Fig. 43.2).

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to result from microtrauma to the affected muscle. This trauma may result from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome.

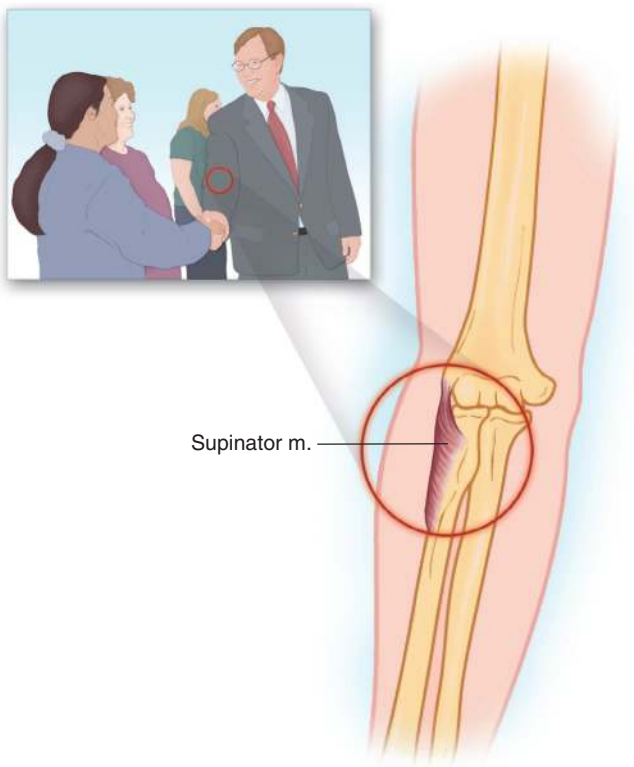

FIG 43.1 Supinator syndrome is usually the result of repetitive microtrauma caused by activities such as turning a screwdriver, prolonged ironing, handshaking, or digging.

For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The supinator muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic

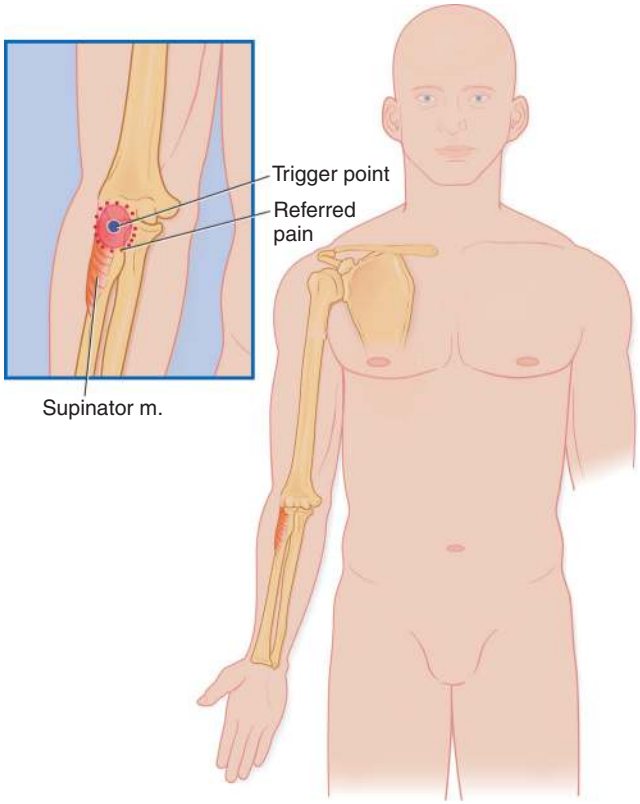

FIG 43.2 Patients with supinator syndrome have a trigger point over the superior portion of the muscle. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:155.)

regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point is the pathologic lesion of supinator syndrome, and it is characterized by a local point of exquisite tenderness in the supinator muscle. This trigger point can best be demonstrated by having the patient supinate the forearm against active resistance. Point tenderness over the lateral epicondyle may also be present and may be amenable to injection therapy.

Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain and referred pain. The jump sign is also characteristic of supinator syndrome, as is pain over the supinator muscle that radiates from the lateral epicondyle and superior portion of the muscle into the forearm.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with supinator syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing of patients suffering from supinator syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic supinator syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of supinator syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from supinator syndrome. The clinician must rule out other coexisting disease processes that may mimic supinator syndrome, including primary inflammatory muscle disease, collagen vascular disease, inflammatory arthritis, tennis elbow, radial tunnel syndrome, tumor, bursitis, tendinitis, and crystal deposition diseases (Fig. 43.3). Radiographic testing, including magnetic resonance imaging of the elbow, can help identify coexisting pathologic processes such as internal derangement of the elbow, tendinitis, and bursitis. Electromyography can rule out cubital and radial tunnel syndromes. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with supinator syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger point and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from supinator syndrome of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor, has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia is selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some

A

FIG 43.3 Idiopathic synovial osteochondromatosis. This 67-year-old woman reported progressive pain and swelling in her elbow over a 6-month period. **A,** The radiograph outlines irregular ossification in the joint (*solid arrows*), with displacement of the anterior fat pad (*arrowhead*), minor osseous erosion (*open arrow*), and osteophytes. **B,** An arthrogram identifies multiple radiolucent filling defects (*arrows*). The diagnosis was confirmed histologically. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3067.)

palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid damage to the underlying neural structures when injecting trigger points in proximity to the elbow and forearm.

CLINICAL PEARLS

Although supinator syndrome is a common disorder, it is often misdiagnosed. Therefore in patients suspected of suffering from supinator syndrome, a careful evaluation to identify underlying disease processes is mandatory. Supinator syndrome commonly coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208(15):87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Goldenberg DL, Clauw DJ, Palmer RE, et al. Opioid use in fibromyalgia: a cautionary tale. *Mayo Clin Proc*. 2016;91(5):640–648.

Hargreaves D, Fetouh D. Common and uncommon nerve compression syndromes around the elbow. *Orthop Trauma*. 2020;34(4):228–234. https://doi.org/10.1016/j.mporth.2020.05.006.

Júnior MH. Uncommon compressive neuropathies of upper limbs. *Best Pract Res Clin Rheumatol*. 2020;34(3):101516. https://doi.org/10.1016/j.berh.2020.101516.

Moffatt R, Thompson L. Peripheral nerve compression syndromes of the upper limb. *Surgery (Oxf)*. 2022;40(3):202–209. https://doi.org/10.1016/j.mpsur.2021.12.002.

Purohit S, Prabhakar A, Raj A, et al. A rare case of posterior interosseous nerve palsy due to ganglion cyst arising from supinator muscle. *J Clin Orthop Trauma*. 2020;11(4):665–667. https://doi.org/10.1016/j.jcot.2020.01.012.

Sluka KA, Clauw DJ. Neurobiology of fibromyalgia and chronic widespread pain. *Neuroscience*. 2016;338(3):114–129.

Waldman SD. Injection technique for supinator syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:214–217.

Wolfe F, Clauw DJ, Fitzcharles MA, et al. 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. *Semin Arthritis Rheum*. 2016;46(3):319–329.

44

Brachioradialis Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The brachioradialis muscle flexes the forearm at the elbow, pronates the forearm when supinated, and supinates the forearm when pronated. It originates at the upper lateral supracondylar ridge of the humerus and the lateral intermuscular septum of the humerus. The muscle inserts on the superior aspect of the styloid process of the radius, the lateral side of the distal radius, and the antebrachial fascia. The muscle is innervated by the radial nerve.

The brachioradialis muscle is susceptible to the development of myofascial pain syndrome. This pain is most often the result of repetitive microtrauma to the muscle from such activities as turning a screwdriver, prolonged ironing, repeated flexing of the forearm at the elbow (e.g., when using exercise equipment), handshaking, or digging with a trowel. Tennis injuries caused by an improper one-handed backhand technique have also been implicated as an inciting factor in myofascial pain syndrome, as has blunt trauma to the muscle (Fig. 44.1).

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the brachioradialis muscle often have referred pain in the ipsilateral forearm and, on occasion, above the elbow.

The trigger point is the pathognomonic lesion of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, is often seen and is characteristic of myofascial pain syndrome. Patients with brachioradialis syndrome have a trigger point over the superior belly of the muscle (Fig. 44.2).

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to result from microtrauma to the affected muscle.

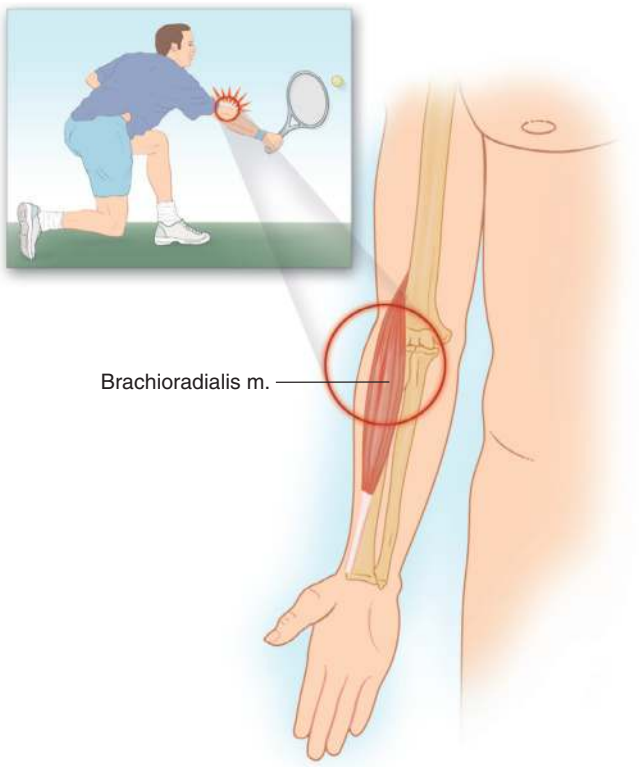

FIG 44.1 Tennis injuries caused by an improper one-handed backhand technique have been implicated in brachioradialis syndrome.

This trauma may occur from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The brachioradialis muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

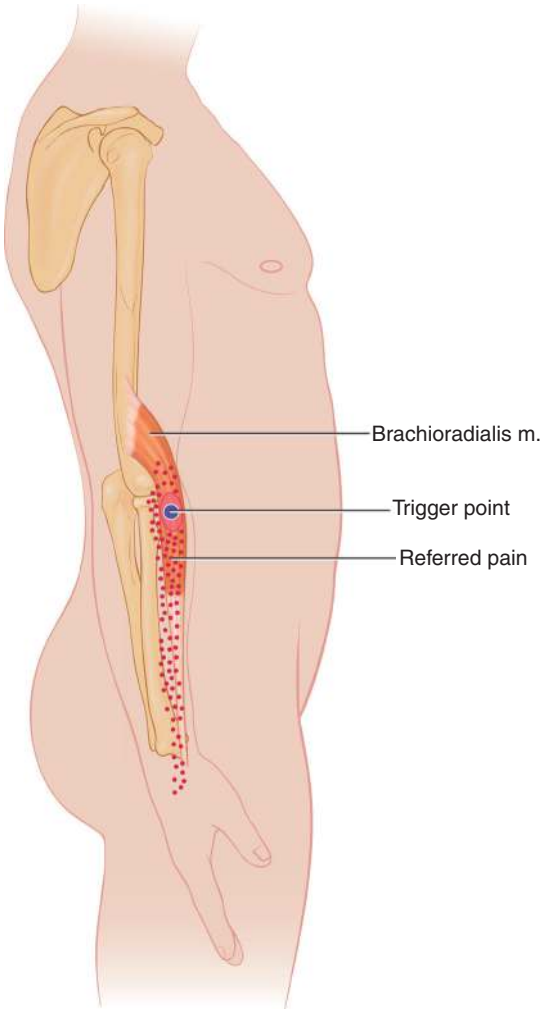

FIG 44.2 Patients with brachioradialis syndrome have a trigger point over the superior belly of the muscle. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007.)

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point is the pathologic lesion of brachioradialis syndrome, and it is characterized by a local point of exquisite tenderness in the brachioradialis muscle. This trigger point can best be demonstrated by having the patient simultaneously flex and pronate the forearm against active resistance. Point tenderness over the lateral supracondylar ridge of the humerus may also be present and may be amenable to injection therapy.

Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain and referred pain. The jump sign is also characteristic of brachioradialis syndrome, as is pain over the brachioradialis muscle that radiates from the lateral epicondyle and superior portion of the muscle into the forearm.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with brachioradialis syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing of patients suffering from brachioradialis syndrome has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic brachioradialis syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of brachioradialis syndrome is made on the basis of clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from brachioradialis syndrome. The clinician must rule out other coexisting disease processes that may mimic brachioradialis syndrome, including primary inflammatory muscle disease and collagen vascular disease. Radiographic testing, including magnetic resonance imaging, can help identify coexisting pathologic processes such as internal derangement of the elbow, tumor, bursitis, tendinitis, crystal deposition diseases, and tennis elbow (Fig. 44.3). Electromyography can rule out cubital and radial tunnel syndromes. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with brachioradialis syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from brachioradialis syndrome of

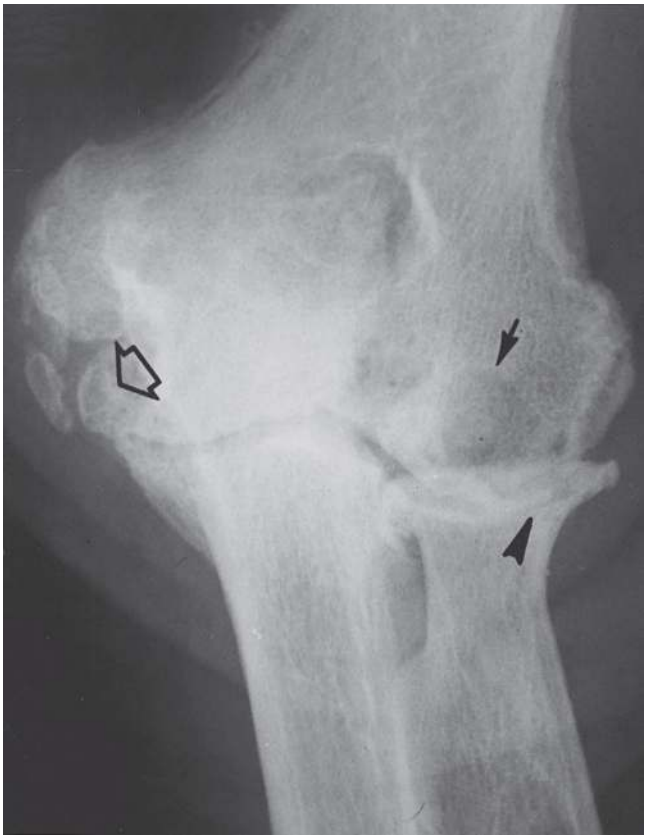

FIG 44.3 Characteristics of pyrophosphate arthropathy, including an unusual articular distribution. Changes in the elbow include joint space narrowing, subchondral cysts (*solid arrow*), deformity of the radial head (*arrowhead*), and fragmentation (*open arrow*). (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:1584.)

the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor, has also shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid damage to the underlying neural structures when injecting trigger points in proximity to the elbow and forearm.

CLINICAL PEARLS

Although brachioradialis syndrome is a common disorder, it is often misdiagnosed. Therefore in patients suspected of suffering from brachioradialis syndrome, a careful evaluation to identify underlying disease processes is mandatory. Brachioradialis syndrome commonly coexists with various somatic and psychological disorders.

SUGGESTED READINGS

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Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Goldenberg DL, Clauw DJ, Palmer RE, et al. Opioid use in fibromyalgia: a cautionary tale. *Mayo Clin Proc*. 2016;91(5):640–648.

Hargreaves D, Fetouh S. Common and uncommon nerve compression syndromes around the elbow. *Orthop Trauma*. 2020;34(4):228–234. https://doi.org/10.1016/j.mporth.2020.05.006.

Júnior MH. Uncommon compressive neuropathies of upper limbs. *Best Pract Res Clin Rheumatol*. 2020;34(3):101516. https://doi.org/10.1016/j.berh.2020.101516.

Moffat R, Thompson L. Peripheral nerve compression syndromes of the upper limb. *Surgery (Oxf)*. 2022;40(3):202–209. https://doi.org/10.1016/j.mpsur.2021.12.002.

Sluka KA, Clauw DJ. Neurobiology of fibromyalgia and chronic widespread pain. *Neuroscience*. 2016;338(3):114–129.

Waldman SD. Injection technique for brachioradialis syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:218–219.

Wolfe F, Clauw DJ, Fitzcharles MA, et al. 2016 Revisions to the 2010/2011 fibromyalgia diagnostic criteria. *Semin Arthritis Rheum*. 2016;46(3):319–329.

Ulnar Nerve Entrapment at the Elbow

ICD-10 CODE G65.20

THE CLINICAL SYNDROME

Ulnar nerve entrapment at the elbow is one of the most common entrapment neuropathies encountered in clinical practice. Causes include compression of the ulnar nerve by an aponeurotic band that runs from the medial epicondyle of the humerus to the medial border of the olecranon, direct trauma to the ulnar nerve at the elbow, and repetitive elbow motion. Ulnar nerve entrapment at the elbow is also called tardy ulnar palsy, cubital tunnel syndrome, and ulnar nerve neuritis. This entrapment neuropathy manifests as pain and associated paresthesias in the lateral forearm that radiate to the wrist and to the ring and little fingers. Some patients also notice pain referred to the medial aspect of the scapula on the affected side. Untreated, ulnar nerve entrapment at the elbow can result in a progressive motor deficit and, ultimately, flexion contracture of the affected fingers. Symptoms usually begin after repetitive elbow motion or repeated pressure on the elbow, such as leaning on the elbow while lying on the floor (Fig. 45.1). Direct trauma to the ulnar nerve as it enters the cubital tunnel may result in a similar clinical presentation. Patients vulnerable to nerve syndromes, such as diabetic and alcoholic patients, are at greater risk for the development of ulnar nerve entrapment at the elbow.

FIG 45.1 The ulnar nerve is susceptible to compression at the elbow.

SIGNS AND SYMPTOMS

Physical findings include tenderness over the ulnar nerve at the elbow. A positive Tinel sign is usually present over the ulnar nerve as it passes beneath the aponeurosis (Fig. 45.2). Weakness of the intrinsic muscles of the forearm and hand that are innervated by the ulnar nerve may be identified with careful manual muscle testing; however, early in the course of cubital tunnel syndrome, the only physical finding other than tenderness over the nerve may be the loss of sensation on the ulnar side of the little finger (Fig. 45.3). Muscle wasting of the intrinsic muscles of the hand can best be identified by viewing the hand from above with the palm down (Fig. 45.4). Patients suffering from ulnar nerve entrapment at the elbow often exhibit a positive Froment sign, which is owing to weakness of the adductor pollicis brevis and flexor pollicis brevis muscles (Fig. 45.5A). Patients with significant muscle weakness secondary to ulnar nerve entrapment at the elbow also exhibit a positive Wartenberg sign, with patients often complaining that the little finger gets caught outside the pants pocket when they reach for car keys (see Fig. 45.5B). Patients suffering from ulnar nerve entrapment at the elbow may also exhibit a positive little finger adduction test (see Fig. 45.5C). The Jeanne test will help confirm the presence of compromise of the ulnar nerve (Fig. 46.6).

FIG 45.2 Tinel's test at the elbow.

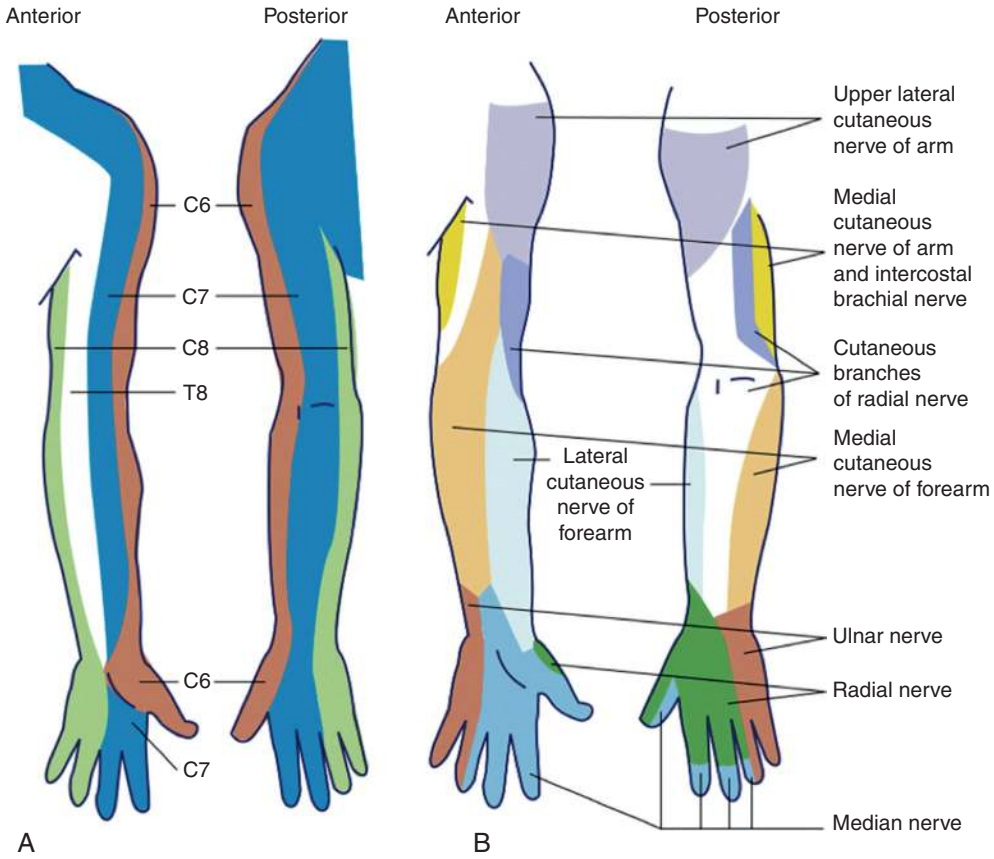

FIG 45.3 Distribution of the ulnar nerve. **A.** Dermatomes **B.** Peripheral nerves. (From Duffy BJ, Tubog TD. The prevention and recognition of ulnar nerve and brachial plexus injuries. *J Perianesth Nurs*. 2017;32(6):636–649.)

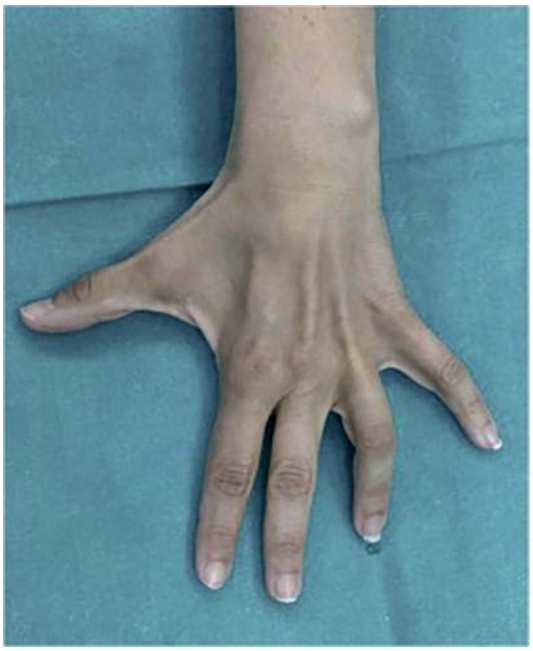

FIG 45.4 Wasting of the intrinsic muscles of the hand in a patient with sever ulnar nerve entrapment at the elbow. (From Lauretti L, D'Alessandris QG, De Simone C, et al. Ulnar nerve entrapment at the elbow. A surgical series and a systematic review of the literature. *J Clin Neurosci*. 2017;46:99–108.)

TESTING

Electromyography and nerve conduction velocity studies are extremely sensitive tests, and a skilled electromyographer can diagnose ulnar nerve entrapment at the elbow with a high degree of accuracy, as well as distinguish other neuropathic causes of pain that may mimic it, including radiculopathy and plexopathy. Plain radiographs are indicated in all patients who present with ulnar nerve entrapment at the elbow to rule out occult bony disorders (Fig. 45.7). Ultrasound imaging is also indicated to identify other abnormalities that may be responsible for compromise of the ulnar nerve at the elbow (Fig. 45.8). If surgery is contemplated, magnetic resonance imaging (MRI) of the affected elbow may further delineate the pathologic process responsible for the nerve entrapment (e.g., bone spur, aponeurotic band thickening) (Figs. 45.9 and 45.10). If Pancoast's tumor or some other tumor of the brachial plexus is suspected, chest radiographs with apical lordotic views may be helpful. If the diagnosis is in question, screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry should be performed to rule out other causes of the patient's pain. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

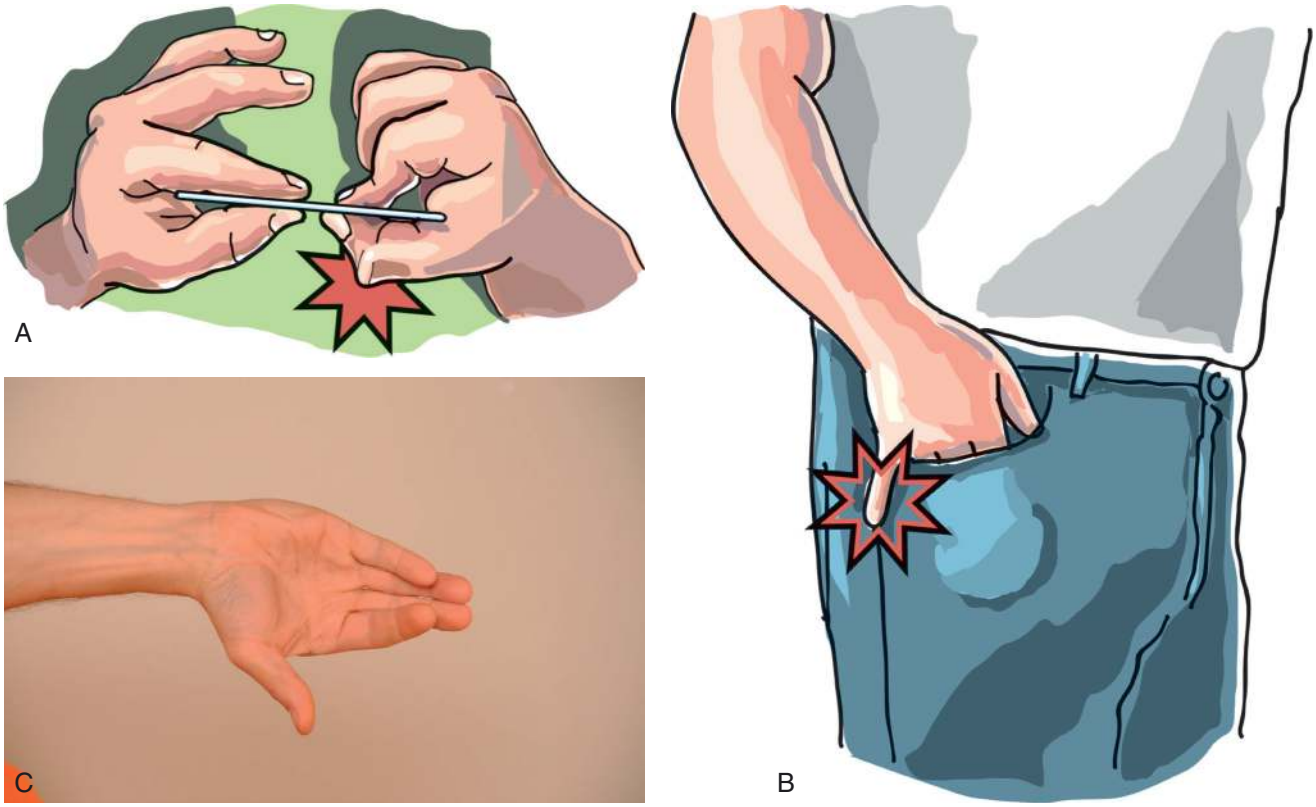

FIG 45.5 **A,** Froment's sign is elicited by asking the patient to grasp a piece of paper lightly between the thumb and index finger of each hand and monitoring flexion of the thumb interphalangeal joint on the affected side. **B,** Wartenberg's sign for ulnar nerve entrapment at the elbow. **C,** The little finger adduction test evaluates the strength in the interosseous muscles of the hand that are innervated by the ulnar nerve. It is performed by asking the patient to touch his or her little finger to the index finger. (From Waldman SD. The little finger adduction test for ulnar nerve entrapment at the elbow. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:126,128.)

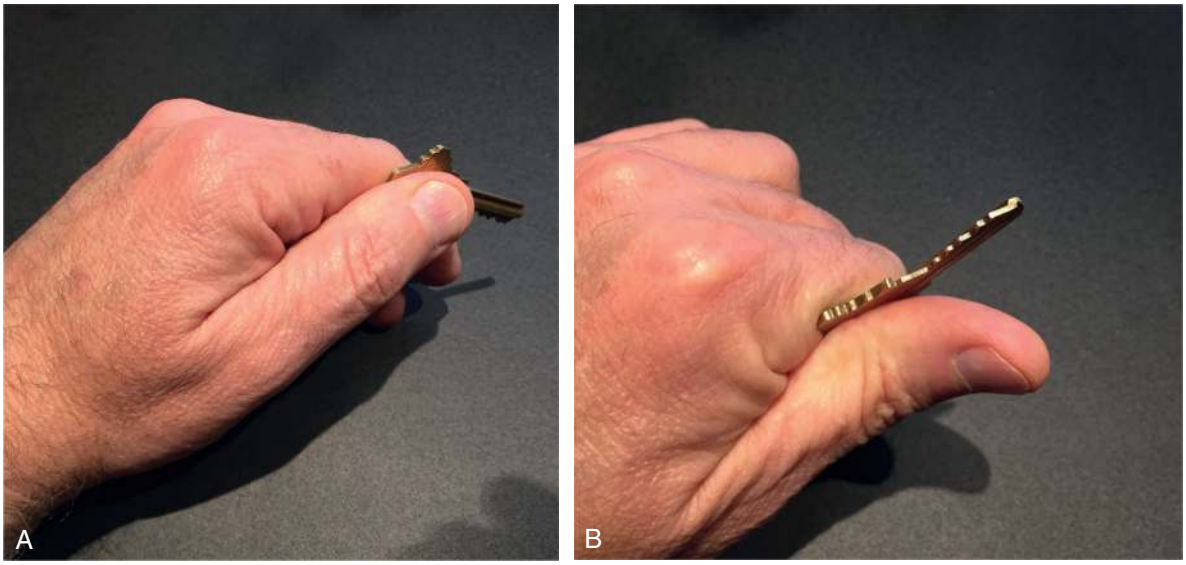

FIG 45.6 **A,** The Jeanne test is performed by asking the patient to lightly grasp a key between the thumb and radial aspect of the index finger of each hand and monitoring the flexion of the thumb interphalangeal joint on the affected side. **B,** The patient is then asked to grasp the key more tightly. The Jeanne test is positive if the metacarpophalangeal joint of the affected thumb hyperextends in order to stabilize the joint to increase grasp pressure.

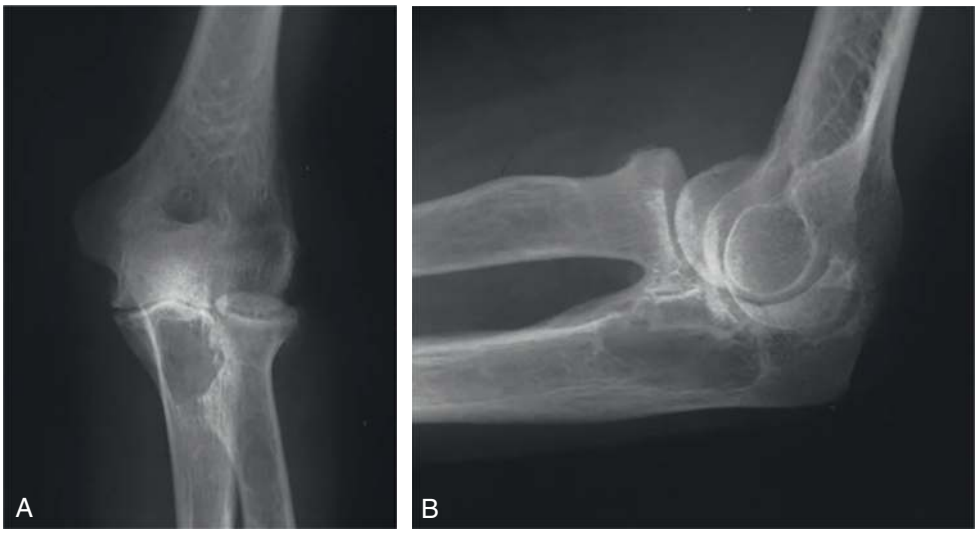

FIG 45.7 AP (A) and lateral (B) radiographs of early rheumatoid involvement of the elbow. There is global joint space narrowing and a large lucent geode is present within the proximal ulna. No erosions are present. *AP*, Anteroposterior. (From Waldman SD, Campbell RSD, eds. Rheumatoid arthritis of the elbow. In: *Imaging of pain*. WB Saunders; 2011: chap 110, 279–280.)

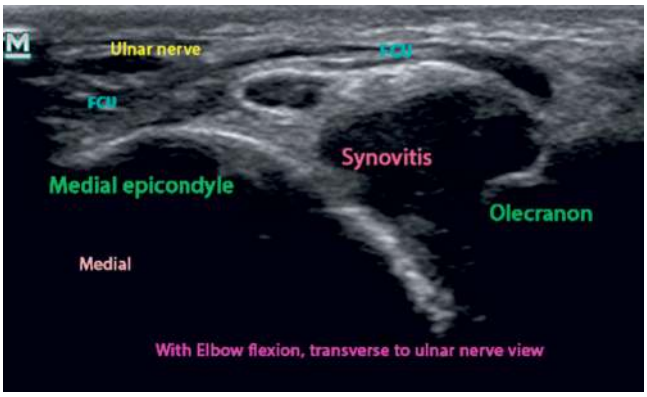

FIG 45.8 Transverse ultrasound image of the elbow in the flexed position demonstrating compression of the ulnar nerve by exuberant synovitis in a patient suffering from rheumatoid arthritis. *FCU*, Flexor carpi ulnaris.

DIFFERENTIAL DIAGNOSIS

Ulnar nerve entrapment at the elbow is often misdiagnosed as golfer's elbow, which explains why many patients with presumed golfer's elbow fail to respond to conservative measures (see Chapter 39). In patients with cubital tunnel syndrome, the maximal tenderness to palpation is over the ulnar nerve 1 inch below the medial epicondyle, whereas with golfer's elbow, the maximal tenderness to palpation is directly over the medial epicondyle. Cubital tunnel syndrome should also be differentiated from cervical radiculopathy involving the C7 or C8 roots. Furthermore, cervical radiculopathy and ulnar nerve entrapment may coexist as the double-crush syndrome. The double-crush syndrome is seen most commonly with median nerve entrapment at the wrist or with carpal tunnel syndrome.

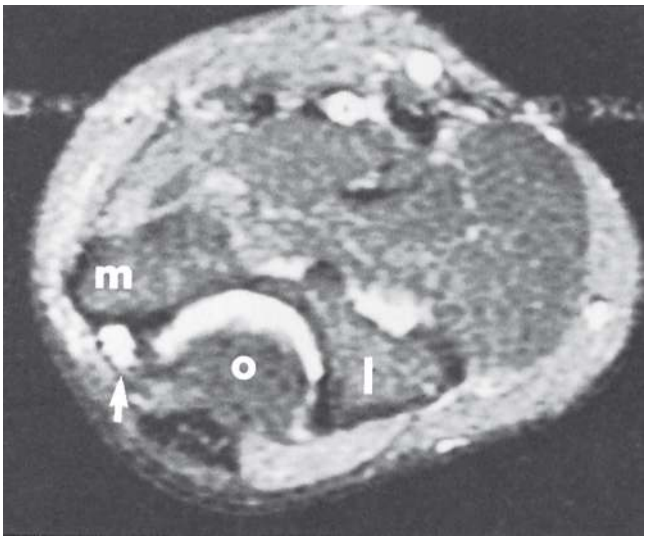

FIG 45.9 Entrapment neuropathy: cubital tunnel syndrome. A transaxial T2-weighted spin-echo magnetic resonance image, obtained with fat suppression, shows increased signal intensity in the ulnar nerve (*arrow*) within the cubital tunnel. The medial (*m*) and lateral (*l*) epicondyles of the humerus and the olecranon process (*o*) of the ulna are indicated. Joint effusion is present. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3065.)

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors, along with splinting to avoid elbow flexion, is indicated in patients who present with ulnar nerve entrapment at the elbow. If no marked reduction in symptoms

FIG 45.10 Magnetic resonance imaging (MRI) of the elbow. Axial T2-weighted **(A)** and T1-weighted **(B)** magnetic resonance (MR) images through the level of proximal radioulnar joint reveal distension of joint capsule with underlying low to intermediate signal fibrous synovial pannus (*asterisks*) and joint effusion (*triangles*). Axial **(C)** and sagittal **(D)** T1-weighted MR image through level of the ulnohumeral joint shows location and degree of marginal erosive changes (*arrows*). **E,** Sagittal T1-weighted MR image through level of the radiocapitellar joint reveals the anterior and posterior capitellar erosion and proximal radial bone marrow edema (*asterisk*). (From Ali Hasan NM, Alam-Eldean MH, Mousa SS. Stiff elbow in adult: MR imaging findings. *Egypt J Radiol Nucl Med*. 2015;46(4):1037–1048.)

occurs within 1 week, careful injection of the ulnar nerve at the elbow using the following technique is a reasonable next step.

Ulnar nerve injection at the elbow is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow slightly flexed, and the dorsum of the hand resting on a folded towel. A total of 5–7 mL local anesthetic is drawn up in a 12-mL sterile syringe. For the first block, 80 mg methylprednisolone is added to the local anesthetic; 40 mg depot steroid is added with subsequent blocks. The clinician identifies the olecranon process and the medial epicondyle of the humerus; the ulnar nerve sulcus is located between these two bony landmarks. After preparing the skin with antiseptic solution, a 5/8-inch, 25-gauge needle is inserted just proximal to the sulcus and is slowly advanced with a slightly cephalad trajectory. When the needle has advanced approximately half an inch, a strong paresthesia in the distribution of the ulnar nerve will be elicited. The patient should be warned to expect this and to say "There!" as soon as the paresthesia is felt. After the paresthesia is elicited and its distribution is identified, gentle aspiration is performed to identify blood. If the aspiration test result is negative and no persistent paresthesia in the distribution of the ulnar nerve remains, 5–7 mL of local anesthetic solution is slowly injected while the patient is monitored closely for signs of local anesthetic toxicity. If no paresthesia can be elicited, a similar amount of solution is slowly injected in a fanlike manner just proximal to the notch, with care taken to avoid intravascular injection.

If the patient does not respond to these treatments or experiences progressive neurologic deficits, surgical decompression of the ulnar nerve is indicated. As mentioned earlier, MRI and ultrasound imaging of the affected elbow can clarify the pathologic process responsible for the ulnar nerve compression.

COMPLICATIONS AND PITFALLS

Failure to identify and treat ulnar nerve entrapment at the elbow promptly can result in permanent neurologic deficit. To avoid harm to the patient, the clinician must also rule out other causes of pain and numbness that may mimic the symptoms of ulnar nerve entrapment, such as Pancoast's tumor.

Ulnar nerve block at the elbow is relatively safe. The major complications are inadvertent intravascular injection into the ulnar artery and persistent paresthesia secondary to needle-induced trauma to the nerve. Because the nerve passes through the ulnar nerve sulcus and is enclosed by a dense fibrous band, care should be taken to inject slowly, just proximal to the sulcus, to avoid additional compromise of the nerve.

CLINICAL PEARLS

Ulnar nerve entrapment at the elbow is often misdiagnosed as golfer's elbow. It must also be differentiated from cervical radiculopathy involving the C8 spinal root; however, cervical radiculopathy and ulnar nerve entrapment may coexist in the double-crush syndrome. Pancoast's tumor invading the medial cord of the brachial plexus may also mimic ulnar nerve entrapment and must be ruled out by apical lordotic chest radiography.

If cubital tunnel syndrome is suspected, injection of the ulnar nerve at the elbow with local anesthetic and steroid provides almost instantaneous relief. This is a simple and safe technique for the evaluation and treatment of ulnar nerve entrapment. Before ulnar nerve block is performed, a careful neurologic examination should be done to identify preexisting neurologic deficits that could later be attributed to the nerve block. Persistent paresthesia tends to develop when the nerve is blocked at this level. The incidence of this complication can be decreased by blocking the nerve proximal to the ulnar nerve sulcus and injecting slowly.

SUGGESTED READINGS

Boone S, Gelberman RH, Calfee RP. The management of cubital tunnel syndrome. *J Hand Surg Am*. 2015;40(9):1897–1904.

Brubacher JW, Leversedge FJ. Ulnar neuropathy in cyclists. *Hand Clin*. 2017;33(1):199–205.

Shen L, Masih S, Patel DB, et al. MR anatomy and pathology of the ulnar nerve involving the cubital tunnel and Guyon's canal. *Clin Imaging*. 2016;40(2):263–274.

Waldman SD. The little finger adduction test for ulnar nerve entrapment at the elbow. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Saunders; 2017:134–135.

Waldman SD. The Wartenberg test for ulnar nerve entrapment at the elbow. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Saunders; 2017:133–134.

Waldman SD. Ulnar nerve entrapment at the elbow. In: *Pain review*. Philadelphia: Saunders; 2016:270–271.

Zacharia B, Pulluparambil Poulose S, Madhu M. Intraneural ganglion cyst of the ulnar nerve causing cubital tunnel syndrome masquerading a peripheral nerve abscess of a neuritic leprosy. *J Clin Orthop Trauma*. 2022;24:101692. https://doi.org/10.1016/j.jcot.2021.101692.

Lateral Antebrachial Cutaneous Nerve Entrapment at the Elbow

ICD-10 CODE G56.80

THE CLINICAL SYNDROME

The lateral antebrachial cutaneous nerve may be entrapped by the biceps tendon or the brachialis muscle (Fig. 46.1). Clinically, patients complain of pain and paresthesias radiating from the elbow to the base of the thumb. Dull aching of the radial aspect of the forearm is also common. The pain of lateral antebrachial cutaneous nerve entrapment at the elbow may develop after an acute twisting injury to the elbow or direct trauma to the soft tissues overlying the lateral antebrachial cutaneous nerve; in other cases, the onset of pain is more insidious, without an obvious inciting factor. The pain is constant and becomes worse with the use of the elbow. Patients with lateral antebrachial cutaneous nerve entrapment often note an increase in pain when using a computer keyboard or playing the piano (Fig. 46.2). Lateral antebrachial cutaneous nerve compromise may also be seen after overuse of the elbow, especially following activities which involve forced extension and maximal pronation of the elbow including tennis and weightlifting. Rupture of the proximal long head of the biceps may displace the nerve laterally, producing a traction neuropathy (Fig. 46.3). Rarely, injury to the nerve during venipuncture with resultant neuropathy can occur. Sleep disturbance is common.

SIGNS AND SYMPTOMS

On physical examination, the patient notes tenderness to palpation of the lateral antebrachial cutaneous nerve at a point just lateral to the biceps tendon (Fig. 46.4). Elbow range of motion is normal. Patients with lateral antebrachial cutaneous nerve entrapment experience pain with active resisted flexion or rotation of the forearm.

TESTING

Electromyography and nerve conduction velocity studies are extremely sensitive tests, and a skilled electromyographer can diagnose lateral antebrachial cutaneous nerve entrapment with a high degree of accuracy, as well as distinguish other neuropathic causes of pain that may mimic it, including radiculopathy and plexopathy. Plain radiographs are indicated in all patients who present with lateral antebrachial cutaneous nerve entrapment to rule out occult bony disorders. If surgery is contemplated, magnetic resonance imaging (MRI) of the affected elbow may further delineate the pathologic process responsible for the nerve entrapment (e.g., bone spur, aponeurotic band thickening). If Pancoast's tumor or some other tumor of the brachial plexus is suspected, chest radiographs with apical lordotic views may be helpful. If the diagnosis is in question, screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, and automated blood chemistry should be performed to rule out other causes of the patient's pain. Injection of the nerve serves as both a diagnostic and a therapeutic maneuver.

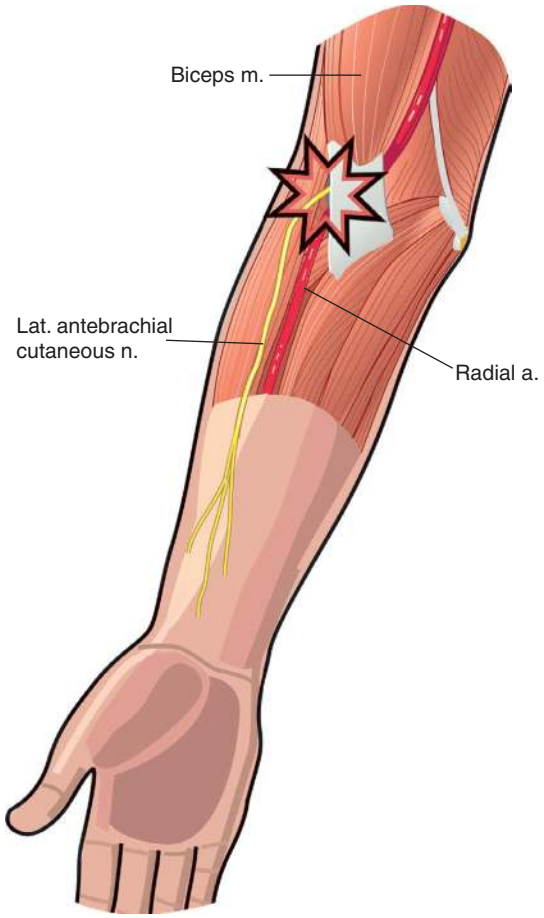

FIG 46.1 Lateral antebrachial cutaneous nerve entrapment: relevant soft tissue anatomy. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms.* Philadelphia: Saunders; 2006:130.)

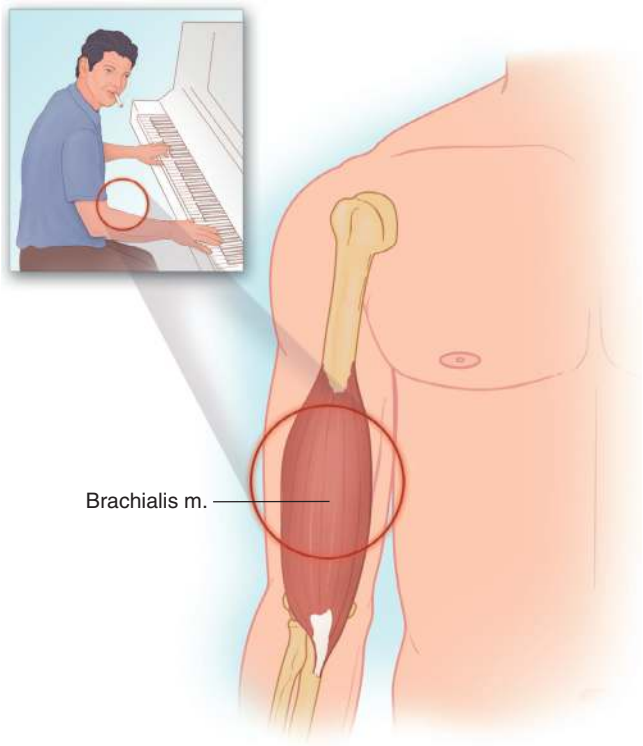

FIG 46.2 Patients with lateral antebrachial cutaneous nerve entrapment at the elbow often note increased pain when using a computer keyboard or playing the piano.

DIFFERENTIAL DIAGNOSIS

Cervical radiculopathy and tennis elbow can mimic nerve entrapment as can entrapment of the superficial radial nerve. In patients with lateral antebrachial cutaneous nerve entrapment, the maximal tenderness to palpation is at the level of the biceps tendon, whereas with tennis elbow, the maximal tenderness to palpation is over the lateral epicondyle (Fig. 46.5, see Chapter 38). Electromyography can distinguish cervical radiculopathy and lateral antebrachial cutaneous nerve entrapment from tennis elbow. Furthermore, cervical radiculopathy and lateral antebrachial cutaneous nerve entrapment may coexist as the double-crush syndrome. The double-crush syndrome is seen most commonly with median nerve entrapment at the wrist or with carpal tunnel syndrome.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors, along with splinting to avoid elbow flexion, is indicated in patients who present with lateral antebrachial cutaneous nerve entrapment at the elbow (Fig. 46.6). If no marked reduction in symptoms occurs within 1 week, careful injection of the lateral antebrachial cutaneous nerve at the elbow is a reasonable next step.

If the patient does not respond to these treatments or experiences progressive neurologic deficits, surgical decompression of the lateral antebrachial cutaneous nerve is indicated.

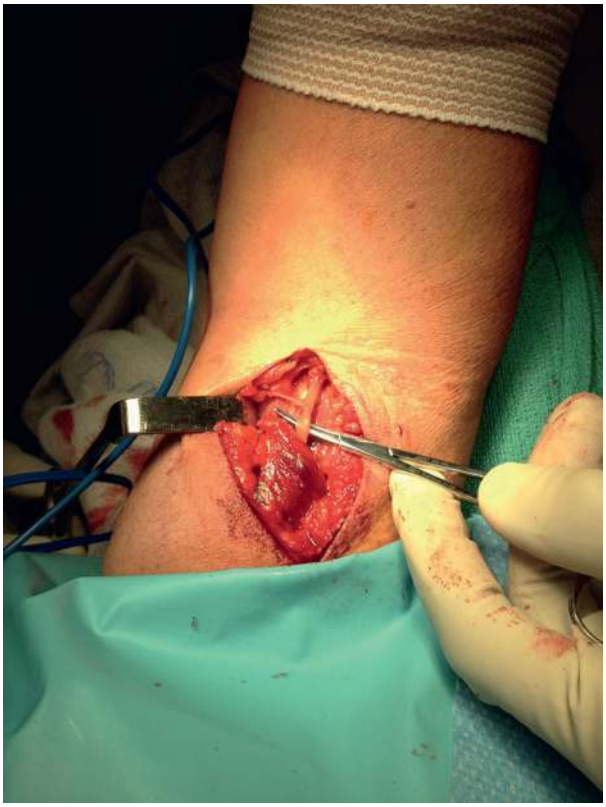

FIG 46.3 Surgical exposure of a lateral antebrachial cutaneous nerve at its point of compression by the biceps tendon. (From Behl AR, Rettig AC, Rettig L. Lateral antebrachial cutaneous nerve compression after traumatic rupture of the long head of the biceps: a case series. *J Shoulder Elbow Surg*. 2014;23(7):919–923.)

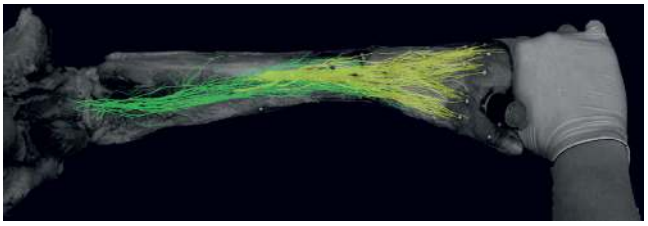

FIG 46.4 Distribution of the lateral antebrachial cutaneous nerve (*green*) and the superficial branch of the radial nerve (*yellow*). (From Poublon AR, Walbeehm ET, Duraku LS, et al. The anatomical relationship of the superficial radial nerve and the lateral antebrachial cutaneous nerve: a possible factor in persistent neuropathic pain. *J Plast Reconstr Aesthet Surg*. 2015;68(2):237–242.)

As mentioned, MRI of the affected elbow can clarify the pathologic process responsible for nerve compression.

COMPLICATIONS AND PITFALLS

Failure to identify and treat lateral antebrachial cutaneous nerve entrapment at the elbow promptly can result in permanent neurologic deficit. To avoid harm to the patient, the clinician must rule out other causes of pain and numbness that may mimic the symptoms of lateral antebrachial cutaneous nerve entrapment, such as Pancoast's tumor.

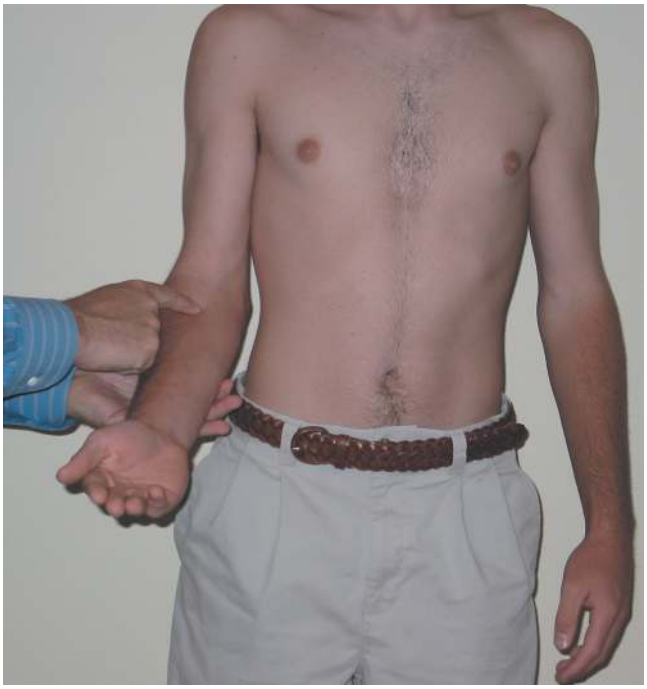

FIG 46.5 Compression test for lateral antebrachial cutaneous nerve entrapment. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:131.)

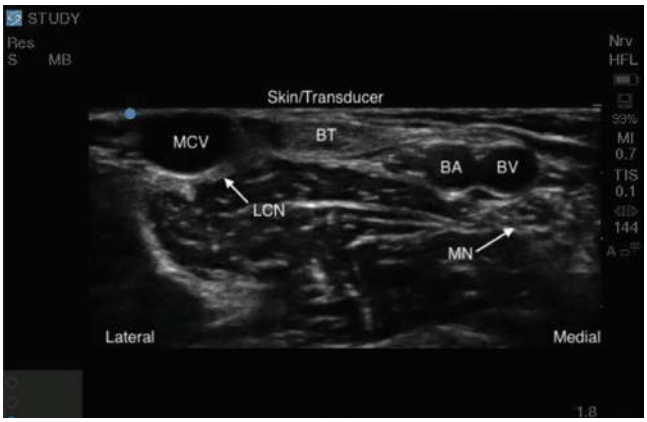

FIG 46.6 Ultrasound image of the distal antecubital fossa where the LCN transitioned from adjacent to the biceps brachii muscle tendon to the terminal branch lying adjacent to the median cubital vein at the level of the venipuncture. *BA*, Brachial artery; *BT*, biceps brachii muscle tendon; *BV*, brachial vein; *LCN*, lateral antebrachial cutaneous nerve; *MCV*, median cubital vein; *MN*, median nerve. (Image taken by the author Christian Falyar, DNAP, CRNA. Reprinted with permission.)

Lateral antebrachial cutaneous nerve block at the elbow is relatively safe. The major complications are inadvertent intravascular injection into the lateral antebrachial cutaneous artery and persistent paresthesia secondary to needle-induced trauma to the nerve. Because the nerve passes through the lateral antebrachial cutaneous nerve sulcus and is enclosed by a dense fibrous band, care should be taken to inject slowly, just proximal to the sulcus, to avoid additional compromise of the nerve.

CLINICAL PEARLS

Lateral antebrachial cutaneous nerve entrapment at the elbow is often misdiagnosed as tennis elbow, which explains why many patients with presumed tennis elbow fail to respond to conservative measures. Lateral antebrachial cutaneous nerve block at the elbow is a simple and safe technique for the evaluation and treatment of this condition. Before the nerve block is performed, a careful neurologic examination should be done to identify preexisting neurologic deficits that could later be attributed to the nerve block. The incidence of persistent paresthesia can be decreased by blocking the nerve proximal to the lateral antebrachial cutaneous nerve sulcus and injecting slowly.

SUGGESTED READINGS

Behl R, Rettig AC, Rettig L. Lateral antebrachial cutaneous nerve compression after traumatic rupture of the long head of the biceps: a case series. *J Shoulder Elbow Surg*. 2014;23(7):919–923.

Belzile E, Cloutier D. Entrapment of the lateral antebrachial cutaneous nerve exiting through the forearm fascia. *J Hand Surg*. 2001;26(1):64–67.

Garon MT, Greenberg JA. Complications of distal biceps repair. *Orthop Clin North Am*. 2016;47(2):435–444.

Pitman JS, Funk EM, Falyar C. Venipuncture-related lateral antebrachial cutaneous nerve injury in primary care settings. *J Nurse Pract*. 2022;18(4):449–451. https://doi.org/10.1016/j.nurpra.2021.12.004.

Poublon AR, Walbeehm ET, Duraku LS, et al. The anatomical relationship of the superficial radial nerve and the lateral antebrachial cutaneous nerve: a possible factor in persistent neuropathic pain. *J Plast Reconstr Aesthet Surg*. 2015;68(2):237–242.

Ramos JA. Venipuncture-related lateral antebrachial cutaneous nerve injury: what to know? *Braz J Anesthesiol*. 2014;64(2):131–133.

Waldman SD. The lateral antebrachial cutaneous nerve. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:105.

Osteochondritis Dissecans of the Elbow

ICD-10 CODE M93.20

THE CLINICAL SYNDROME

Although osteochondritis dissecans was first described in the late 1800s, its exact etiology remains unknown. Current thinking is that osteochondritis dissecans is the result of continual microtrauma to the articular cartilage of the elbow. Studies suggest that this repetitive microtrauma causes an ischemic insult to the cartilage and supporting structures that results in the characteristic localized separation of the articular cartilage and subchondral bone. Osteochondritis dissecans most often affects the elbow of the dominant upper extremity in young male athletes, with bilateral findings occurring in approximately 5% of those affected. Sports most often associated with the development of osteochondritis dissecans include racquetball, baseball, weight lifting, cheerleading, and competitive gymnastics (Fig. 47.1). Pain on the use of the affected elbow is universally present in patients suffering with osteochondritis dissecans and eases at rest. The pain is often deep, dull, and poorly defined. Loose bodies of the joint are common. Bilateral disease has been reported.

SIGNS AND SYMPTOMS

The pain of osteochondritis dissecans is generally the patient's first indication of an elbow problem. The pain is poorly localized, and the patient often rubs his or her elbow when trying to describe it. If joint mice are present, the patient may complain of grating or popping sensations with flexion and extension of the affected elbow. Sleep disturbance is common.

Patients suffering from osteochondritis dissecans may exhibit a decreased ability to extend the affected elbow fully. Active compression across the radiocapitellar joint from muscular forces may reproduce the patient's pain, as will an active radiocapitellar compression test, which is performed by having the patient pronate and supinate the forearm while flexing and extending the elbow (Fig. 47.2A and B). Physical examination may also reveal tenderness to palpation of the elbow. If the patient has an associated acute injury to the elbow, swelling

FIG 47.1 Competitive gymnastics is one of the sports most often associated with the development of osteochondritis dissecans. (From *Dorling Kindersley/Getty images,* New York, 2010.)

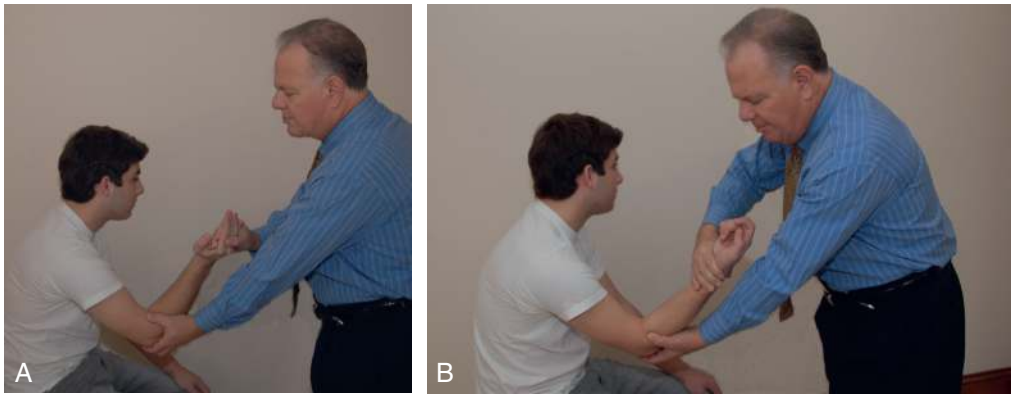

FIG 47.2 The active radiocapitellar compression test is performed by having the patient pronate and supinate the forearm while flexing and extending the elbow.

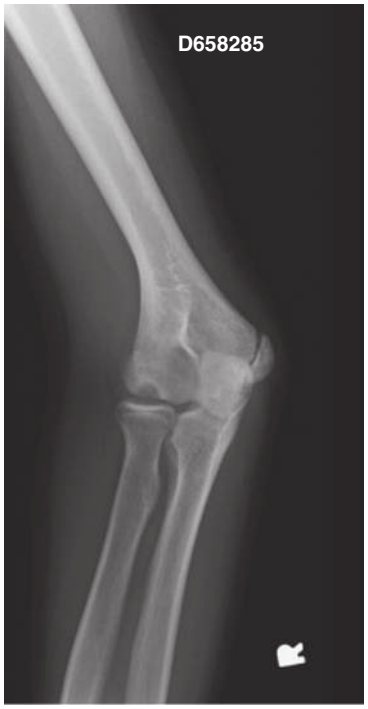

FIG 47.3 Lateral radiograph demonstrating radiolucency and rarefaction typical of osteochondritis dissecans of the capitellum. (From Savoie FH III. Osteochondritis dissecans of the elbow. *Oper Tech Sports Med*. 2008;16(4):187–193.)

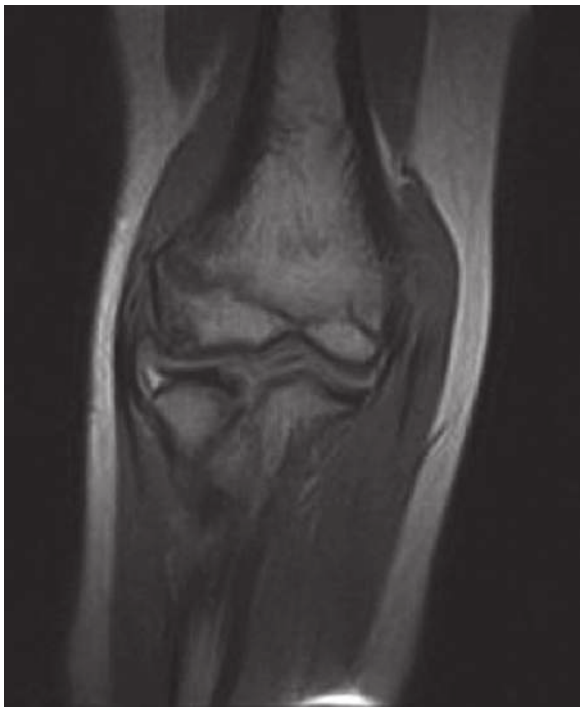

FIG 47.4 Coronal magnetic resonance image of osteochondritis dissecans of the capitellum. Increased signal intensity of the T2-weighted image indicates a detachment of the primary fragment. (From Savoie FH III. Osteochondritis dissecans of the elbow. *Oper Tech Sports Med*. 2008;16(4):187–193.)

and ecchymosis may be present. Flexion contracture, which may also be present, results in a loss of full elbow extension (see Fig. 41.3). In some high-performing athletes, these range-of-motion abnormalities represent adaptive changes and are not often the sole cause of the patient's pain symptoms. As with thrower's elbow, patients suffering from osteochondritis dissecans often suffer from other coexisting disorders of the elbow including tendinitis, ligamentous injury, myofascial pain syndromes, nerve entrapments, and bursitis.

TESTING

Plain radiographs should be obtained in all patients who present with elbow pain, to rule out joint mice and other occult bony disorders such as avulsion fractures of the olecranon (Fig. 47.3). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging of the elbow is indicated if joint instability is suspected or if the symptoms of osteochondritis dissecans persist (Fig. 47.4). Electromyography (EMG) is indicated to diagnose entrapment neuropathy at the elbow and to exclude cervical radiculopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Occasionally, cervical radiculopathy mimics osteochondritis dissecans; however, patients suffering from cervical radiculopathy usually have neck pain and proximal upper extremity pain in addition to symptoms below the elbow. As noted earlier, EMG can distinguish radiculopathy from osteochondritis dissecans. Bursitis, arthritis, tendinitis, and gout may also mimic osteochondritis dissecans and confuse the diagnosis. The olecranon bursa lies in the posterior aspect of the elbow joint and may become inflamed as a result of direct trauma to the joint or its overuse. Other bursae susceptible to the development of bursitis are located between the insertion of the biceps and the head of the radius, as well as in the antecubital and cubital areas.

TREATMENT

Initial treatment of the pain and functional disability associated with osteochondritis dissecans includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

Injection for osteochondritis dissecans is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow fully extended, and the dorsum of the hand resting on a folded towel to relax the affected tendons. A total of 1-mL local anesthetic and 40 mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the medial

aspect of the joint, the medial epicondyle is identified. Using strict aseptic technique, a 1-inch, 25-gauge needle is inserted perpendicular to the medial epicondyle through the skin and into the subcutaneous tissue overlying the affected tendon. If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. Little resistance to injection should be felt. If significant resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can proceed with less resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for elbow pain. A Velcro band placed around the flexor tendons may also help relieve the symptoms. Occupational therapy for activities of daily living education may also be beneficial. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms, and return to play should not occur until the patient's symptoms are completely resolved.

COMPLICATIONS AND PITFALLS

The major complications associated with this injection technique are related to trauma to the inflamed and previously damaged tendon, which may rupture if it is injected directly. Therefore the needle position should be confirmed to be outside the tendon before the clinician proceeds with the injection. Another complication of the injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Injection is safe if careful attention is paid to the clinically relevant anatomy; in particular, the ulnar nerve is susceptible to damage at the elbow. Approximately 25% of patients complain of a transient increase in pain after this injection technique, and patients should be warned of this possibility.

CLINICAL PEARLS

A complete understanding of the biomechanics of overhead throwing is essential if the clinician is to diagnose and treat osteochondritis dissecans successfully. The injection technique described is extremely effective in the treatment of pain secondary to osteochondritis dissecans. Coexistent bursitis and tendinitis may contribute to elbow pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with this injection technique. Cervical radiculopathy and other abnormalities of the elbow may mimic osteochondritis dissecans and must be excluded.

SUGGESTED READINGS

Ellington MD, Edmonds EW. Pediatric elbow and wrist pathology related to sports participation. *Orthop Clin North Am*. 2016; 47(4):743–748.

Eygendaal D, Bain G, Pederzini L, Poehling G. Osteochondritis dissecans of the elbow: state of the art. *J ISAKOS*. 2017;2(1): 47–57. https://doi.org/10.1136/jisakos-2015-000008.

Logli AL, Leland DP, Bernard CD, et al. Capitellar osteochondritis dissecans lesions of the elbow: a systematic review of osteochondral graft reconstruction options. *Arthroscopy*. 2020;36(6):1747–1764.

Macken AA, van Bergen CJA, Eygendaal D. The B, diagnosis and treatment of osteochondritis dissecans of the elbow. *Orthop Trauma*. 2020;34(4):213–218. https://doi.org/10.1016/j.mporth.2020.05.004.

Takahara M, Uno T, Maruyama M, et al. Staging of osteochondritis dissecans of the elbow based on pathologic progression in the partially detached articular fragment. *J Shoulder Elbow Surg*. 2022;31(2):391–401. https://doi.org/10.1016/j.arthro.2020.01.037.

Olecranon Bursitis

ICD-10 CODE M70.20

THE CLINICAL SYNDROME

Olecranon bursitis may develop gradually as a result of repetitive irritation of the olecranon bursa or acutely as a result of trauma or infection. The olecranon bursa lies in the posterior aspect of the elbow between the olecranon process of the ulna and the overlying skin. It may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. With overuse or misuse, these bursae may become inflamed, enlarged, and, on rare occasions, infected. The swelling associated with olecranon bursitis may be quite impressive, and the patient may complain about being unable to wear a long-sleeved shirt.

The olecranon bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are often caused by direct trauma to the elbow in patients who play sports, such as hockey, or who fall directly onto the olecranon process. Repeated pressure from leaning on the elbow, such as when working long hours at a drafting table, may result in inflammation and swelling of the olecranon bursa (Fig. 48.1). Rarely, gout or bacterial infection precipitates acute olecranon bursitis. If inflammation of the olecranon bursa becomes chronic, rice bodies may occur as may cords, as well as calcification of the bursa resulting in residual calcified nodules called gravel (Fig. 48.2).

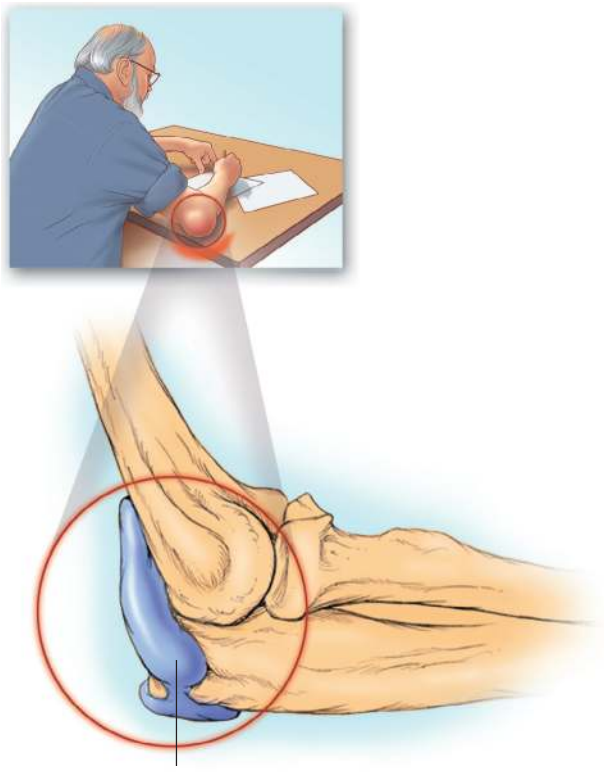

FIG 48.1 Olecranon bursitis is often caused by repeated pressure on the elbow.

SIGNS AND SYMPTOMS

Patients suffering from olecranon bursitis, which is also known as dialysis elbow frequently, complain of swelling and pain with any movement of the elbow, but especially with extension. (Box 48.1). The pain is localized to the olecranon area, with referred pain often noted above the elbow joint. Frequently, the patient is more concerned about the swelling than about the pain. Physical examination reveals point tenderness over the olecranon and swelling of the bursa that may be extensive (Fig. 48.3). Passive extension and resisted flexion reproduce the pain, as does any pressure over the bursa. Fever and chills usually accompany infection of the bursa.

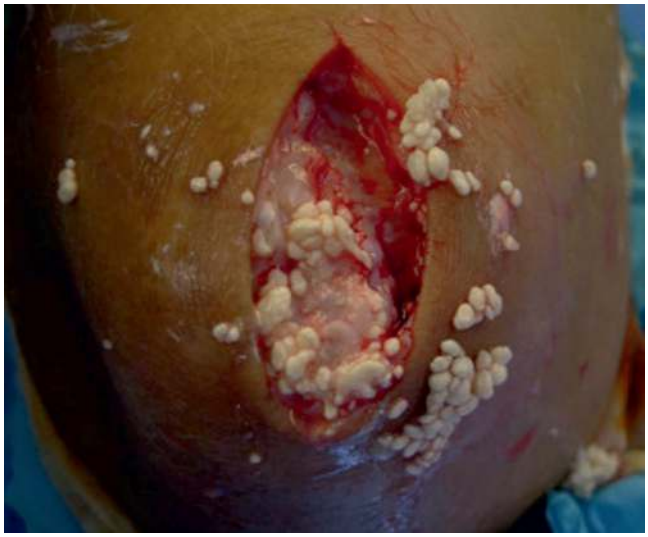

FIG 48.2 Intraoperative photograph of a patient with rheumatoid arthritis and chronic olecranon bursitis. Abundant rice bodies were found when the bursa was excised. (From Reilly D, Kamineni S. Olecranon bursitis. *J Shoulder Elbow Surg*. 2016;25(1):158–167. Waldman SD. Olecranon bursitis. In: Waldman SD, ed. *Atlas of common pain syndromes*. 4th ed. 2019: chap 48, 187–190.)

TESTING

The diagnosis of olecranon bursitis is usually made on clinical grounds alone. Plain radiographs of the posterior elbow are indicated if the patient has a history of elbow trauma or if arthritis of the elbow is suspected. Plain radiographs may also reveal calcification of the bursa and associated structures, consistent with chronic inflammation. Magnetic resonance imaging and ultrasound imaging are indicated if joint instability is suspected, to further characterize the nature of masses of the posterior elbow (e.g., solid or cystic) and to clarify the diagnosis of olecranon bursitis if it is in question (Figs. 48.4 and 48.5). A complete blood count, automated chemistry profile including uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing should be performed if collagen vascular disease is suspected (see Fig. 48.2). If infection is suspected, aspiration, Gram stain, and culture of the bursal fluid, followed by treatment with appropriate antibiotics, are required on an emergency basis (Fig. 48.6).

BOX 48.1 Other Names For Olecranon Bursitis

Draftsman's elbow
Plumber's elbow
Student's elbow
Miner's elbow
Dialysis elbow
Old Man's bursitis

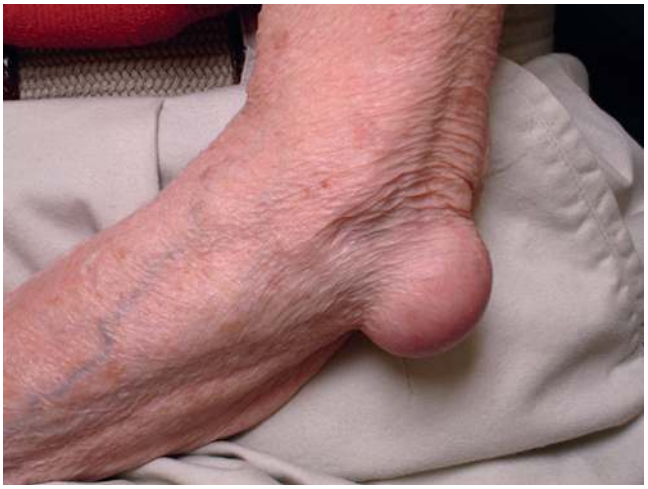

FIG 48.3 Photograph of an enlarged olecranon bursa consistent with nonseptic olecranon bursitis. (From DeLee JC, Drez DD, Miller M, eds. *DeLee and Drez's orthopaedic sports medicine: principles and practice*. 3rd ed. Philadelphia: Saunders; 2010:1247.)

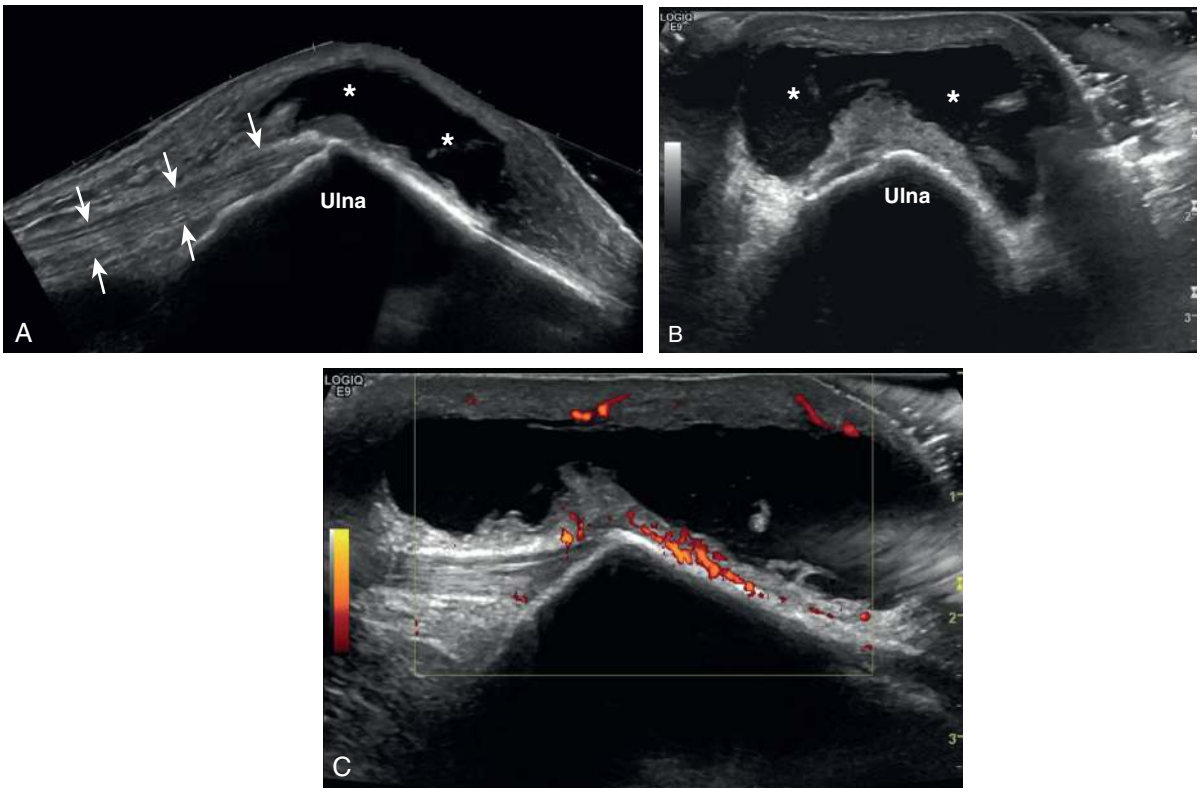

FIG 48.4 Longitudinal **(A)** and axial **(B)** ultrasound (US) images of a patient with olecranon bursitis. There is a low-echo, fluid-filled bursa (*asterisks*) superficial to the proximal ulna, and the distal triceps tendon is visualized on the longitudinal image (*white arrows*). **C,** The Doppler US image demonstrates increased vascularity in the periphery of the bursa consistent with mild inflammatory synovitis. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011:274.)

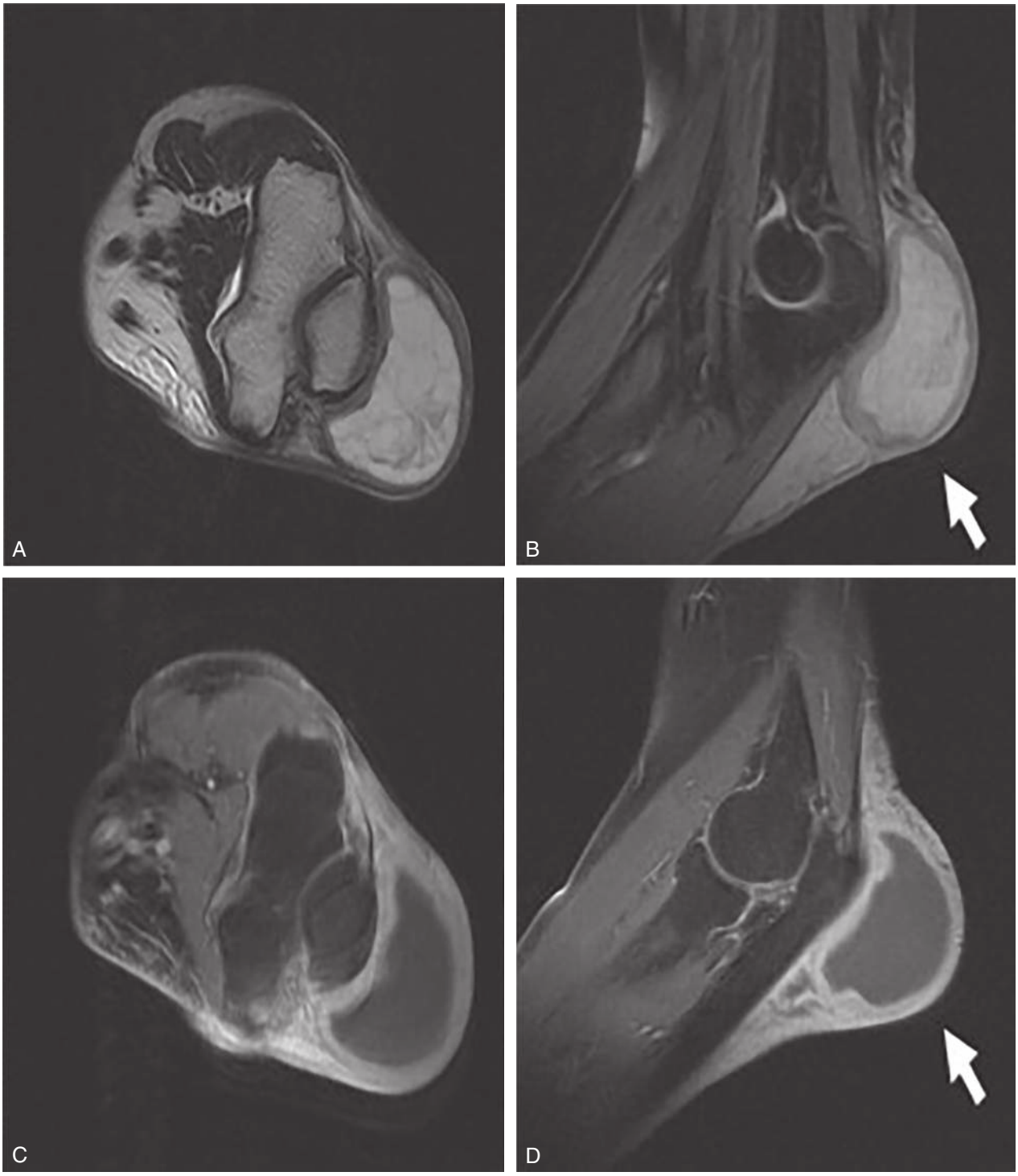

FIG 48.5 Case 1: **(A)** axial T2 FSE weighted MR image showing distended olecranon bursa with thickened walls, **(B)** sagittal T2 FAT SAT showing the same features with subcutaneous edema, **(C)** axial, **(D)** sagittal T1 FAT SAT postcontrast showing enhancing margins with fluid distension and peribursal subcutaneous inflammation. *FSE*, fast spin echo; *MR*, magnetic resonance. (From Emad Y, Ragab Y, El-Shaarawy N, Rasker JJ. Olecranon bursitis as initial presentation of gout in asymptomatic normouricemic patients. *Egypt Rheumatol*. 2014;36(1):47–50.)

DIFFERENTIAL DIAGNOSIS

Olecranon bursitis is usually a straightforward clinical diagnosis. Occasionally, rheumatoid nodules or gouty arthritis of the elbow may confuse the clinical picture. Synovial cysts of the elbow may also mimic olecranon bursitis. Coexistent tendinitis (e.g., tennis elbow, golfer's elbow) may require additional treatment. Rarely, pyoderma gangrenosa may mimic the clinical presentation of olecranon bursitis.

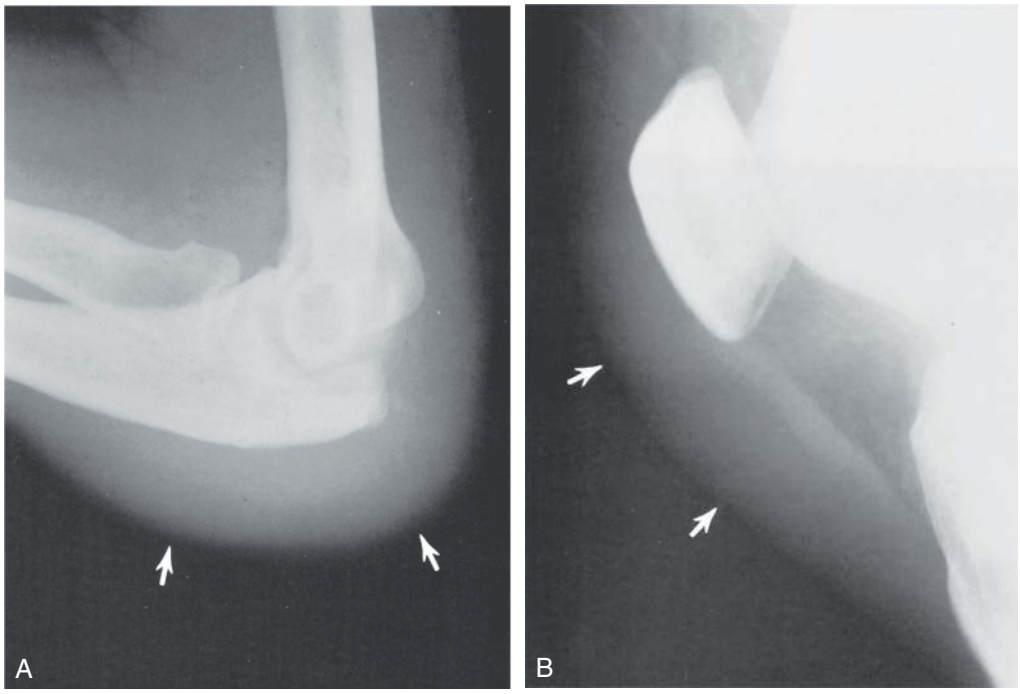

FIG 48.6 Septic bursitis. **A,** Olecranon bursitis. Note the olecranon swelling (*arrows*) and soft tissue edema resulting from *Staphylococcus aureus* infection. Previous surgery and trauma caused the adjacent bony abnormalities. **B,** Prepatellar bursitis. This 28-year-old carpenter who worked on his knees for prolonged periods developed tender swelling in front of the knee (*arrows*). Inflammatory fluid that was culture positive for *S. aureus* was recovered from the bursa. (From Reilly D, Kamineni S. Olecranon bursitis. *J Shoulder Elbow Surg.* 2016;25(1):158–167.)

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs), or cyclooxygenase-2 inhibitors, along with an elbow protector to prevent further trauma, is the initial treatment for patients suffering from olecranon bursitis. If rapid improvement fails to occur, the following injection technique is a reasonable next step.

The patient is placed in the supine position with the arm fully adducted at the patient's side, the elbow flexed, and the palm of the hand resting on the patient's abdomen. A total of 2 mL local anesthetic and 40 mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the posterior aspect of the joint, the olecranon process and overlying bursa are identified. Using strict aseptic technique, the clinician inserts a 1-inch, 25-gauge needle through the skin and subcutaneous tissues directly into the bursa in the midline. If bone is encountered, the needle is withdrawn into the bursa. The contents of the syringe are gently injected; little resistance to injection should be felt. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance may be useful when draining complex loculated or multisegmented olecranon bursa (Fig. 48.7).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after injection for elbow pain. A compression dressing may also help prevent the reaccumulation of fluid following aspiration.

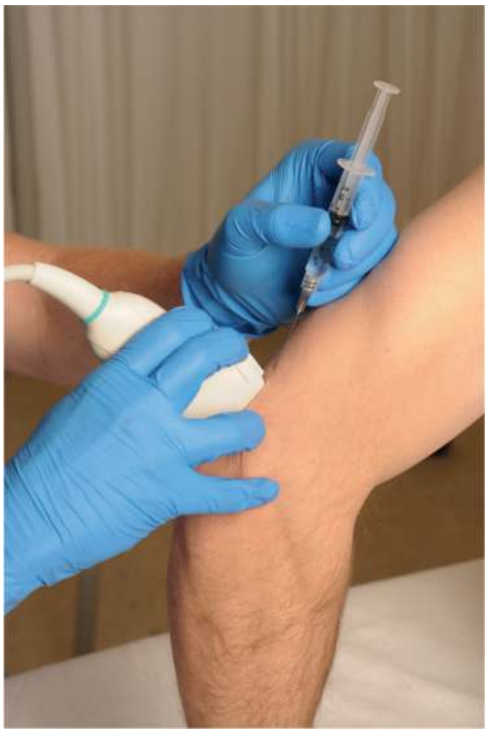

FIG 48.7 Ultrasound-guided injection of the olecranon bursa.

Rarely, surgical removal of the inflamed bursa is required to relieve the pain and functional disability. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

Failure to treat olecranon bursitis adequately may result in chronic pain and loss of elbow range of motion. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. In particular, the ulnar nerve is susceptible to damage at the elbow; such damage can be avoided by keeping the needle trajectory in the midline. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. The major complication of bursal injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection of the olecranon bursa, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain and swelling secondary to olecranon bursitis. Coexistent tendinitis and epicondylitis may contribute to elbow pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with the injection technique.

SUGGESTED READINGS

Reilly D, Kamineni S. Olecranon bursitis. *J Shoulder Elb Surg*. 2016;25(1):158–167.

Waldman SD. Olecranon bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:269–275.

Waldman SD. Olecranon bursa injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:207–210.

Waldman SD. Olecranon bursitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:257–258.

Waldman SD, Campbell RSD. Olecranon bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:273–274.

Working S, Tyser A, Levy D. *Mycobacterium avium* complex olecranon bursitis resolves without antimicrobials or surgical intervention: a case report and review of the literature. *IDCases*. 2015;2(2):59–62.

Yari SS, Reichel LM. Case report: misdiagnosed olecranon bursitis: pyoderma gangrenosum. *J Shoulder Elb Surg*. 2014;23(9):e207–e211.

49

Arthritis Pain of the Wrist

ICD-10 CODE M19.90

THE CLINICAL SYNDROME

Arthritis of the wrist is a common complaint that can cause significant pain and suffering. The wrist joint is susceptible to the development of arthritis from various conditions that have in common the ability to damage joint cartilage. Patients with arthritis of the wrist present with pain, swelling, and decreasing function of the wrist. Decreased grip strength is also a common finding. Osteoarthritis is the most common form of arthritis that results in wrist joint pain. However, rheumatoid arthritis, posttraumatic arthritis, and psoriatic arthritis are also common causes of arthritic wrist pain. These types of arthritis can result in significant alteration in the biomechanics of the wrist, because they affect not only the joint but also the tendons and other connective tissues that make up the functional unit.

SIGNS AND SYMPTOMS

Most patients presenting with wrist pain secondary to osteoarthritis or posttraumatic arthritis complain of pain that is localized around the wrist and hand. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with use of the joint, and crepitus and pain on palpation of the dorsal aspect of the wrist may be present on physical examination (Fig. 49.1). If the pain and dysfunction are secondary to rheumatoid arthritis, the metacarpophalangeal joints are often involved, with characteristic deformity.

In addition to pain, patients suffering from arthritis of the wrist joint often experience a gradual reduction in functional ability because of decreasing wrist range of motion that makes simple everyday tasks such as using a computer keyboard, holding a coffee cup, turning a doorknob, or unscrewing a bottle cap quite difficult (Fig. 49.2). With continued disuse, muscle wasting may occur and adhesive capsulitis with subsequent ankylosis may develop (Fig. 49.3).

TESTING

Plain radiographs are indicated in all patients who present with wrist pain (see Fig. 49.3). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and/or ultrasound imaging of the wrist is indicated if joint instability is thought to be present, as well as to further characterize the causes of pain and functional disability (Figs. 49.4 and 49.5). If infection is suspected, Gram stain and culture of the synovial fluid should be performed on an emergency basis and treatment with appropriate antibiotics should be started.

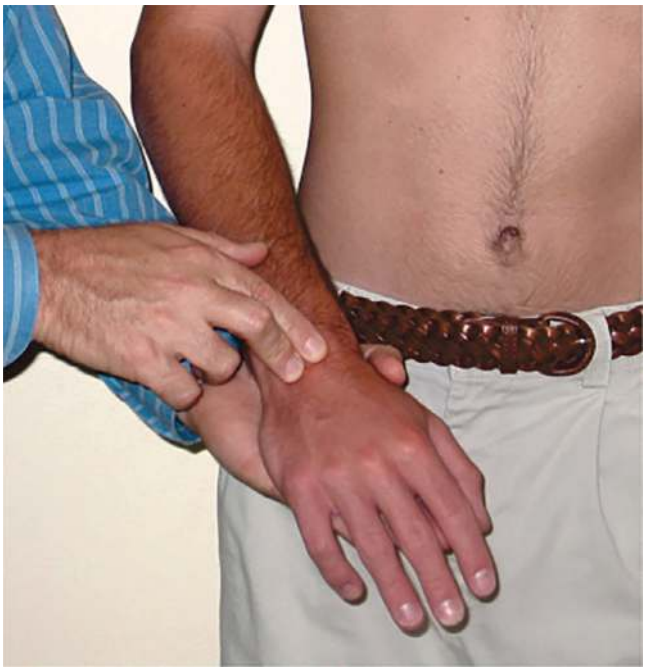

FIG 49.1 Palpation of the dorsal aspect of the wrist. (From Waldman S. *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2021, Fig. 108–1.)

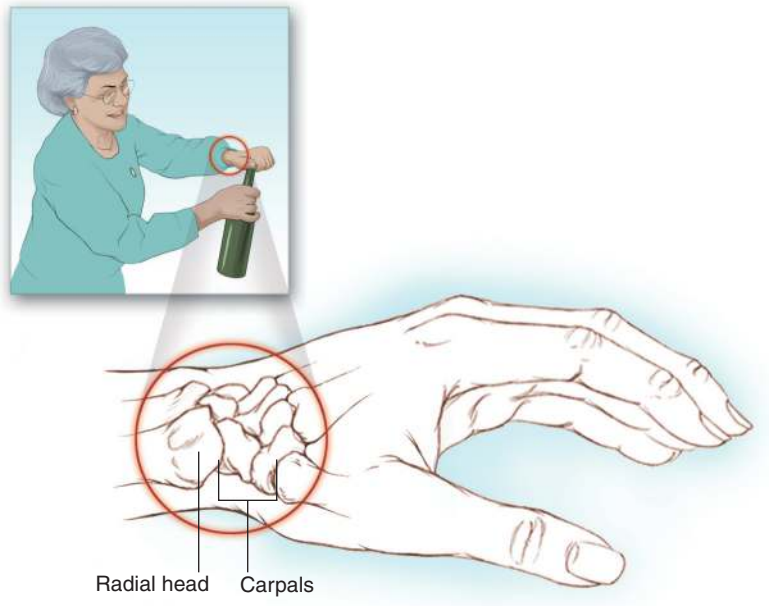

FIG 49.2 Arthritis of the wrist often makes simple everyday tasks such as opening a bottle painful.

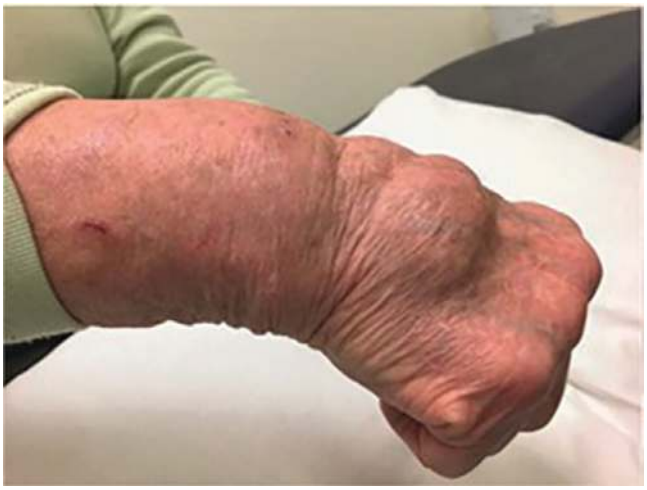

FIG 49.3 Positive Tuck sign in a patient with severe extensor tenosynovitis of the wrist. Prominent 3 × 3 × 4 cm swelling over dorsum of hand with associated distal forearm swelling and intrinsic muscle wasting. (Reproduced from Achilleos KM, Gaffney K. The Tuck sign-proliferative extensor tenosynovitis of the wrist. *Jt Bone Spine*. 2018 [Fig. 1a]. ISSN 1297-319X, https://doi.org/10.1016/j.jbspin.2018.11.007, Copyright © 2018 Elsevier Masson SAS. All rights reserved.)

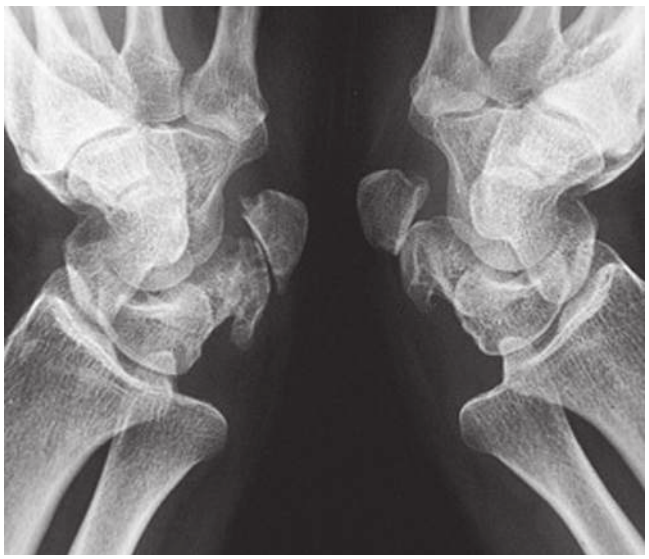

FIG 49.4 Pisotriquetral joint osteoarthritis. Comparative ulnar side radiograph shows bilateral pisotriquetral joint osteoarthritis. (From Feydy A, Pluot E, Guerini H, Drapé J-L. Role of imaging in spine, hand, and wrist osteoarthritis. *Rheum Dis Clin North Am*. 2009:35(3):605–649.)

DIFFERENTIAL DIAGNOSIS

Osteoarthritis is the most common form of arthritis that results in wrist joint pain. However, rheumatoid arthritis and posttraumatic arthritis are also common causes of wrist pain (Figs. 49.6 and 49.7). Less common causes of arthritis-induced wrist pain include collagen vascular diseases, infection, villonodular synovitis, and Lyme disease (Box 49.1). Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized and treated with antibiotics. Collagen vascular diseases generally manifest as polyarthropathy rather than as monarthropathy limited to the wrist joint; however, wrist pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described here.

TREATMENT

Initial treatment of the pain and functional disability associated with osteoarthritis of the wrist includes a combination of

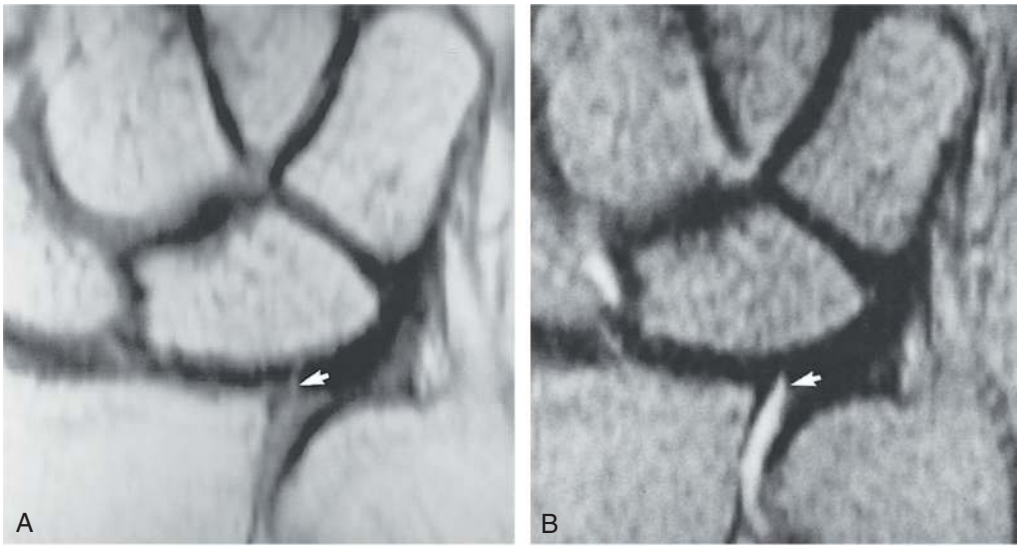

FIG 49.5 Triangular fibrocartilage complex with a communicating defect. **A,** Coronal intermediate-weighted spin-echo magnetic resonance imaging (MRI). Note the linear region of increased signal intensity (*arrow*) in the triangular fibrocartilage. **B,** T2-weighted spin-echo MRI. Fluid of high signal intensity is present in the defect (*arrow*) in the triangular fibrocartilage and in the distal radioulnar joint. Fluid is also present in the midcarpal joint. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3033.)

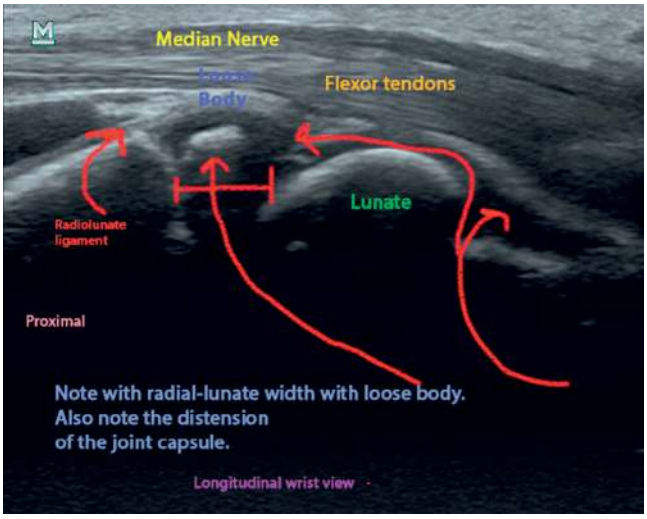

FIG 49.6 Longitudinal ultrasound image demonstrating a joint mouse in the radiolunate joint. Note the distention of the joint capsule.

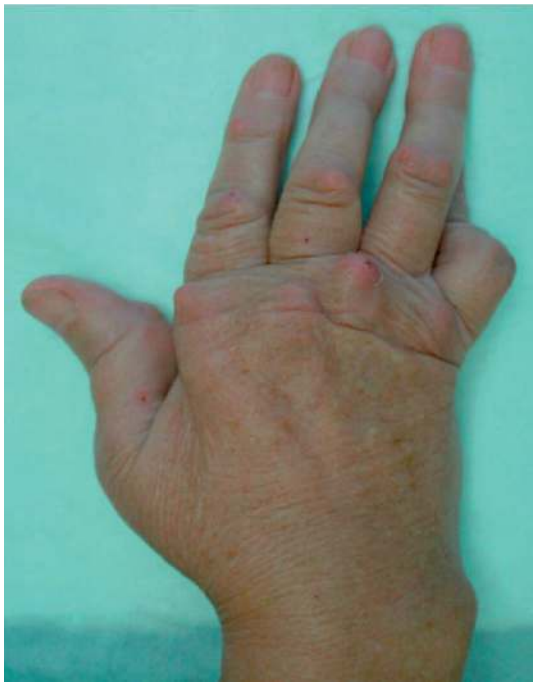

FIG 49.7 Photograph of a rheumatoid hand; note axial deviation of the wrist. (From Trieb K. Treatment of the wrist in rheumatoid arthritis. *J Hand Surg*. 2008;33(1):113–123.)

nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Splinting the wrist in the neutral position may provide symptomatic relief and protect the joint from additional trauma. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

Intraarticular injection of the wrist is performed by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow slightly flexed, and the palm of the hand resting on a folded towel. A total of 1.5-mL local anesthetic and 40-mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the dorsal joint, the midcarpus proximal to the indentation of the capitate bone is identified. Just proximal to the capitate bone is an indentation that allows

BOX 49.1 Causes of Wrist Pain

- Bony Abnormalities
  - Fracture
  - Tumor
  - Osteomyelitis
  - Osteonecrosis
  - Kienböck disease and Preiser disease
- Articular Abnormalities
  - Osteoarthritis
  - Rheumatoid arthritis
- Collagen Vascular Diseases
  - Reiter syndrome
  - Psoriatic arthritis
- Crystal Deposition Diseases
  - Gout
  - Pseudogout
  - Pigmented villonodular synovitis
  - Sprain
  - Strain
  - Hemarthrosis
- Periarticular Abnormalities
  - Tendon sheath disorders
  - Trigger finger
  - Flexor tenosynovitis
  - Extensor tenosynovitis
  - de Quervain tenosynovitis
  - Dupuytren contracture
  - Ganglion cyst
  - Gouty tophi
  - Subcutaneous nodules associated with rheumatoid arthritis
  - Glomus tumor
- Neurologic Abnormalities
  - Median nerve entrapment
  - Carpal tunnel syndrome
  - Pronator syndrome
  - Anterior interosseous nerve syndrome
  - Ulnar nerve entrapment
  - Ulnar tunnel syndrome
  - Cubital tunnel syndrome
  - Cheiralgia paresthetica
  - Lower brachial plexus lesions
  - Cervical nerve root lesions
  - Spinal cord lesions
  - Syringomyelia
  - Spinal cord tumors
  - Reflex sympathetic dystrophy
  - Causalgia
- Vascular Abnormalities
  - Vasculitis
  - Raynaud syndrome
  - Takayasu arteritis
  - Scleroderma
- Referred Pain
  - Shoulder-hand syndrome
  - Angina

From Waldman SD. Painful conditions of the wrist and hand. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016 chap 102: 166.

easy access to the wrist joint. Using strict aseptic technique, the clinician inserts a 1-inch, 25-gauge needle in the center of the midcarpal indentation through the skin, subcutaneous tissues, and joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. The injection of platelet-rich plasma and/or stem cells have been advocated to reduce the pain and functional disability of arthritis of the wrist.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient begins treatment for arthritis of the wrist. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

Joint protection is especially important in patients suffering from inflammatory arthritis of the wrist, because repetitive trauma can result in further damage to the joint, tendons, and connective tissues. The major complication of intraarticular injection of the wrist is infection, although it should be exceedingly rare if strict aseptic technique is followed. The injection technique is safe if careful attention is paid to the clinically relevant anatomy; the ulnar nerve is especially susceptible to damage at the wrist. Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the wrist joint, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to arthritis of the wrist joint. Simple analgesics and NSAIDs can be used concurrently with the injection technique. Coexistent bursitis and tendinitis may contribute to wrist pain and necessitate additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Bay COA, Willacy RA, Moses AR, Coleman TE, Wilson RH. Nonspecific wrist pain in pediatric patients: a systematic review. *J Orthop*. 2020;22:308–315.

Brewer PE, Storey PA. The hand and wrist in rheumatoid and osteoarthritis. *Surgery (Oxf)*. 2016;34(3):144–151.

Dineen HA, Greenberg JA. Ulnar-sided wrist pain in the athlete. *ClSports Med*. 2020;39(2):373–400.
Laulan J, Marteau E, Bacle G. Wrist osteoarthritis. *Orthop Traumatol Surg Res*. 2015;101(1):S1–S9.
Ul Islam S, Brown D, Cheung G. Management of osteoarthritis of the wrist and hand. *Orthop Trauma*. 2019;33(1):30–37.
Waldman SD. Abnormalities of the distal radioulnar joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:353–360.
Waldman SD. Clinical correlates: functional anatomy of the wrist. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2016:158–159.
Waldman SD. Intra-articular injection of the wrist. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:250–253.
Waldman SD. Arthritis pain of the wrist. In: *Atlas of common pain syndromes*. 4th ed. Philadelphia: Elsevier; 2019: 190–194.

Carpal Tunnel Syndrome

ICD-10 CODE G56.00

THE CLINICAL SYNDROME

Carpal tunnel syndrome is the most common entrapment neuropathy encountered in clinical practice. It is caused by compression of the median nerve as it passes through the carpal canal at the wrist. The most common causes of compression of the median nerve at this location include flexor tenosynovitis, rheumatoid arthritis, pregnancy, amyloidosis, and other space-occupying lesions that compromise the median nerve as it passes through this closed space. It occurs more commonly in women. This entrapment neuropathy presents as pain, numbness, paresthesias, and associated weakness in the hand and wrist that radiate to the thumb, index finger, middle finger, and radial half of the ring finger. These symptoms may also radiate proximal to the entrapment into the forearm. Untreated, progressive motor deficit and, ultimately, flexion contracture of the affected fingers can result. Symptoms usually begin after repetitive wrist motions or repeated pressure on the wrist, such as resting the wrists on the edge of a computer keyboard (Fig. 50.1, Box 50.1). Direct trauma to the median nerve as it enters the carpal tunnel may result in a similar clinical presentation. Recent studies have suggested a higher incidence of abnormalities of connective tissue coding genes in patients suffering from carpal tunnel syndrome when compared with normal controls.

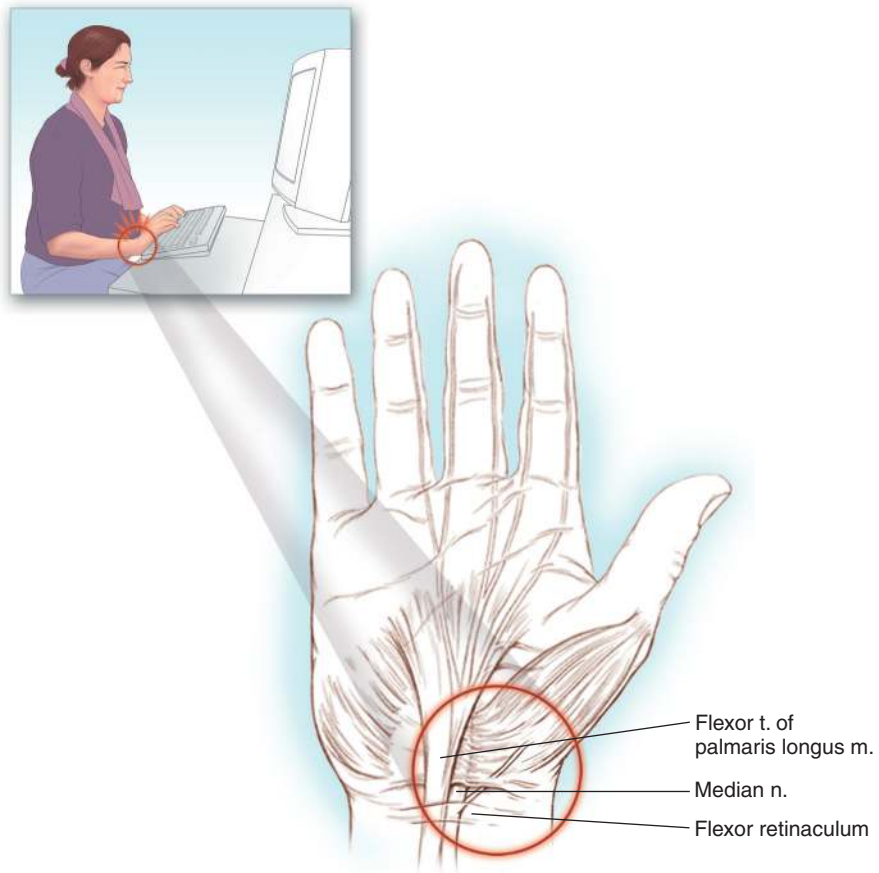

FIG 50.1 Poor positioning of the hand and wrist during keyboarding can result in carpal tunnel syndrome.

BOX 50.1 Conditions Associated With Carpal Tunnel Syndrome

Structural/Anatomic
- Persistent median artery
- Aneurysm
- Lipoma
- Ganglion
- Neuroma
- Acromegaly
- Fracture

Inflammatory
- Tenosynovitis
- Collagen vascular disease
  - Rheumatoid arthritis
  - Scleroderma
- Gout
- Crystal deposition disease

Neuropathic/Ischemic
- Diabetes
- Alcoholism
- Vitamin abnormalities
- Ischemic neuropathies
- Peripheral neuropathies
- Amyloidosis

Shifts in Fluid Balance
- Pregnancy
- Hypothyroidism
- Obesity
- Kidney failure
- Menopause

Repetitive Stress Related
- Abnormal hand and wrist position
- Excessive flexion
- Microtrauma
- Vibration

SIGNS AND SYMPTOMS

Physical findings include tenderness over the median nerve at the wrist. A positive Tinel sign is usually present over the median nerve as it passes beneath the flexor retinaculum (Fig. 50.2). A positive Phalen maneuver is highly suggestive of carpal tunnel syndrome. The Phalen maneuver is performed by having the patient place the wrists in complete unforced flexion for at least 30 seconds (Fig. 50.3). If the median nerve is entrapped at the wrist, this maneuver reproduces the symptoms of carpal tunnel syndrome. Weakness of thumb opposition and wasting of the thenar eminence are often seen in advanced cases of carpal tunnel syndrome; however, because of the complex motion of the thumb, subtle motor deficits can easily be missed (Fig. 50.4). Early in the course of carpal tunnel syndrome, the only physical finding other than tenderness over the median nerve may be the loss of sensation in the foregoing fingers. Untreated, muscle wasting of the thenar eminence can be seen (Fig. 50.5).

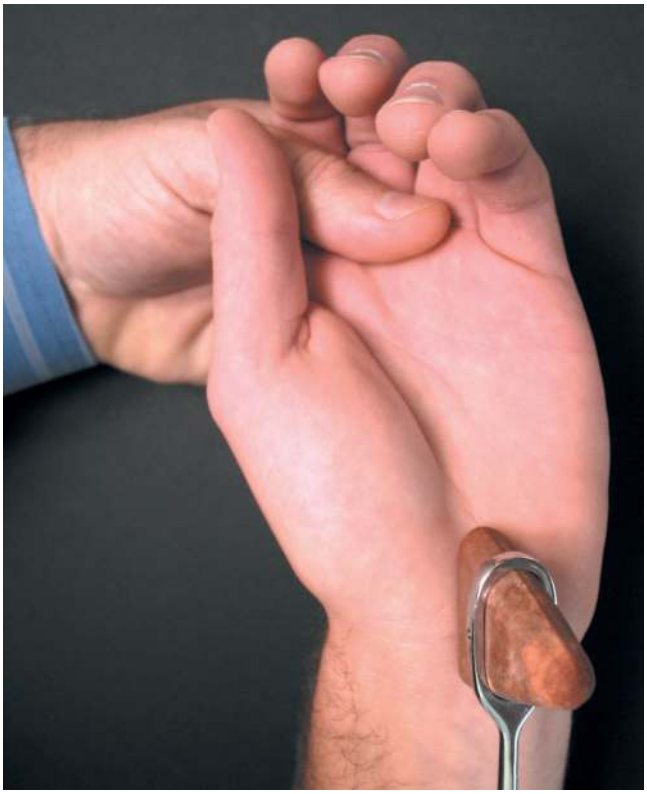

FIG 50.2 Tinel's sign for carpal tunnel syndrome. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:178.)

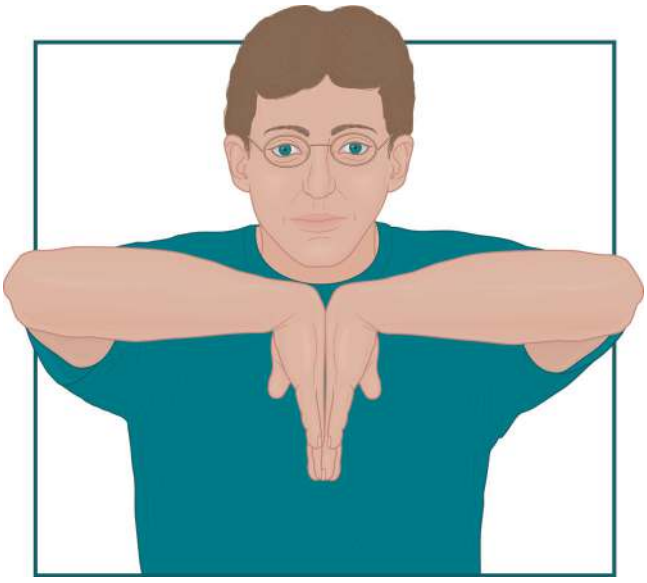

FIG 50.3 A positive Phalen maneuver is highly indicative of carpal tunnel syndrome. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

TESTING

Electromyography can distinguish cervical radiculopathy and diabetic polyneuropathy from carpal tunnel syndrome. Plain radiographs are indicated in all patients who present with carpal tunnel syndrome, to rule out occult bony

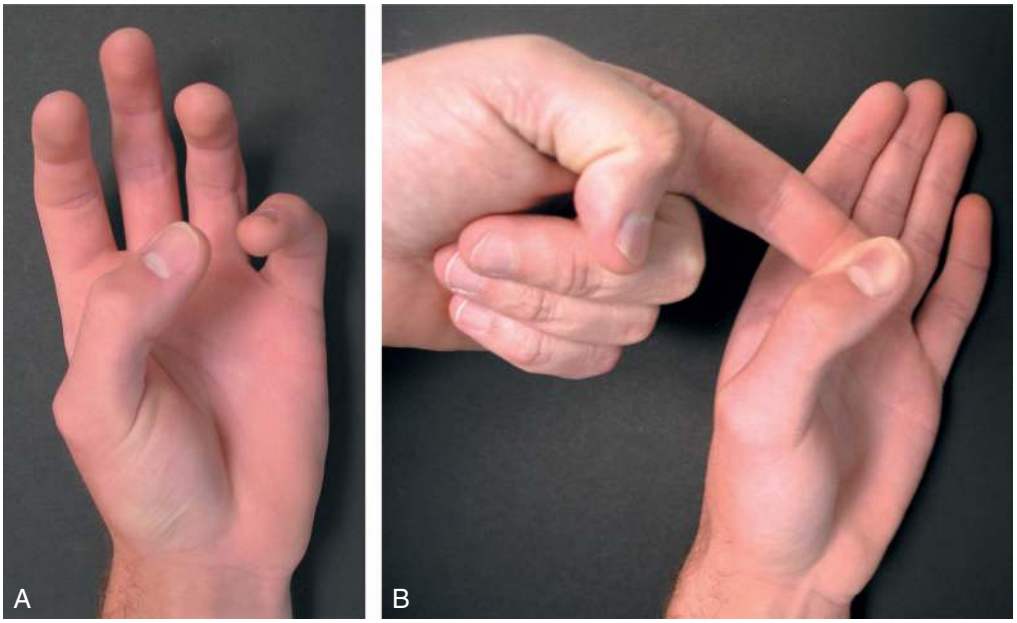

FIG 50.4 (A&B) The opponens weakness test for carpal tunnel syndrome. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:180.)

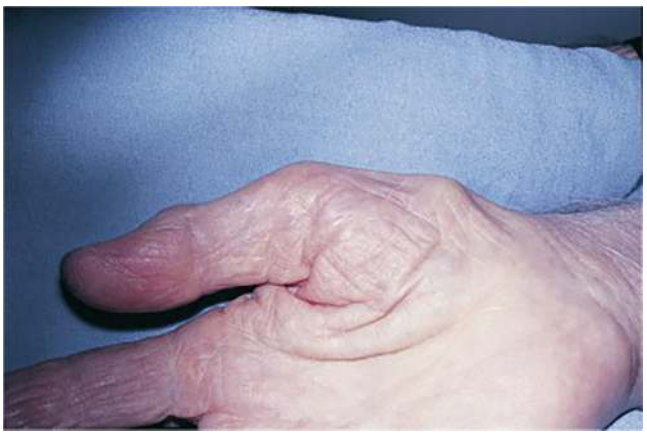

FIG 50.5 Wasting of the thenar eminence is seen as carpal tunnel syndrome progresses. (From Nahsel DJ: Soft tissue. In Klippel JH, Dieppe PA [eds]: *Rheumatology*, 2nd ed. London, Mosby, 1998, p 4–16.7)

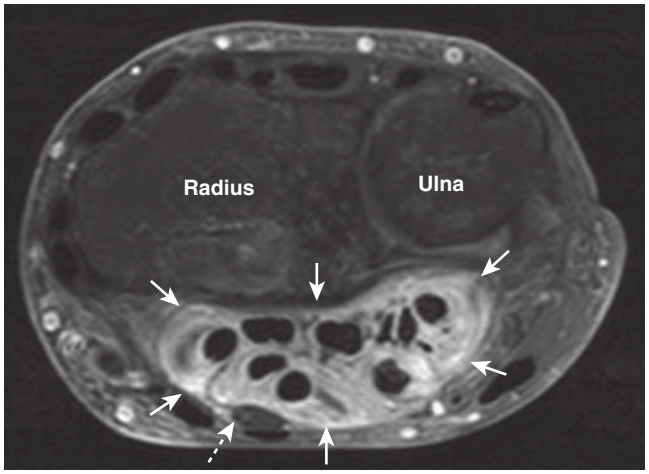

FIG 50.6 Axial fast-spin T2-weighted (FST2W) magnetic resonance (MR) image of the wrist in a patient with rheumatoid arthritis and symptoms of carpal tunnel syndrome. Extensive high-SI tenosynovitis surrounds the flexor tendons just proximal to the carpal tunnel (*white arrows*). The median nerve (*broken white arrow*) lies between the inflamed flexor tendons and the normal palmaris longus tendon. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011: Fig. 125.2.)

disorders. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging of the wrist is indicated if joint instability or a space-occupying lesion is suspected or to confirm the actual cause of median nerve compression (Fig. 50.6). Ultrasound imaging may also be useful in the evaluation of the median nerve as it passes through the carpal tunnel (Fig. 50.7). Studies have suggested a strong correlation between the cross-sectional area of the nerve and clinical carpal tunnel syndrome (Fig. 50.8). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Carpal tunnel syndrome is often misdiagnosed as arthritis of the carpometacarpal joint of the thumb, cervical radiculopathy, or diabetic polyneuropathy. Patients with arthritis of the carpometacarpal joint of the thumb have a positive Watson test and radiographic evidence of arthritis. Most patients suffering from cervical radiculopathy have reflex, motor,

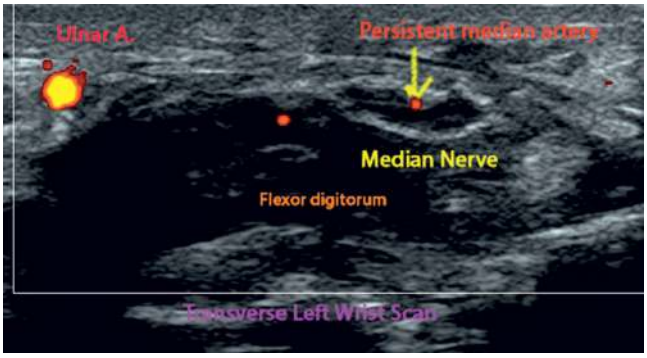

FIG 50.7 Transverse image in a woman with carpal tunnel syndrome shows a persistent median artery (*arrow*).

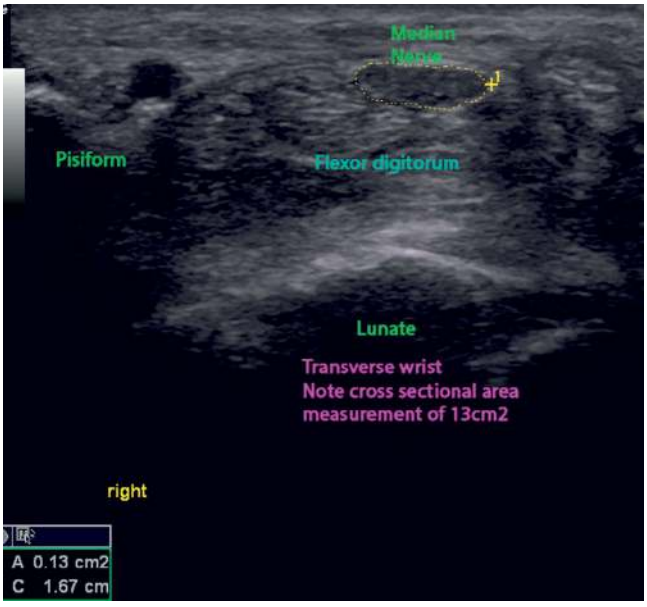

FIG 50.8 Transverse ultrasound image of the median nerve at the proximal wrist crease demonstrating an increased cross-sectional area of the 13 cm^2 which is highly suggestive of carpal tunnel syndrome. Note the loss of the normal neural echo texture of the median nerve.

and sensory changes associated with neck pain; in contrast, patients with carpal tunnel syndrome have no reflex changes, and motor and sensory changes are limited to the distal median nerve. Diabetic polyneuropathy generally manifests as a symmetric sensory deficit involving the entire hand, rather than being limited to the distribution of the median nerve. Cervical radiculopathy and median nerve entrapment may coexist as the double-crush syndrome. Furthermore, carpal tunnel syndrome is commonly seen in patients with diabetes, and it is not uncommon for diabetic polyneuropathy to be present as well.

TREATMENT

Mild cases of carpal tunnel syndrome usually respond to conservative therapy; surgery should be reserved for more severe cases. Initial treatment of carpal tunnel syndrome consists of

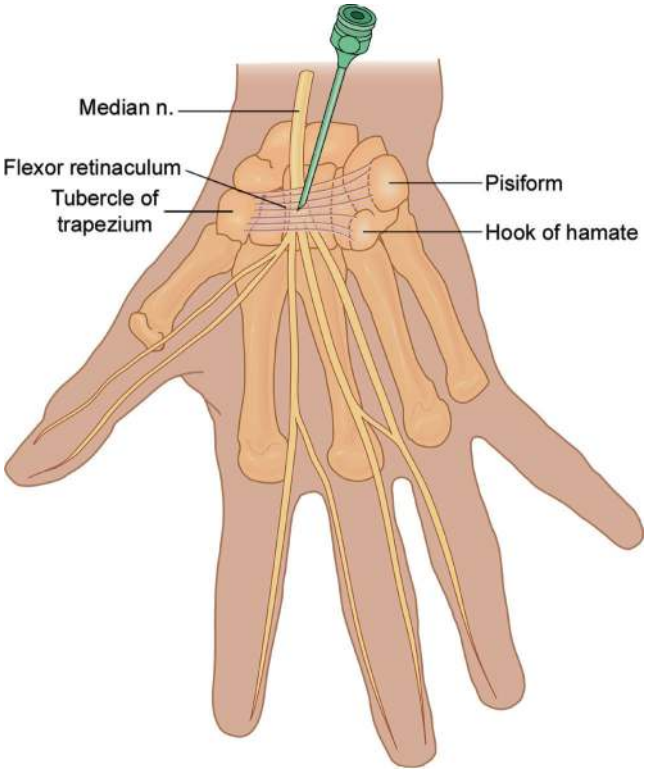

FIG 50.9 Proper needle placement for injection of the carpal tunnel. (From Waldman SD. *Atlas of pain management injection techniques.* Philadelphia: Saunders; 2000.)

simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors and splinting of the wrist. At a minimum, the splint should be worn at night, but wearing it for 24 hours a day is ideal. Avoidance of the repetitive activities that are thought to be responsible for carpal tunnel syndrome (e.g., keyboarding, hammering) can also help ameliorate the patient's symptoms. If the patient fails to respond to these conservative measures, a next reasonable step is injection of the carpal tunnel with local anesthetic and steroid.

Carpal tunnel injection is performed by placing the patient in the supine position with the arm fully abducted at the patient's side, the elbow slightly flexed, and the dorsum of the hand resting on a folded towel. A total of 3 mL local anesthetic and 49 mg methylprednisolone is drawn up in a 5-mL sterile syringe. The patient is then told to make a fist and at the same time flex his or her wrist to aid in identifying the palmaris longus tendon. After the preparation of the skin with antiseptic solution, a ⅝-inch, 25-gauge needle is inserted just medial to the tendon and just proximal to the crease of the wrist at a 30-degree angle (Fig. 50.9). The needle is slowly advanced until the tip is just beyond the tendon. Paresthesia in the distribution of the median nerve is often elicited, and the patient should be warned to expect this and to say "There!" as soon as the paresthesia is felt. If a paresthesia is elicited, the needle is withdrawn slightly away from the median nerve. Gentle aspiration is then carried out to identify blood. If the aspiration test result is negative and no persistent paresthesia is noted in the distribution of the median nerve, 3 mL of solution is slowly

FIG 50.10 Proper needle placement for ultrasound-guided injection for carpal tunnel syndrome utilizing an out of plane approach at the wrist.

injected while the patient is monitored closely for signs of local anesthetic toxicity. If no paresthesia is elicited and the needle tip hits bone, the needle is withdrawn out of the periosteum, and, after careful aspiration, 3 mL of solution is slowly injected. Ultrasound needle guidance may improve the accuracy of needle placement and help avoid needle-induced trauma to the median nerve (Fig. 50.10).

When these treatment modalities fail, surgical release of the median nerve at the carpal tunnel is indicated. Endoscopic techniques appear to result in less postoperative pain and dysfunction. Recent studies suggest that a trial of extracorporeal shock wave therapy may be considered as an alternative treatment for carpal tunnel syndrome.

COMPLICATIONS AND PITFALLS

Failure to treat carpal tunnel syndrome adequately can result in permanent pain, numbness, and functional disability. The problem can be exacerbated if coexistent reflex sympathetic dystrophy is not aggressively treated with sympathetic neural blockade. Injection of the carpal tunnel is a relatively safe technique. The major complications are inadvertent intravascular injection and persistent paresthesia secondary to needle-induced trauma to the nerve. This technique can be safely performed in the presence of anticoagulation by using a 25- or 27-gauge needle, albeit with an increased risk of hematoma formation. The incidence of this complication can be decreased if manual pressure is applied to the area immediately after injection. The application of cold packs for 20 minutes after injection can also decrease the degree of postprocedure pain and bleeding.

CLINICAL PEARLS

Carpal tunnel syndrome should always be differentiated from cervical radiculopathy involving the cervical nerve roots, which may mimic median nerve compression. Furthermore, cervical radiculopathy and median nerve entrapment may coexist in the double-crush syndrome.

Carpal tunnel injection is a simple and safe technique. Before median nerve block at the wrist is initiated, a careful neurologic examination should be performed to identify preexisting neurologic deficits that may later be attributed to the nerve block, especially in patients with clinical symptoms of diabetes or clinically significant carpal tunnel syndrome.

Care should be taken to place the needle just beyond the flexor retinaculum and to inject slowly, to allow the solution to flow easily into the carpal tunnel without further compromising the median nerve.

SUGGESTED READINGS

Borire AA, Hughes AR, Lueck CJ, et al. Sonographic differences in carpal tunnel syndrome with normal and abnormal nerve conduction studies. *J Clin Neurosci*. 2016;34:77–80.

Csillik A, Bereczki D, Bora L, Arányi Z. The significance of ultrasonographic carpal tunnel outlet measurements in the diagnosis of carpal tunnel syndrome. *Clin Neurophysiol*. 2016;127(12):3516–3523.

Raissi GR, Ghazaei F, Forogh B, et al. The effectiveness of radial extracorporeal shock waves for treatment of carpal tunnel syndrome: a randomized clinical trial. *Ultrasound Med Biol*. 2017;43(2):453–460.

Waldman SD. Carpal tunnel syndrome and other abnormalities of the median nerve at the wrist. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:370–385.

Waldman SD. Injection technique for carpal tunnel syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:269–273.

Waldman SD. Carpal tunnel syndrome. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:259–260.

Waldman SD, Campbell RSD. Carpal tunnel syndrome. In: *Imaging of pain*. Philadelphia: Saunders; 2011:319–321.

51

Flexor Carpi Ulnaris Tendinitis

ICD-10 M65.849

THE CLINICAL SYNDROME

The flexor carpi ulnaris tendon of the hand may develop tendinitis after overuse or misuse, especially when performing activities that require repeated flexion and adduction of the hand. Acute flexor carpi ulnaris tendinitis has been seen in clinical practice with increasing frequency owing to the increasing popularity of racquet sports such as tennis, baseball, and golf (Fig. 51.1). Improper stretching of flexor carpi ulnaris muscle and flexor carpi ulnaris tendon before exercise has also been implicated in the development of flexor carpi ulnaris tendinitis, as well as acute tendon rupture. Injuries ranging from partial to complete tears of the tendon can occur when the distal tendon sustains direct trauma while it is fully flexed under load or when the wrist is forcibly flexed while the hand is full radial deviation.

SIGNS AND SYMPTOMS

The pain of flexor carpi ulnaris tendinitis is constant and severe and is localized to the dorsoulnar aspect of the wrist. The patient suffering from flexor carpi ulnaris tendinitis often complains of sleep disturbance owing to pain. Patients with flexor carpi ulnaris tendinitis exhibit pain with active resisted flexion of the hand and with radial deviation of the wrist. In an effort to decrease pain, patients suffering from flexor carpi ulnaris tendinitis often splint the inflamed tendon by limiting hand flexion and radial deviation of the wrist to remove tension from the inflamed tendon. If untreated, patients suffering from flexor carpi ulnaris tendinitis may experience difficulty in performing any task that requires flexion and adduction of the wrist and hand such as using a hammer or lifting a heavy coffee mug. Over time, if the tendinitis is not treated, muscle atrophy and calcific tendinitis may result or the distal musculotendinous unit may suddenly rupture (Fig. 51.2). Patients who experience complete rupture of the flexor carpi ulnaris tendon will not be able to fully and forcefully flex the hand or fully adduct the wrist.

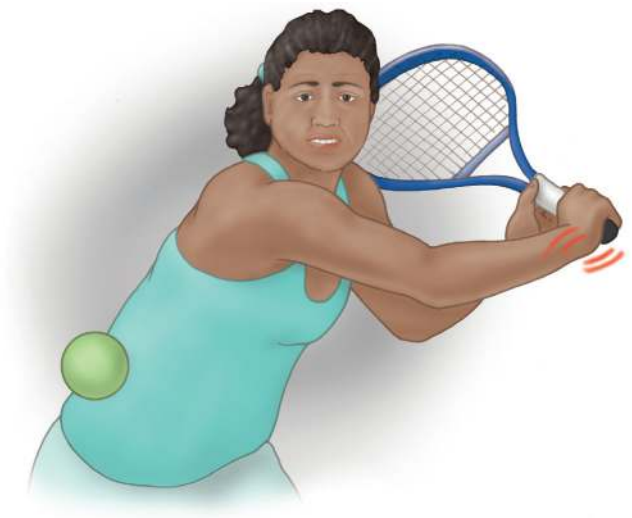

FIG 51.1 The flexor carpi ulnaris tendon of the hand may develop tendinitis after overuse or misuse, especially when performing activities that require repeated flexion and adduction of the hand as when playing tennis, baseball, or racquetball.

TESTING

Plain radiographs are indicated in all patients who present with wrist and hand pain (Fig. 51.2). Based on the patient's clinical presentation, additional testing may be indicated, including complete blood cell count, sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging or ultrasound imaging of the wrist and hand is indicated if flexor carpi ulnaris tendinopathy or tear is suspected. Magnetic resonance imaging or ultrasound evaluation of the affected area may also help delineate the presence of calcific tendinitis or other hand pathology (Figs. 51.3–51.5).

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of ulnar-sided wrist pain should include extensor carpi ulnaris tendonitis, flexor carpi ulnaris tendonitis, pisotriquetral arthritis, triangular fibrocartilage complex lesions, ulnar impaction, lunotriquetral instability, hook of the hamate fracture, hypothenar hammer syndrome, and distal radioulnar joint instability (Box 51.1). Flexor carpi ulnaris is localized to the dorsoulnar aspect of the wrist and will worsen with flexion, distinguishing it from extensor carpi ulnaris tendonitis.

TREATMENT

Initial treatment of the pain and functional disability associated with flexor carpi ulnaris tendinitis includes a combination

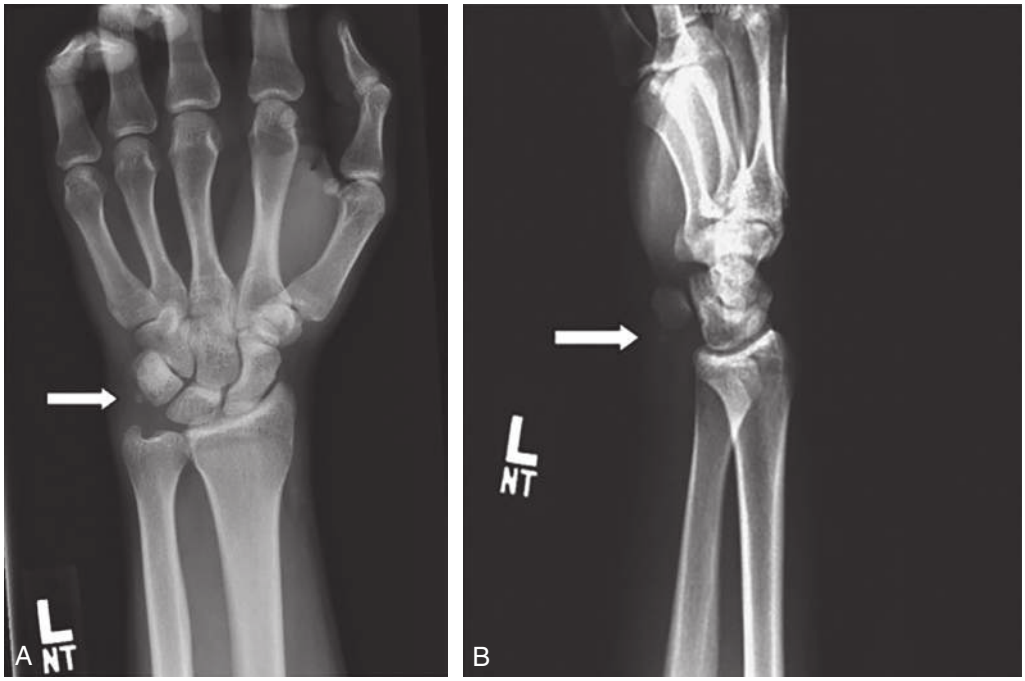

FIG 51.2 Anteroposterior **(A)** and oblique **(B)** radiographs demonstrate a small area of calcification (*arrow*) just proxymal to the pisiform consistent with calcific tendinitis of the flexor carpi ulnaris tendon. (From Torbati SS, Bral D, Geiderman JM. Acute calcific tendinitis of the wrist. *J Emerg Med*. 2013;44(2):352–354.)

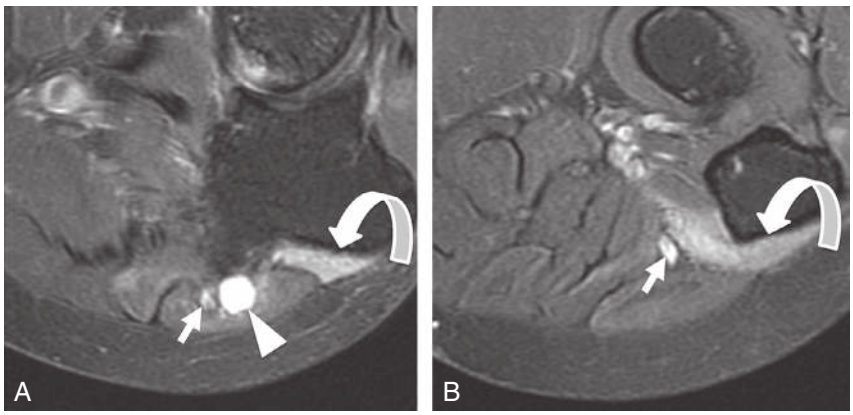

FIG 51.3 Axial T2-weighted fat-saturated images at proximal forearm demonstrate an extraneural ganglion cyst arising from the ulnotrochlear margin of the joint (**A,** *arrowhead*). The cyst compresses the T2-weighted hyperintense and enlarged ulnar nerve (**A, B,** *arrows*) against the adjacent flexor carpi ulnaris. Denervation change is present in the proximal flexor digitorum profundus muscle (**A, B,** *curved arrows*). (From Howe BM, Spinner RJ, Felmlee JP, Frick MA. MR imaging of the nerves of the upper extremity: elbow to wrist. *Magn Reson Imaging Clin North Am*. 2015;23(3):469–478.)

of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities, injection of local anesthetic and steroid is a reasonable next step.

Injection for flexor carpi ulnaris tendinitis is performed by placing the patient in the supine position with the arm fully adducted at the patient's side, the elbow flexed, and the dorsum of the hand resting on a folded towel to relax the affected tendons. A total of 1 mL local anesthetic and 40 mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the insertion of the flexor carpi ulnaris tendon, the patent is asked to forcefully extend his or her wrist to simplify the identification of the tendon. Using strict aseptic technique, a 1-inch, 25-gauge needle is inserted through the skin and into the subcutaneous tissue

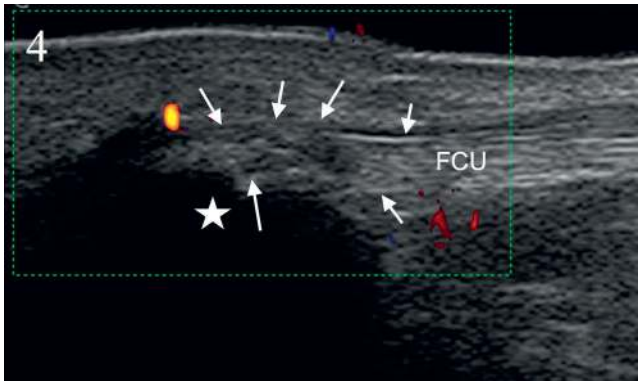

FIG 51.4 Sagittal high-frequency ultrasound image of an active flexi carpi ulnaris (FCU) tendon enthesopathy (i.e., the simultaneous sonomorphologic occurrence of tendon thickening and tendon hypoechogenity with loss of the fibrillar echotexture [*arrows*], in a 45-year-old woman with painful pisiform overuse). The ★ indicates the pisiform. (From Wick MC, Weiss RJ, Arora R, et al. Enthesopathy of the flexor carpi ulnaris at the pisiform: findings of high-frequency sonography. *Eur J Radiol*. 2011;77(2):240–244.)

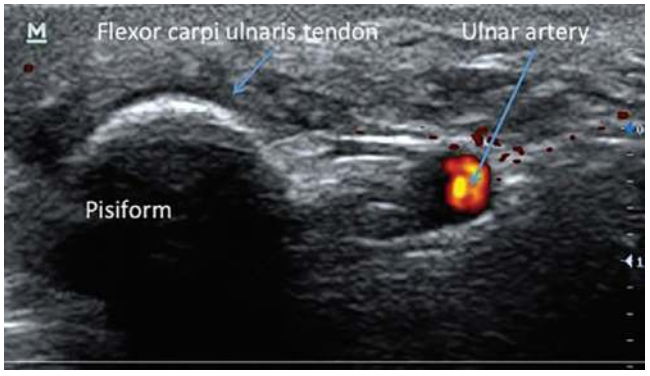

FIG 51.5 Color Doppler ultrasound image demonstrating the flexor carpi ulnaris insertion on the pisaform. Note the relationship of the flexor carpi ulnaris tendon to the ulnar artery.

BOX 51.1 Common Causes of Ulnar-Sided Wrist Pain

- Extensor carpi ulnaris tendonitis
- Flexor carpi ulnaris tendonitis
- Pisotriquetral arthritis
- Triangular fibrocartilage complex lesions
- Ulnar impaction
- Lunotriquetral instability
- Hook of the hamate fracture
- Hypothenar hammer syndrome
- Distal radioulnar joint instability

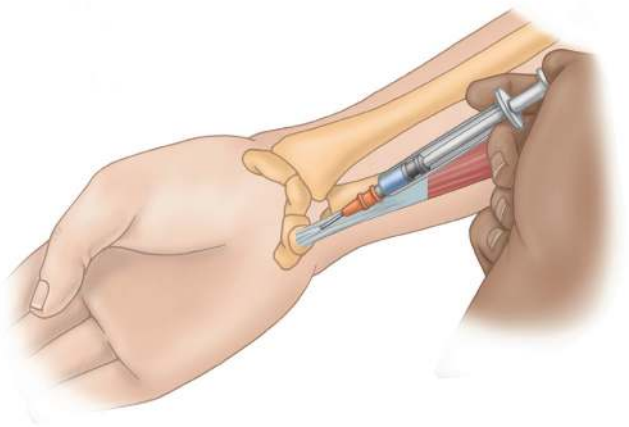

FIG 51.6 Injection technique for flexor carpi ulnaris injection.

overlying the affected tendon (Fig. 51.6). If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Recent clinical experience suggests that the injection of type A botulinum toxin and platelet-rich plasma and/or stem cells may provide improved symptom relief and healing of flexor carpi ulnaris tendinitis. Ultrasound guidance may improve the accuracy of needle placement in patients in whom anatomic landmarks are hard to identify.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for flexor carpi ulnaris tendinitis. Low-level laser therapy may also be beneficial. A Velcro counterforce orthotic band placed around the extensor tendons may also help relieve the symptoms. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The major complication associated with flexor carpi ulnaris tendinitis is rupture of the inflamed tendon either from repetitive trauma or from injection directly into the tendon. To prevent inflamed and previously damaged tendons from rupturing, the needle position should be confirmed to be outside the tendon before the clinician proceeds with the injection. Another complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. The injection technique is safe if careful attention is paid to the clinically relevant anatomy; in particular, the ulnar nerve is susceptible to damage at the elbow. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The distal musculotendinous unit of the flexor carpi ulnaris muscle is subjected to an amazing variation of stresses as it performs its function of flexing and adducting the hand. The relatively poor blood supply of the distal musculotendinous unit limits the ability of the muscle and tendon to heal when traumatized. Over time, muscle tears and tendinopathy develop, further weakening the musculotendinous unit and making it susceptible to additional damage and ultimately complete rupture.

Wrist pathology, including osteophytes of the scaphoid and pisiform bones, bursitis, osteoarthritis, avascular necrosis, and entrapment neuropathies including carpal tunnel syndrome, may coexist with flexor carpi ulnaris tendinitis and may contribute to the patient's pain symptomatology. Universal precautions should always be observed to protect the operator, and strict adherence to sterile technique must be used to avoid infection. Gentle physical therapy and local heat should be introduced following ultrasound-guided injection of flexor carpi ulnaris tendinitis to reduce pain and improve function. Simple analgesics and nonsteroidal antiinflammatory agents or COX-2 inhibitors may be used concurrently with this injection technique.

SUGGESTED READINGS

Fedorczyk JM. Tendinopathies of the elbow, wrist, and hand: histopathology and clinical considerations. *J Hand Ther*. 2012;25(2):191–201.

Henderson CJ, Kobayashi KM. Ulnar-sided wrist pain in the athlete. *Orthopedic Clin North Am*. 2016;47(4):789–798.

Patrick NC, Warren C. Hammert, hand and wrist tendinopathies. *ClSports Med*. 2020;39(2):247–258.

Pirolo JM, Yao J. Minimally invasive approaches to ulnar-sided wrist disorders. *Hand Clin*. 2014;30(1):77–89.

Torbati SS, Bral D, Geiderman JM. Acute calcific tendinitis of the wrist. *J Emerg Med*. 2013;44(2):352–354.

Waldman SD. Flexor carpi ulnaris and other abnormalities of the flexor carpi ulnaris tendon. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:413–416.

Waldman SD. Injection technique for flexor carpi ulnaris tendinitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2017:262–264.

Waldman SD, Campbell RSD. Biceps tendinopathy. In: *Imaging of pain*. Philadelphia: Saunders; 2011:329–330.

Wick MC, Weiss RJ, Arora R, et al. Enthesopathy of the flexor carpi ulnaris at the pisiform: findings of high-frequency sonography. *Eur J Radiol*. 2011;77(2):240–244.

52

de Quervain's Tenosynovitis

ICD-10 CODE M65.4

THE CLINICAL SYNDROME

de Quervain's tenosynovitis is caused by inflammation and swelling of the tendons of the abductor pollicis longus and extensor pollicis brevis tendons at the level of the radial styloid process. This painful condition occurs most commonly between the ages of 30 and 50 years. It occurs more frequently in women. It is usually the result of trauma to the tendon from repetitive twisting motions. This condition is often associated with inflammatory arthritis including rheumatoid arthritis. There is also an association with pregnancy and baby care as lifting an infant requires using the thumbs for leverage. If the inflammation and swelling become chronic, the tendon sheath thickens, resulting in its constriction. A triggering phenomenon may occur, with the tendon catching within the sheath and causing the thumb to lock, or "trigger." Arthritis and gout of the first metacarpal joint may coexist with de Quervain's tenosynovitis and exacerbate the associated pain and disability.

de Quervain's tenosynovitis occurs in patients engaged in repetitive activities such as handshaking or high-torque wrist turning (e.g., when scooping ice cream). de Quervain's tenosynovitis may also develop without obvious antecedent trauma.

The pain of de Quervain's tenosynovitis is localized to the region of the radial styloid. It is constant and is made worse with active pinching activities of the thumb or ulnar deviation of the wrist (Fig. 52.1). Patients note an inability to hold a coffee cup or turn a screwdriver. Sleep disturbance is common.

SIGNS AND SYMPTOMS

On physical examination, the patient has tenderness and swelling over the tendons and tendon sheaths along the distal radius, with point tenderness over the radial styloid (Fig. 52.2). Many patients with de Quervain's tenosynovitis note a creaking sensation with flexion and extension of the thumb. A catching or stop-and-go sensation may be present when moving the thumb. A range of motion of the thumb may be decreased by the pain, and a trigger thumb phenomenon may be noted. Patients with de Quervain's tenosynovitis demonstrate a positive Finkelstein test result (Fig. 52.3).

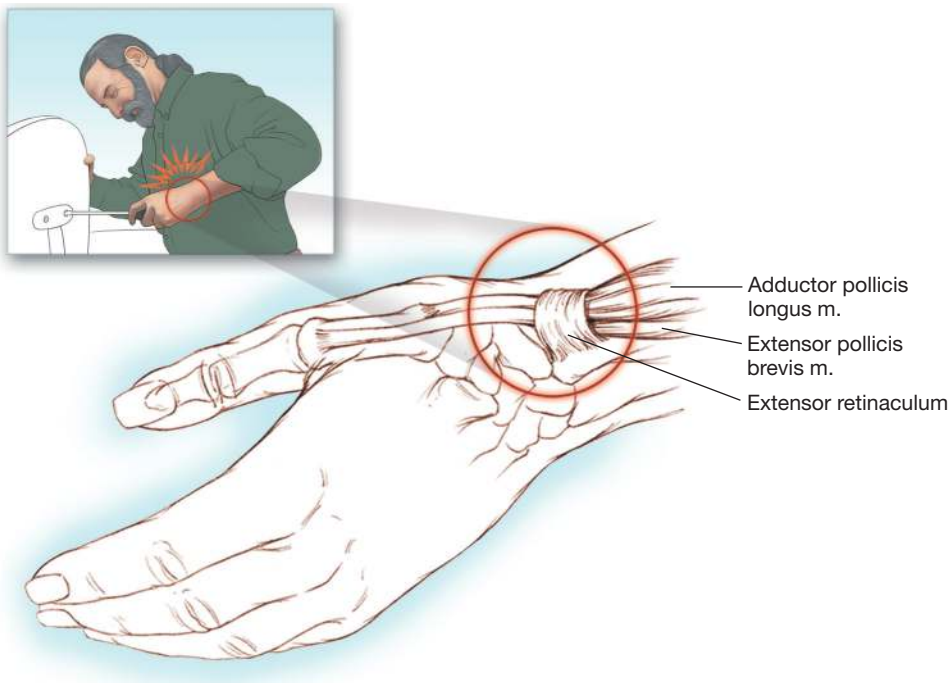

FIG 52.1 Repetitive microtrauma to the wrist can result in de Quervain's tenosynovitis.

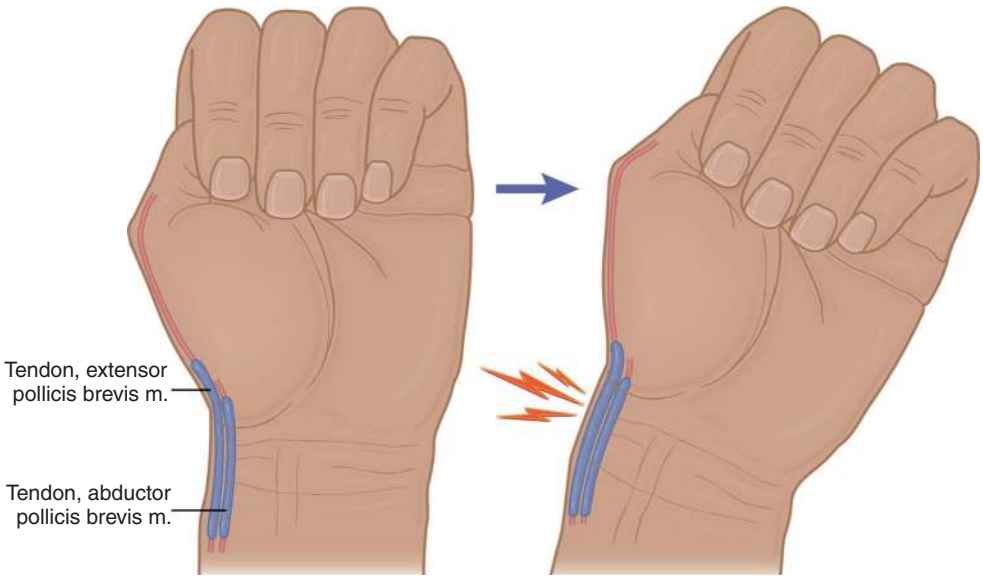

FIG 52.2 Example of de Quervain tenosynotis of the left wrist. Note the thickening of the tendon. (From Waldman S. *Atlas of pain management injection techniques*. 4th ed. St. Louis: Elsevier; 2017: Fig. 77.1.)

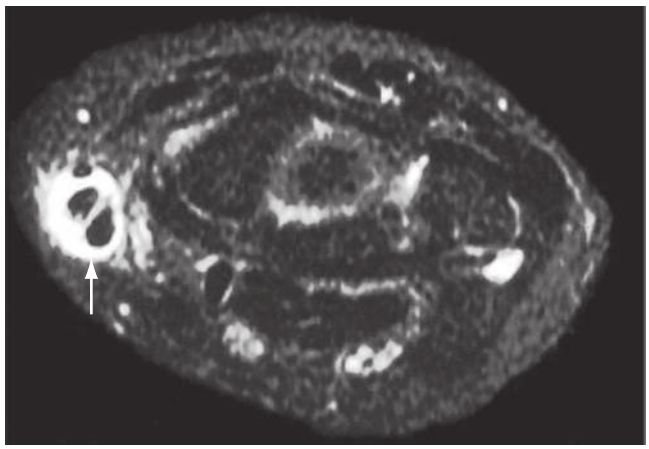

FIG 52.3 A positive Finkelstein test is indicative of de Quervain's tenosynovitis. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

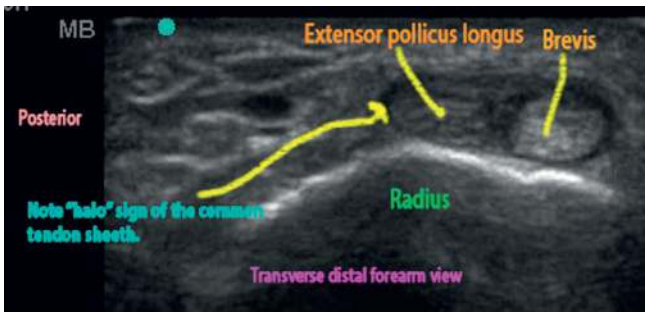

FIG 52.4 Axial short tau inversion recovery magnetic resonance image demonstrating de Quervain's tenosynovitis. Note the thickened first extensor compartment tendons, with prominent tendon sheath fluid (*arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3357.)

The Finkelstein test is performed by stabilizing the patient's forearm, having the patient fully flex his or her thumb into the palm, and then actively forcing the wrist toward the ulna. Sudden severe pain is highly suggestive of de Quervain's tenosynovitis.

TESTING

The diagnosis is generally made on clinical grounds, but magnetic resonance imaging (MRI) can confirm the presence of tenosynovitis (Fig. 52.4). Electromyography can distinguish de Quervain's tenosynovitis from neuropathic processes such as cervical radiculopathy and cheiralgia paresthetica. Plain radiographs are indicated in all patients who present with de Quervain's tenosynovitis, to rule out occult bony disease. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Plain radiographs may reveal osteopenia of the distal radius (Fig. 52.5). MRI and ultrasound imaging of the wrist are also indicated if joint instability is suspected and to clarify the clinical pathology responsible for the patient's symptoms (Fig. 52.5). Ultrasound evaluation will also help determine if the abductor pollicis longus and extensor pollicis tendons are separated by a septum, necessitating repositioning of the needle to determine accurate placement of corticosteroid and local anesthetic (Fig. 52.6). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

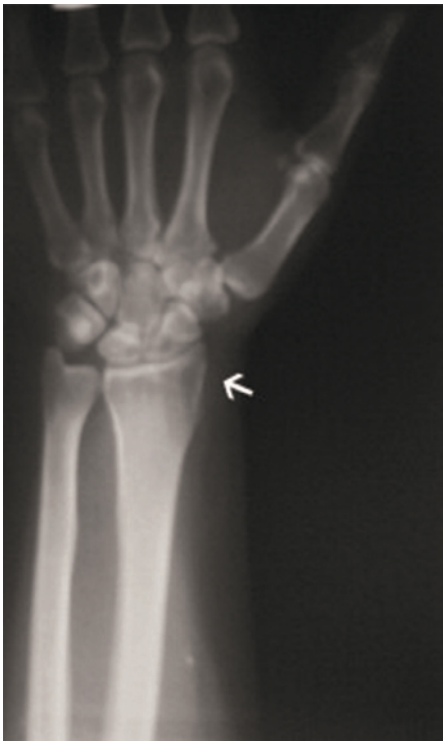

FIG 52.5 Localized osteopenia on the anteroposterior roentgenogram of the wrist in a patient with de Quervain tenosynovitis (*arrow*). Note mild soft tissue swelling over the radial aspect of the wrist. (From Altay MA, Erturk C, Isikan UE. De Quervain's disease treatment using partial resection of the extensor retinaculum: a short-term results survey. *Orthop Traumatol Surg Res*. 2011;97(5):489–493 [Fig. 1]. ISSN 1877-0568.)

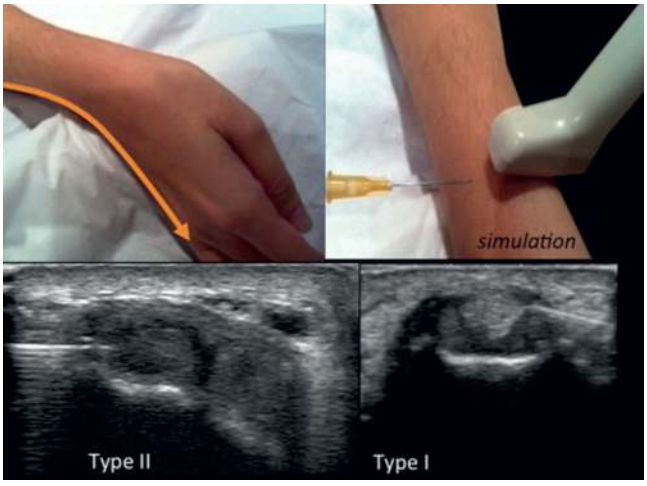

FIG 52.6 de Quervain's tenosynovitis. Transverse ultrasound image of the first dorsal compartment tendons (abductor pollicis longus and extensor pollicis brevis) showing tenosynovitis. Note the halo sign around tendons due to fluid surrounding the inflamed tendons.

DIFFERENTIAL DIAGNOSIS

Entrapment of the lateral antebrachial cutaneous nerve, arthritis of the first metacarpal joint, gout, cheiralgia paresthetica (caused by entrapment of the superficial branch of the radial nerve at the wrist), and occasionally C6–C7 radiculopathy can mimic de Quervain's tenosynovitis. All these painful conditions can also coexist with de Quervain's tenosynovitis (Box 52.1).

BOX 52.1 Differential Diagnosis of de Quervain Tenosynovitis

- Abnormalities of the radial styloid
- Fractures of the scaphoid
- Intersection syndrome
- C6 radiculopathy
- Keinboch disease
- Arthritis of the carpophalangeal joint of the thumb
- Cheiragia paresthetica (wristwatch palsy)
- Tumor
- Infection
- Ganglion cysts
- Entrapment neuropathies of the wrist
- Synostosis of the scaphoid and trapezium

TREATMENT

Initial treatment of the pain and functional disability associated with de Quervain's tenosynovitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. Nighttime splinting of the affected thumb may help avoid the trigger phenomenon that can occur on awakening in many patients suffering from this condition. For patients who do not respond to these treatment modalities, the following injection technique is a reasonable next step.

Injection for de Quervain's tenosynovitis is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side and the ulnar surface of the wrist and hand resting on a folded towel to relax the affected tendons. A total of 2-mL local anesthetic and 40-mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the affected tendons, the radial styloid is identified. Using strict aseptic technique, the clinician inserts a 1-inch, 25-gauge needle at a 45-degree angle toward the radial styloid through the skin and into the subcutaneous tissue overlying the affected tendon. If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. The injection of platelet-rich plasma and/or stem cells around the inflamed tendons may aid in the resolution of symptoms associated with de Quervain's tenosynovitis. Ultrasound-guided needle placement will aid in the accurate needle placement in patients suffering from de Quervain's tenosynovitis (Fig. 52.7).

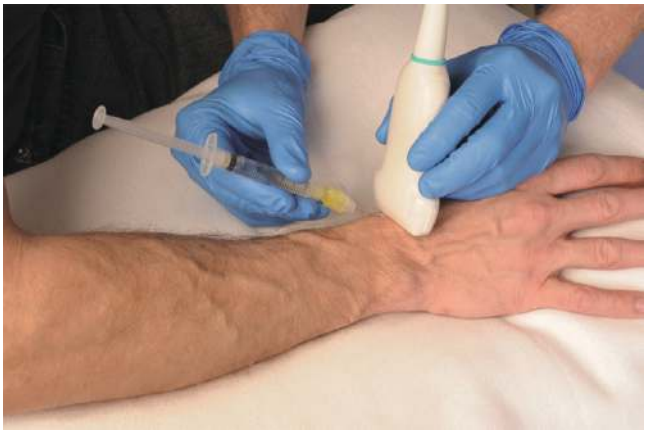

FIG 52.7 Ultrasound-guided injection of de Quervain tenosynovitis.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Splinting may provide symptomatic relief. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The radial artery and superficial branch of the radial nerve are susceptible to damage if the needle is placed too medially, so care must be taken to avoid these structures. The major complications associated with injection are related to trauma to the inflamed and previously damaged tendons. These tendons may rupture if they are injected directly, so the needle position should be confirmed to be outside the tendon before injection. Another complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed, as well as universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to de Quervain's tenosynovitis. A hand splint to immobilize the thumb may also help relieve the symptoms. Simple analgesics and NSAIDs can be used concurrently with the injection technique. Coexistent arthritis and gout may contribute to the pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Arthritis of the first metacarpal joint, gout, cheiralgia paresthetica, and cervical radiculopathy may mimic de Quervain's tenosynovitis and must be excluded.

SUGGESTED READINGS

O'Neill CJ. de Quervain tenosynovitis. In: Frontera WR, Silver JK, Rizzo TD, eds. *Essentials of physical medicine and rehabilitation*. 4th ed. Philadelphia: Elsevier; 2020:149–153.

Waldman SD. de Quervain's tenosynovitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:261–263.

Waldman SD. de Quervain tenosynovitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:417–424.

Waldman SD. Functional anatomy of the wrist. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:105–106.

Waldman SD. Injection technique for de Quervain tenosynovitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2017:278–281.

Waldman SD. Intra-articular injection of the wrist joint. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:250–253.

Waldman SD. Painful conditions of the wrist and hand. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2021:184.

Waldman SD. The Finkelstein test for de Quervain tenosynovitis. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2021:187–189.

53

Arthritis Pain at the Carpometacarpal Joints

ICD-10 CODE M18.9

THE CLINICAL SYNDROME

The carpometacarpal joints of the fingers are synovial plane joints that serve as the articulation between the carpals and the metacarpals and allow the bases of the metacarpal bones to articulate with one another. Movement of the joints is limited to a slight gliding motion, with the carpometacarpal joint of the little finger possessing the greatest range of motion. The primary function of these joints is to optimize the grip function of the hand. Most patients have a common joint space.

Pain and dysfunction from arthritis of the carpometacarpal joints are common complaints. These joints are susceptible to the development of arthritis from various conditions that share the ability to damage joint cartilage. Osteoarthritis is the most common form of arthritis that results in carpometacarpal joint pain. It occurs more often in female patients, and although the thumb is most frequently affected, arthritis may develop in the other carpometacarpal joints as well, especially after trauma. Rheumatoid arthritis, posttraumatic arthritis, and psoriatic arthritis are also common causes of carpometacarpal pain. Less frequent causes of arthritis-induced carpometacarpal pain include collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular diseases generally manifest as polyarthropathy rather than as monarthropathy limited to the carpometacarpal joint; however, carpometacarpal pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described here.

SIGNS AND SYMPTOMS

Most patients presenting with carpometacarpal pain secondary to osteoarthritis or posttraumatic arthritis complain of pain that is localized to the dorsum of the wrist. Activity associated with flexion, extension, and ulnar deviation of the carpometacarpal joints exacerbates the pain, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical examination. If the carpometacarpal joint of the thumb is affected, the patient will exhibit a positive Watson test. The test is performed by having the patient place the dorsum of the affected hand against a table with the fingers fully extended. The examiner then pushes the thumb toward the table. The test is positive if it reproduces the patient's pain (Fig. 53.1).

In addition to pain, patients suffering from arthritis of the carpometacarpal joint often experience a gradual reduction in functional ability because of decreasing pinch and grip strength that makes everyday tasks such as using a pencil or opening a jar quite difficult (Fig. 53.2). With continued disuse, muscle wasting may occur and adhesive capsulitis with subsequent ankylosis may develop.

TESTING

Plain radiographs are indicated in all patients who present with carpometacarpal pain (Fig. 53.3). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) and ultrasound imaging of the affected carpometacarpal joint is indicated if joint instability is thought

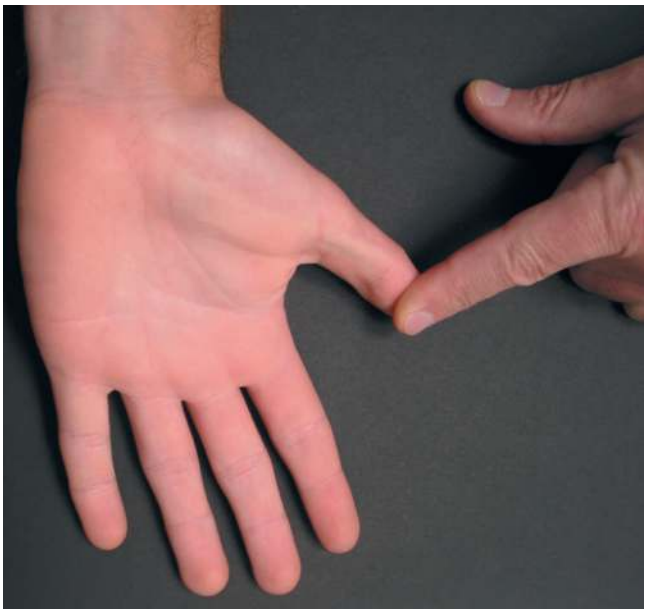

FIG 53.1 The Watson stress test for arthritis of the carpometacarpal joint of the thumb. (From Waldman SD. *Physical diagnosis of pain*. 3rd ed. Philadelphia: Elsevier; 2016: Fig. 106.2.)

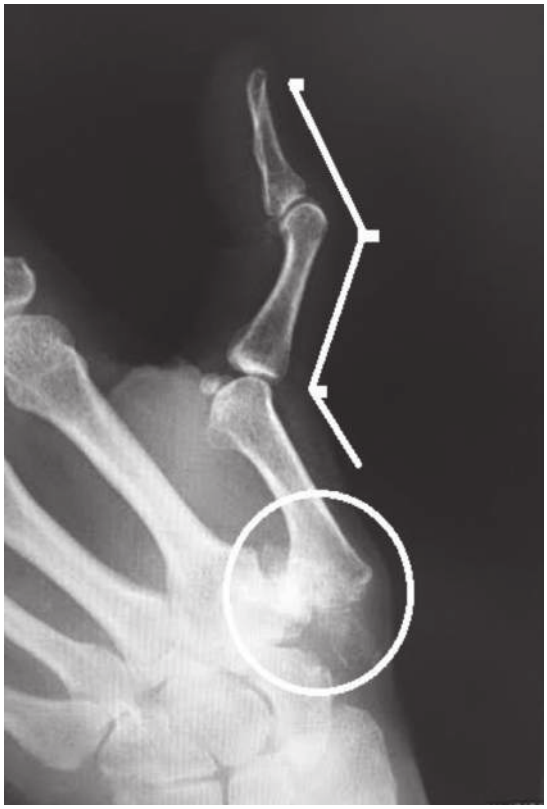

FIG 53.2 Radiographic example of severe osteoarthritis of the thumb carpometacarpal (CMC) joint. The zigzag deformity, common in advanced stages of thumb CMC joint osteoarthritis, can be visualized. (From Grenier ML, Mendonca R, Dalley P. The effectiveness of orthoses in the conservative management of thumb CMC joint osteoarthritis: an analysis of functional pinch strength. *J Hand Ther*. 2016;29(3):307–313.)

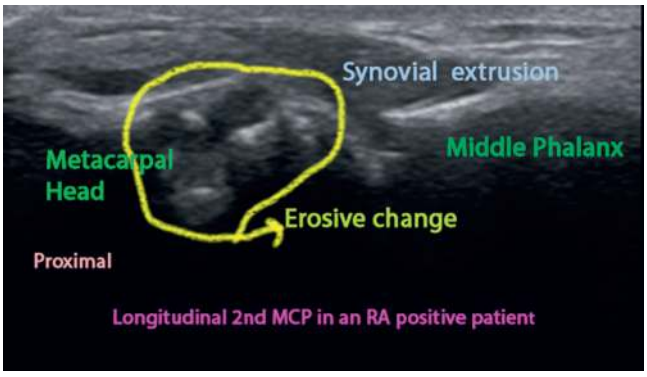

FIG 53.3 Longitudinal ultrasound image demonstrating erosive changes of the second carpometacarpal joint. Note the synovial extrusion caused by exuberant synovitis.

to be present as well as to clarify the cause of joint pain and functional disability. If infection is suspected, Gram stain and culture of the synovial fluid should be performed on an emergency basis and treatment with appropriate antibiotics should be started. If the patient has a history of trauma (Fig. 53.4), MRI or radionuclide bone scanning may be useful (Figs. 53.5 and 53.6), because fractures of the navicular bone are often missed on plain radiographs of the wrist.

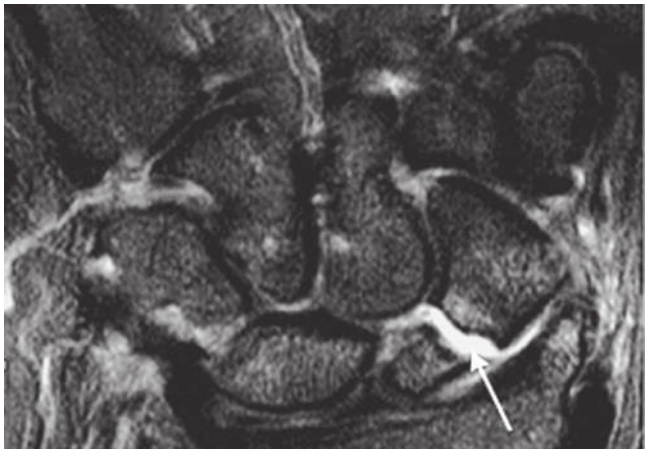

FIG 53.4 Coronal long repetition time/echo time (TR/TE) fast spin-echo magnetic resonance image with fat saturation shows nonunion of a proximal pole scaphoid fracture (*arrow*), outlined by fluid signal in the fracture. (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3344.)

DIFFERENTIAL DIAGNOSIS

Arthritis pain of the carpometacarpal joints is usually diagnosed on clinical grounds, and plain radiographs confirm the clinical findings (Fig. 53.7). Occasionally, arthritis pain of the carpometacarpal joints may be confused with de Quervain's tenosynovitis or other forms of tendinitis involving the wrist and fingers. These painful conditions, as well as gout, may coexist and make the diagnosis more difficult. If the patient has a history of trauma, occult fractures of the metacarpals should always be considered.

TREATMENT

Initial treatment of the pain and functional disability associated with osteoarthritis of the carpometacarpal joints includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Splinting the wrist in the neutral position may provide symptomatic relief and protect the joint from additional trauma. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

Intraarticular injection of the carpometacarpal joint is performed by placing the patient in the supine position with the arm fully adducted at the patient's side and the hand in a neutral position, with the palmar aspect resting on a folded towel. A total of 1.5-mL local anesthetic and 40-mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the affected carpometacarpal joint, the space between the carpal and metacarpal joints is identified. The joint can be more easily identified by gliding it back and forth. Using strict aseptic technique, the clinician inserts a 1-inch, 25-gauge needle through the

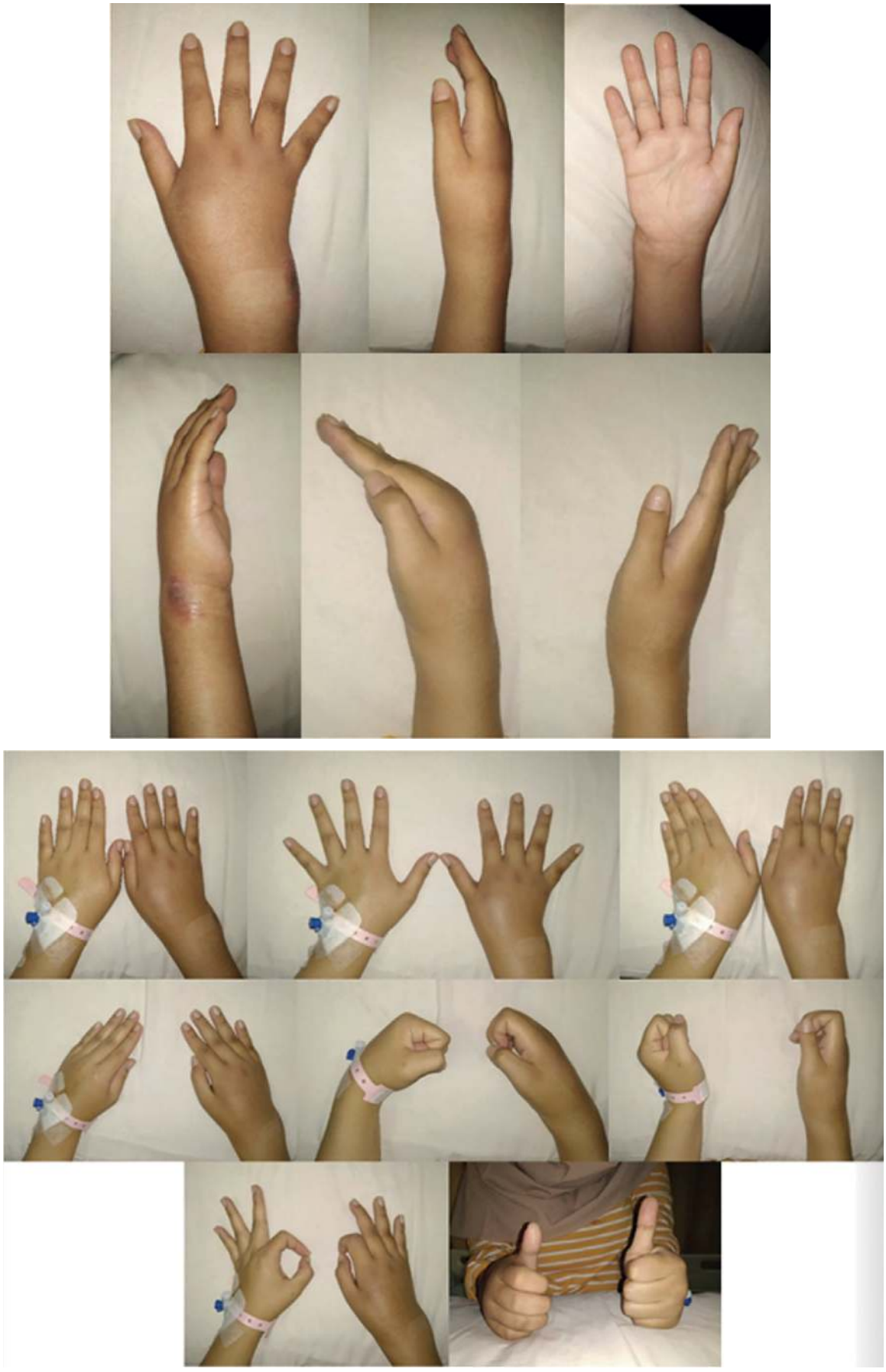

FIG 53.5 Clinical appearance of septic arthritis of the wrist. (From Latief W, Asril E. Tuberculosis of the wrist mimicking rheumatoid arthritis—a rare case. *Int J Surg Case Rep*. 2019;63:13–18.)

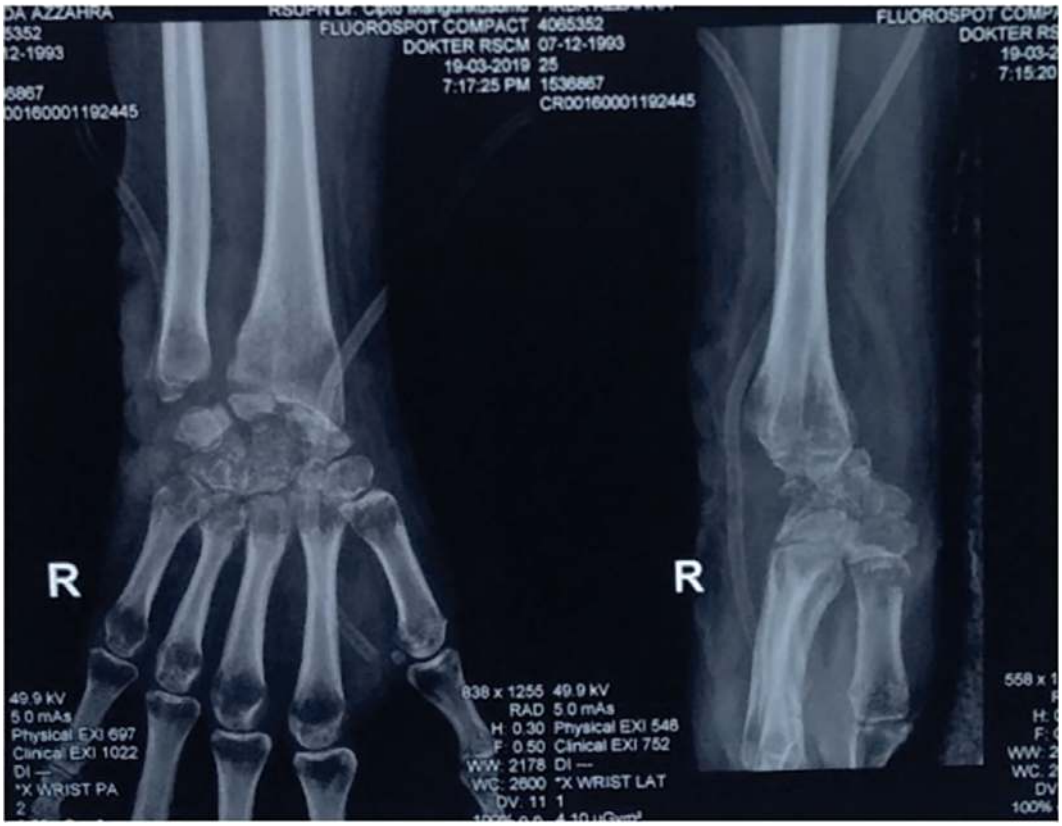

FIG 53.6 Radiograph of the right wrist demonstrating diffuse demineralization and destruction of joint architecture in a patient with proven septic arthritis of the wrist. (From Latief W, Asril E. Tuberculosis of the wrist mimicking rheumatoid arthritis—a rare case. *Int J Surg Case Rep*. 2019;63:13–18.)

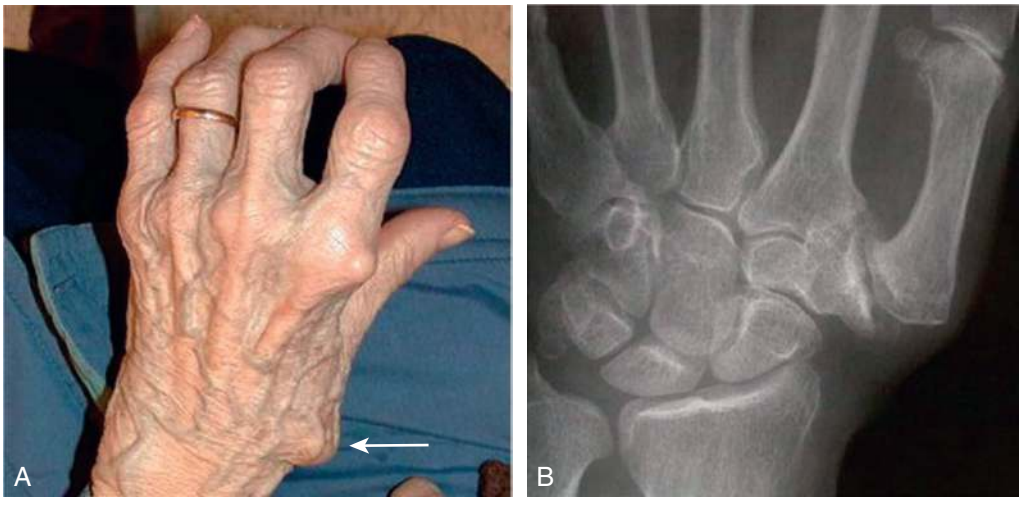

FIG 53.7 First carpometacarpal arthritis. **A,** Radial subluxation of the base of the first metacarpal giving the "shoulder sign" (*arrow*). **B,** Anteroposterior radiograph of the same hand. (From Young D, Papp S, Giachino A. Physical examination of the wrist. *Orthop Clin North Am*. 2007;38(2):149–165.)

skin, subcutaneous tissues, and joint capsule and into the center of the joint (Fig. 53.8). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected medially. After entering the joint space, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site.

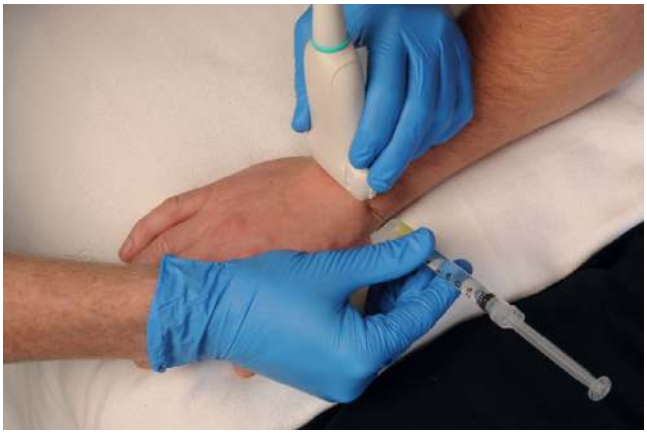

FIG 53.8 Proper needle placement for ultrasound-guided injection of the carpometacarpal joints.

Clinical experience has suggested that injection of platelet-rich plasma and/or stem cells may hasten resolution of the patient's joint symptomatology. Ultrasound needle guidance will aid in accurate needle placement.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient begins treatment for arthritis of the carpometacarpal joints. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

Joint protection is especially important in patients suffering from inflammatory arthritis of the carpometacarpal joints, because repetitive trauma can result in further damage to the joints, tendons, and connective tissues. The major complication associated with the intraarticular injection technique is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the carpometacarpal joints, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of arthritis pain of the carpometacarpal joints, and it is safe as long as careful attention is paid to the clinically relevant anatomy. Simple analgesics and NSAIDs can be used concurrently with the injection technique. Coexistent bursitis and tendinitis may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Bay COA, Willacy RA, Moses AR, et al. Nonspecific wrist pain in pediatric patients: a systematic review. *J Orthop*. 2020;22:308–315.

Brewer PE, Storey PA. The hand and wrist in rheumatoid and osteoarthritis. *Surgery (Oxford)*. 2016;34(3):144–151.

Dineen HA, Greenberg JA. Ulnar-sided wrist pain in the athlete. *ClSports Med*. 2020;39(2):373–400.

Laulan J, Marteau E, Bacle G. Wrist osteoarthritis. *Orthop Traumatol Surg Res*. 2015;101(Suppl_1):S1–S9.

Ul Islam S, Brown D, Cheung G. Management of osteoarthritis of the wrist and hand. *Orthop Trauma*. 2019;33(1):30–37.

Waldman SD. Clinical correlates: functional anatomy of the wrist. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2016:158–159.

Waldman SD. Abnormalities of the distal radioulnar joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:353–360.

Waldman SD. Arthritis and other disorders of the carpometacarpal joints. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:446–450.

Waldman SD. Intra-articular injection of the wrist. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:250–253.

Waldman SD. Intra-articular injection of the carpometacarpal joint. In: *Atlas of pain management injection techniques*. 4th ed. 2017:290–292.

Waldman SD. The Watson stress test for arthritis of the carpometacarpal joint of the thumb. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. 2017:173–174.

Ganglion Cysts of the Wrist

ICD-10 CODEM67.40

THE CLINICAL SYNDROME

The dorsum of the wrist is especially susceptible to the development of ganglion cysts in the area overlying the extensor tendons or the joint space, with a predilection for the joint space of the lunate or from the tendon sheath of the extensor carpi radialis (Figs. 54.1 and 54.2). These cysts are thought to form as the result of herniation of synovial-containing tissues from joint capsules or tendon sheaths. This tissue may then become irritated and begin producing increased amounts of synovial fluid, which can pool in cyst-like cavities overlying the tendons and joint space. A one-way valve phenomenon may cause these cyst-like cavities to expand, because the fluid cannot flow freely back into the synovial cavity. Ganglion cysts may also occur on the volar aspect of the wrist. Occurring three times more commonly in women than in men, ganglion cysts of the wrist represent 65%–70% of all soft tissue tumors of the hand and wrist (Fig. 54.3). Ganglion cysts occur in all age groups, with a peak incidence in the fourth to sixth decades.

SIGNS AND SYMPTOMS

Activity, especially extreme flexion and extension, makes the pain worse; rest and heat provide some relief (Fig. 54.4). The pain is constant and is characterized as aching. Occasionally, the ganglion will cause a trigger wrist. Often, the unsightly nature of the ganglion cyst, rather than the pain, causes the patient to seek medical attention. The ganglion is smooth to palpation and transilluminates with a penlight, in contradistinction to solid tumors, which do not transilluminate. Palpation of the ganglion may increase the pain (Fig. 54.5).

TESTING

Plain radiographs of the wrist are indicated in all patients who present with ganglion cysts, to rule out bony abnormalities, including tumors. Ultrasound imaging will help determine whether a soft tissue mass of the wrist is cystic or solid (Figs. 54.6 and 54.7). Based on the patient's clinical presentation, additional testing may be indicated, including complete blood count, sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) and ultrasound imaging of the wrist are indicated if the cause of the wrist mass is suspect (Figs. 54.8–54.10).

DIFFERENTIAL DIAGNOSIS

Although ganglion cysts are the most common soft tissue tumor of the wrist, many other pathologic processes can mimic this disorder (Box 54.1). Infection, tenosynovitis,

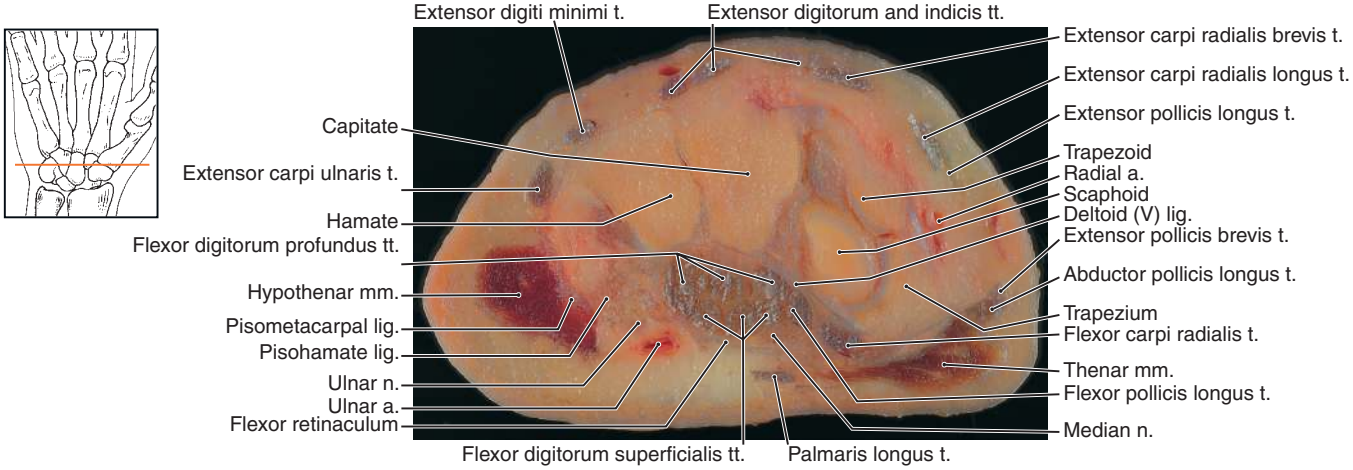

FIG 54.1 Ganglion cysts of the wrists are thought to form as the result of herniation of synovial-containing tissues from joint capsules or tendon sheaths. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas.* 2nd ed. Philadelphia: Saunders; 2002:178.)

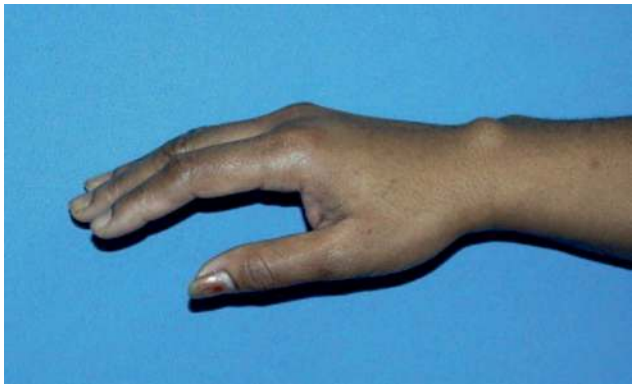

FIG 54.2 A typical dorsal ganglion cyst.

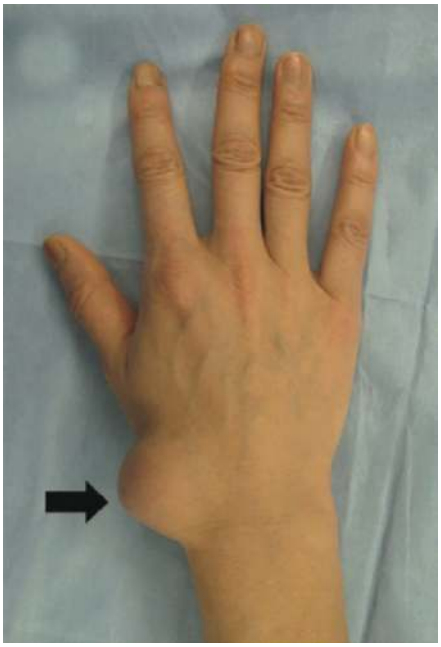

FIG 54.3 Large dorsal ganglion cyst of the right wrist. (From Yamamoto M, Kurimoto S, Okui N, Tatebe M, Shinohara T, Hirata H. Sonography-assisted arthroscopic resection of volar wrist ganglia: a new technique. *Arthrosc Tech*. 2012;1(1):e31–e35.)

lipomas, and carpal bosses are among the more common diseases that may mimic ganglion cysts of the wrist. Less commonly, malignant tumors including sarcomas and metastatic disease may confuse the diagnosis (Fig. 54.11).

TREATMENT

Initial treatment of the pain and functional disability associated with osteoarthritis of the wrist includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may also be beneficial. Splinting the wrist in the neutral position may provide symptomatic relief and

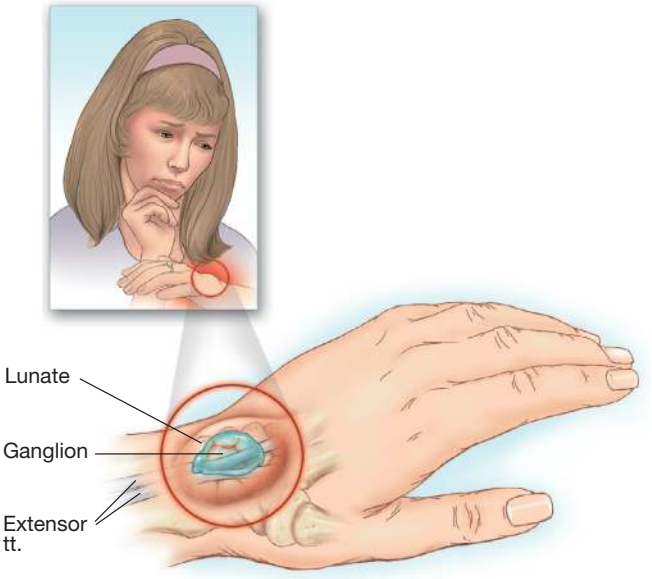

FIG 54.4 Ganglion cysts usually appear on the dorsum of the wrist, overlying the extensor tendon or joint space. Patients often seek medical attention out of a fear of cancer.

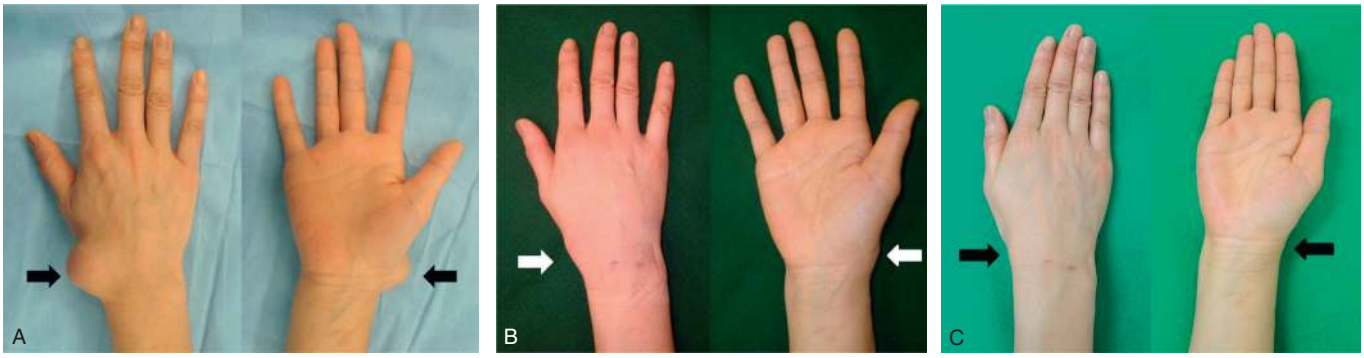

FIG 54.5 Ganglion cysts of the wrist. **(A)** Preoperative gross view of hand. This patient underwent aspiration at a previous hospital more than 20 times, but the ganglion cyst (*arrows*) did not heal, and she complained of wrist pain and an abnormal appearance. **(B)** Postoperative gross view of hand. After 1 week, the wound had healed and the patient was allowed to perform light work. The ganglion became smaller, but slight swelling at the lesion (*arrows*) remained because of residual cysts. **(C)** One month after surgery, there was no swelling (*arrows*) and no pain. The patient could fully return to work and sporting activities. (From Yamamoto M, Kurimoto S, Okui N, Tatebe M, Shinohara T, Hirata H. Sonography-assisted arthroscopic resection of volar wrist ganglia: a new technique. *Arthrosc Tech*. 2012;1(1):e31–e35.)

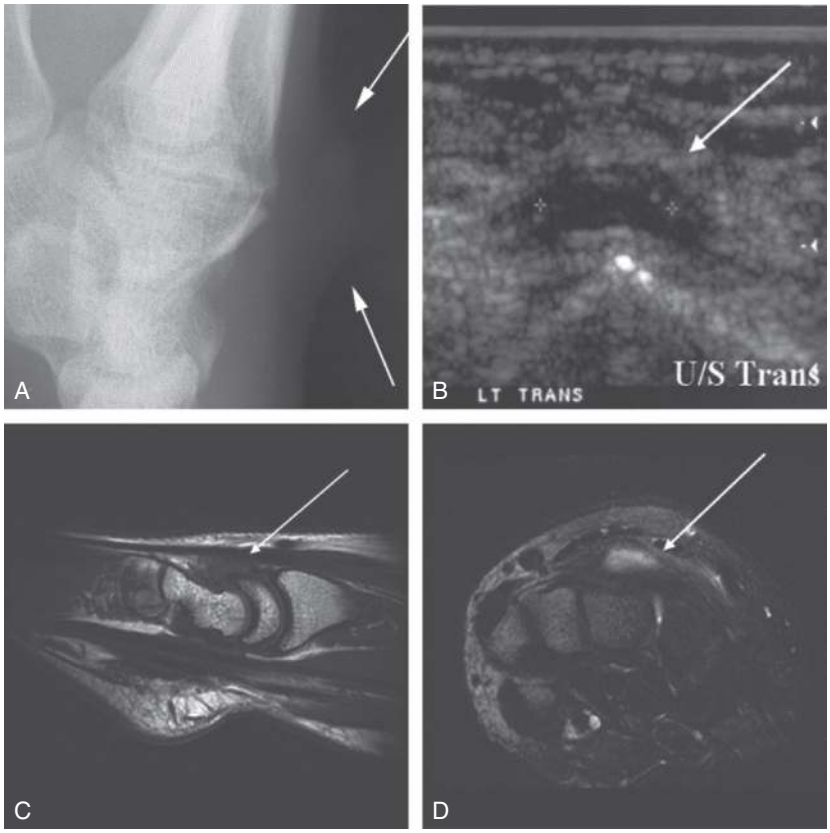

FIG 54.6 Dorsal wrist ganglion (*arrows*). Lateral radiograph **(A)** demonstrates a soft tissue mass on the dorsum of the wrist. Ultrasound **(B)** in a second patient shows the typical anechoic cystic appearance of a ganglion. Sagittal T1-weighted **(C)** and axial T2-weighted fat-saturation **(D)** magnetic resonance images through the distal carpal row in a third patient show a circumscribed cystic mass. (From Nguyen V, Choi J, Davis KW. Imaging of wrist masses. *Curr Probl Diagn Radiol*. 2004;33(4):147–160.)

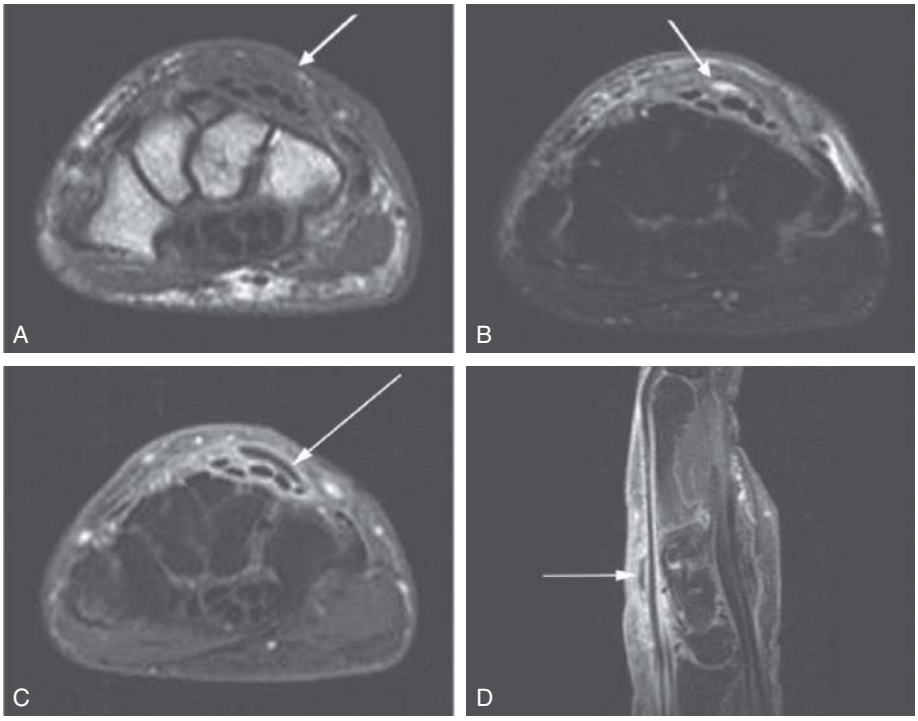

FIG 54.7 Abscess. Axial T1-weighted **(A)** and axial T2-weighted fat-saturation **(B)** magnetic resonance imaging (MRI) scans demonstrate a small focus of fluid (*arrows*) dorsal to the extensor tendons. Postgadolinium axial **(C)** and sagittal T1-weighted **(D)** fat-saturation MRI scans show rim enhancement. (From Nguyen V, Choi J, Davis KW. Imaging of wrist masses. *Curr Probl Diagn Radiol*. 2004;33(4):147–160.)

protect the joint from additional trauma. For patients who do not respond to these treatment modalities, injection of the ganglion cyst with local anesthetic and steroid is a reasonable next step. If symptoms persist, surgical excision of the ganglion is recommended.

To inject a ganglion cyst of the wrist, the patient is placed in a supine position with the arm fully adducted at the patient's side and the elbow slightly flexed with the palm of the hand resting on a folded towel. A total of 1.5-mL of local anesthetic and 40-mg of methylprednisolone is drawn up in a 5-mL sterile syringe.

After sterile preparation of skin overlying the ganglion, a 1-inch, 22-gauge needle is inserted in the center of the ganglion, and the contents of the cyst are aspirated (Fig. 54.12). If bone is encountered, the needle is withdrawn back into the ganglion cyst and aspiration is performed. After the ganglion cyst is aspirated, the contents of the syringe are gently injected. Little resistance to injection should be felt. The needle is then removed, and a sterile pressure dressing and ice pack are placed at the injection site. If the ganglion reappears, surgical treatment ultimately may be required.

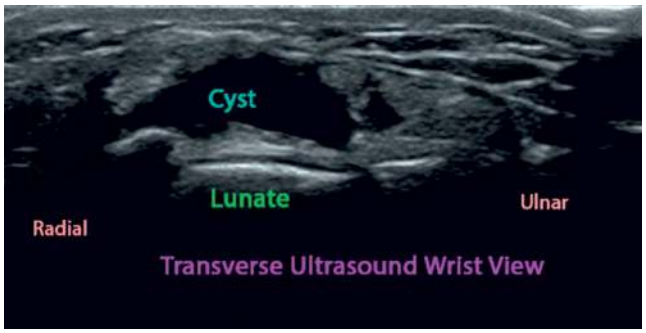

FIG 54.8 Dorsal wrist ganglion. Transverse ultrasound image demonstrating classic ganglion cyst.

COMPLICATIONS AND PITFALLS

The injection technique described here is safe if careful attention is paid to the clinically relevant anatomy; the ulnar nerve is especially susceptible to damage at the wrist. Care must be taken to avoid injecting directly into tendons that may already be inflamed from irritation caused by rubbing of the ganglion against the tendon. Approximately 25% of patients complain of a transient increase in pain after this injection technique, and patients should be warned of this possibility.

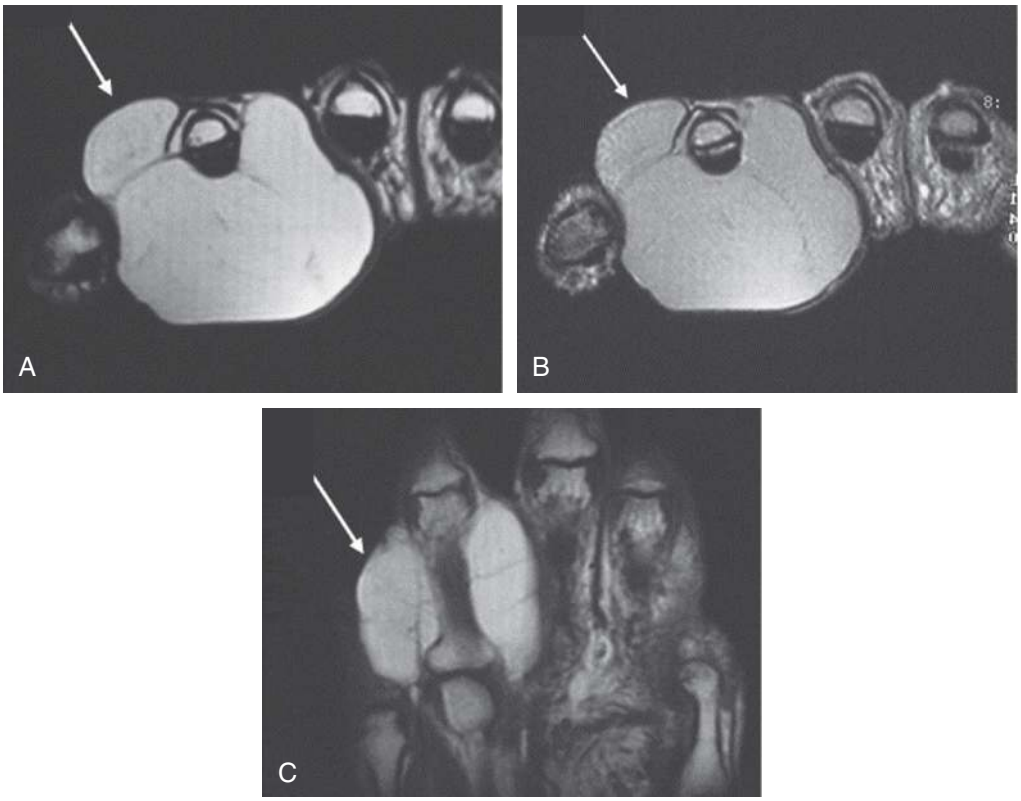

FIG 54.9 Lipoma (*arrows*). Axial T1-weighted **(A),** axial T2-weighted **(B),** and coronal T1-weighted **(C)** magnetic resonance images demonstrate a large mass surrounding the index finger. The lesion has identical signal to fat on all sequences. (From Nguyen V, Choi J, Davis KW. Imaging of wrist masses. *Curr Probl Diagn Radiol*. 2004;33(4):147–160.)

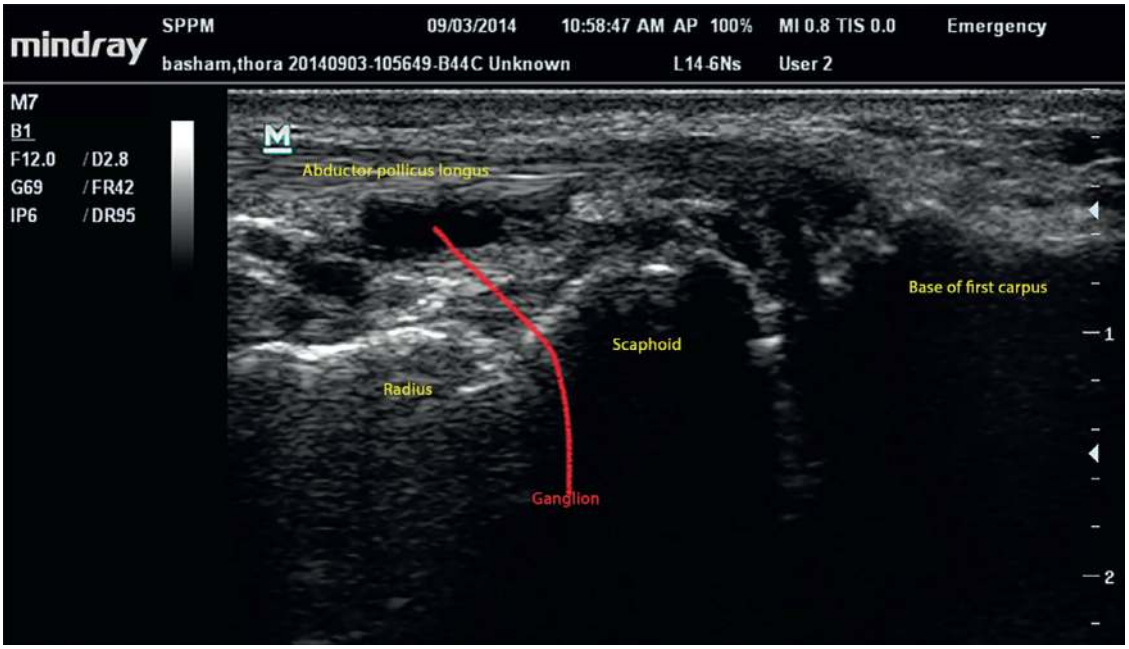

FIG 54.10 Longitudinal ultrasound image demonstrating a ganglion cyst of the wrist lying beneath the abductor pollicis longus.

BOX 54.1 Diseases That May Mimic Ganglion Cyst of the Wrist

- Infection
- Lipoma
- Tenosynovitis
- Carpal boss
- Neuroma
- Hypertrophied extensor digitorum brevis manus muscle belly
- Instability of the scaphoid
- Instability of the lunate
- Scaphotrapezial arthritis
- Vascular aneurysm
- Sarcoma

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to arthritis of the wrist joint. Simple analgesics and NSAIDs can be used concurrently with the injection technique. Coexistent bursitis and tendinitis may contribute to wrist pain and necessitate additional treatment with more localized injection of local anesthetic and methylprednisolone.

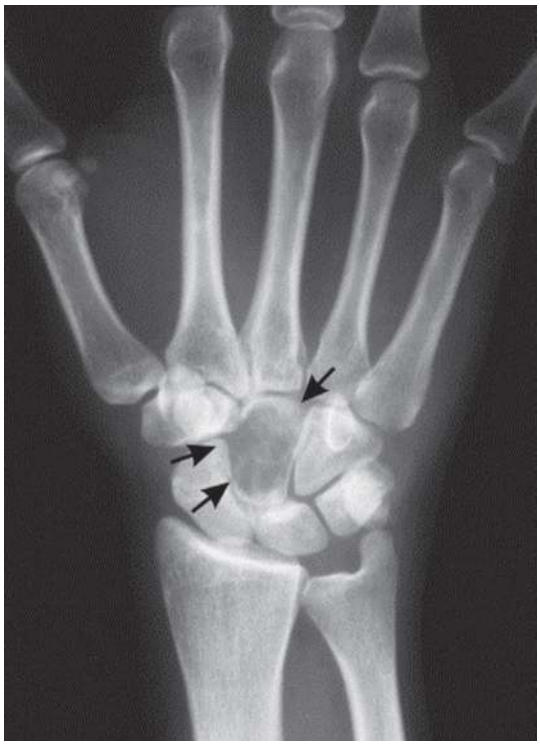

FIG 54.11 Posteroanterior radiograph of the right hand demonstrating a lytic lesion of the capitate (*arrows*) with cortical destruction secondary to metastatic malignant melanoma. (From Tomas X, Conill C, Combalia A, et al. Malignant melanoma with metastasis into the capitate. *Eur J Radiol*. 2005;56(3):362–364.)

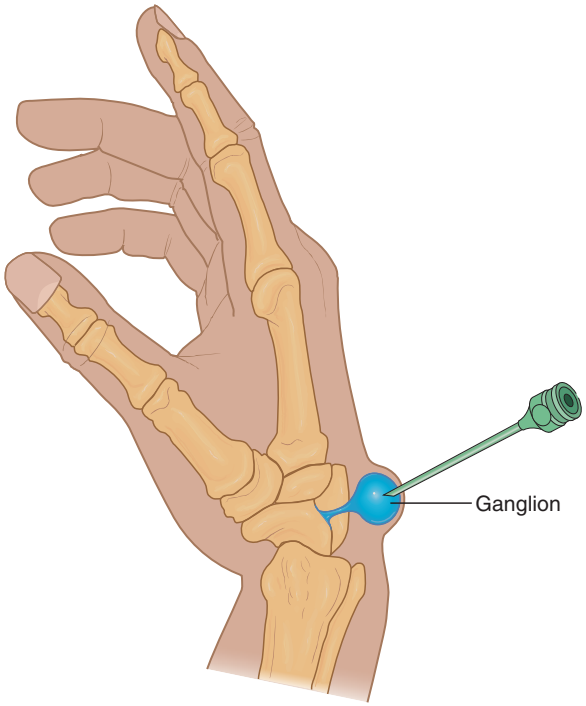

FIG 54.12 Injection technique for the treatment of ganglion cysts of the wrist. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:273.)

SUGGESTED READINGS

Al-Qattan MM, Elshamma NA, Alqabbani A. Trigger wrist and carpal tunnel syndrome caused by a flexor tendon-related ganglion in a teenager: a case report. *Int J Surg Case Rep*. 2017;30:86–88.

Balazs GC, Dworak TC, Tropf J, et al. Incidence and risk factors for volar wrist ganglia in the U.S. military and civilian populations. *J Hand Surg Am*. 2016;41(11):1064–1070.

Head L, Gencarelli JR, Allen M, et al. Wrist ganglion treatment: systematic review and meta-analysis. *J Hand Surg Am*. 2015; 40(3):546–553.e8.

Waldman SD. Ganglion cysts of the wrist. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:432–438.

Waldman SD. Injection technique for ganglion cysts of the wrist and hand. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:323–329.

Waldman SD. Injection technique for ganglion cysts of the wrist and hand. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:323–329.

Waldman SD, Campbell RSD. Ganglion cyst of the wrist. In: *Imaging of pain*. Philadelphia: Saunders; 2011:227–229.

55

Trigger Thumb

ICD-10 CODE M65.30

THE CLINICAL SYNDROME

Trigger thumb is caused by inflammation and swelling of the tendon of the flexor pollicis longus tendon as a result of compression by the head of the first metacarpal bone. Sesamoid bones in this region may also compress and cause trauma to the tendon. Trauma is usually caused by repetitive motion or pressure on the tendon as it passes over these bony prominences. If the inflammation and swelling become chronic, the tendon sheath may thicken, resulting in constriction (Fig. 55.1). Frequently, nodules develop on the tendon, and they can often be palpated when the patient flexes and extends the thumb. Such nodules may catch in the tendon sheath and produce a triggering phenomenon that causes the thumb to catch or lock. Pathologic changes of the pulley mechanism also contribute to the triggering phenomenon (Fig. 55.2). Trigger thumb occurs in patients engaged in repetitive activities, such as handshaking by politicians, or activities that require repetitive pinching movements of the thumb, such as playing video games, texting, or frequent card playing (Fig. 55.3). It occurs more commonly in females and in those patients suffering from diabetes.

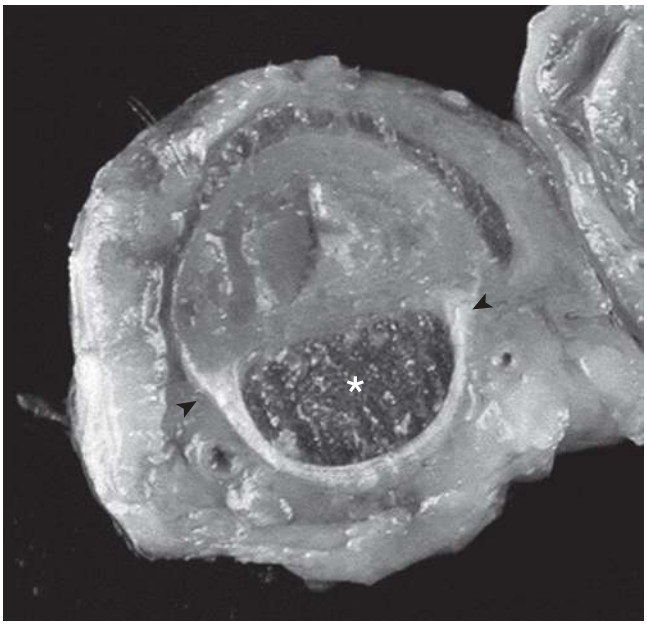

FIG 55.1 Axial specimen photograph demonstrates the flexor tendon (*asterisk*) as it resides in a fibroosseous canal, anchored to the bones of the fingers by thickened fibrous sheaths called the annular pulleys (*arrowheads*). (From Ragheb D, Stanley A, Gentili A, et al. MR imaging of the finger tendons: normal anatomy and commonly encountered pathology. *Eur J Radiol*. 2005;56(3):296–306.)

SIGNS AND SYMPTOMS

The pain of trigger thumb is localized to the palmar aspect of the base of the thumb (unlike the pain of de Quervain's tenosynovitis, which is most pronounced more proximally, over the radial styloid). The pain of trigger thumb is constant and is made worse with active pinching of the thumb. Patients note the inability to hold a coffee cup or a pen. Sleep disturbance is common, and patients often awaken to find that the thumb has become locked in a flexed position.

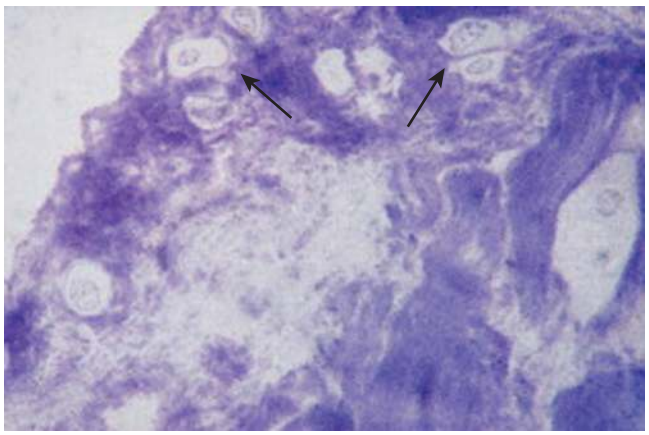

FIG 55.2 Specimen from a pathologic pulley showing chondroid metaplasia (*arrows*) with cells that look like cartilaginous cells organized in nests. (Semithin section, toluidine blue stain; original magnification ×250.) (From Sbernardori MC, Bandiera P. Histopathology of the A1 pulley in adult trigger fingers. *J Hand Surg Eur*. 2007;32(5):556–559.)

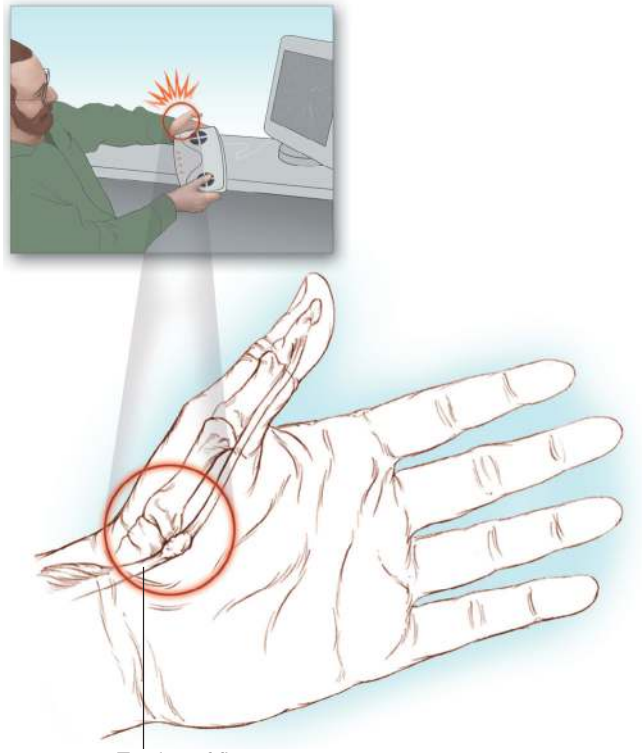

FIG 55.3 Trigger thumb is caused by microtrauma from repetitive pinching movements of the thumb.

On physical examination, tenderness and swelling are noted over the tendon, with maximal point tenderness over the base of the thumb. Many patients with trigger thumb experience a creaking sensation with flexion and extension of the thumb. A range of motion of the thumb may be decreased because of pain, and a triggering phenomenon may be present. As mentioned earlier, patients with trigger thumb often have nodules on the flexor pollicis longus tendon.

TESTING

Plain radiographs are indicated in all patients who present with trigger thumb, to rule out occult bony disease (Fig. 55.4). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the hand are indicated if first metacarpal joint instability is suspected or if the diagnosis of trigger thumb is in question. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

The diagnosis of trigger thumb is usually made on clinical grounds. Arthritis or gout of the first metacarpal joint may accompany trigger thumb and exacerbate the patient's pain. The nidus of pain from trigger thumb is the flexor pollicis longus tendon at the level of the base of the first metacarpal; occasionally, this may be confused with de Quervain's tenosynovitis.

TREATMENT

Initial treatment of the pain and functional disability associated with trigger thumb includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. A quilter's glove to protect the thumb may also help relieve the patient's symptoms. If these treatments fail, the following injection technique is a reasonable next step.

Injection of trigger thumb is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side and the dorsal surface of the hand resting on a folded towel. A total of 2-mL local anesthetic and 40-mg methylprednisolone is drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the affected tendon, the metacarpophalangeal joint of the thumb is identified. Using strict aseptic technique, at a point just proximal to the joint, the clinician inserts a 1-inch, 25-gauge needle at a 45-degree angle parallel to the affected tendon through the skin and into the subcutaneous tissue overlying the tendon. If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. The tendon sheath should distend as the injection proceeds. Little resistance to injection should be felt; if resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can be accomplished without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound guidance for needle placement may improve the accuracy of needle placement. Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Splinting of the thumb may also be of benefit. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Surgical release of the affected tendon(s) should be reserved for patients who do not respond to conservative therapy and injection of local anesthetic and corticosteroids.

COMPLICATIONS AND PITFALLS

Failure to treat trigger thumb early and adequately in its course can result in permanent pain and functional disability caused by continued trauma to the tendon and tendon sheath. The major complications associated with injection are related to trauma to the inflamed and previously damaged tendon. The tendon may rupture if it is injected directly, so a needle position outside the tendon should be confirmed before proceeding with the injection. Furthermore, the radial artery and superficial branch of the radial nerve are susceptible to damage if the needle is placed too far medially. Another complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

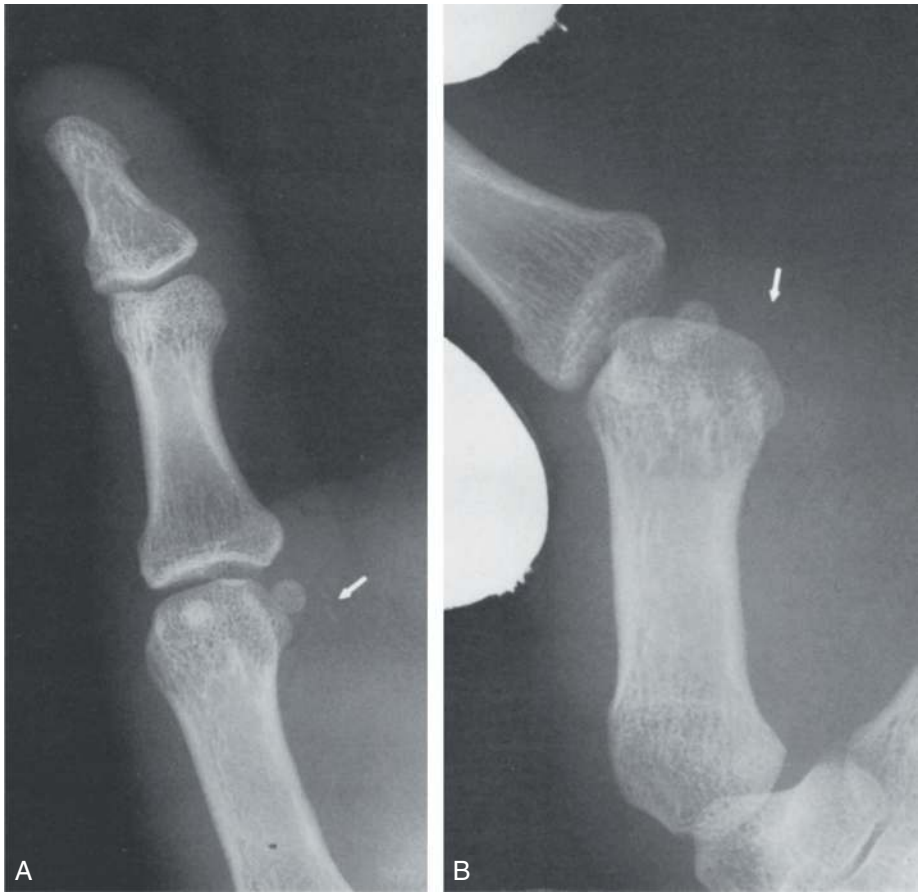

FIG 55.4 Gamekeeper's thumb. **A,** The initial radiograph outlines small osseous fragments adjacent to the first metacarpophalangeal joint (*arrow*). **B,** A radiograph obtained during radial stress reveals subluxation of the phalanx on the metacarpal bone. The fracture fragments are shown (*arrow*). (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:2583.)

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to trigger thumb. Coexistent arthritis and gout may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique is safe if careful attention is paid to the clinically relevant anatomy, including the radial artery and the superficial branch of the radial nerve. de Quervain's tenosynovitis may be confused with trigger thumb, but it can be distinguished by the location of the pain and the motions that cause the triggering phenomenon.

SUGGESTED READINGS

Ayhan E, Cevik K. Triggering thumb is not always a trigger thumb. *J Hand Surg Glob Online*. 2022. https://doi.org/10.1016/j.jhsg.2022.04.004.

Castellanos J, Muñoz-Mahamud E, Domínguez E, et al. Long-term effectiveness of corticosteroid injections for trigger finger and thumb. *J Hand Surg Am*. 2015;40(1):121–126.

Dbeis A, Ngo J, Chiang E, Ishak A. Trigger thumb preceding flexor pollicis longus tendon rupture after distal radius volar plate fixation: a case report. *Int J Surg Case Rep*. 2022;94:107050. https://doi.org/10.1016/j.ijscr.2022.107050.

Giugale JM, Fowler JR. Trigger finger: adult and pediatric treatment strategies. *Orthop Clin North Am*. 2015;46(4):561–569.

Hutchinson DT, Ran AAe, Montanez A. The natural history of pediatric trigger thumb in the United States. *J Hand Surg*. 2021;46(5):424.e1–424.e7. https://doi.org/10.1016/j.jhsa.2020.10.016.

Schubert MF, Shah VS, Craig CL, et al. Varied anatomy of the thumb pulley system: implications for successful trigger thumb release. *J Hand Surg Am*. 2012;37(11):2278–2285.

Waldman SD. Digitorum superficialis and profundus injection for tendinitis and trigger finger. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:323–329.

Waldman SD. Trigger finger. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:451–457.

56

Trigger Finger

ICD-10 CODE M65.30

THE CLINICAL SYNDROME

Trigger finger is caused by inflammation and swelling of the tendon of the flexor digitorum superficialis resulting from compression by the head of the metacarpal bone. Sesamoid bones in this region may also compress and cause trauma to the tendon. Trauma is usually the result of repetitive motion or pressure on the tendon as it passes over these bony prominences. If the inflammation and swelling become chronic, the tendon sheath may thicken, resulting in constriction. Frequently, nodules develop on the tendon, and they can often be palpated when the patient flexes and extends the fingers. Such nodules may catch in the tendon sheath as they pass under a restraining tendon pulley, thus producing a triggering phenomenon that causes the finger to catch or lock (Fig. 56.1). Trigger finger occurs more commonly in females and in patients with diabetes. Patients engaged in repetitive activities such as hammering, gripping a steering wheel, or holding a horse's reins too tightly also have a higher incidence of trigger finger (Fig. 56.2).

SIGNS AND SYMPTOMS

The pain of trigger finger is localized to the distal palm, and tender nodules can often be palpated. The pain is constant and is made worse with active gripping motions of the hand. Patients note significant stiffness when flexing the fingers. Sleep disturbance is common, and patients often awaken to find that the finger has become locked in a flexed position.

On physical examination, tenderness and swelling are noted over the tendon, with maximal point tenderness over the head of the metacarpal. Many patients with trigger finger experience a creaking sensation with flexion and extension of the fingers. The range of motion of the fingers may be decreased because of pain, and a triggering phenomenon may be noted. A catching tendon sign may also be elicited by having the patient clench the affected hand for 30 seconds and then relax but not open the hand. The examiner then passively extends the affected finger, and if he or she appreciates a locking, popping, or catching of the tendon as the finger is straightened, the sign is positive (Fig. 56.3).

TESTING

Plain radiographs are indicated in all patients who present with trigger finger, to rule out occult bony disease (Fig. 56.4). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance, computerized tomography, and ultrasound imaging of the hand are indicated if joint instability or some other boney abnormality is suspected (Figs. 56.5–56.9). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

The diagnosis of trigger finger is usually made on clinical grounds. Arthritis or gout of the metacarpal or interphalangeal joints may accompany trigger finger and exacerbate the patient's pain. Occult fractures occasionally confuse the clinical presentation.

TREATMENT

Initial treatment of the pain and functional disability associated with trigger finger includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. A nighttime splint to protect the fingers may also help relieve the symptoms. If these treatments fail, the following injection technique is a reasonable next step.

Injection of trigger finger is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side and the dorsal surface of the hand resting on a folded towel. A total of 2-mL local anesthetic and 40-mg methylprednisolone are drawn up in a 5-mL sterile syringe. After sterile preparation of the skin overlying the affected tendon, the head of the metacarpal beneath the tendon is identified. Using strict aseptic technique, at a point just proximal to the joint, a 1-inch, 25-gauge needle is injected at a 45-degree angle parallel to the affected tendon through the skin and into the subcutaneous tissue overlying the tendon. If bone is encountered, the needle is withdrawn into the subcutaneous tissue. The contents of the syringe are then gently injected. The tendon sheath should distend as the injection proceeds. Little resistance to injection should be felt; if resistance is encountered, the needle is probably in the tendon and should be withdrawn until the injection can be accomplished without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site (Fig. 56.10).

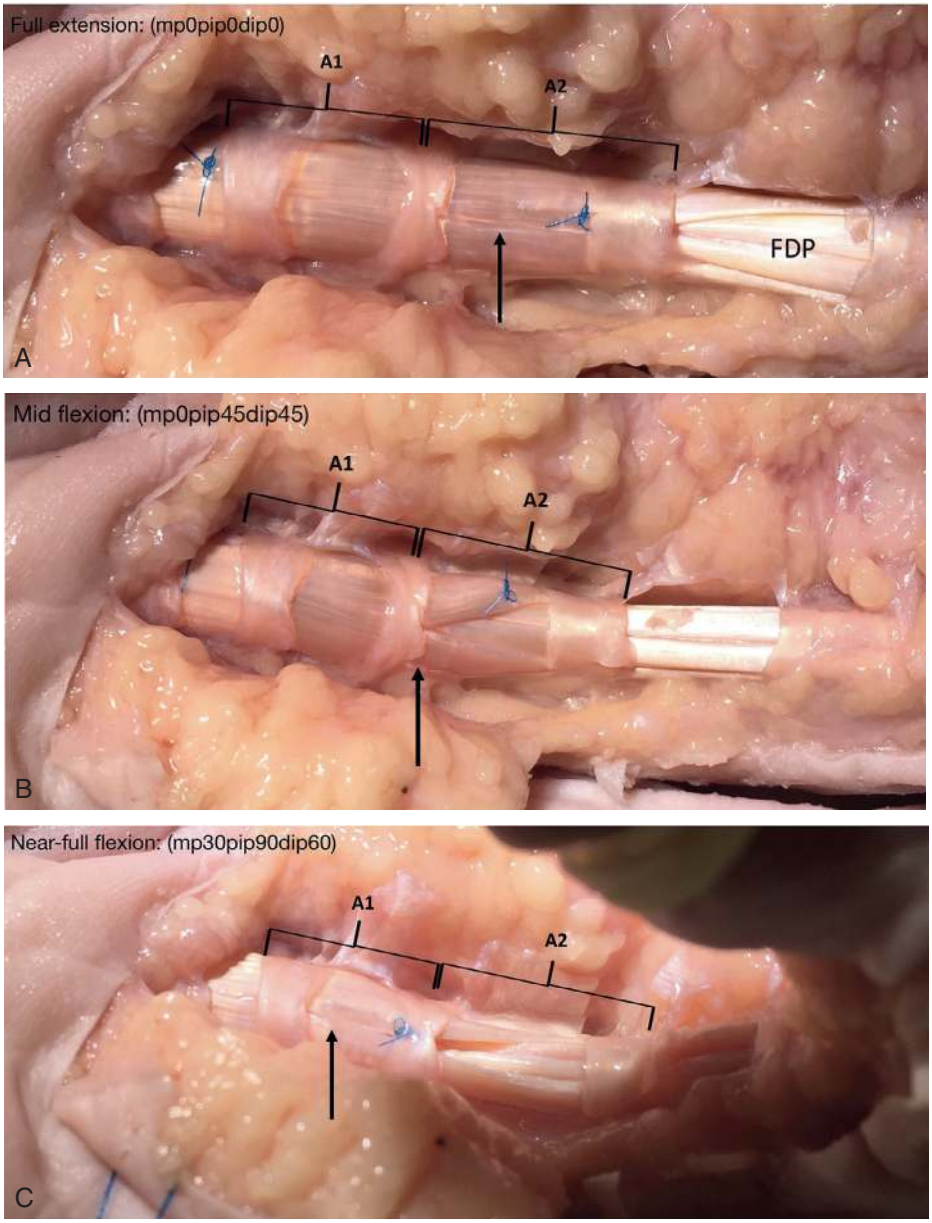

FIG 56.1 Anatomic dissection of the flexor digitorum superficialis (FDS). **A,** Windows have been removed from the distal half of A1 and the proximal A2 preserving a narrow band at the A1/A2 junction. In this posture, marker sutures on FDS overlie locations 1 and 5. An *arrow* indicates location 4. **B,** Tensioning of the FDS has separated the bifurcation with a tendency for the FDS slips to bunch and to rotate around the FDP to a more lateral position. The FDP under tension increases the separation of the FDS slips. The whole tendon mass is thickened in the region of the FDS bifurcation. **C,** The thickened FDS bifurcation now lies within the A1 pulley. Further metacarpophalangeal joint flexion would deliver the thickened tendon mass proximal to A1. *Arrow* indicates the point on the tendon, which commenced at location 4. (From Chuang XL, Ooi CC, Chin ST, et al. What triggers in trigger finger? The flexor tendons at the flexor digitorum superficialis bifurcation. *J Plast Reconstr Aesth Surg.* 2017;70(10):1411–1419.)

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

Surgical treatment should be considered for patients who fail to respond to the aforementioned treatment modalities.

COMPLICATIONS AND PITFALLS

Failure to treat trigger finger early and adequately in its course can result in permanent pain and functional disability because of continued trauma to the tendon and tendon sheath. The major complications associated with injection are related to trauma to the inflamed and previously damaged

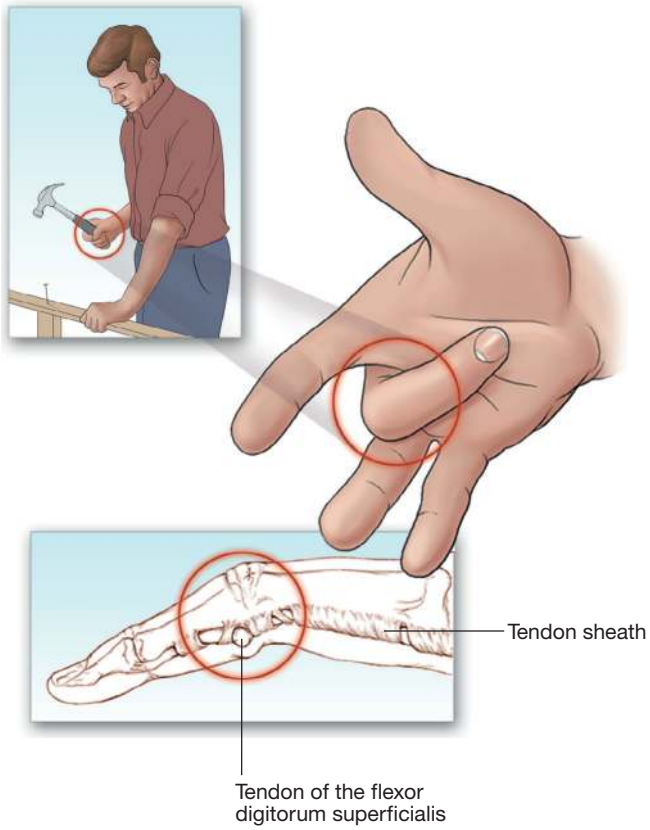

FIG 56.2 Trigger finger is caused by repetitive microtrauma from repeated clenching of the hand.

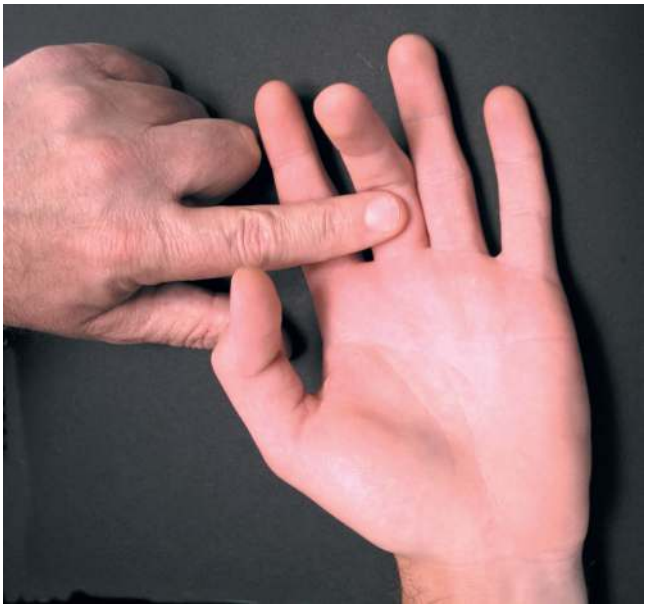

FIG 56.3 The catching tendon sign for trigger finger. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:195.)

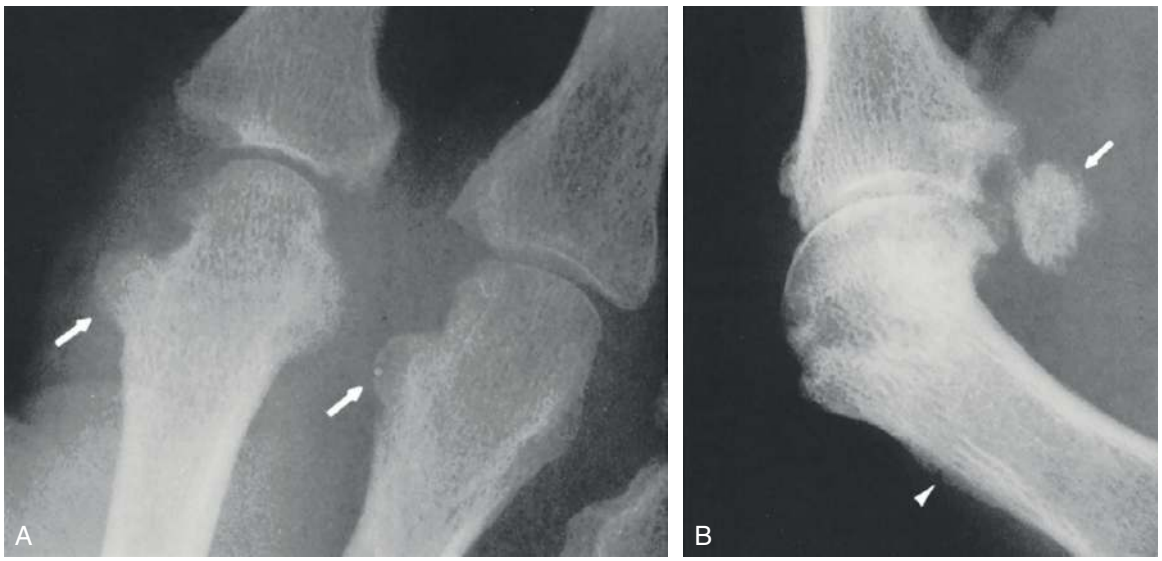

FIG 56.4 Radiographic abnormalities of the metacarpophalangeal joints. **A,** Startling osseous excrescences (*arrows*) around the metacarpal heads are associated with soft tissue swelling, joint space narrowing, and bony erosion and proliferation in the phalanges. **B,** At the first metacarpophalangeal joint, irregular bone formation in the metacarpal head, proximal phalanx, and adjacent sesamoid (*arrow*) can be seen. Periostitis of the metacarpal diaphysis is also evident (*arrowhead*). (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2000:1087.)

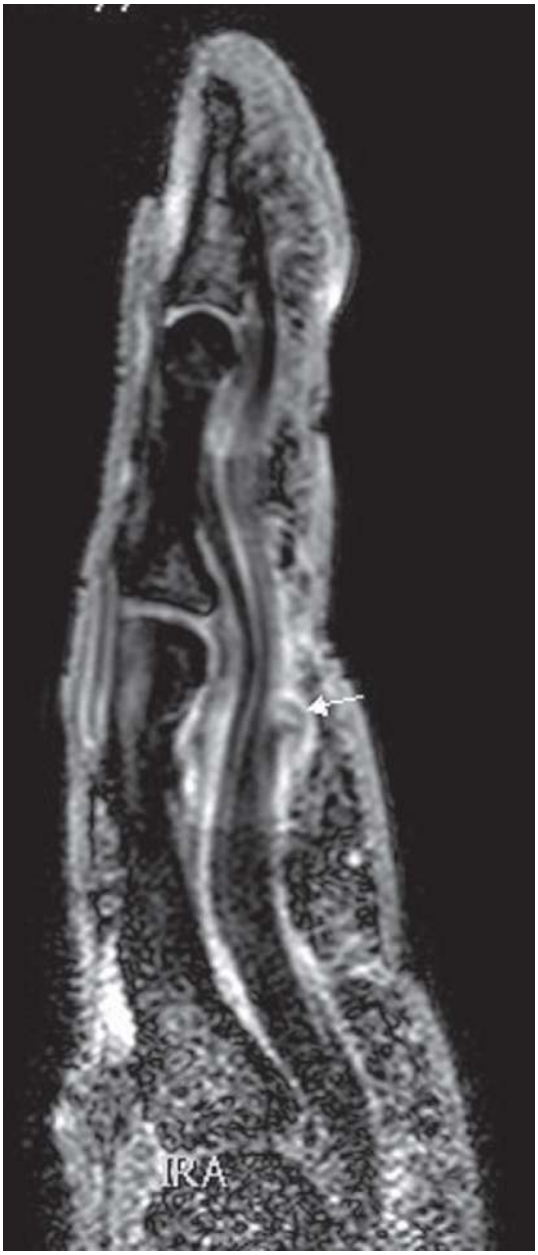

FIG 56.5 Magnetic resonance T2-weighted sagittal image showing the impinging flexor tendon tag (*arrow*). (From Couceiro J, Fraga J, Sanmartin M. Trigger finger following partial flexor tendon laceration: magnetic resonance imaging-assisted diagnosis. *Int J Surg Case Rep*. 2015;9:112–114.)

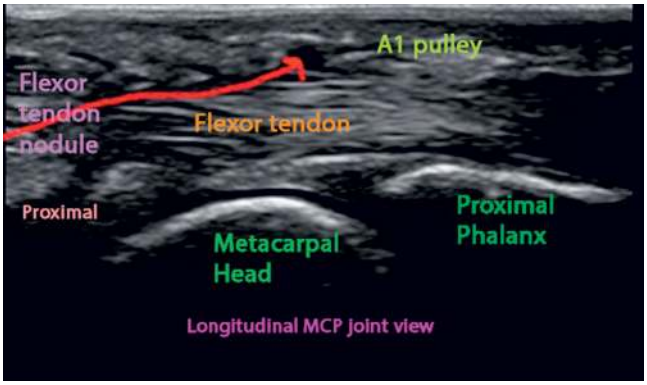

FIG 56.6 Longitudinal ultrasound image demonstrating a nodule of the flexor tendon in a patient with trigger finger.

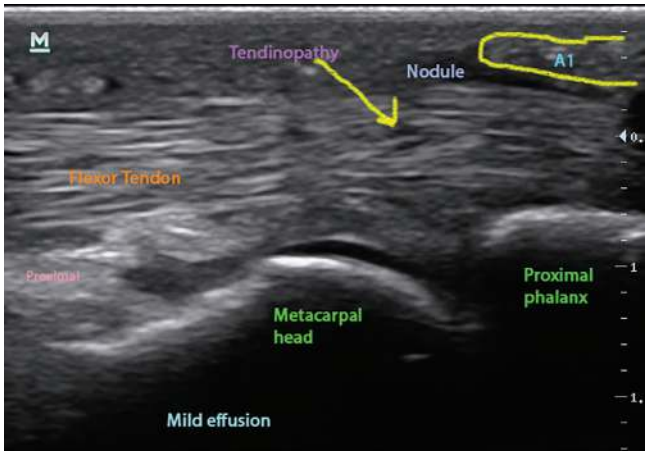

FIG 56.7 Transverse ultrasound image demonstrating a large nodule of the lateral superficial portion of the flexor tendon in a patient with trigger finger.

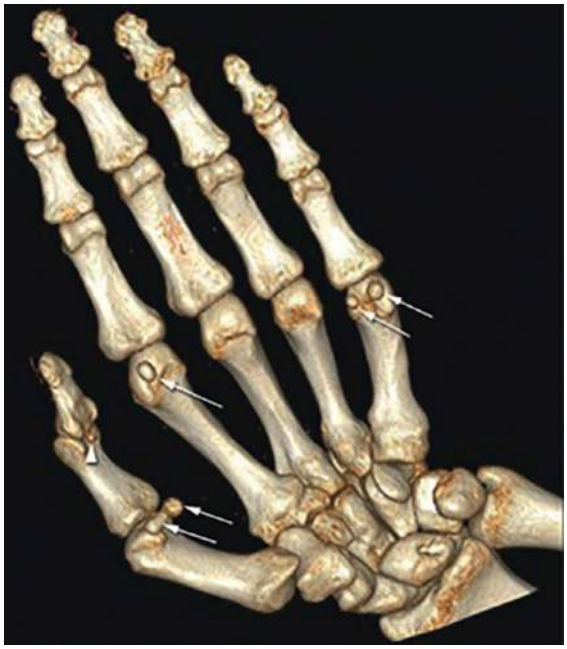

FIG 56.8 Three-dimensional computed tomography scan demonstrating the sesamoid bones of the hand. (From Ozcanli H, Sekerci R, Keles N. Sesamoid disorders of the hand. *J Hand Surg Am* 2015;40(6):1231–2123.)

FIG 56.9 Proper needle placement for ultrasound-guided trigger finger injection.

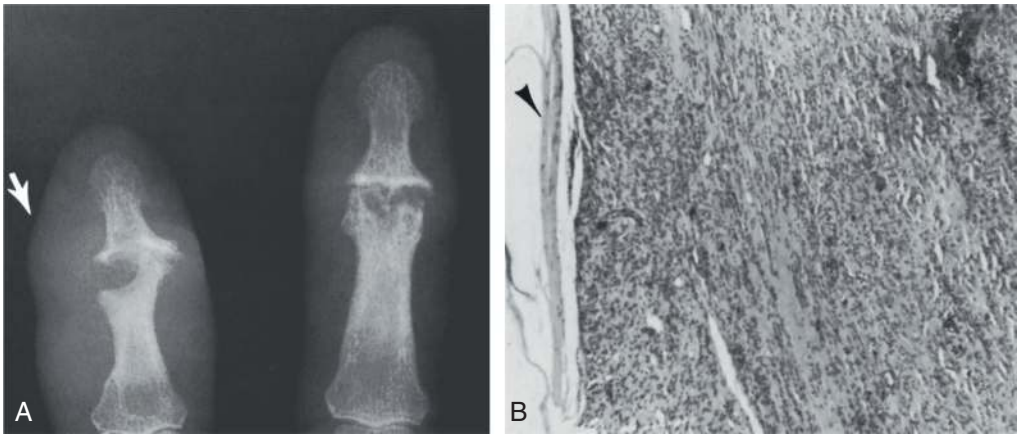

FIG 56.10 Giant cell tumor of the tendon sheath. **A,** In this 56-year-old woman with a 2-year history of pain and gradual swelling of the fingers, a soft tissue mass *(arrow)* can be identified at one distal interphalangeal joint. Underlying inflammatory osteoarthritis of the articulations is evident, and this combination of findings would suggest that the mass is a mucous cyst. However, biopsy of the affected joint demonstrated a giant cell tumor of the tendon sheath. **B,** Photomicrograph (×86) in a different patient reveals a tendon capsule tumor *(arrowhead)* associated with moderately vascularized stroma, plump spindle-shaped or ovoid cells, and multinucleated giant cells. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:42–48.)

tendon. The tendon may rupture if it is injected directly, so a needle position outside the tendon should be confirmed before proceeding with the injection. Further, the radial artery and superficial branch of the radial nerve are susceptible to damage if the needle is placed too far medially. Another complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is used, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of pain secondary to trigger finger. Coexistent arthritis or gout may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. A hand splint to protect the fingers may also help relieve the symptoms of trigger finger. Simple analgesics and NSAIDs can be used concurrently with the injection technique.

SUGGESTED READINGS

Kuczmarski AS, Harris AP, Gil JA, et al. Management of diabetic trigger finger. *J Hand Surg Am*. 2019;44(2):150153.

Matzon JL, Lebowitz C, Graham JG, et al. Risk of infection in trigger finger release surgery following corticosteroid injection. *J Hand Surg Am*. 2020;45(4):310–316.

Quinet MT, Raghavan M, Morris E, et al. Effectiveness of amniotic fluid injection in the treatment of trigger finger: a pilot study. *J Hand Surg Glob Online*. 2020;2(5):301–305.

Waldman SD. Flexor digitorum superficialis and profundus injection for tendinitis and trigger finger. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier.

Waldman SD. Trigger finger. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:451–456. Elsevier; 2017:296300.

Waldman SD. Ultrasound-guided injection technique for trigger finger 303–304. In: *Waldman's comprehensive atlas of ultrasound guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters-Kluwer; 2019:551–558.

Yamazaki A, Matsuura Y, Kuniyoshi K, et al. A1 pulley stretching treats trigger finger: A1 pulley luminal region under digital flexor tendon traction. *Clin Biomech*. 2020;72:136–140.

Sesamoiditis of the Hand

ICD-10 CODES M89.8 x 9

THE CLINICAL SYNDROME

Sesamoid bones are small, rounded structures embedded in the flexor tendons of the hand, usually in close proximity to the joints (Fig. 57.1). The name sesamoid was first used by second-century physician Galen to underscore these bones' resemblance to sesame seeds. These bones serve to decrease the friction and pressure of the flexor tendon as it passes in proximity to a joint. Most patients have two sesamoid bones at the metacarpal phalangeal joint, one at the interphalangeal joint of the thumb, one at the metacarpophalangeal joint of the index finger, and one at the metacarpophalangeal joint of the little finger. These bones are subject to fracture, dislocation, tumors, avascular necrosis, inflammation, and associated tendinitis, all of which can cause hand pain and functional disability (Fig. 57.2).

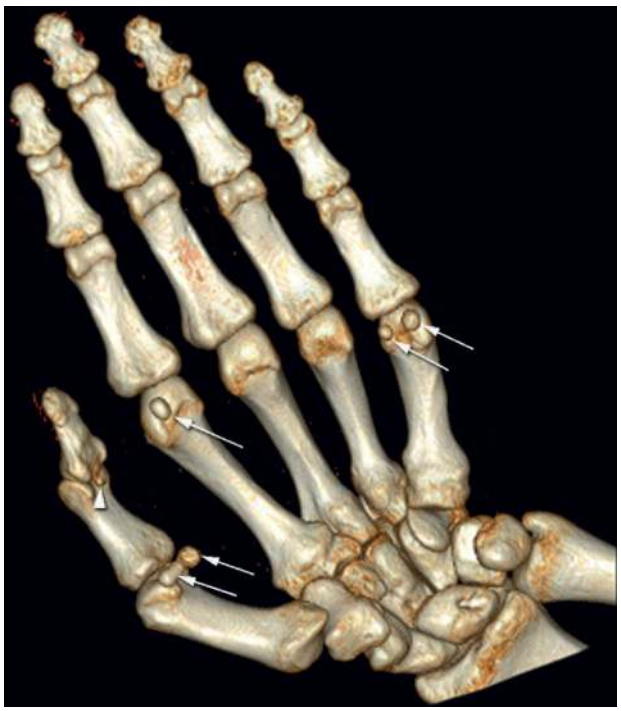

FIG 57.1 The sesamoid bones of the hand *(arrows)*. (From Ozcanli H, Sekerci R, Keles N. Sesamoid disorders of the hand. *J Hand Surg Am* 2015;40(6):1231–2123.)

Sesamoiditis is characterized by tenderness and pain over the flexor aspect of the thumb or, much less commonly, the index finger (Fig. 57.3). When grasping something, the patient often feels that he or she has a foreign body embedded in the affected digit. The pain of sesamoiditis worsens with repeated flexion and extension of the affected digit. When the thumb is affected, it is usually on the radial side, where the condyle of the adjacent metacarpal is less obtrusive. Patients suffering from psoriatic arthritis may have a higher incidence of sesamoiditis of the hand.

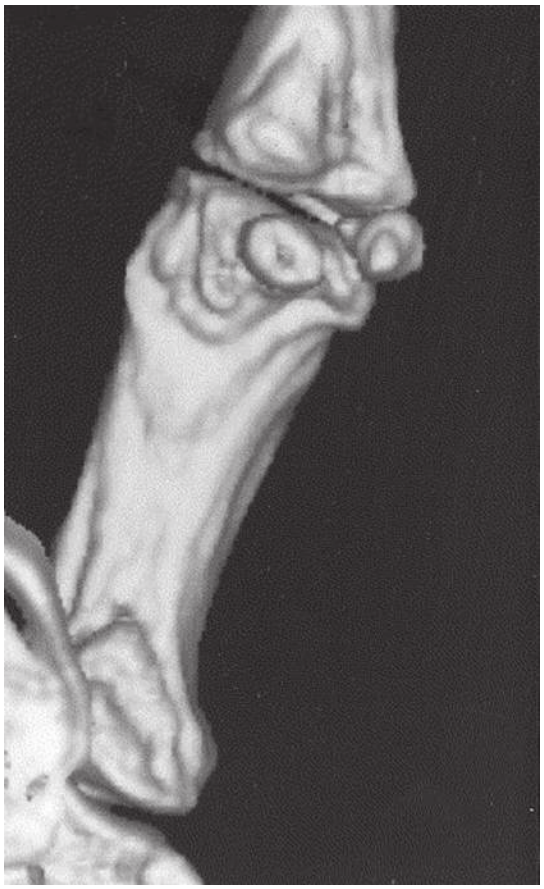

FIG 57.2 Computed tomography and reconstruction views showing proximal displacement of the radial sesamoid of the thumb. (From Deshmukh NV, Saikia AN, Norton ER, Sonanis SV. Sesamoid displacement: a rare cause of "clicking thumb." *Injury.* 1999;30(2):141–143.)

SIGNS AND SYMPTOMS

On physical examination, pain can be reproduced by pressure on the sesamoid bone. In patients with sesamoiditis, the tender area moves with the flexor tendon when the patient actively flexes the thumb or finger, whereas with occult bony disease of the phalanges, the tender area remains over the pathologic area. Clicking and triggering of the affected digit may be present on passive and active flexion and extension. With acute trauma to the sesamoid, ecchymosis over the flexor surface of the affected digit may be present.

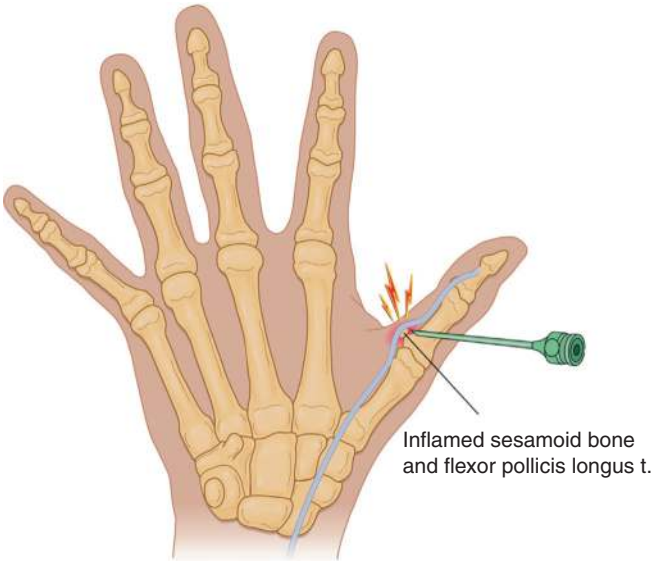

FIG 57.3 Sesamoiditis is characterized by tenderness and pain over the flexor aspect of the thumb. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:238.)

TESTING

Plain radiographs and magnetic resonance imaging (MRI) are indicated in all patients who present with sesamoiditis, to rule out fractures and identify sesamoid bones that may have become inflamed (Figs. 57.4 and 57.5). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. MRI, ultrasound imaging, and/or computed tomography of the fingers and wrist is indicated if joint instability, occult mass, occult fracture, dislocation, avascular necrosis, infection, or tumor is suspected (Fig. 57.6). Radionuclide bone scanning may be useful to identify stress fractures of the thumb and fingers or sesamoid bones that may be missed on plain radiographs of the hand.

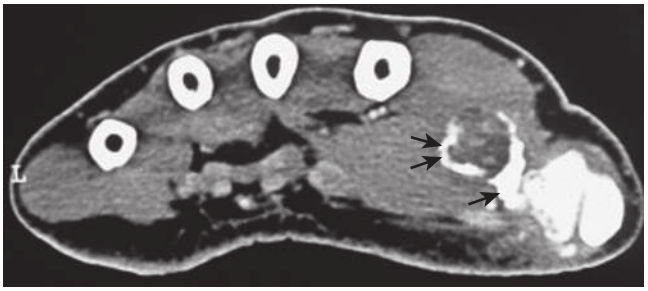

FIG 57.5 Computed tomography image of a chondroma *(double arrows)* and the ulnar sesamoid bone *(single arrow)*. (From Louaste J, Amhajji L, Eddine EC, et al. Chondroma in a sesamoid bone of the thumb: case report. *J Hand Surg Am*. 2008;33(8):1378–1379.)

FIG 57.4 Radiographic abnormalities of the metacarpophalangeal joints. **A,** Startling osseous excrescences *(arrows)* around the metacarpal heads are associated with soft tissue swelling, joint space narrowing, and bony erosion and proliferation in the phalanges. **B,** At the first metacarpophalangeal joint, irregular bone formation in the metacarpal head, proximal phalanx, and adjacent sesamoid *(arrow)* can be seen. Periostitis of the metacarpal diaphysis is also evident *(arrowhead)*. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2000:1087.)

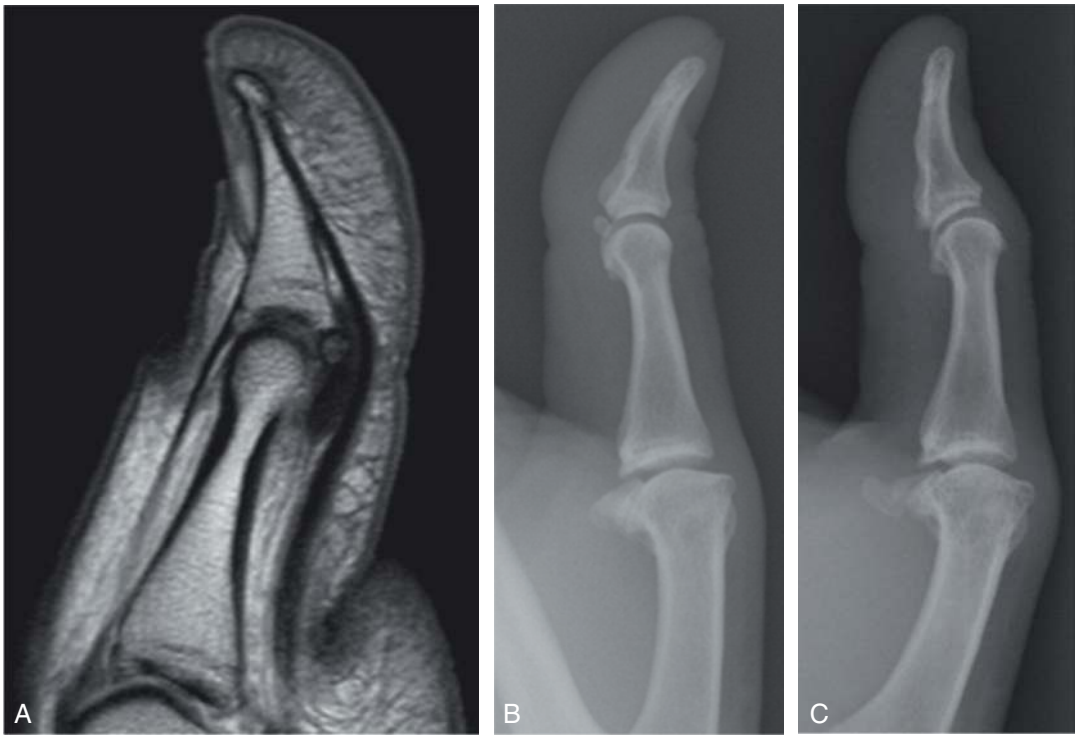

FIG 57.6 Preoperative (A) magnetic resonance imaging scan and (B) x-ray for case 2, demonstrating a thumb interphalangeal joint sesamoid bone, with (C) subsequent postoperative x-ray following excision. (From Ecker JO, Edwick SJ, Ebert JR. Painful clicking of the thumb interphalangeal joint caused by a sesamoid bone: a report of three cases. *J Hand Surg Am.* 2012; 37(3):423–426.)

DIFFERENTIAL DIAGNOSIS

The tentative diagnosis of sesamoiditis is made on clinical grounds and is confirmed by radiographic testing. Arthritis, tenosynovitis, or gout of the affected digit may accompany sesamoiditis and exacerbate the patient's pain. Occult fractures and dislocations occasionally confuse the clinical presentation. Occasionally, osseous neoplasm can occur within a sesamoid bone and can further confuse the diagnosis.

TREATMENT

Initial treatment of the pain and functional disability associated with sesamoiditis of the hand includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. A nighttime splint to protect the fingers may also help relieve the symptoms. If the patient does not respond to these conservative measures, a trial of injection therapy with local anesthetic and steroid is a reasonable next step.

To perform injection of the sesamoid bone, the patient is placed in the supine position with the palmar surface of the hand exposed. The skin overlying the tender sesamoid bone is prepared with antiseptic solution. A sterile syringe containing 2-mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 5/8-inch, 25-gauge needle using strict aseptic technique. The needle is carefully advanced through the palmar surface of the affected digit until the needle tip rests against the sesamoid bone (see Fig. 57.3). The needle is then withdrawn slightly out of the periosteum and substance of the tendon. Once the needle is in the correct position next to the affected sesamoid bone and aspiration for blood is negative, the contents of the syringe are gently injected. Slight resistance to injection may be felt, given the closed nature of the space. If significant resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. The ice should not be left on for longer than 10 minutes to avoid freezing injuries.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, which should be exceedingly rare if strict aseptic technique is followed; in addition, universal precautions should be used to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection of sesamoid bones, and patients should be warned of this possibility.

CLINICAL PEARLS

Pain emanating from the hand is a common problem. Sesamoiditis must be distinguished from stress fractures and other occult diseases of the phalanges, as well as fractures of the sesamoid bones. Although the injection technique described provides pain relief, patients often require resting hand splints to aid in rehabilitation of the affected finger. Padded gloves may be useful to take pressure off the affected sesamoid bone and overlying soft tissue. Coexisting bursitis and tendinitis may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and steroid. Simple analgesics and NSAIDs can be used concurrently with the injection technique.

SUGGESTED READINGS

Chin AYH, Sebastin SJ, Wong M, et al. Long-term results using a treatment algorithm for chronic sesamoiditis of the thumb metacarpophalangeal joint. *J Hand Surg Am*. 2013;38(2):316–321.

Deshmukh NV, Saikia AN, Norton ER, et al. Sesamoid displacement: a rare cause of 'clicking thumb'. *Injury*. 1999;30(2):141–143.

Ecker JO, Edwick SJ, Ebert JR. Painful clicking of the thumb interphalangeal joint caused by a sesamoid bone: a report of three cases. *J Hand Surg Am*. 2012;37(3):423–426.

Waldman SD. Painful conditions of the wrist and hand. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2016:166.

Waldman SD. Injection technique for sesamoiditis of the hand. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2017:304–306.

Plastic Bag Palsy

ICD-10 CODE G56.90

THE CLINICAL SYNDROME

Plastic bag palsy is an entrapment neuropathy of the digital nerves caused by compression of the nerves against the bony phalanges by the handles of a plastic bag. The common digital nerves arise from fibers of the median and ulnar nerves. The thumb also has contributions from superficial branches of the radial nerve. The common digital nerves pass along the metacarpal bones and divide as they reach the distal palm. The volar digital nerves supply the majority of sensory innervation to the fingers and run along the ventrolateral aspect of the finger beside the digital vein and artery. The smaller dorsal digital nerves contain fibers from the ulnar and radial nerves and supply the dorsum of the fingers as far as the proximal joints.

Plastic bag palsy has increased in frequency as stores have switched from paper to plastic bags. Compression by the handles of a heavy plastic bag is the inciting cause, and the most common clinical feature is the presence of painful digital nerves at the point of compression (Fig. 58.1). Plastic bag palsy may present in either an acute or a chronic form. Pain may develop from an acute injury to the nerves after carrying a heavy bag on too few fingers, or it may occur from direct trauma to the soft tissues overlying the digital nerves if the fingers become caught in a bag handle twisted around them. Plastic bag palsy is occasionally seen in homeless people who carry their possessions around in bags and who use the same hand day after day. The affected nerves may be thickened, and inflammation of the nerve and overlying soft tissues may be seen. In addition to pain, patients may complain of paresthesias and numbness just below the point of nerve compromise.

SIGNS AND SYMPTOMS

The pain of plastic bag palsy is constant and is made worse with compression of the affected digital nerves. Patients often note the inability to hold objects with the affected fingers. Sleep disturbance is common.

On physical examination, the patient has tenderness to palpation of the affected digital nerves. Palpation can also cause paresthesias, and continued pressure on the nerves may induce numbness distal to the point of compression. The range of motion of the thumb is normal. With acute trauma to the sesamoid, ecchymosis of the skin overlying the affected digital nerves may be present.

TESTING

Plain radiographs are indicated in all patients who present with plastic bag palsy, to rule out occult bony disorders such as bone spurs or cysts that may be compressing the digital nerves. Electromyography can distinguish other causes of hand numbness. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the hand can rule out soft tissue abnormalities including tumors that may be compressing the digital nerves (Fig. 58.2). Injection of the nerve serves as both a diagnostic and a therapeutic maneuver.

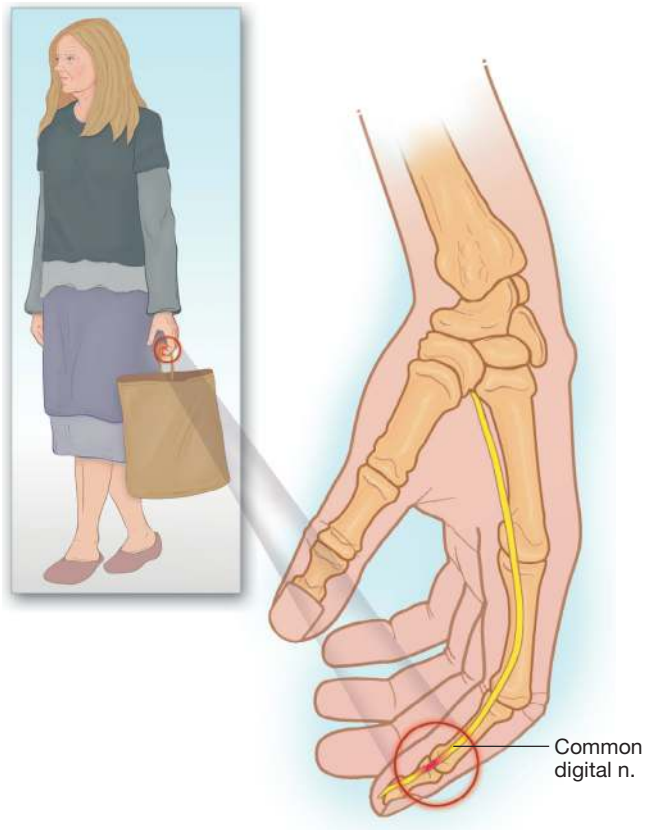

FIG 58.1 Compression by the handles of a heavy plastic bag can cause plastic bag palsy.

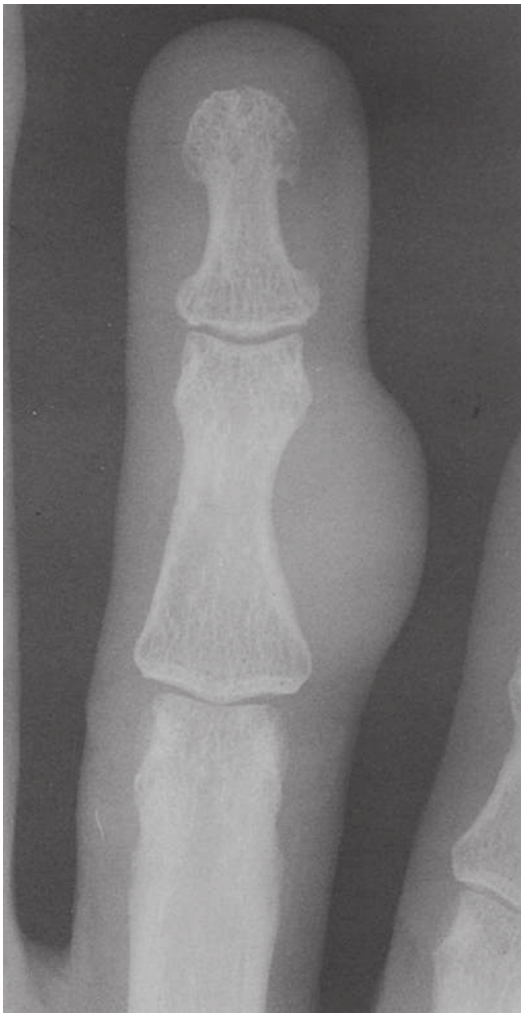

FIG 58.2 Fibrous xanthoma. A soft tissue mass adjacent to the middle phalanx has produced erosion of the adjacent bone. This pattern of bony resorption is indicative of pressure atrophy and is not a sign of malignancy. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:4190.)

DIFFERENTIAL DIAGNOSIS

The tentative diagnosis of plastic bag palsy is made on clinical grounds and is confirmed by electromyography. Arthritis, tenosynovitis, or gout of the affected digits may accompany plastic bag palsy and exacerbate the patient's pain. Occult fractures occasionally confuse the clinical presentation as can tumors of the digital nerves (Fig. 58.3).

TREATMENT

The first step in the treatment of the pain and functional disability associated with plastic bag palsy is to remove the offending compression of the digital nerves. Nonsteroidal antiinflammatory drugs, simple analgesics, or cyclooxygenase-2 inhibitors may be prescribed as well. If the patient complains of significant dysesthesias or paresthesias, the addition of gabapentin should be considered. Gabapentin is started at a bedtime dose of 300 mg; it is then titrated upward to 3600 mg in divided doses, as side effects allow. Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced to avoid loss of function. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. A nighttime splint to protect the fingers may be helpful, and wearing padded gloves can take pressure off the affected digital nerves and overlying soft tissue. If sleep disturbance is present, low-dose tricyclic antidepressants are indicated. If the patient does not respond to these conservative modalities, a trial of injection therapy with local anesthetic and steroid is a reasonable next step. Rarely, surgical exploration and neuroplasty of the affected nerves are required for symptomatic relief.

COMPLICATIONS AND PITFALLS

Injection of the affected digital nerves is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, as well as universal

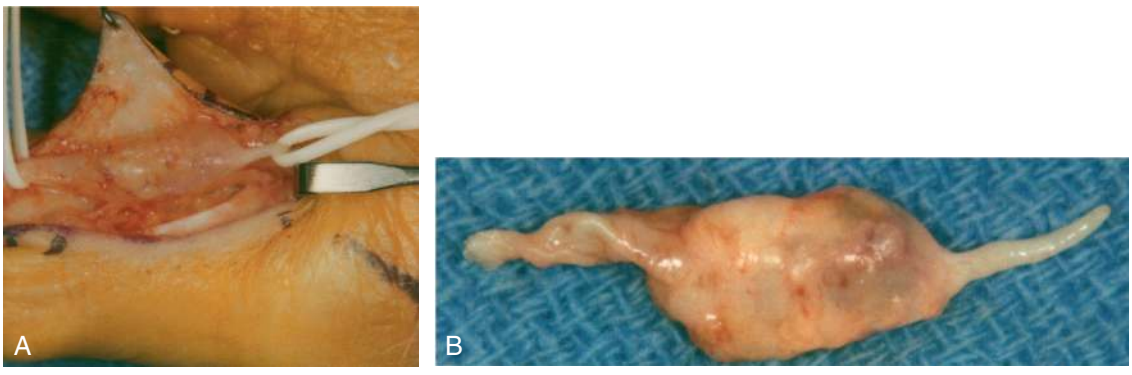

FIG 58.3 **A,** Intraneural ganglion of the dorsal branch of the radial digital nerve of the ring finger. **B,** The ganglion was excised with the nerve. (From Naam NH, Carr SB, Massoud AHA. Intraneural ganglions of the hand and wrist. *J Hand Surg Am*. 2015;40(8):1625–1630.)

precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complication of injection is infection, which should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection of the affected digital nerves, and patients should be warned of this possibility.

CLINICAL PEARLS

Pain emanating from the hand is a common problem. Plastic bag palsy must be distinguished from stress fractures and other occult disorders of the phalanges, as well as sesamoiditis and occult fractures of the sesamoid bones. Coexistent bursitis and tendinitis may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and steroid.

SUGGESTED READINGS

El Sayed L, Teboul F, Asmar G, Goubier J-N. The first case of hourglass-like constriction neuropathy of a digital nerve. *Hand Surg Rehabil*. 2018;37(2):114–116. https://doi.org/10.1016/j.hansur.2017.11.005.

Hill EJR, Patterson JMM, Yee A, et al. What is operative? Conceptualizing neuralgia: neuroma, compression neuropathy, painful hyperalgesia, and phantom nerve pain. *J Hand Surg Glob Online*. 2022. https://doi.org/10.1016/j.jhsg.2021.11.004.

Júnior MH. Uncommon compressive neuropathies of upper limbs. *Best Pract Res Clin Rheumatol*. 2020;34(3):101516. https://doi.org/10.1016/j.berh.2020.101516.

Khadilkar SV, Patil SB, Shetty VP. Neuropathies of leprosy. *J Neurol Sci*. 2021;420:117288. https://doi.org/10.1016/j.jns.2020.117288.

Naam NH, Carr SB, Massoud AHA. Intraneural ganglions of the hand and wrist. *J Hand Surg Am*. 2015;40(8):1625–1630.

Pinski J, Guss M, Coccoluto N, et al. Ulnar-sided digital nerve tumor of the thumb in the professional baseball player who uses grip-adjusting batting equipment: a report of 2 cases. *J Hand Surg Am*. 2021;46(10):932.e1–932.e5. https://doi.org/10.1016/j.jhsa.2020.10.028.

Rosenbaum AJ, Leonard G, Mulligan M, et al. Nerve entrapments in musicians. In: Tubbs RS, Rizk E, Shoja MM, eds. *Nerves and nerve injuries*. San Diego: Academic Press; 2015:665–675.

Waldman SD. Painful conditions of the wrist and hand. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2016:166.

59

Carpal Boss Syndrome

ICD-10 CODE M25.70

THE CLINICAL SYNDROME

Carpal boss syndrome, or os styloideum, is characterized by localized tenderness and sharp pain over the junction of the second and third carpometacarpal joints. The pain of carpal boss syndrome results from exostosis of the second and third carpometacarpal joints or, more uncommonly, a loose body involving the intraarticular space (Fig. 59.1). Patients often report that the pain is worse *after* rigorous physical activity involving the hand rather than during the activity itself. The pain of carpal boss syndrome may also radiate locally, thus confusing the clinical presentation. The disease typically affects the dominant hand, although carpal bossing is present approximately 15% of the time in patients suffering from carpal boss syndrome. Carpal boss syndrome has a slight male predominance and a peak incidence in the middle of the third decade of life. Trauma is often the common denominator in the development of carpal boss syndrome.

SIGNS AND SYMPTOMS

On physical examination, the carpal boss appears as a bony protuberance that can be seen more easily by having the patient flex his or her wrist (Figs. 59.2 and 59.3). The pain

A

B

FIG 59.1 Radiographic manifestations of os styloideum. A lateral radiograph of the hand **(A)** demonstrates the osteophytic appearance of the extra ossification center *(arrow)*. Clinically, a painless soft tissue lump is often evident. In another patient, a similar outgrowth *(arrows)* is evident on lateral **(B)** and frontal **(C)** radiographs. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:1312.)

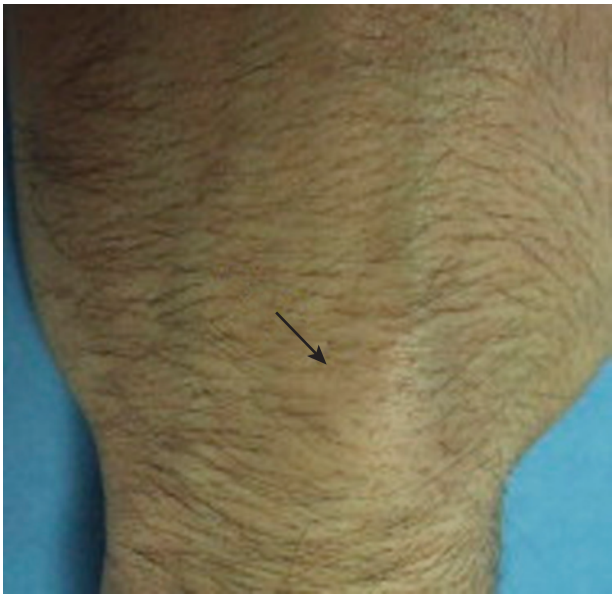

FIG 59.2 The carpal boss is frequently confused initially with a dorsal ganglion on viewing the dorsal wrist. It generally feels harder with palpation, is positioned more distally than wrist ganglion, and overlies the index and middle finger carpometacarpal joints *(arrow)*. (From Park MJ, Namdari S, Weiss AP. The carpal boss: review of diagnosis and treatment. *J Hand Surg Am*. 2008;33(3):446–449.)

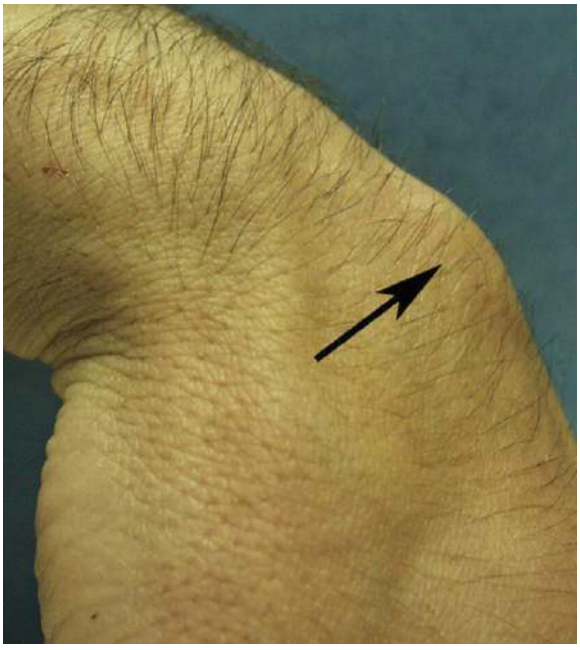

FIG 59.3 With wrist flexion, the prominence of the carpal boss becomes strikingly evident (*arrow*). (From Park MJ, Namdari S, Weiss AP. The carpal boss: review of diagnosis and treatment. *J Hand Surg*. 2008;33(3):446–449.)

associated with this action can be reproduced by applying pressure to the soft tissue overlying the carpal boss. Patients with carpal boss syndrome demonstrate a positive hunchback sign; that is, the examiner can appreciate a bony prominence when he or she palpates the carpal boss

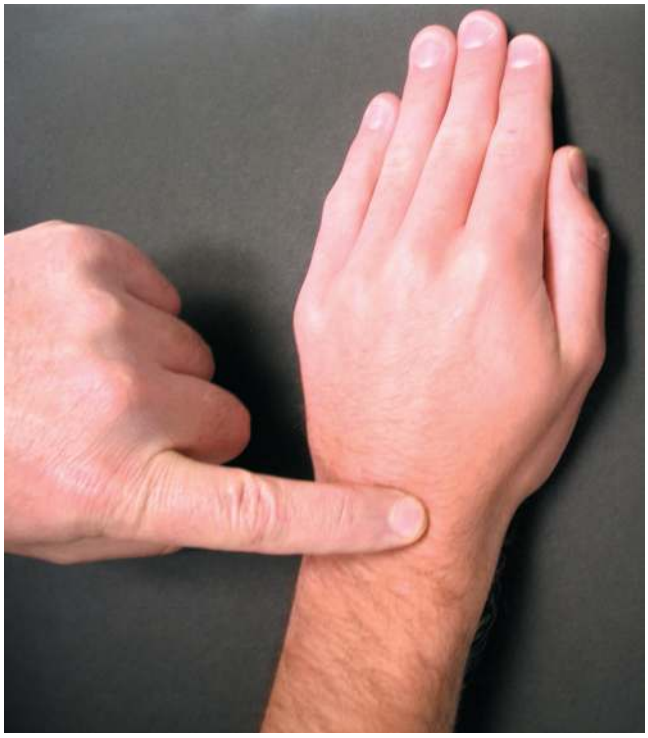

FIG 59.4 The hunchback sign for carpal boss. (From Waldman SD. Painful conditions of the wrist and hand. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:189.)

(Fig. 59.4). Occasionally an inflamed bursa may overlie the bony excrudescence. With acute trauma to the dorsum of the hand, ecchymosis over the carpal boss of the affected joint or joints may be present.

TESTING

Plain radiographs are indicated in all patients who present with a carpal boss, to rule out fractures and to identify exostoses responsible for the symptoms (Box 59.1; see Figs. 59.5 and 59.6).Based on the patient's clinical presentation, additional testing may be warranted to exclude inflammatory arthritis, including a complete blood count, erythrocyte sedimentation rate, uric acid level, and antinuclear antibody testing. Magnetic resonance imaging, computerized tomography, and ultrasound imaging of the fingers and wrist are indicated if joint instability, occult mass, occult fracture, infection, or tumor is suspected, as well as to further assess the condition of the overlying tendons (Figs. 59.7 and 59.6). Radionuclide bone scanning may be useful to identify carpal boss as well as occult stress fractures (see Fig. 59.7).

DIFFERENTIAL DIAGNOSIS

The tentative diagnosis of carpal boss syndrome is made on clinical grounds and is confirmed by radiographic testing. Arthritis, tenosynovitis, or gout of the affected digits

BOX 59.1 Differential Diagnosis of Carpal Boss

- Ganglion cyst
- Inflamed bursa
- Synovitis
- Fracture callus
- Exuberant synovium
- Fibroma
- Giant cell tumor
- Aneurysmal bone cyst
- Unicameral bone cyst
- Lipoma
- Neural tumors
- Interosseous ganglions
- Osteoid osteoma
- Osteochondroma
- Osteosarcoma
- Metastatic disease

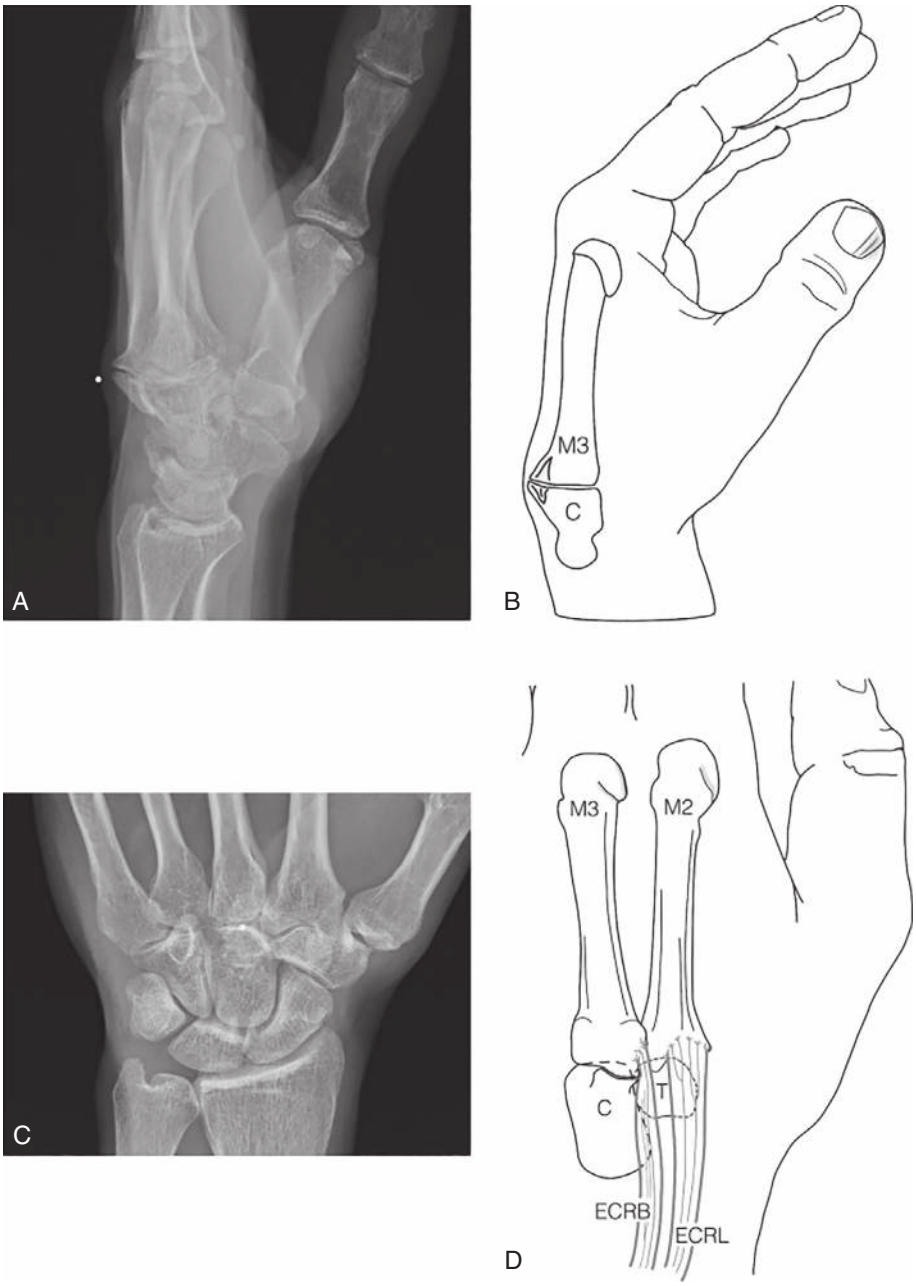

FIG 59.5 Lateral radiograph **(A)** metallic marker and **(B)** diagram demonstrate a carpal boss at the base of the third metacarpal and capitate. Frontal radiograph **(C)** metallic marker and **(D)** diagram demonstrate the carpal boss and a theoretic depiction of associated displacement of the extensor carpi radialis brevis tendon. *C*, Capitate; *ECRB*, extensor carpi radialis brevis; *ECRL*, extensor carpi radialis longus; *M2*, second metacarpal; *M3*, third metacarpal; *T*, trapezoid. (From Porrino J, Maloney E, Chew FS. Current concepts of the carpal boss: pathophysiology, symptoms, clinical or imaging diagnosis, and management. *Curr Prob Diag Radiol*. 2015;44(5):462–468.)

FIG 59.6 A 36-year-old man with an asymptomatic palpable mass at the dorsal surface of his right wrist. Transverse **(A)** and longitudinal **(B)** ultrasound images obtained using a 12–5 MHz linear transducer on a Philips iu22 machine demonstrate a fragmented carpal boss at the dorsal base of the third metacarpal *(arrowheads)*, adjacent to the trapezoid ("T") and capitate ("C"). A radiograph from the same patient obtained with partial supination and ulnar deviation **(C)** improves conspicuity of the carpal boss *(arrowheads)* relative to the routine lateral view **(D)**. (From Porrino J, Maloney E, Chew FS. Current concepts of the carpal boss: pathophysiology, symptoms, clinical or imaging diagnosis, and management. *Curr Probl Diagn Radiol*. 2015;44(5):462–468.)

may accompany carpal boss syndrome and exacerbate the patient's pain. Occult fractures occasionally confuse the clinical presentation.

TREATMENT

Initial treatment of the pain and functional disability associated with a carpal boss consists of nonsteroidal antiinflammatory drugs, simple analgesics, or cyclooxygenase-2 inhibitors. Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced to avoid loss of function. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. A nighttime splint to protect the fingers may be helpful. If sleep disturbance is present, low-dose tricyclic antidepressants are indicated. If the patient does not respond to these conservative modalities, a trial of injection therapy with local anesthetic and steroid is a reasonable next step (Fig. 59.8). Rarely, surgical exploration and removal of the carpal boss are required for symptomatic relief.

COMPLICATIONS AND PITFALLS

The major complication of injection with local anesthetic and steroid is infection, which should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility. The clinician should always keep in mind that occult fracture or tumor may mimic the clinical symptoms of a carpal boss.

FIG 59.7 A 39-year-old woman with dorsal wrist pain. Bone scan **(A)**, acquired 3 hours after injection of 32 mCi Tc-99m MDP, demonstrates increased radiotracer uptake in the left wrist *(asterisk)* compared with the right. Sagittal noncontrast computed tomography (CT) scan **(B)** and noncontrast proton-density fat-saturated magnetic resonance (MR) **(C)** image demonstrates a carpal boss at the base of the third metacarpal *(arrowhead)*. Noncontrast axial T2 fat-saturated MR image **(D)** just proximal to the carpal boss demonstrates abnormal fluid in the tendon sheath of the extensor carpi radialis brevis *(arrowhead)*, consistent with tenosynovitis. (From Porrino J, Maloney E, Chew FS. Current concepts of the carpal boss: pathophysiology, symptoms, clinical or imaging diagnosis, and management. *Curr Probl Diagn Radiol*. 2015;44(5):462–468.)

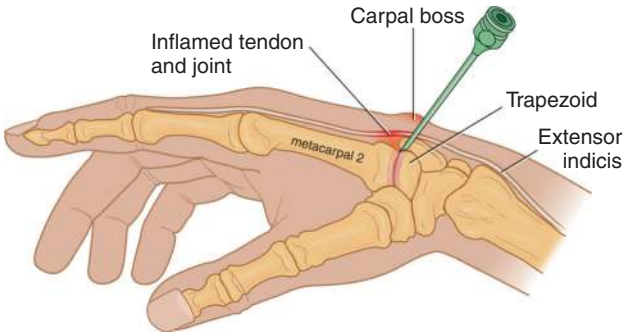

FIG 59.8 Injection technique for carpal boss, or os styloideum. (From Waldman SD. Carpal boss. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:268.)

CLINICAL PEARLS

Pain emanating from the hand is a common problem. Carpal boss must be distinguished from stress fracture, arthritis, and other occult disorders of the wrist and hand. Although injection with local anesthetic and steroid palliates the pain of carpal boss, patients may ultimately require surgical removal of the exostosis to obtain long-lasting relief. Coexistent bursitis and tendinitis may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and steroid.

SUGGESTED READINGS

Alemohammad AM, Nakamura K, El-Sheneway M, et al. Incidence of carpal boss and osseous coalition: an anatomic study. *J Hand Surg Am*. 2009;34(1):1–6.

Fusi S, Watson HK, Cuono CB. The carpal boss: a 20-year review of operative management. *J Hand Surg Am*. 1995;20(3):405–408.

Ghatan AC, Carlson EJ, Athanasian EA, et al. Attrition or rupture of digital extensor tendons due to carpal boss: report of 2 cases. *J Hand Surg Am*. 2014;39(5):919–922.

Melone CP Jr, Polatsch DB, Beldner S. Disabling hand injuries in boxing: boxer's knuckle and traumatic carpal boss. *Clin Sports Med*. 2009;28(4):609–621.

Park MJ, Namdari S, Weiss AP. The carpal boss: review of diagnosis and treatment. *J Hand Surg Am*. 2008;33(3):446–449.

Porrino J, Maloney E, Chew FS. Current concepts of the carpal boss: pathophysiology, symptoms, clinical or imaging diagnosis, and management. *Curr Probl Diagn Radiol*. 2015;44(5):462–468.

Waldman SD. Carpal boss. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:267–279.

Waldman SD. Painful conditions of the wrist and hand. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:153–154.

60

Dupuytren's Contracture

ICD-10 CODE M72.0

THE CLINICAL SYNDROME

Dupuytren's contracture is a common complaint. Although it is initially painful, the pain seems to decrease as the condition progresses. As a result, patients suffering from Dupuytren's contracture generally seek medical attention for functional disability rather than for pain.

Dupuytren's contracture is caused by progressive fibrosis of the palmar fascia. Initially, the patient may notice fibrotic nodules along the course of the flexor tendons of the hand that are tender to palpation. As the disease advances, these nodules coalesce and form fibrous bands that gradually thicken and contract around the flexor tendons; this has the effect of drawing the affected fingers into flexion. Although any finger can develop Dupuytren's contracture, the ring and little fingers are most commonly affected (Fig. 60.1). If untreated, the fingers can develop permanent flexion contractures. The plantar fascia may be affected concurrently.

Dupuytren's contracture is thought to have a genetic basis and occurs most frequently in male patients of northern Scandinavian descent. It has an autosomal-dominant inheritance pattern with variable penetrance. Siblings of patients suffering from Dupuytren's contracture have three times the risk of developing the disease than the general population. Recent research has demonstrated that there are nine susceptibility loci with the strongest association with the clinical expression of Dupuytren's contracture located on an intron of *EPDR1*, the gene responsible for encoding the ependymin-related 1 protein. The disease may also be associated with trauma to the palm,

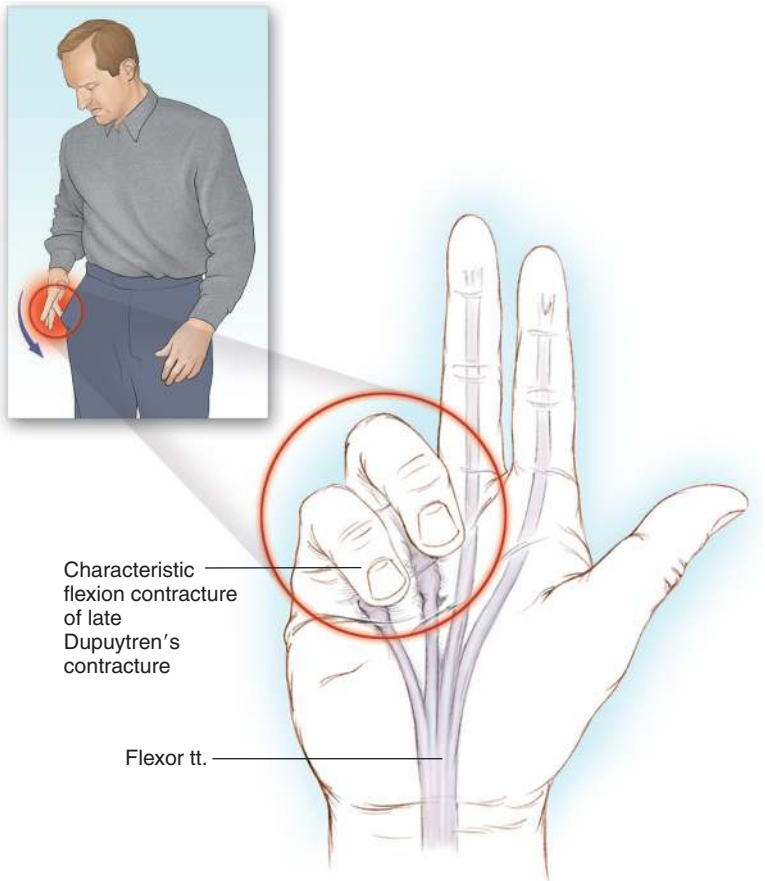

FIG 60.1 Dupuytren's contracture usually affects the fourth and fifth digits in men older than 40 years.

diabetes, alcoholism, anticonvulsants, and long-term barbiturate use. The disease rarely occurs before the fourth decade.

SIGNS AND SYMPTOMS

In the early stages of the disease, hard fibrotic nodules known as Garrod's pads along the path of the flexor tendons can be palpated (Fig. 60.2). These nodules are often misdiagnosed as calluses or warts. At this early stage, pain is invariably present. As the disease progresses, taut fibrous bands form; they may cross the metacarpophalangeal joint and ultimately the proximal interphalangeal joint (Fig. 60.3). These bands are not painful to palpation, and, although they limit finger extension, finger flexion remains relatively normal. At this point, patients often seek medical advice because of difficulty putting on gloves and reaching into their pockets (Fig. 60.4).

In the final stages of the disease, flexion contracture develops, with its negative impact on function (Fig. 60.5). Arthritis, gout of the metacarpal and interphalangeal joints, and trigger finger may coexist with Dupuytren's contracture and may exacerbate the patient's pain and disability.

TESTING

Plain radiographs are indicated for all patients who present with Dupuytren's contracture, to rule out occult bony disease (Fig. 60.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the hand are indicated if joint instability or tumor is suspected as well as to further evaluate the extent of

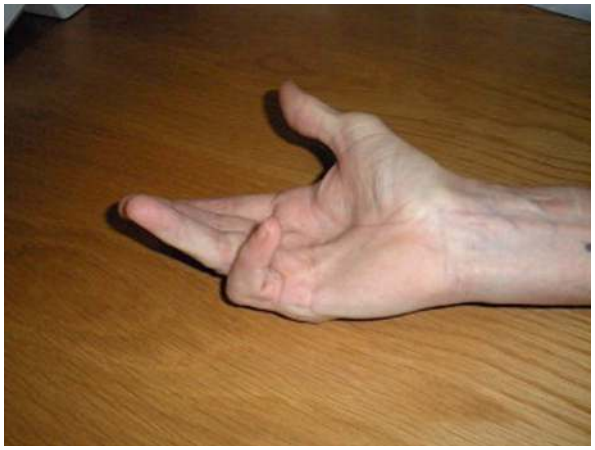

FIG 60.2 This clinical photograph demonstrates the classic presentation of Dupuytren's disease. The ulnar fingers show flexion contractures. (From Birks M, Bhalla A. Dupuytren's disease. *Surgery (Oxf)*. 2013;31(4):177–180.)

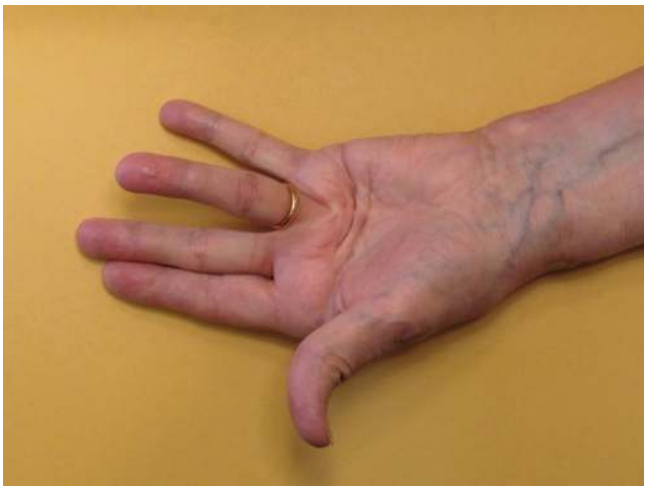

FIG 60.3 Stage 1 of Dupuytren's contracture presents as band in the palmar aponeurosis with skin tethering, puckering, and pitting. (From Bogdanov I, Payne CR. Dupuytren contracture as a sign of systemic disease. *Clin Dermatol*. 2019;37(6):675–678.)

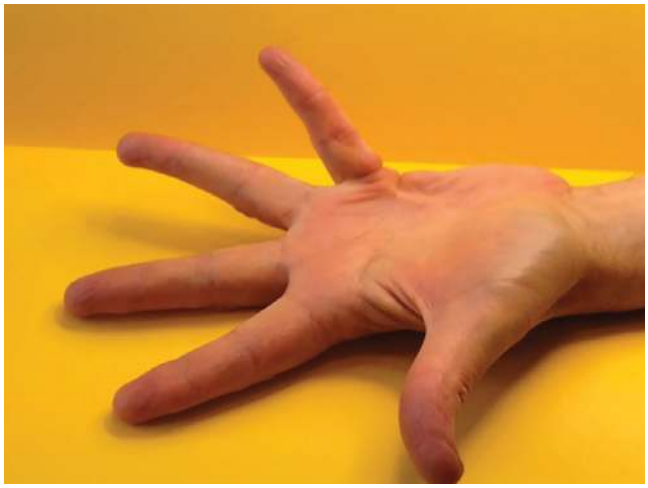

FIG 60.4 Stage 2 presents as a peritendinous band, and extension of the affected finger is limited. (From Bogdanov I, Payne CR. Dupuytren contracture as a sign of systemic disease. *Clin Dermatol*. 2019;37(6):675–678.)

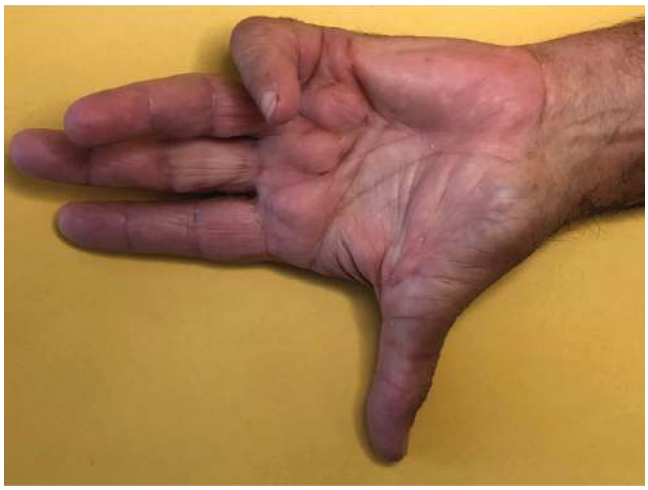

FIG 60.5 Stage 3 of Dupuytren's contracture presents as flexion contracture of the fifth digit. (From Bogdanov I, Payne CR. Dupuytren contracture as a sign of systemic disease. *Clin Dermatol*. 2019;37(6):675–678.)

A B

FIG 60.6 Radiographic manifestations of Dupuytren's contracture. **A,** Flexion deformities of the metacarpophalangeal joints of the four ulnar digits are demonstrated. **B,** Severe flexion contracture is evident in the fifth finger, with minor changes in the other digits. (From Resnick D. *Diagnosis of bone and joint disorders.* 4th ed. Philadelphia: Saunders; 2002:4667.)

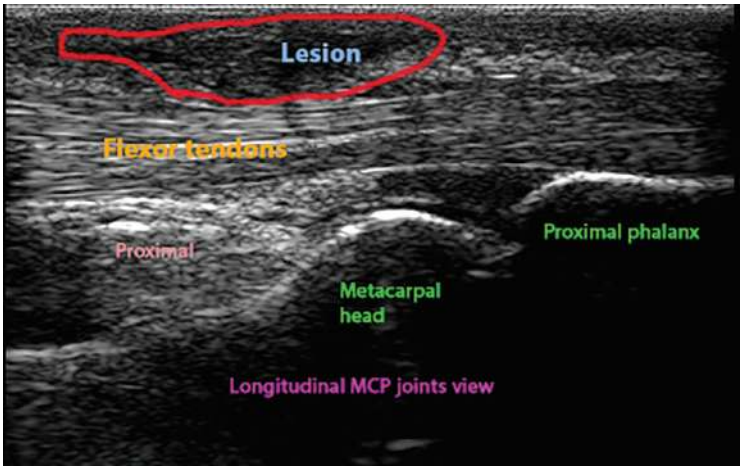

FIG 60.7 Longitudinal ultrasound image demonstrating stage 1 Dupuytren's contracture (palmar fibromatosis). Rounded hypoechoic solid fibroma (*arrows*) on the palmar aspect of the flexor tendon is the earliest sign of Dupuytren's contracture of the hand. A plantar fibroma of the foot would be intimately related to the plantar fascia and have a similar appearance on ultrasound.

the disease prior to surgical treatment (Figs. 60.7 and 60.8). Electromyography is indicated if coexistent ulnar or carpal tunnel syndrome is a possibility.

DIFFERENTIAL DIAGNOSIS

Dupuytren's contracture is a clinically distinct entity that is rarely misdiagnosed once the syndrome is well established. Rarely, trigger finger or the claw hand associated with ulnar nerve pathology is misdiagnosed as Dupuytren's contracture. Coexisting flexor tendinitis or trigger finger may be confused with Dupuytren's contracture early in the course of the disease (Box 60.1).

TREATMENT

Initial treatment of the pain and functional disability associated with Dupuytren's contracture includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. A nighttime splint

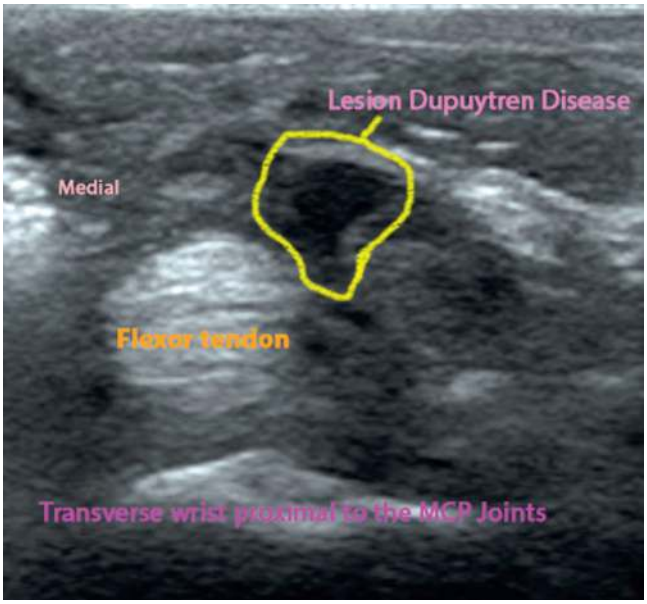

FIG 60.8 Transverse ultrasound image demonstrating a large palmar fibromatosis associated with Dupuytren's disease.

BOX 60.1 Diseases That May Mimic Dupuytren's Disease

- Ganglion cysts
- Callus formation
- Hyperkeratosis
- Rheumatoid nodules
- Palmar fibromatosis
- Pigmented villonodular synovitis
- Giant cell tumors
- Epithelioid sarcomas

to protect the fingers may be helpful. If greater symptomatic relief is required, the following injection technique is a reasonable next step.

Injection of Dupuytren's contracture is carried out by placing the patient in the supine position with the arm fully adducted at the patient's side and the dorsal surface of the hand resting on a folded towel. A total of 2-mL local anesthetic and 40-mg methylprednisolone are drawn up in a 5-mL sterile syringe. The skin overlying the fibrous band or nodule is prepared with antiseptic solution. At a point just lateral to the fibrosis, a 1-inch, 25-gauge needle is inserted at a 45-degree angle parallel to the fibrosis, through the skin, and into the subcutaneous tissue overlying the fibrotic area (Fig. 60.9). If bone is encountered, the needle is withdrawn into the subcutaneous tissue and is advanced again in proximity to the fibrosis. The contents of the syringe are then gently injected. The clinician may feel some resistance to injection because of fibrosis of the surrounding tissue. If significant resistance is encountered, the needle is probably in the tendon or nodule and should be withdrawn until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance may improve the accuracy of needle placement. This technique can be used for the injection of collagenase clostridium histolyticum which is rapidly gaining acceptance as a nonsurgical treatment for Dupuytren's contracture.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

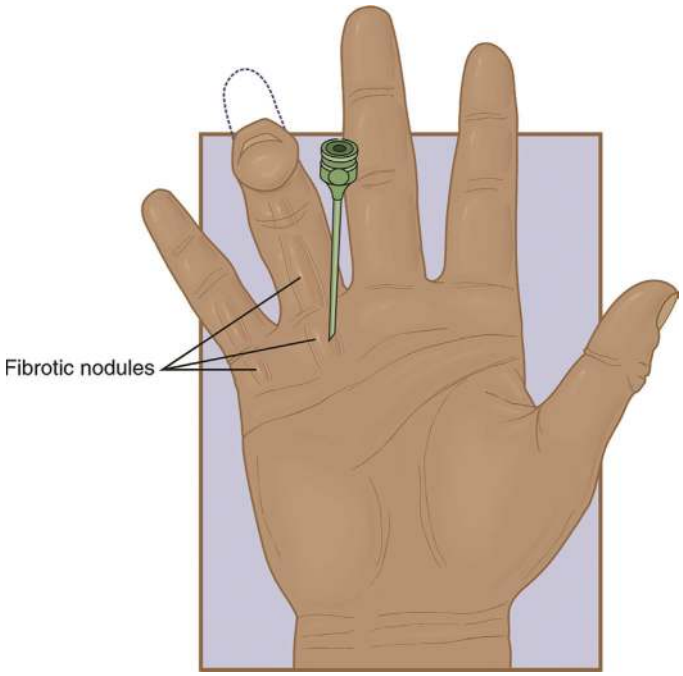

FIG 60.9 Injection technique for the treatment of Dupuytren's contracture. (From Waldman SD. *Atlas of pain management injection techniques*. 4th ed. St. Louis: Elsevier; 2017.)

Although the foregoing treatment modalities provide symptomatic relief, Dupuytren's contracture usually requires surgical treatment.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, as well as universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The major complications associated with injection are related to trauma to an inflamed or previously damaged tendon. Such tendons may rupture if they are injected directly, so a needle position outside the tendon should be confirmed before the clinician proceeds with the injection. Another complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The aforementioned treatment modalities are useful in providing symptomatic relief of the pain and disability of Dupuytren's contracture. However, most patients ultimately require surgical treatment. Coexistent arthritis or gout may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. Simple analgesics and NSAIDs can be used concurrently with this injection technique. This disease can occur in the feet and penis and is known as lederhosen disease and Peyronie's disease, respectively.

SUGGESTED READINGS

Bogdanov I, Payne CR. Dupuytren contracture as a sign of systemic disease. *Clin Dermatol*. 2019;37(6):675–678.

Dutta A, Jayasinghe G, Deore S, et al. Dupuytren's contracture current concepts. *J Clin Orthop Trauma*. 2020;11(4):590–596.

Fei TT, Chernoff E, Monacco NA, et al. Collagenase Clostridium histolyticum for the treatment of distal interphalangeal joint contractures in Dupuytren disease. *J Hand Surg Am*. 2019;44(5):417.e1–417.e4.

Kadhum M, Smock E, Khan A, et al. Radiotherapy in Dupuytren's disease: a systematic review of the evidence. *J Hand Surg Eur Vol*. 2017;42(7):689–692.

Mella JR, Guo L, Hung V. Dupuytren's contracture: an evidence-based review. *Ann Plast Surg*. 2018;81(6):S97–S101.

Nordenskjöld J, Englund M, Zhou C, et al. Prevalence and incidence of doctordiagnosed Dupuytren's disease: a population-based study. *J Hand Surg Eur Vol*. 2017;42(7):673–677.

Waldman SD. Dupuytren's contracture. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:263–264.

Waldman SD. Dupuytren contracture injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2017:325–327.

Waldman SD. Dupuytren's contracture. In: *Atlas of common pain syndromes*. 4th ed. Philadelphia: Elsevier; 2019:236–238.

Zhang D, Earp BE, Blazar P. Risk factors for skin tearing in collagenase treatment of Dupuytren contractures. *J Hand Surg Am*. 2019;44(12):1021–1025.

61

Angina Pectoris

ICD-10 CODE 120.9

THE CLINICAL SYNDROME

Angina pectoris is a constellation of symptoms that include substernal chest pain that radiates into one or both upper extremities, shoulders, and into the neck and jaw (Fig. 61.1). Usually occurring with exertion, the pain of angina pectoris is described as a pressure or heaviness in the chest with an associated sensation of tightness or squeezing. Occasionally, angina pectoris will manifest as dyspnea and/or nausea and vomiting in the absence of significant chest pain. The pain of angina pectoris is ill-defined due to the broad number of dermatomes subserving the pain, with C7 to T4 the most prominent, but with contributions to other sensory pathways as evidenced by the frequent radiation of pain associated with angina pectoris into the teeth and lower face which are subserved via the trigeminal nerve. Occasionally, the pain of angina pectoris may occur at rest.

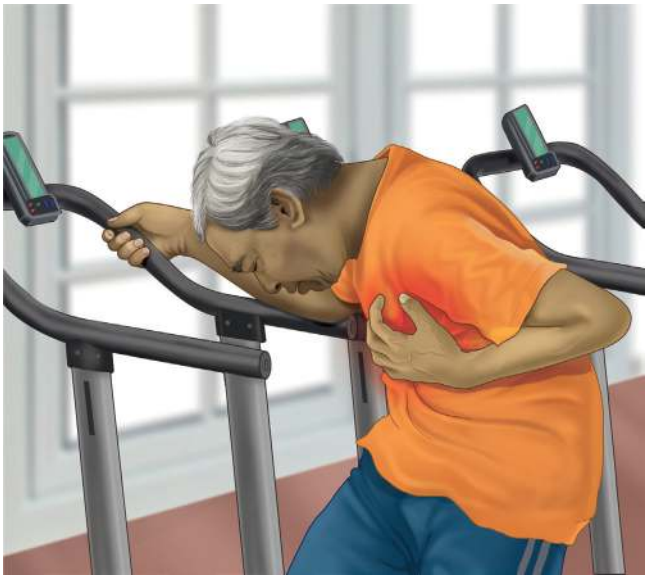

FIG 61.1 The pain of angina pectoris is described as a pressure or heaviness in the chest with an associated sensation of tightness or squeezing.

SIGNS AND SYMPTOMS

The diagnosis of angina pectoris is almost always made on the basis of the patient history rather than the physical examination, as there are no specific or pathognomonic findings that suggest the diagnosis. For this reason, the focus of the physical examination in the patient with a clinical history suggesting of angina pectoris should be to identify comorbidities that may place the patient at increased risk for anginal symptoms. Specific comorbidities would include aortic stenosis, mitral valvular disease, pulmonary disease, acute cholecystitis, peripheral vascular disease, carotid artery disease, to name a few (Figs. 61.2 and 61.3). It should be noted that hyperventilation associated with panic attacks can mimic the presentation of angina pectoris.

TESTING

The purpose of testing in patients suspected of suffering from angina pectoris is twofold: (1) to identify abnormalities that point to cardiac ischemia; and (2) to identify other pathologic processes that may mimic the clinical presentation of angina pectoris. Electrocardiograms may show ST segment depression suggesting cardiac ischemia or acute ST segment elevation suggesting myocardial infarction (Figs. 61.4 and 61.5). Cardiac enzyme testing will be useful in identifying myocardial ischemia, and troponin levels will identify myocardia injury. Chest radiography will help rule out intrathoracic diseases that may mimic angina pectoris such as pneumonia and pneumothorax as well as diseases below the diaphragm that may cause a pleural effusion such as acute pancreatitis (Fig. 61.6). Cardiac stress testing and echocardiography and stress cardiac magnetic resonance imaging may help clarify the diagnosis in questionable cases but should be used with caution in patients with significant anginal symptoms (Fig. 61.7). Coronary angiography may be necessary to define the extent of coronary artery disease responsible for the patient's pain symptomatology.

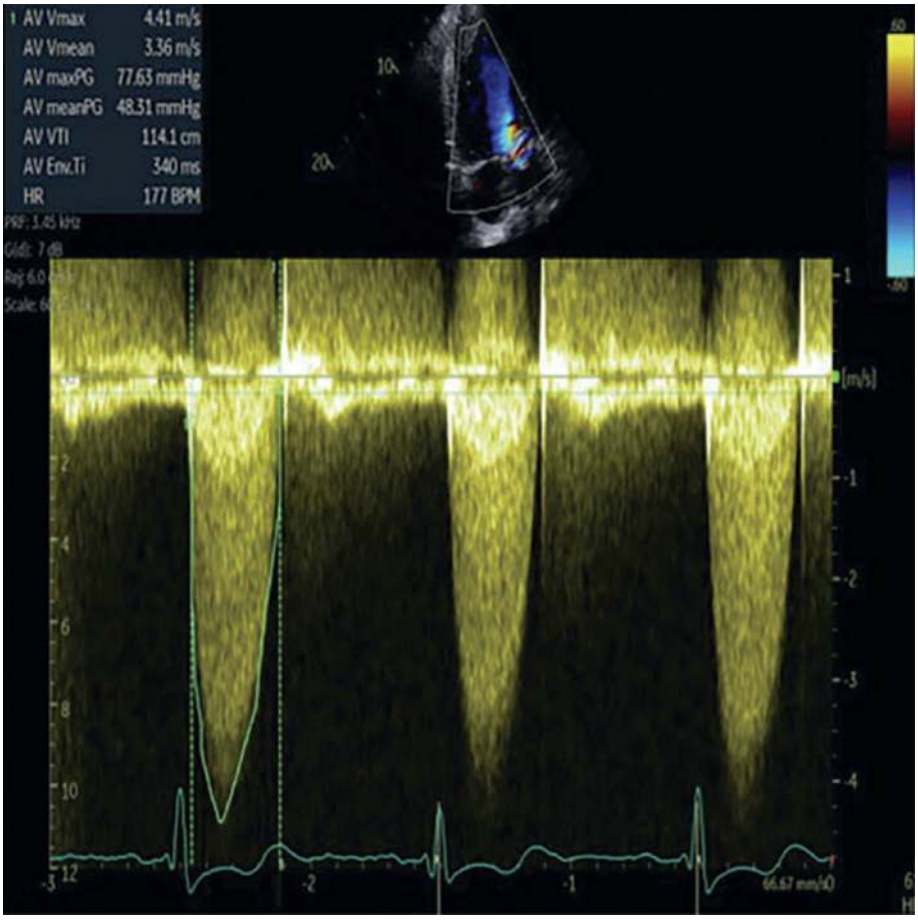

FIG 61.2 Severe aortic stenosis on transthoracic echocardiography: aortic bioprosthetic stenosis with a mean pressure gradient of 48 mm Hg. (From Jdaidani J, Iskandarani DZ, Chaabo O, et al. Iatrogenic aortic stenosis during a case of aortic paravalvular leak closure. *Struct Heart*. 2022:100022.)

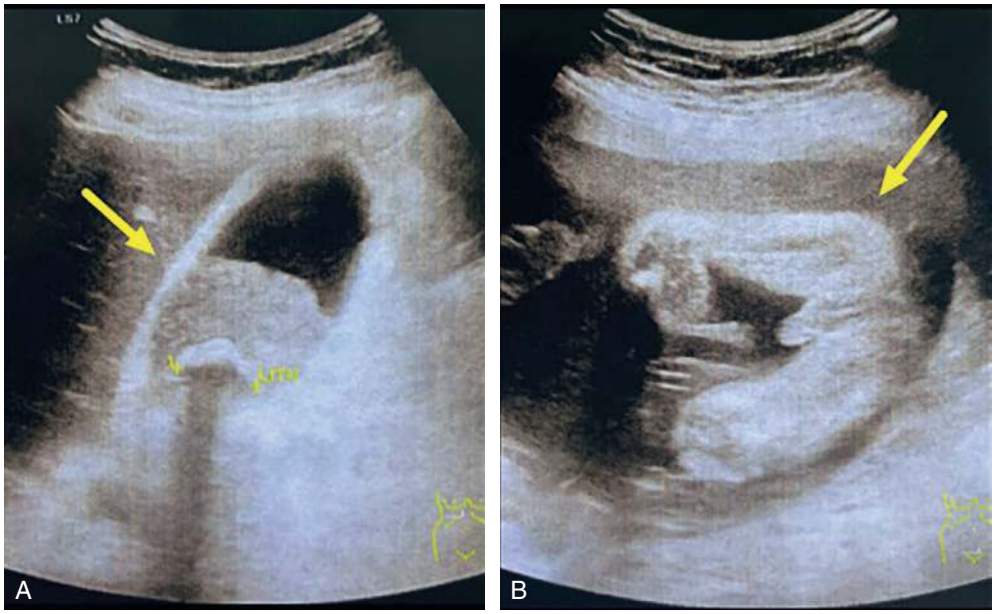

FIG 61.3 Acute cholecystitis mimicking angina pectoris. Abdominal ultrasound: **A,** thickened gallbladder wall with an accumulation of fluid inside gallbladder lumen and posterior acoustic shadow suggesting of gallbladder inflammation and gallbladder stone; **B,** fetus located inside the uterus. (From Handaya AY, Fauzi AR, Andrew J, et al. Management of gallstone-induced severe acute cholecystitis and pancreatitis in the second trimester of pregnancy during covid-19 pandemic: a case report. *Ann Med Surg*. 2021;68:102563.)

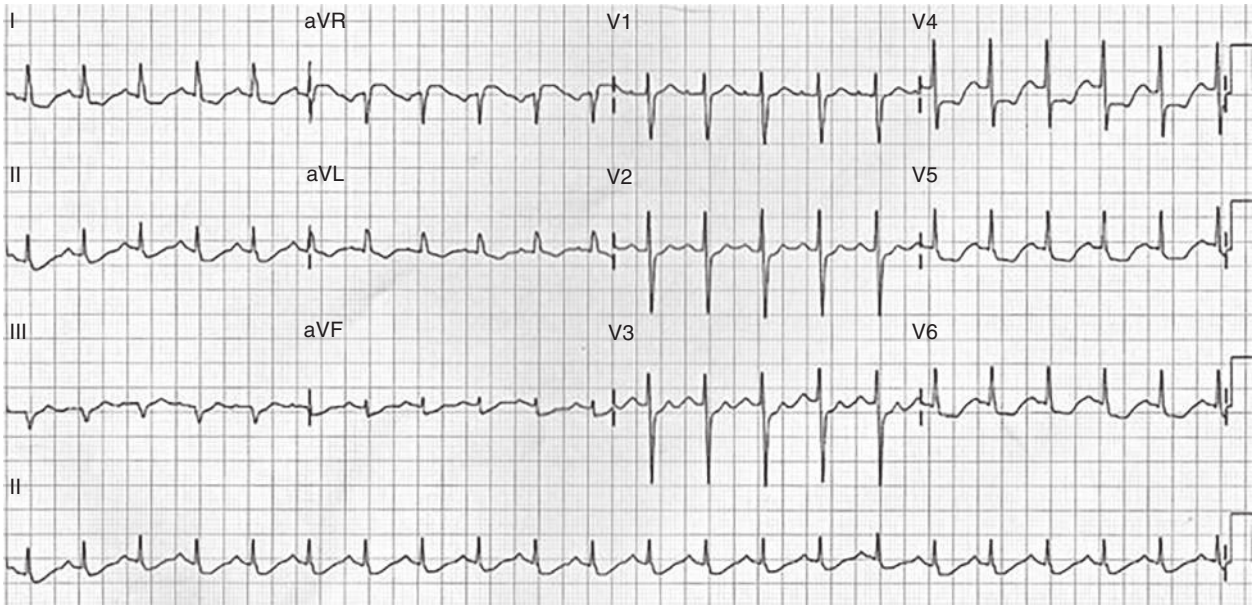

FIG 61.4 Electrocardiogram demonstrating ST segment depression consistent with myocardial ischemia in patient with angina.

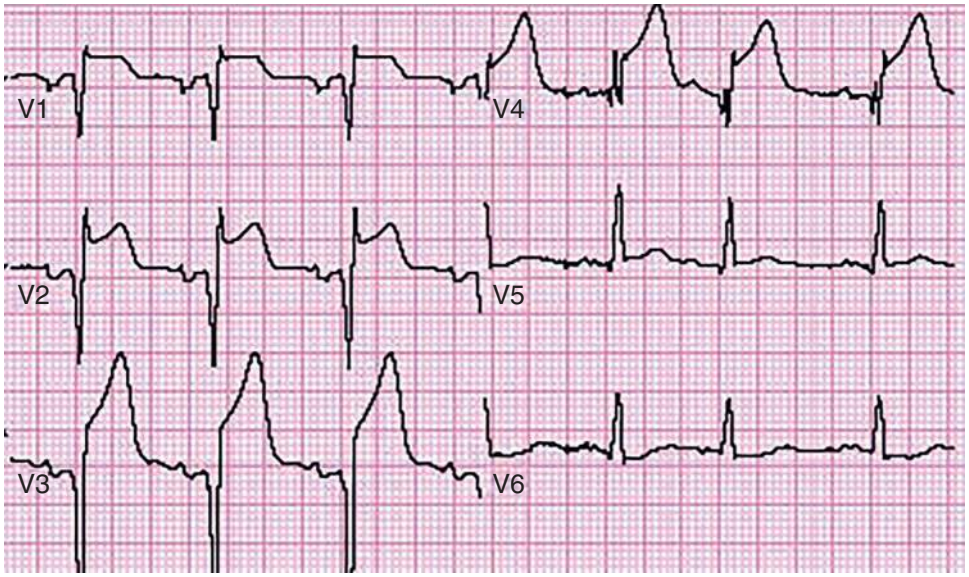

FIG 61.5 Electrocardiogram demonstrating a STEMI. *STEMI,* ST segment elevation suggesting myocardial infarction.

DIFFERENTIAL DIAGNOSIS

Because the classic triad of substernal chest pressure, occurring with exertion, and pain radiating into the left upper extremity does not occur in many patients with angina pectoris, a high index of suspicion for the disease is indicated combined with a heightened awareness of diseases that may mimic angina pectoris, for example, pericarditis and pulmonary embolus (Box 61.1).

TREATMENT

Prehospital treatment of patients suspected of suffering from angina pectoris includes the administration of nitroglycerin, aspirin, and oxygen. Emergency room care should include the administration of morphine and beta blockers, and if the presence of abnormal markers suggestive of myocardial injury is identified, for example, elevated troponin or creatine phosphokinase - myocardial band (CPK-MB) level, and there

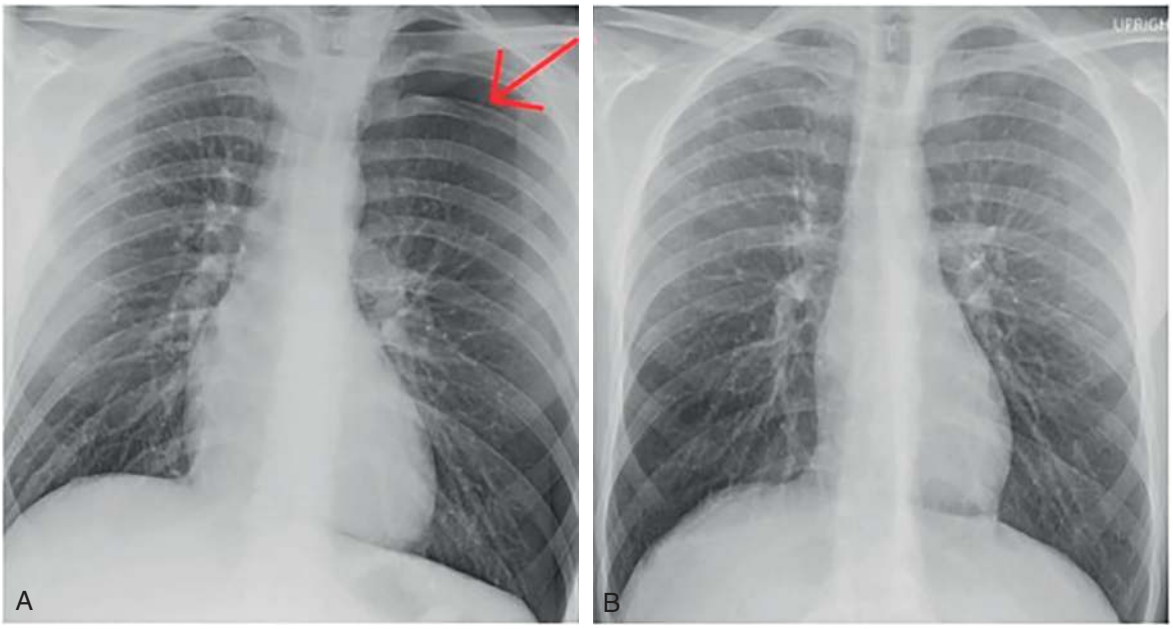

FIG 61.6 Spontaneous pneumothorax may mimic angina pectoris. Chest X-rays showing pneumothoraces upon presentation **(A)** and resolution of pneumothoraces at the time of discharge **(B)**. (From Ashraf O, Nasrullah A, Karna R, Alhajhusain A. Vaping associated spontaneous pneumothorax—a case series of an enigmatic entity! *Respir Med Case Rep*. 2021;34:101535.)

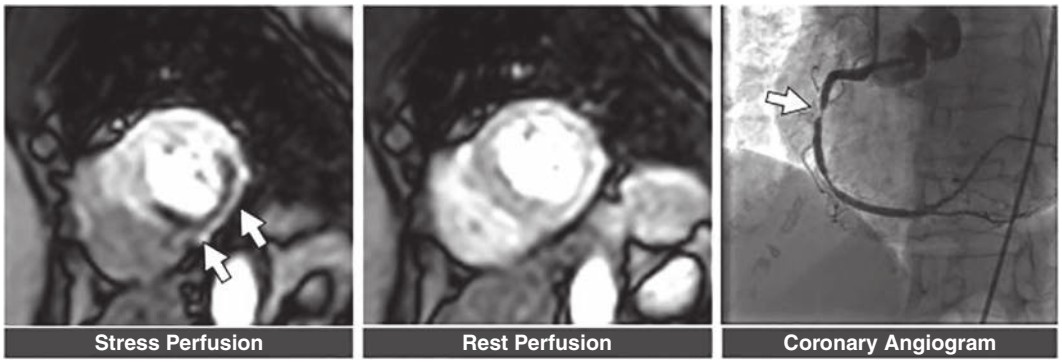

FIG 61.7 Abnormal stress cardiac magnetic resonance perfusion. (**Left**) Perfusion defect in mid-ventricular short axis slice denoted by *arrows*. (**Middle**) Normal perfusion noted in same slice position during resting conditions. (**Right**) Severe right coronary artery stenosis corresponding to perfusion defect. (From Patel AR, Salerno M, Kwong RY, et al. Stress cardiac magnetic resonance myocardial perfusion imaging: JACC review topic of the week. *J Am Coll Cardiol*. 2021;78(16):1655–1668.)

is no contraindication, consideration should be given as to the administration of low-molecular-weight heparin, clopidogrel, or other antiplatelet medications including the glycoprotein inhibitors abciximab, tirofiban, and eptifibatide. If pain persists, percutaneous coronary artery angiography and angioplasty or thrombolytics are a reasonable next step.

COMPLICATIONS AND PITFALLS

The failure to promptly diagnose a patient presenting with chest pain can lead to significant unnecessary testing as well as significant morbidity and mortality. Given that many patients suffering from angina pectoris do not present with the classic triad of ischemic heart disease coupled with the fact that many serious diseases can mimic angina pectoris, the potential for diagnostic misadventures remains large.

CLINICAL PEARLS

Almost 10 million Americans experience angina pectoris annually with approximately 500,000 new cases diagnosed each year. In spite of the high incidence of this disease, diagnosis can be difficult. A high index of suspicion is critical to avoid missing this life-threatening diagnosis.

BOX 61.1 Differential Diagnoses of Angina Pectoris

- Abdominal aortic aneurysm
- Acute cholecystitis and biliary colic
- Acute gastritis
- Acute pancreatitis
- Acute pericarditis
- Anxiety disorders
- Aortic dissection
- Aortic stenosis
- Boerhaave syndrome
- Cholecystitis
- Cocaine toxicity
- Coronary artery anomalies
- Coronary artery atherosclerosis
- Coronary artery vasospasm
- Esophageal spasm
- Gastroesophageal reflux disease
- Herpes zoster
- Hiatal hernia
- Hyperventilation syndrome
- Kawasaki disease
- Mediastinitis
- Mitral regurgitation
- Mitral valve prolapse
- Panic disorder
- Peptic ulcer disease
- Pericardial effusion
- Pleurodynia
- Pneumonia
- Pneumothorax
- Polyarteritis nodosa
- Pulmonary embolism (PE)
- Takayasu arteritis
- Thyroid storm

SUGGESTED READINGS

Al-Mohaissen MA. Echocardiographic assessment of primary microvascular angina and primary coronary microvascular dysfunction. *Trends Cardiovasc Med*. 2022 Feb 19; S1050–1738(22)00036–6. https://doi.org/10.1016/j.tcm.2022.02.007.

Gallone G, Baldetti L, Tzanis G, et al. Refractory angina: from pathophysiology to new therapeutic nonpharmacological technologies. *JACC Cardiovasc Interv*. 2020;13(1):1–19. https://doi.org/10.1016/j.jcin.2019.08.055.

Madeira S, Brízido C, Raposo L, et al. Non-pharmacological treatment of refractory angina: the coronary sinus reducer, the new kid on the block. *Rev Port Cardiol (English Ed)*. 2021;40(5):371–382. https://doi.org/10.1016/j.repce.2020.09.003.

Makowski M, Samanta Makowska J, Zielińska M. Refractory angina—unsolved problem. *Cardiol Clin*. 2020;38(4):629–637. https://doi.org/10.1016/j.ccl.2020.07.009.

Manolis AJ, Boden WE, Collins, et al. State of the art approach to managing angina and ischemia: tailoring treatment to the evidence. *Eur J Intern Med*. 2021;92:40–47. https://doi.org/10.1016/j.ejim.2021.08.003.

Vargas K, Messman A, Levy PD. Nuances in the evaluation of chest pain in women. *JACC Case Rep*. 2021;3(17):1793–1797. https://doi.org/10.1016/j.jaccas.2021.07.035.

62

Costosternal Joint Pain

ICD-10 CODE R07.9

THE CLINICAL SYNDROME

Many patients with noncardiogenic chest pain suffer from costosternal joint pain. Most commonly, the costosternal joints become painful in response to inflammation as a result of overuse or misuse or in response to trauma secondary to acceleration–deceleration injuries or blunt trauma to the chest wall (Fig. 62.1). With severe trauma, the joints may subluxate or dislocate. The costosternal joints are susceptible to the development of osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, and psoriatic arthritis. The joints are also subject to invasion by tumor from primary malignant tumors, including thymoma, or from metastatic disease.

SIGNS AND SYMPTOMS

Physical examination reveals that the patient vigorously attempts to splint the joints by keeping the shoulders stiffly in a neutral position. Pain is reproduced with active protraction or retraction of the shoulder, deep inspiration, and full elevation of the arm. Shrugging off the shoulder may also reproduce the pain. Coughing may be difficult, leading to inadequate pulmonary toilet in patients who have sustained trauma to the anterior chest wall. The costosternal joints and adjacent intercostal muscles may be tender to palpation and the patient may exhibit a positive shoulder retraction test (Figs. 62.2 and 62.3). The patient may also complain of a clicking sensation with joint movement.

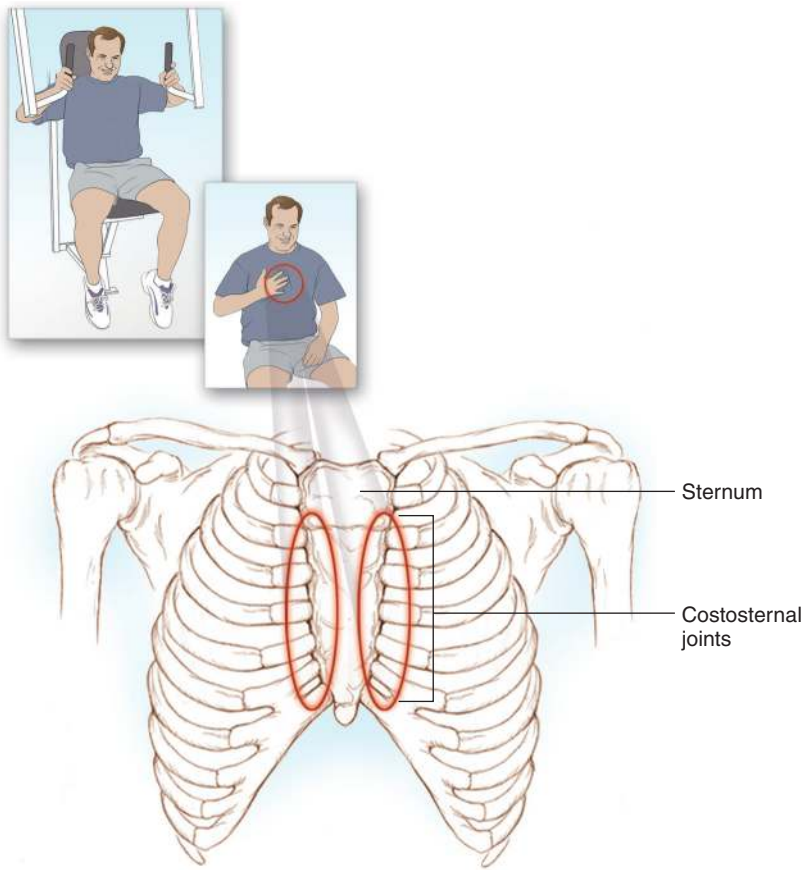

FIG 62.1 Irritation of the costosternal joints from overuse of exercise equipment can cause costosternal syndrome.

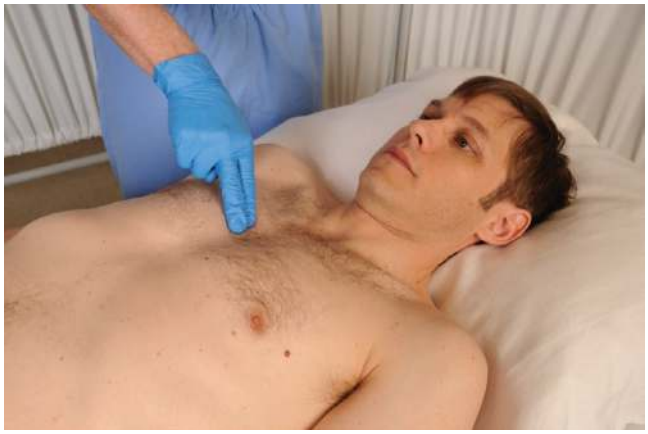

FIG 62.2 Palpation of the costosternal joint.

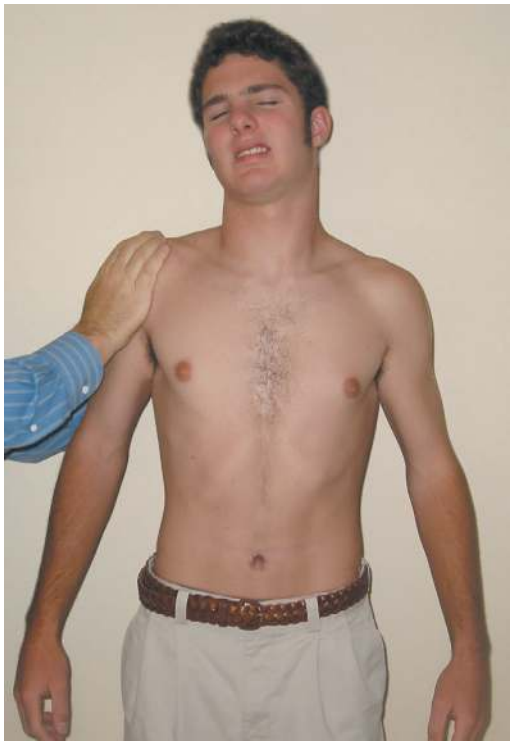

FIG 62.3 To elicit a shoulder retraction test in patients who are suspected of suffering from costosternal syndrome, the patient is placed in the standing position with the shoulders in neutral position, facing the examiner. The patient is then asked to retract the shoulder vigorously. The shoulder retraction test is considered positive if the retraction maneuver reproduces the patient's anterior chest wall pain. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2021: Fig. 143-1.)

TESTING

Plain radiographs are indicated for all patients who present with pain that is thought to be emanating from the costosternal joints, to rule out occult bony disorders, including tumor (Fig. 62.4). If trauma is present, radionuclide bone scanning may be useful to exclude occult fractures of the ribs or sternum. Based on the patient's clinical presentation, additional

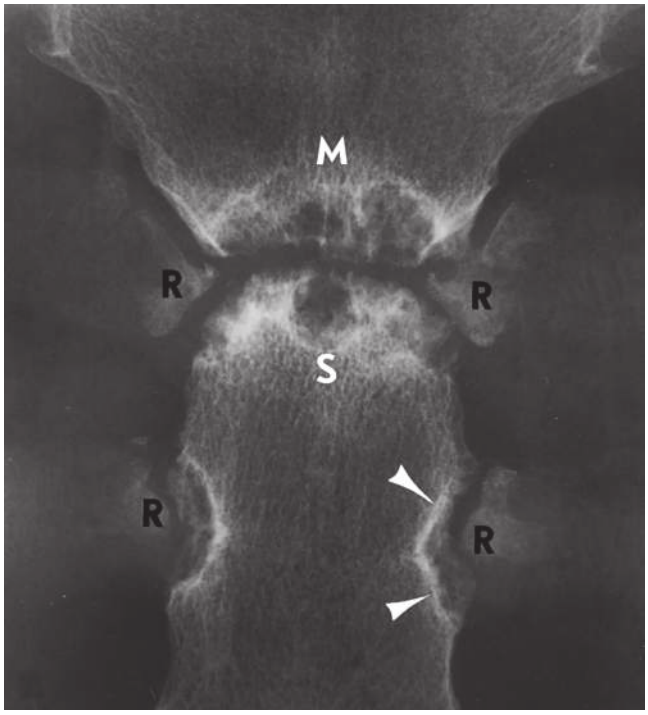

FIG 62.4 Abnormalities of manubriosternal and sternocostal joints in rheumatoid arthritis. Radiograph of a sternum from a cadaver with rheumatoid arthritis shows large erosions of the articular surface of both the manubrium *(M)* and the body of the sternum *(S)*. Subtle irregularities of the second and third sternocostal joints are evident, most prominently in the sternal facet of the left third sternocostal joint *(arrowheads)*. *R*, Ossified costal cartilage. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:854.)

testing may be indicated, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Laboratory evaluation for collagen vascular disease is indicated in patients suffering from costosternal joint pain if other joints are involved. Computerized tomography, magnetic resonance, and ultrasound imaging of the joints are indicated if joint instability or occult mass is suspected or to elucidate the cause of the pain further (Figs. 62.5–62.7). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

As mentioned, the pain of costosternal syndrome is often mistaken for pain of cardiac origin, and it leads to visits to the emergency department and unnecessary cardiac workups (Box 62.1). If trauma has occurred, costosternal syndrome may coexist with fractured ribs or fractures of the sternum itself, which can be missed on plain radiographs and may require radionuclide bone scanning for proper identification (Fig. 62.8). Tietze's syndrome, which is painful enlargement of the upper costochondral cartilage associated with viral infection, may be confused with costosternal syndrome.

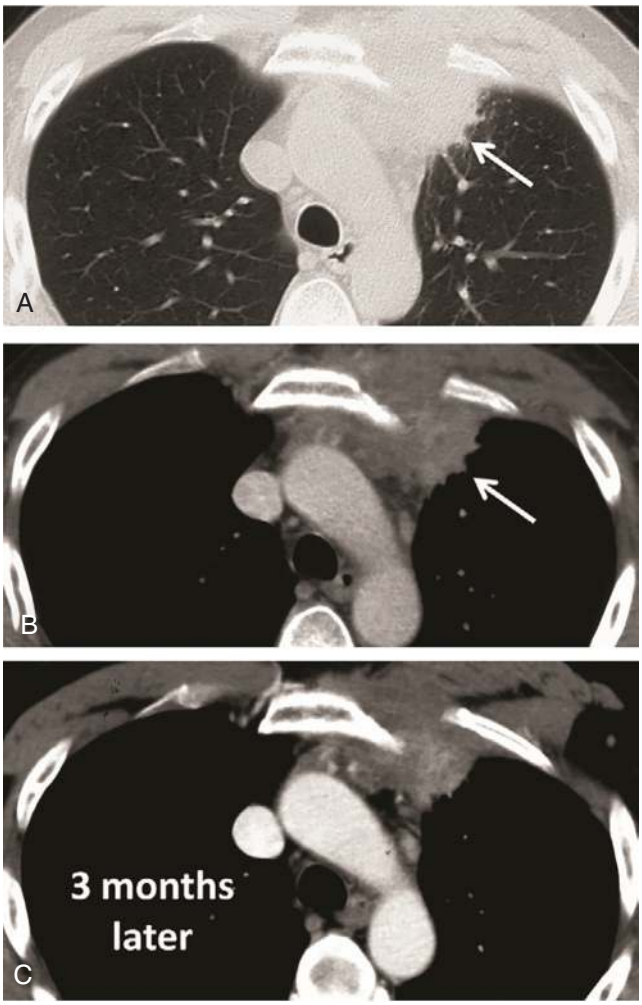

FIG 62.5 Computed tomography (CT) scan of a patient with anterior chest wall pain. CT scanning of anterior upper mediastinal mass after iodinated contrast administration. Lung window (**A**). Mediastinum window (**B**) shows inhomogeneous contrast enhancement of the mass. Enhanced multidetector CT follow-up after 3 months (**C**) shows decrease in volume of mediastinal mass. (From De Filippo M, Albini A, Castaldi V, et al. MRI findings of Tietze's syndrome mimicking mediastinal malignancy on MDCT. *Eur J Radiol Extra*. 2008;65(1):33–35.)

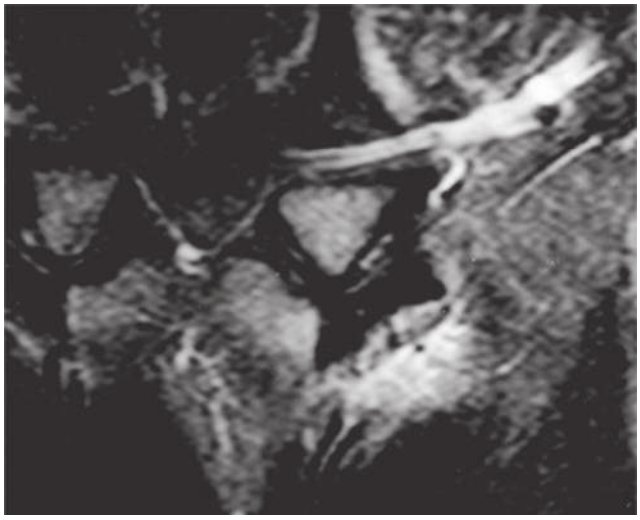

FIG 62.6 Magnetic resonance imaging of Tietze's syndrome. A coronal short tau inversion recovery (STIR) magnetic resonance (MR) image of the thorax, showing high-intensity signal at the costosternal joint. (From Resnick D, ed. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:2605.)

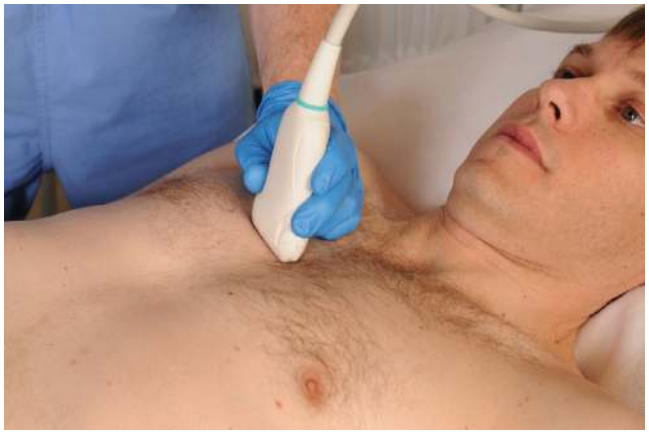

FIG 62.7 Proper placement of the high-frequency linear ultrasound probe for ultrasound evaluation of the costosternal joint.

BOX 62.1 Musculoskeletal Chest Wall Pain

Pain Arising From the Joints of the Chest Wall
Strain of the costosternal joint
Manubriosternal arthritis
Tietze syndrome
Costochondritis
Xiphodynia
Costovertebral joint disorders
Septic arthritis

Pain Arising From the Ribs
Rib trauma
Rib fracture
Primary and metastatic neoplasm of the rib infection
Slipping rib syndrome

Pain Arising From the Soft Tissues
Myositis
Muscle strain
Fibromyalgia
Myofascial pain

Miscellaneous Sources of Pain
Precordial catch syndrome
Acute herpes zoster
Zoster sine herpete
Somatoform disorders

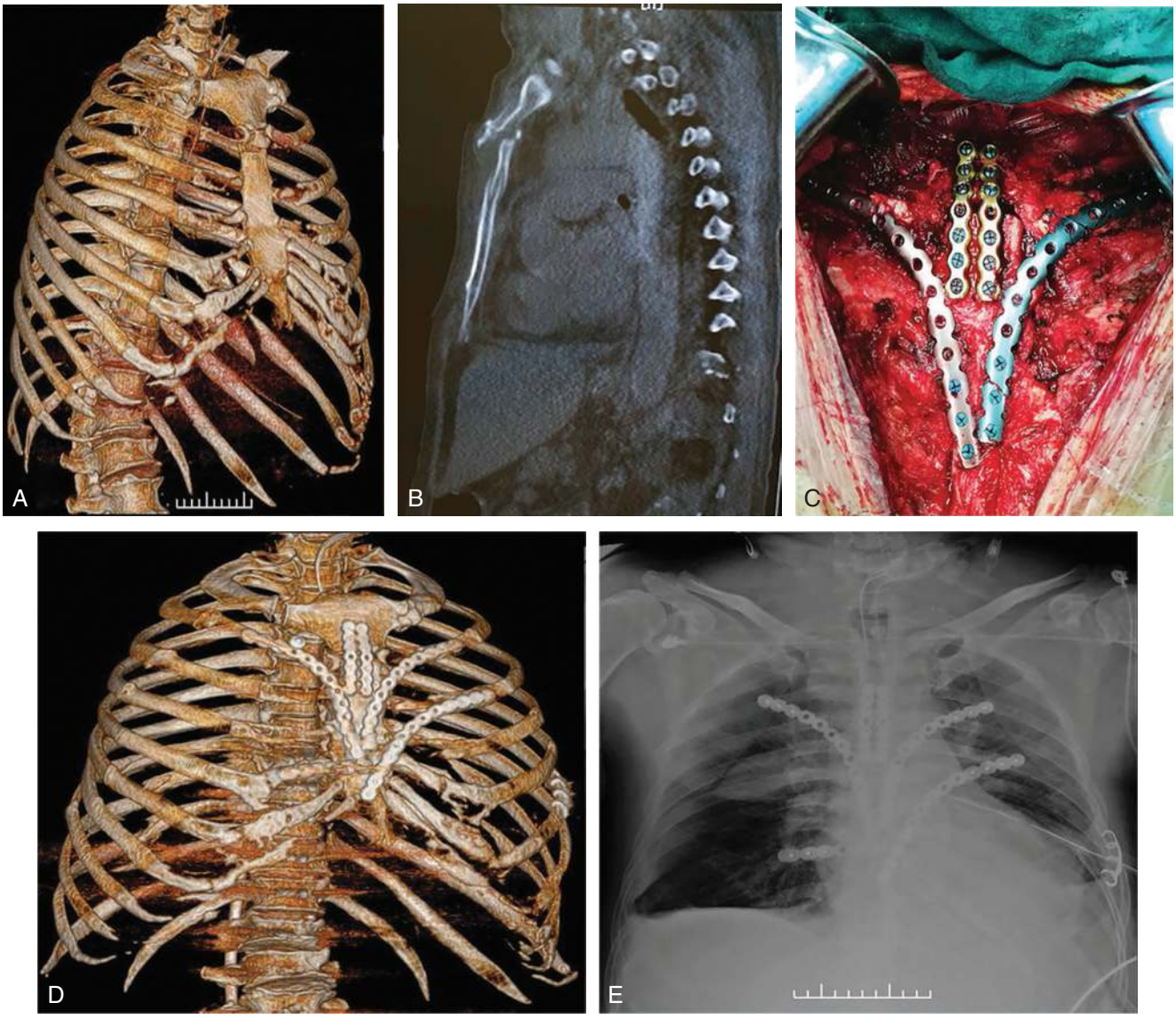

FIG 62.8 Crush injury of the chest with massive chest wall trauma. **A,** Computed tomographic (CT) three-dimensional reconstruction image of the chest wall on admission. **B,** CT image of the sternum. **C,** Surgical procedure: fixation of sternal and multiple costal cartilage fractures. **D,** CT three-dimensional reconstruction image of the chest wall after operation. **E,** Chest film obtained 4 days after operation. (From Gao E, Li Y, Zhao T, et al. Simultaneous surgical treatment of sternum and costal cartilage fractures. *Ann Thorac Surg.* 2019;107(2):e119–e120.)

Neuropathic pain involving the chest wall may also be confused or coexist with costosternal syndrome. Examples of such neuropathic pain syndromes include diabetic polyneuropathies and acute herpes zoster involving the thoracic nerves. Diseases of the structures of the mediastinum are possible and may be difficult to diagnose. Pathologic processes that inflame the pleura, such as pulmonary embolus, infection, and Bornholm disease, may also confuse the diagnosis and complicate treatment.

TREATMENT

Initial treatment of the pain and functional disability associated with costosternal syndrome includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors. The local application of heat and cold may also be beneficial. The use of an elastic rib belt may provide symptomatic relief and protect the costosternal joints from additional trauma. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

Intraarticular injection of the costosternal joint is performed by placing the patient in the supine position. The skin overlying the affected costosternal joints is prepared with antiseptic solution. A sterile syringe containing 1 mL of 0.25% preservative-free bupivacaine for each joint to be injected and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The costosternal joints

FIG 62.9 Proper needle placement for ultrasound-guided injection of the costosternal joint.

are identified; they should be easily palpable as a slight bulging at the point where the rib attaches to the sternum. The needle is then carefully advanced through the skin and subcutaneous tissues medially, with a slight cephalad trajectory, into proximity with the joint (Fig. 62.9). If bone is encountered, the needle is withdrawn from the periosteum. After the needle is in proximity to the joint, 1 mL of solution is gently injected. The clinician should feel limited resistance to injection. If significant resistance is encountered, the needle should be withdrawn slightly until the injection can proceed with only limited resistance. This procedure is repeated for each affected joint. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance may improve the safety and accuracy of needle placement when injecting the costosternal joint.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for costosternal joint pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Because many pathologic processes can mimic the pain of costosternal syndrome, the clinician must carefully rule out underlying cardiac disease and diseases of the lung and structures of the mediastinum. Failure to do so could lead to disastrous results. The major complication of the injection technique is pneumothorax if the needle is placed too laterally or deeply and invades the pleural space. Infection, although rare, can occur if strict aseptic technique is not followed. Trauma to the contents of the mediastinum is also a possibility. The risk of this complication can be greatly decreased with strict attention to accurate needle placement.

CLINICAL PEARLS

Patients suffering from pain emanating from the costosternal joints often believe that they are having a heart attack. Reassurance is required, but the clinician should remember that musculoskeletal pain syndromes and coronary artery disease can coexist. Tietze's syndrome may be confused with costosternal syndrome, although both respond to the aforementioned injection technique.

SUGGESTED READINGS

Ayloo A, Cvengros T, Marella S. Evaluation and treatment of musculoskeletal chest pain. *Prim Care*. 2013;40(4):863–887.

Hillen TJ, Wessell DE. Multidetector CT scan in the evaluation of chest pain of nontraumatic musculoskeletal origin. *Thorac Surg Clin*. 2010;20(1):167–173.

Lu CH, Hsieh SC, Li KJ. Tophi in anterior chest wall. *Joint Bone Spine*. 2014;81(4):366.

Stochkendahl MJ, Christensen HW. Chest pain in focal musculoskeletal disorders. *Med Clin North Am*. 2010;94(2):259–273.

Waldman SD. Costosternal joint injection. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:462–463.

Waldman SD. Ultrasound-guided injection technique for costosternal joint pain. In: *Waldman's comprehensive atlas of ultrasound guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:591–594.

Waldman SD. Costosternal joint injection for Tietze syndrome. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:450–455.

Manubriosternal Syndrome

ICD-10 CODE R07.2

THE CLINICAL SYNDROME

The manubrium articulates with the body of the sternum by way of the manubriosternal joint at the angle of Louis. The manubriosternal joint is a fibrocartilaginous joint or synchondrosis, which lacks a true joint cavity. The joint allows protraction and retraction of the thorax.

Pain originating from the manubriosternal joint can mimic pain of cardiac origin. The manubriosternal joint is susceptible to the development of osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, and psoriatic arthritis. The joint can also be traumatized during acceleration–deceleration injuries and blunt trauma to the chest (Figs. 63.1 and 63.2). With severe trauma, the joint may subluxate or dislocate. Overuse or misuse can result in acute inflammation of the manubriosternal joint, which can be quite debilitating. The joint is also subject to invasion by tumor from primary malignant tumors, including thymoma, or from metastatic disease. Rarely, septic arthritis of the manubriosternal joint can occur (Fig. 63.3).

SIGNS AND SYMPTOMS

Physical examination reveals that the patient vigorously attempts to splint the joint by keeping the shoulders stiffly in a neutral position. Pain is reproduced with active protraction or retraction of the shoulder, deep inspiration, and full elevation of the arm. Shrugging off the shoulder may also

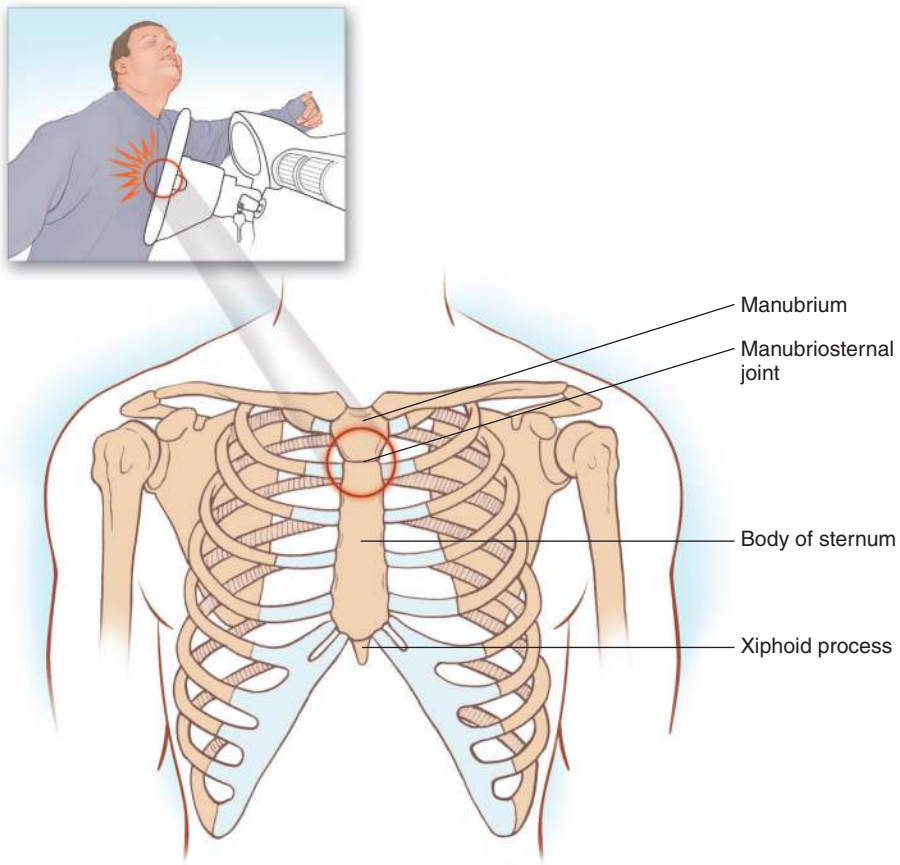

FIG 63.1 The manubriosternal joint is susceptible to the development of arthritis. It is often traumatized during acceleration–deceleration injuries and blunt trauma to the chest.

reproduce the pain. Coughing may be difficult, leading to inadequate pulmonary toilet in patients who have sustained trauma to the anterior chest wall. The manubriosternal joint may be tender to palpation. The patient may also complain of a clicking sensation with movement of the joint.

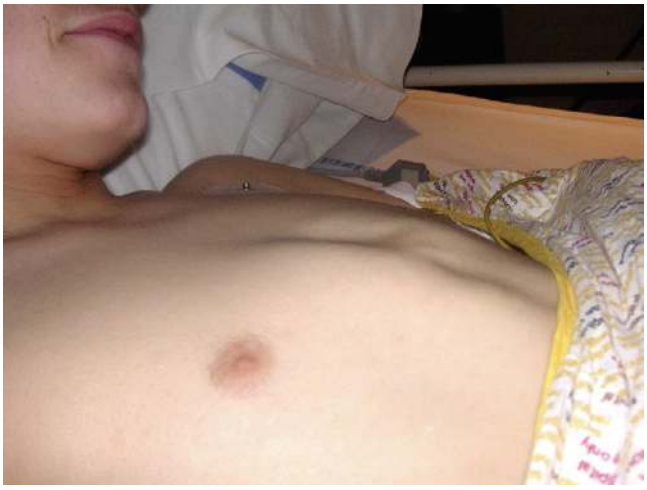

FIG 63.2 Photograph showing an obvious step-off in the manubriosternal joint following dislocation. (From Lyons I, Saha S, Arulampalam T. Manubriosternal joint dislocation: an unusual risk of trampolining. *J Emerg Med*. 2010;39:596–598.)

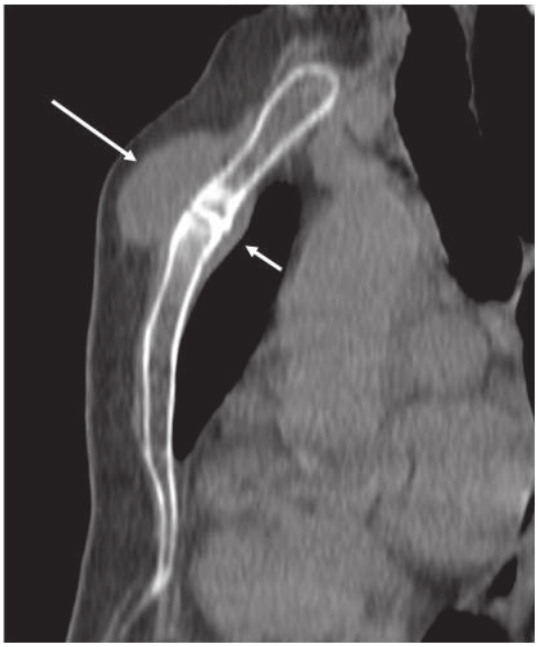

FIG 63.3 Sagittal reformat image of the sternum in mediastinal window demonstrates a more prominent soft-tissue presternal mass *(long arrow)* and a smaller posterior mass *(short arrow)* centered on the manubriosternal joint. (From Gorospe L, Ayala-Carbonero AM, Rodríguez-Díaz R, Latorre RG, Muñoz-Molina GM, Cabañero-Sánchez A. Tuberculosis of the manubriosternal joint and concurrent asymptomatic active pulmonary tuberculosis in a patient presenting with a chest wall mass. *Clin Imaging*. 2015;39(2):311–314.)

TESTING

Plain radiographs are indicated for all patients who present with pain thought to be emanating from the manubriosternal joint, to rule out occult bony disorders, including tumor. If trauma is present, radionuclide bone scanning may be useful to exclude occult fractures of the ribs or sternum. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Laboratory evaluation for collagen vascular disease is indicated in patients suffering from manubriosternal joint pain if other joints are involved. Magnetic resonance imaging, ultrasound imaging, and/or computed tomography (CT) of the joint is indicated if joint instability, infection, or occult mass is suspected or to further elucidate the cause of the pain (Figs. 63.4–63.6). The use of multidetector CT for patients presenting to the emergency room with acute chest pain has led to more rapid and accurate diagnosis of chest wall pain syndromes. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

As mentioned, the pain of manubriosternal syndrome is often mistaken for pain of cardiac origin, and it leads to visits to the emergency department and unnecessary cardiac workups. If trauma has occurred, manubriosternal syndrome may coexist with fractured ribs or fractures of the sternum itself, which can be missed on plain radiographs and may require radionuclide bone scanning for proper identification. Tietze's syndrome, which is painful enlargement of the upper

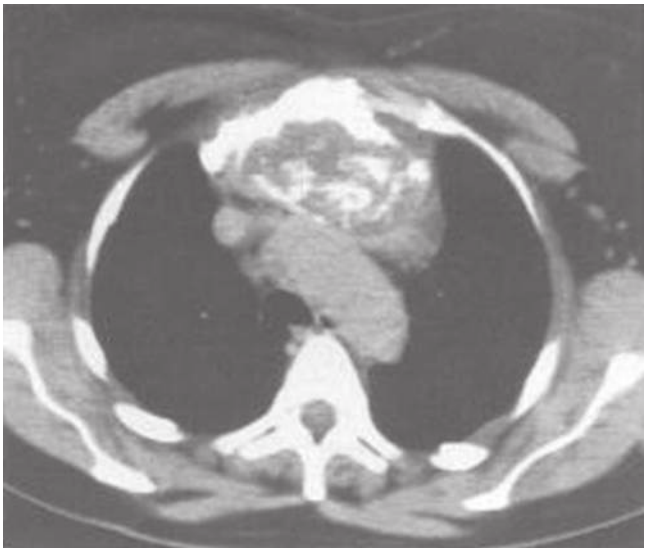

FIG 63.4 Chondrosarcoma of the sternum. Computed tomography clearly demonstrates manubrial irregularity and a preaortic soft tissue mass with chondral calcification. Nearly all sternal tumors are malignant. (From Grainger RG, Allison DJ, Adam A, Dixon AK. *Grainger & Allison's diagnostic radiology: a textbook of medical imaging*. 4th ed. Philadelphia: Churchill Livingstone; 2002:253.)

costochondral cartilage associated with viral infection, may be confused with manubriosternal syndrome.

Neuropathic pain involving the chest wall may also be confused or coexist with manubriosternal syndrome. Examples of such neuropathic pain syndromes include diabetic polyneuropathies and acute herpes zoster involving the thoracic nerves. Diseases of the structures of the mediastinum are possible and can be difficult to diagnose. Pathologic processes that inflame the pleura, such as pulmonary embolus, infection, and Bornholm disease, may also confuse the diagnosis and complicate treatment.

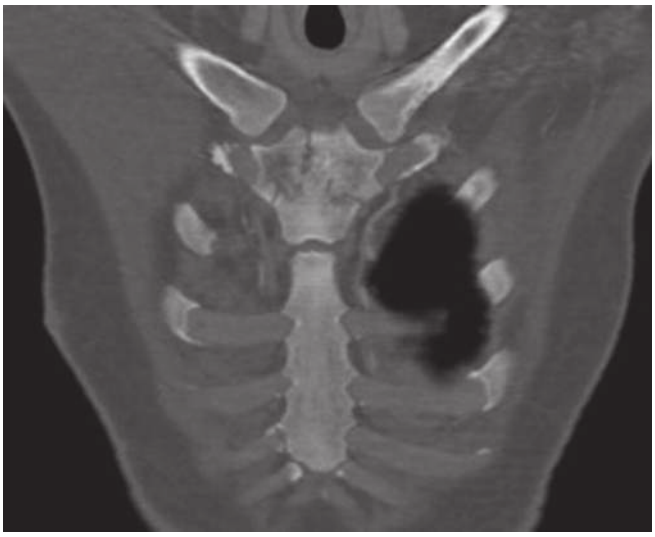

FIG 63.5 Pulmonary embolus (PE) protocol multidetector computed tomography (MDCT) examination for acute chest pain in a patient with lung cancer metastatic to the manubrium with a pathologic fracture. The patient had a known history of lung cancer, and the MDCT examination was obtained to evaluate for suspected PE. Although the presence of the lesion was evident on the straight transverse (*axial*) images, the extent of the lesion and the associated pathologic fracture are better elucidated on the coronal reformats. (From Hillen TJ, Wessell DE. Multidetector CT scan in the evaluation of chest pain of nontraumatic musculoskeletal origin. *Thorac Surg Clin*. 2010;20(1):167–173.)

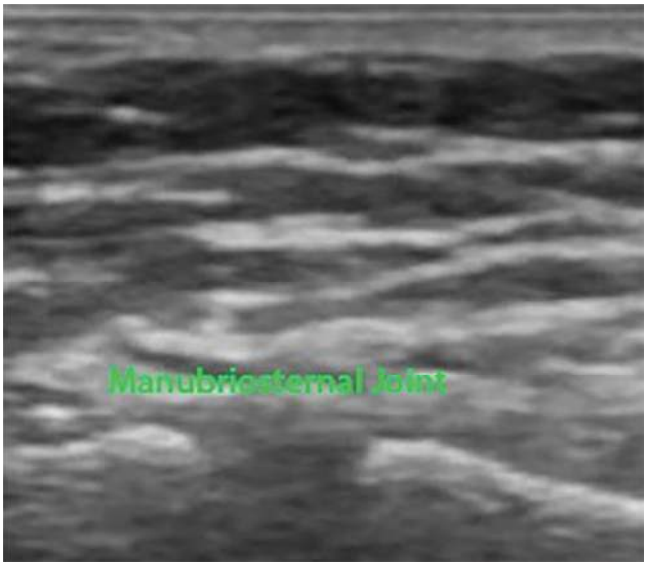

FIG 63.6 Longitudinal ultrasound image of the manubriosternal joint.

TREATMENT

Initial treatment of the pain and functional disability associated with manubriosternal syndrome includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors. The local application of heat and cold may also be beneficial. The use of an elastic rib belt may also provide symptomatic relief and protect the manubriosternal joint from additional trauma. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

The patient is placed in the supine position, and the skin overlying the angle of the sternum is prepared with antiseptic solution. A sterile syringe containing 1 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The angle of the sternum is identified; the manubriosternal joint should be easily palpable as a slight indentation at this point. The needle is then carefully advanced through the skin and subcutaneous tissues medially, with a slight cephalad trajectory, into the joint (Fig. 63.7). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected slightly more cephalad. After the needle enters the joint, the contents of the syringe are gently injected. The clinician should feel some resistance to injection because of the fibrocartilaginous nature of the joint. If significant resistance is encountered, the needle should be advanced or withdrawn slightly into the joint until the injection can proceed with only limited resistance. The needle is

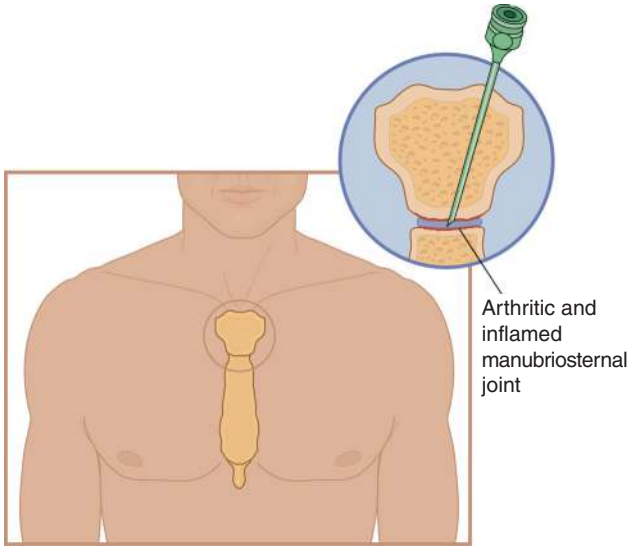

FIG 63.7 Correct needle placement for injection of the manubriosternal joint. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2000.)

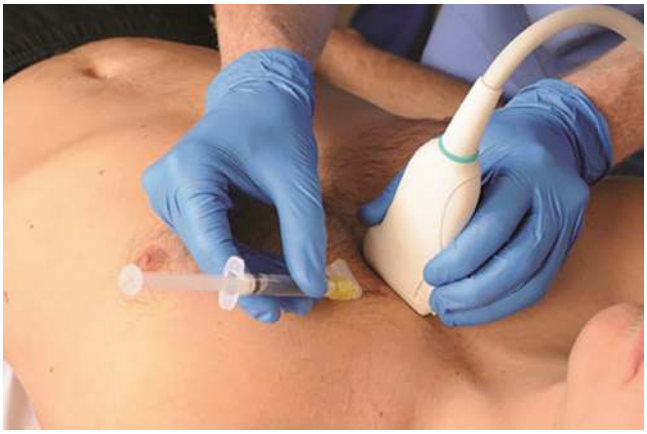

FIG 63.8 Proper needle placement for out-of-plane ultrasound-guided injection of the manubriosternal joint.

then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance may improve the accuracy of needle placement when performing injection of the manubriosternal joint (Fig. 63.8).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for manubriosternal joint pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Because many pathologic processes can mimic the pain of manubriosternal syndrome, the clinician must carefully rule out underlying cardiac disease and diseases of the lung and structures of the mediastinum. Failure to do so could lead to disastrous results. The major complication of the injection technique is pneumothorax if the needle is placed too laterally or deeply and invades the pleural space. Infection, although rare, can occur if strict aseptic technique is not followed. Trauma to the contents of the mediastinum is also a possibility. The incidence of complication can be greatly decreased with strict attention to accurate needle placement.

CLINICAL PEARLS

Patients suffering from pain emanating from the manubriosternal joint often believe that they are having a heart attack. Reassurance is required, but the clinician should remember that musculoskeletal pain syndromes and coronary artery disease can coexist. Tietze's syndrome may be confused with manubriosternal syndrome, although both respond to the aforementioned injection technique.

SUGGESTED READINGS

Carnevale A, Righi R, Maniscalco P, et al. Primary septic arthritis of the manubriosternal joint in an immunocompetent young patient: a case report. *Radiol Case Rep*. 2017;12(4):682–685.

Gorospe L, Ayala-Carbonero AM, Rodríguez-Díaz R, et al. Tuberculosis of the manubriosternal joint and concurrent asymptomatic active pulmonary tuberculosis in a patient presenting with a chest wall mass. *Clin Imaging*. 2015;39(2):311–314.

Kim DK, Kim KH, Park BK, et al. Atypical SAPHO syndrome with isolated manubriosternal inflammation: a multi-image demonstration. *PM&R*. 2016;8(7):716–717.

Kothari M, Saini P, Shethna S, et al. Manubriosternal dislocation with spinal fracture: a rare cause for delayed haemothorax. *Chin J Traumatol*. 2015;18(4):245–248.

Lyons I, Saha S, Arulampalam T. Manubriosternal joint dislocation: an unusual risk of trampolining. *J Emerg Med*. 2010;39(5):596–598.

Sarkeshik AA, Jamal A, Perry PA. Manubriosternal joint dislocation due to blunt force trauma. *Trauma Case Rep*. 2019;21:100187.

Stochkendahl MJ, Christensen HW. Chest pain in focal musculoskeletal disorders. *Med Clin North Am*. 2010;94(2):259–273.

Waldman SD. Arthritis and other abnormalities of the manubriosternal joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:519–525.

Waldman SD. Ultrasound-guided injection technique for the manubriosternal joint. In: *Waldman's comprehensive atlas of ultrasound-guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:595–599.

Waldman SD. Manubriosternal joint pain. In: *Atlas of uncommon pain syndromes*. 4th ed. Philadelphia: Elsevier; 2021:233–236.

Intercostal Neuralgia

ICD-10 CODE G54.8

THE CLINICAL SYNDROME

Whereas most other causes of chest wall pain are musculoskeletal, the pain of intercostal neuralgia is neuropathic. As with costosternal joint pain, Tietze's syndrome, and rib fractures, many patients who suffer from intercostal neuralgia seek medical attention because they believe they are having a heart attack. If the subcostal nerve is involved, gallbladder disease may be suspected. The pain of intercostal neuralgia is the result of damage to or inflammation of the intercostal nerves. The pain is constant and burning, and it may involve any of the intercostal nerves, as well as the subcostal nerve of the twelfth rib. The pain usually begins at the posterior axillary line and radiates anteriorly into the distribution of the affected intercostal or subcostal nerves, or both (Fig. 64.1). Deep inspiration or movement of the chest wall may slightly increase the pain of intercostal neuralgia, but to a much lesser extent than with musculoskeletal causes of chest wall pain.

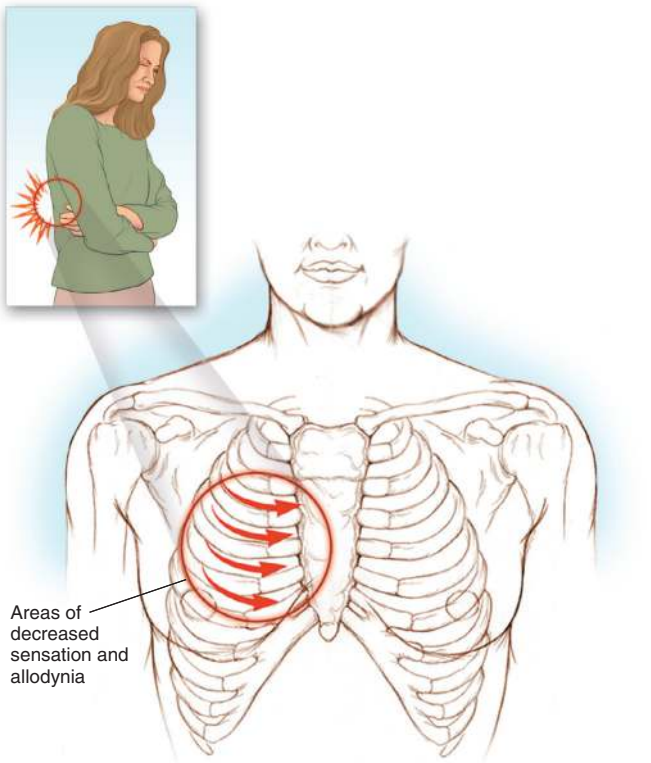

FIG 64.1 The pain of intercostal neuralgia is neuropathic rather than musculoskeletal in origin.

SIGNS AND SYMPTOMS

Physical examination generally reveals minimal findings unless the patient has a history of previous thoracic or subcostal surgery or cutaneous evidence of herpes zoster involving the thoracic dermatomes (Fig. 64.2). Unlike patients with musculoskeletal causes of chest wall and subcostal pain, those with intercostal neuralgia do not attempt to splint or protect the affected area. Careful sensory examination of the affected dermatomes may reveal decreased sensation or allodynia. When motor involvement of the subcostal nerve is significant, the patient may complain that his or her abdomen bulges outward.

TESTING

Plain radiographs are indicated for all patients who present with pain thought to be emanating from the intercostal nerve, to rule out occult bony disorders, including tumor (Fig. 64.3). If trauma is present, radionuclide bone scanning may be useful to exclude occult fractures of the ribs or sternum. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Computed tomography of the thoracic contents is indicated if an occult mass is suspected

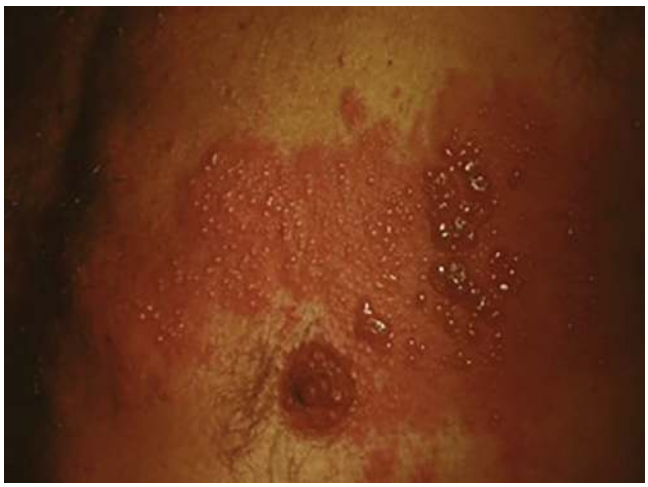

FIG 64.2 Acute herpes zoster of the thoracic dermatome. (From Waldman SD. *Atlas of pain management injection techniques*. 4th ed. St. Louis: Elsevier; 2017: Fig. 106-1.)

(Fig. 64.4). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

As mentioned, the pain of intercostal neuralgia is often mistaken for pain of cardiac or gallbladder origin, and it leads to visits to the emergency department and unnecessary cardiac and gastrointestinal workups (Box 64.1). If trauma has occurred, intercostal neuralgia may coexist with fractured ribs or fractures of the sternum itself, which can be missed on plain radiographs and may require radionuclide bone scanning for proper identification. Tietze's syndrome, which is painful enlargement of the upper costochondral cartilage associated with viral infection, may be confused with intercostal neuralgia.

Other types of neuropathic pain involving the chest wall may be confused or coexist with intercostal neuralgia.

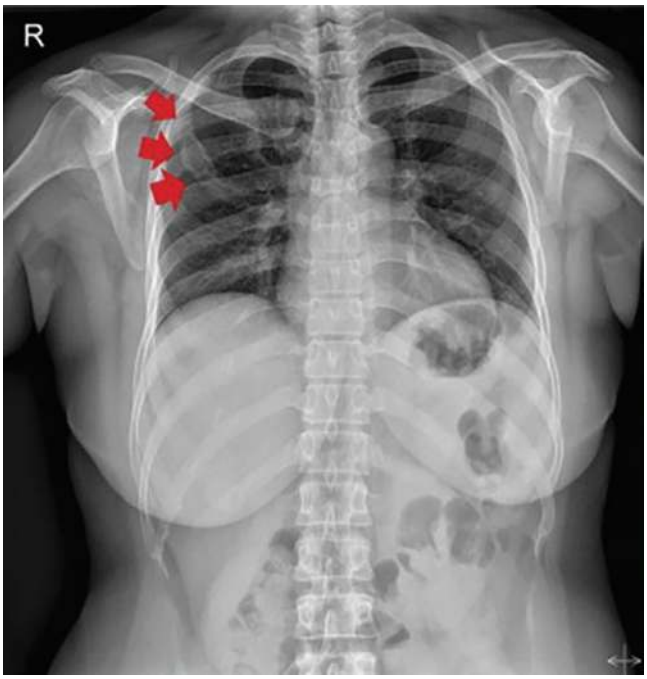

FIG 64.3 Chest roentgenogram showing an expansile and osteolytic lesion (*red arrows*) in the right second rib. (From Sa YJ, Hwang SJ, Sim SB, et al. Cholesterol granuloma: a rare benign rib tumor. *Ann Thor Surg*. 2013;95(5):1801–1803.)

BOX 64.1 Differential Diagnosis of Intercostal Neuralgia

Rib fracture
Rib tumors (primary or metastatic)
Chest wall contusion
Thoracic radiculopathy
Acute herpes zoster
Postherpetic neuralgia
Vertebral compression fracture
Costochondritis
Tietze syndrome
Spondylitis
Pleurisy
Nephrolithiasis
Pyelonephritis
Pulmonary embolism
Pneumothorax
Cholecystitis
Esophageal disorders
Peptic ulcer disease
Referred cardiac pain
Referred gastrointestinal pain
Referred pulmonary pain

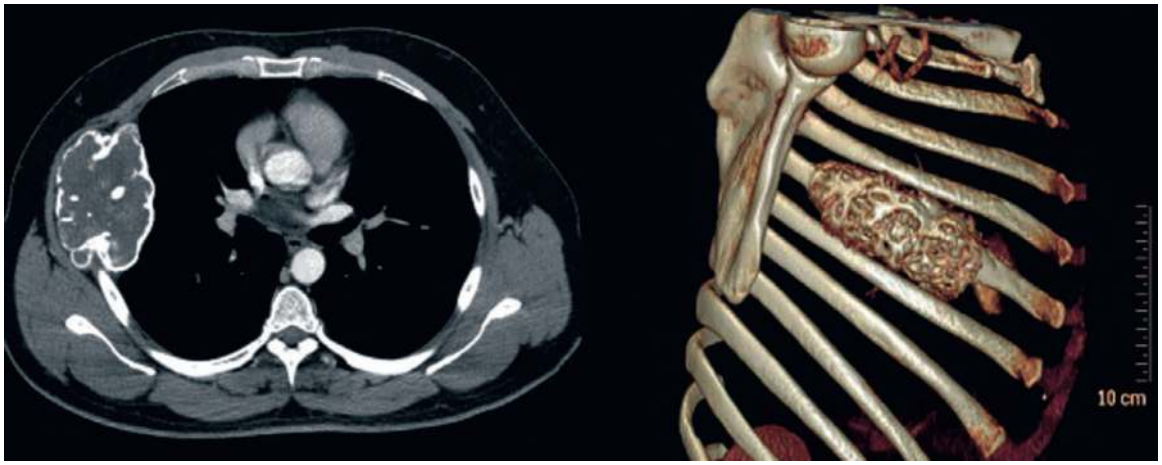

FIG 64.4 Right chest wall mass approximately 11 cm in diameter with slightly rounded morphology, which depends on the anterior segment of the fifth rib. The profile of the mass is relatively sharp, and it has linear and anfractuous punctate calcifications inside. The rest of the content has homogenous attenuation. There was no soft tissue component, although the tumor displaces the serratus anterior muscle and pleural folds. There was no pleural effusion or mediastinal lymphadenopathy. (From Rambalde E, Parra A, Santapau A, et al. SPECT/CT with 99mTc-MDP in a patient with monostotic fibrous dysplasia of the rib. *Rev Esp Med Nucl Imag Mol*. 2013;32(2):126–127)

Examples of such neuropathic pain syndromes include diabetic polyneuropathies and acute herpes zoster involving the thoracic nerves. Diseases of the structures of the mediastinum and thoracic aorta are possible and can be difficult to diagnose (Fig. 64.5). Pathologic processes that inflame the pleura, such as pulmonary embolus, infection, and Bornholm disease, may also confuse the diagnosis and complicate treatment.

TREATMENT

Initial treatment of intercostal neuralgia includes a combination of simple analgesics and nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors. If these medications do not adequately control the patient's symptoms, a tricyclic antidepressant or gabapentin should be added.

Traditionally, tricyclic antidepressants have been a mainstay in the palliation of pain caused by intercostal neuralgia. Controlled studies have demonstrated the efficacy of amitriptyline, and nortriptyline and desipramine have also proved to be clinically useful. Unfortunately, this class of drugs is associated with significant anticholinergic side effects, including dry mouth, constipation, sedation, and urinary retention. These drugs should be used with caution in patients suffering from glaucoma, cardiac arrhythmia, and prostatism. To minimize side effects and encourage compliance, the physician should start amitriptyline or nortriptyline at a 10-mg dose at bedtime; the dose can then be titrated upward to 25-mg at bedtime, as side effects allow. Subsequently, upward titration in 25-mg increments can be carried out each week, as side effects allow. Even at lower doses, patients generally report a rapid reduction in sleep disturbance and begin to experience some pain relief in 10–14 days. If the patient does not show any reduction in pain as the dose is being titrated upward, the addition of gabapentin alone or in combination with nerve blocks is recommended (see later). The selective serotonin reuptake inhibitors such as

FIG 64.5 Coarctation of the thoracic aorta with intercostal collateral vessels in a 27-year-old man evaluated for a ruptured aneurysm. **A,** Frontal chest radiograph shows rib notching *(arrow)* and abnormal contour of the thoracic aorta. **B,** Axial T1-weighted magnetic resonance imaging (MRI) shows large paraspinous and intercostal collateral vessels with associated flow-related signal voids. **C,** Oblique sagittal maximum-intensity projected MRI shows severe coarctation of the thoracic aorta with large intercostal and internal thoracic collateral vessels. (From Lee TJ, Collins J. MR imaging evaluation of disorders of the chest wall. *Magn Reson Imaging Clin N Am.* 2008;16(2):355–379.)

fluoxetine have also been used to treat the pain of intercostal neuralgia; although these drugs are better tolerated than tricyclic antidepressants, they appear to be less efficacious.

If the antidepressant compounds are ineffective or contraindicated, gabapentin is a reasonable alternative. Gabapentin is started at a 300-mg dose at bedtime for 2 nights. The patient should be cautioned about potential side effects, including dizziness, sedation, confusion, and rash. The drug is then increased in 300-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dose of 2400-mg/day is reached. At this point, if the patient has experienced partial pain relief, blood values are measured, and the drug is carefully titrated upward using 100-mg tablets. Rarely is a dose greater than 3600-mg/day required.

The local application of heat and cold or the use of an elastic rib belt may also provide symptomatic relief. For patients who do not respond to these treatment modalities, injection using local anesthetic and steroid is a reasonable next step.

The patient is placed in the prone position with the arms hanging loosely off the sides of the table. Alternatively, this block can be done with the patient in the sitting or lateral position. The rib to be blocked is identified by palpating its path at the posterior axillary line. The operator's index and middle fingers are then placed on the rib, thus bracketing the site of needle insertion, and the skin is prepared with antiseptic solution. A 1½-inch, 22-gauge needle is attached to a 12-mL syringe and is advanced perpendicular to the skin while aiming for the middle of the rib between the operator's index and middle fingers. The needle should impinge on bone after being advanced approximately ¾ inch. Once bony contact is made, the needle is withdrawn into the subcutaneous tissues, and the skin and subcutaneous tissues are retracted with the operator's palpating fingers inferiorly. This technique allows the needle to be walked off the inferior margin of the rib. As soon as bony contact is lost, the needle is slowly advanced approximately 2-mm deeper. This maneuver places the needle in proximity to the costal groove, which contains the intercostal nerve as well as the intercostal artery and vein. After careful aspiration reveals no blood or air, the operator injects 3–5 mL of 1% preservative-free lidocaine. If the pain has an inflammatory component, the local anesthetic is combined with 80-mg methylprednisolone and injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40-mg methylprednisolone for the initial 80-mg dose. Because of the overlapping innervation of the chest and upper abdominal wall, the intercostal nerves above and below the nerve suspected of causing the pain must also be blocked. Ultrasound needle guidance will decrease the incidence of complications, including pneumothorax, and increase the accuracy of needle placement when performing intercostal nerve block (Fig. 64.6).

FIG 64.6 Proper needle position for performing ultrasound-guided intercostal nerve block.

COMPLICATIONS AND PITFALLS

The major problem in the treatment of patients thought to be suffering from intercostal neuralgia is failure to identify potentially serious disease of the thorax or upper abdomen. Given the proximity of the pleural space, pneumothorax after intercostal nerve block is a distinct possibility. The incidence of this complication is less than 1%, but it occurs with greater frequency in patients with chronic obstructive pulmonary disease. Owing to the proximity to the intercostal nerve and artery, the clinician must carefully calculate the total dosage of local anesthetic administered, because vascular uptake through these vessels is high. Although uncommon, infection is always a possibility, especially in immunocompromised patients with cancer. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Intercostal neuralgia is a commonly encountered cause of chest wall and thoracic pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious intrathoracic or intraabdominal disease. Pharmacologic agents generally provide adequate pain control. If necessary, intercostal nerve block is a simple technique that can produce dramatic pain relief, but the proximity of the intercostal nerve to the pleural space makes careful attention to technique mandatory.

SUGGESTED READINGS

Ellis H. Anterior abdominal wall. *Anaesth Intens Care Med*. 2006; 7:36–37.

Rahn DD, Phelan JN, Roshanravan SM, et al. Anterior abdominal wall nerve and vessel anatomy: clinical implications for gynecologic surgery. *Am J Obstet Gynecol*. 2010;202:234. e1234.e5.

Vishy M. Anatomy of the anterior abdominal wall and groin. *Surgery (Oxford)*, 2006;24:221–223.

Waldman SD. Abnormalities of the rib and intercostal space. In: *Pain Management*. 2nd ed. Elsevier Philadelphia 2009;27:251–254.

Waldman SD. Anterior cutaneous nerve block. In: *Pain review*. Philadelphia: Saunders; 2009:497–498.

Waldman SD. Ultrasound-guided intercostal nerve block. In: *Atlas of Pain Management Injection Techniques*. Philadelphia: W.B. Saunders; 2014:185–188.

Waldman SD. *Comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:535–544.

Waldman SD. Pneumothorax, pulmonary embolism, and other abnormalities of the lung. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:545–560.

Waldman SD. Intercostal nerve block. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:454–455.

Waldman SD. Fractured ribs. In: *Atlas of common pain syndromes*. 4th ed. Philadelphia: Elsevier; 2019:260–264.

Waldman SD. *Comprehensive atlas of ultrasound-guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:653–661.

Waldman SD. Intercostal nerve block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021: 404–412.

Waldman SD. Postmastectomy pain. In: *Atlas of uncommon pain syndromes*. 3rd ed. Philadelphia: Elsevier.

Waldman SD. The intercostal nerves. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:113–114.

65

Diabetic Truncal Neuropathy

ICD-10 CODE G58.7

THE CLINICAL SYNDROME

Diabetic neuropathy is a term used by clinicians to describe a heterogeneous group of diseases that affect the autonomic and peripheral nervous systems of patients suffering from diabetes mellitus. Diabetic neuropathy is now thought to be the most common form of peripheral neuropathy that afflicts humankind, with an estimated 220 million people worldwide suffering from this malady.

One of the most commonly encountered forms of diabetic neuropathy is diabetic truncal neuropathy. In this condition, pain and motor dysfunction are often incorrectly attributed to intrathoracic or intraabdominal disorders and lead to extensive workups for appendicitis, cholecystitis, renal calculi, and so on. The onset of symptoms frequently coincides with periods of extreme hypoglycemia or hyperglycemia or with weight loss or gain. Patients who present with diabetic truncal neuropathy complain of severe dysesthetic pain with patchy sensory deficits in the distribution of the lower thoracic or upper thoracic dermatomes. The pain is often worse at night and causes significant sleep disturbance. The symptoms of diabetic truncal neuropathy often resolve spontaneously over 6–12 months. However, because of the severity of symptoms, aggressive treatment with pharmacotherapy and neural blockade is indicated.

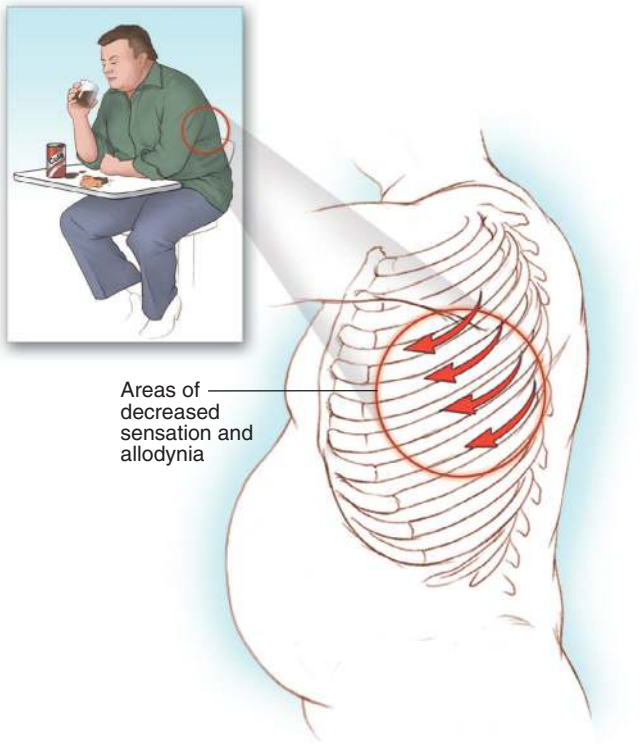

FIG 65.1 The pain of diabetic truncal neuropathy is neuropathic and is often made worse by poorly controlled blood glucose levels.

SIGNS AND SYMPTOMS

Physical examination generally reveals minimal findings unless the patient has a history of previous thoracic or subcostal surgery or cutaneous evidence of herpes zoster involving the thoracic dermatomes. Unlike patients with musculoskeletal causes of chest wall and subcostal pain, patients with diabetic truncal neuropathy do not attempt to splint or protect the affected area. Careful sensory examination of the affected dermatomes may reveal decreased sensation or allodynia. With significant motor involvement of the subcostal nerve, the patient may complain that his or her abdomen bulges outward (Fig. 65.1). This abnormal bulging is known as pseudohernia (Fig. 65.2). The clinician should be aware that the neurological impact of diabetes is varied and can confuse the diagnosis (Fig. 65.3).

TESTING

The presence of diabetes should lead to a high index of suspicion for diabetic truncal neuropathy. If the diagnosis of diabetic truncal neuropathy is suspected based on the targeted history and physical examination, screening laboratory tests, including a complete blood count, chemistry profile, erythrocyte sedimentation rate, thyroid function studies, antinuclear antibody testing, and urinalysis, should rule out most other peripheral neuropathies that are easily treatable. Electromyography and nerve conduction velocity testing are indicated in all patients suffering from peripheral neuropathy, to identify treatable entrapment neuropathies and further delineate the type of peripheral neuropathy present; these tests may also be able to quantify the severity of peripheral or entrapment neuropathy. Additional laboratory testing is indicated as the clinical situation dictates (e.g., Lyme disease titers, heavy metal screens). Magnetic resonance imaging of the spinal canal and cord should be performed if myelopathy is suspected. Nerve or skin biopsy is occasionally indicated if no cause of the peripheral neuropathy can be ascertained.

Lack of response to the therapies discussed later should lead to a reconsideration of the diagnosis and the repetition of testing, as clinically indicated.

DIFFERENTIAL DIAGNOSIS

Diseases other than diabetic neuropathy may cause peripheral neuropathies in diabetic patients (Box 65.1). These diseases may exist alone or may coexist with diabetic truncal neuropathy, thus making identification and treatment difficult.

Although uncommon in the United States, Hansen's disease is a common cause of peripheral neuropathy worldwide that may mimic or coexist with diabetic truncal neuropathy.

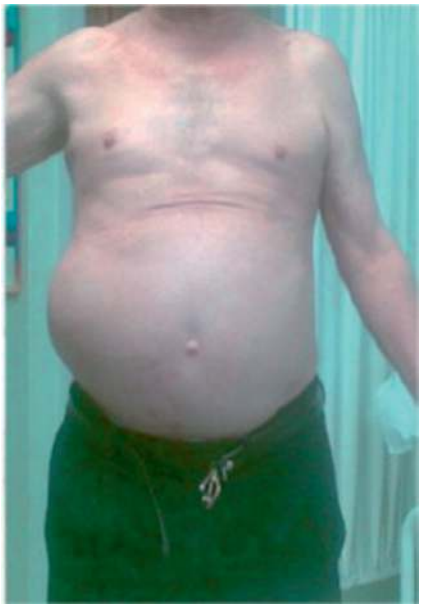

FIG 65.2 With significant motor involvement of the subcostal nerve, a patient suffering from diabetic truncal neuropathy may complain that his or her abdomen bulges outward.

BOX 65.1 The Impact of Diabetes Mellitus on the Nervous System

- Sensorimotor neuropathy
  - Distal symmetric polyneuropathy
  - Diabetic mononeuropathy
  - Carpal tunnel syndrome
  - Tarsal tunnel syndrome
  - Ulnar neuropathy
  - Lateral femoral cutaneous nerve entrapment syndrome
  - Focal neuropathy (usually confined to the distribution of a single cranial or peripheral nerve)
  - Mononeuritis multiplex (can involve the distribution of several peripheral nerves)
  - Diabetic amyotrophy
- Diabetic autonomic neuropathy
  - Hypoglycemic unawareness
  - Silent myocardial ischemia
  - Orthostatic hypotension and abnormal circadian blood pressures
  - Altered exercise tolerance
  - Genitourinary
  - Neurogenic bladder
  - Frequent urinary tract infections
  - Urinary incontinence
  - Sexual dysfunction
  - Gastropathy
  - Gastroparesis
  - Abdominal pain and bloating
  - Diarrhea and constipation
  - Fecal incontinence
  - Malabsorption
  - Dysphagia
  - Sudomotor abnormalities
  - Gustatory sweating
  - Sweating only from the chest down
  - High risk of Charcot arthropathy

Modified from Unger J, Cole BE. Recognition and management of diabetic neuropathy. *Prim Care Clin Off Pract.* 2007;34(4):887–913. ISSN 0095-4543. https://doi.org/10.1016/j.pop.2007.07.00, http://www.sciencedirect.com/science/article/pii/S0095454307000632.

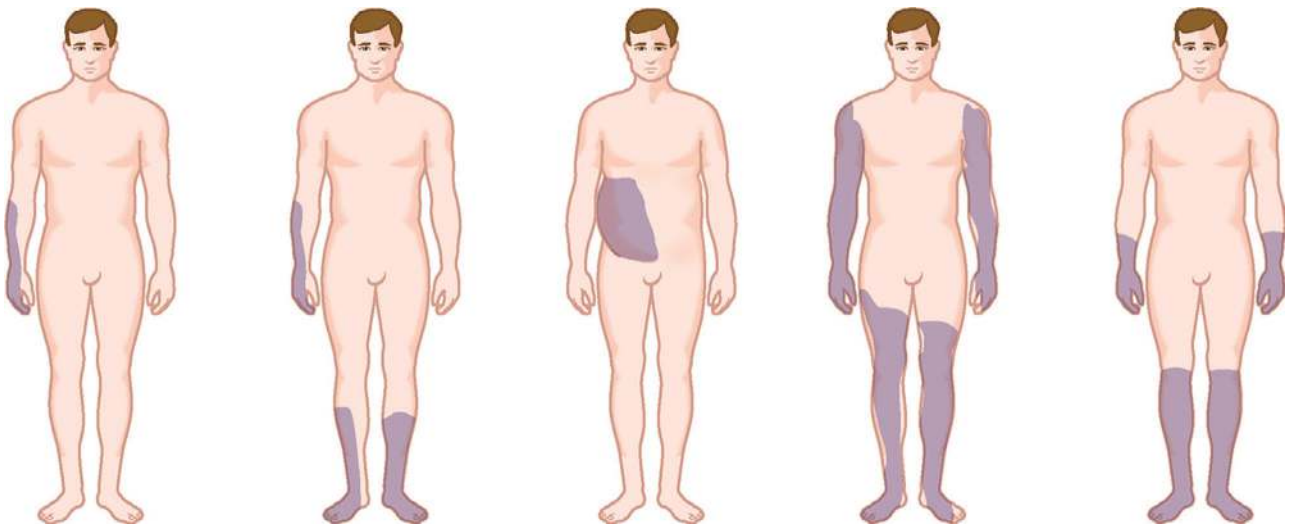

FIG 65.3 Classifications of diabetic neuropathy.

Other infectious causes of peripheral neuropathies include Lyme disease and human immunodeficiency virus infection. Substances that are toxic to nerves, including alcohol, heavy metals, chemotherapeutic agents, and hydrocarbons, may also cause peripheral neuropathies that are indistinguishable from diabetic neuropathy on clinical grounds. Heritable disorders such as Charcot–Marie–Tooth disease and other familial diseases of the peripheral nervous system must also be considered, although treatment options are somewhat limited. Metabolic and endocrine causes of peripheral neuropathy that must be ruled out include vitamin deficiencies, pernicious anemia, hypothyroidism, uremia, and acute intermittent porphyria. Other causes of peripheral neuropathy that may confuse the clinical picture include Guillain–Barré syndrome, amyloidosis, entrapment neuropathies, carcinoid syndrome, paraneoplastic syndromes, and sarcoidosis. Because many of these diseases are treatable, it is imperative that the clinician exclude them before attributing a patient's symptoms solely to diabetes.

Intercostal neuralgia and musculoskeletal causes of chest wall and subcostal pain may also be confused with diabetic truncal neuropathy. In all these conditions, the patient's pain may be erroneously attributed to cardiac or upper abdominal disease, thus leading to unnecessary testing and treatment.

TREATMENT

Control of Blood Glucose Levels

Current thinking is that the better the patient's glycemic control is, the less severe the symptoms of diabetic truncal neuropathy will be. Significant swings in blood glucose levels seem to predispose diabetic patients to the development of clinically significant diabetic truncal neuropathy. Some investigators believe that, although oral hypoglycemic agents control blood glucose levels, they do not protect patients from diabetic truncal neuropathy as well as does insulin. In fact, some patients taking hypoglycemic agents experience a reduction in the symptoms of diabetic truncal neuropathy when they are switched to insulin.

Pharmacologic Treatment

Antidepressants

Traditionally, tricyclic antidepressants have been a mainstay in the palliation of pain caused by diabetic truncal neuropathy. Controlled studies have demonstrated the efficacy of amitriptyline, and nortriptyline and desipramine have also proved to be clinically useful. Unfortunately, this class of drugs is associated with significant anticholinergic side effects, including dry mouth, constipation, sedation, and urinary retention. These drugs should be used with caution in patients suffering from glaucoma, cardiac arrhythmia, and prostatism. To minimize side effects and encourage compliance, the physician should start amitriptyline or nortriptyline at a 10-mg dose at bedtime; the dose can then be titrated upward to 25-mg at bedtime, as side effects allow. Subsequently, upward titration in 25-mg increments can be carried out each week, as side effects allow. Even at lower doses, patients generally report a rapid reduction in sleep disturbance and begin to experience some pain relief in 10–14 days. If the patient does not show any reduction in pain as the dose is being titrated upward, the addition of gabapentin alone or in combination with nerve blocks is recommended (see later). The selective serotonin reuptake inhibitors, such as fluoxetine, have also been used to treat the pain of diabetic truncal neuropathy, and although these drugs are better tolerated than tricyclic antidepressants, they appear to be less efficacious.

Anticonvulsants

Anticonvulsants have long been used to treat neuropathic pain, including that of diabetic truncal neuropathy. Both phenytoin and carbamazepine have been used with varying degrees of success, either alone or in combination with antidepressants. Unfortunately, the side effects of these drugs have limited their clinical usefulness.

The anticonvulsant gabapentin is highly efficacious in the treatment of various painful neuropathic conditions, including postherpetic neuralgia and diabetic truncal neuropathy. Used properly, gabapentin is extremely well tolerated, and in most pain centers, it has become the adjuvant analgesic of choice in the treatment of diabetic truncal neuropathy. Gabapentin has a large therapeutic window, but the physician is cautioned to start at the low end of the dosage spectrum and titrate upward slowly to avoid central nervous system side effects, including sedation and fatigue. The following recommended dosage schedule can minimize side effects and encourage compliance: A single bedtime dose of 300-mg for 2 nights is followed by 300-mg twice a day for an additional 2 days. If the patient is tolerating this twice-daily dosing, the dosage may be increased to 300-mg three times a day. Most patients begin to experience pain relief at this dosage. Additional titration upward can be carried out in 300-mg increments, as side effects allow. A total greater than 3600-mg/day in divided doses is not currently recommended. The use of 600- or 800-mg tablets can simplify maintenance dosing after titration has been completed.

Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50-mg three times a day and maybe titrated upward to 100-mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Antiarrhythmics

Mexiletine is an antiarrhythmic drug that may be effective in the management of diabetic truncal neuropathy. Some pain specialists believe that mexiletine is especially useful in patients with primarily sharp, lancinating pain or burning pain. Unfortunately, this drug is poorly tolerated by most patients and should be reserved for those who do not respond to first-line pharmacologic treatments such as gabapentin or nortriptyline alone or in combination with neural blockade.

Topical Agents

Some clinicians have reported the successful treatment of diabetic truncal neuropathy with topical application of

capsaicin. An extract of chili peppers, capsaicin is thought to relieve neuropathic pain by depleting substance P. The side effects of capsaicin include significant burning and erythema, however, and thus limit its use.

Topical lidocaine administered by transdermal patch or in a gel can also provide short-term relief of the pain of diabetic truncal neuropathy. This drug should be used with caution in patients taking mexiletine, because of the potential for cumulative local anesthetic toxicity. Whether topical lidocaine has a role in the long-term treatment of diabetic truncal neuropathy remains to be seen.

Analgesics

In general, neuropathic pain responds poorly to analgesic compounds. The simple analgesics, including acetaminophen and aspirin, can be used in combination with antidepressants and anticonvulsants, but care must be taken not to exceed the recommended daily dose, because renal or hepatic side effects may occur. The nonsteroidal antiinflammatory drugs may also provide a modicum of pain relief when they are used with antidepressants and anticonvulsants. Because of the nephrotoxicity of this class of drugs, however, they should be used with extreme caution in diabetic patients, given the high incidence of diabetic nephropathy even early in the course of the disease. The role of cyclooxygenase-2 inhibitors in the palliation of the pain of diabetic truncal neuropathy has not been adequately studied.

The pain of diabetic truncal neuropathy responds poorly to treatment with opioid analgesics. Given the significant central nervous system and gastrointestinal side effects, coupled with the problems of tolerance, dependence, and addiction, opioid analgesics should rarely, if ever, be used as a primary treatment for the pain of diabetic truncal neuropathy. If an opioid analgesic is being considered, however, tramadol may be a reasonable choice; it binds weakly to the opioid receptors and may provide some symptomatic relief. Tramadol should be used with care in combination with antidepressants to avoid the increased risk of seizures.

Cannabinoids

Considerable anecdotal evidence and advocacy support a possible beneficial role for cannabinoids in the treatment of diabetic peripheral neuropathy. Large-scale trials are underway to determine if cannabinoids offer advantages over other traditional treatments for this debilitating condition.

Neural Blockade

Neural blockade with local anesthetic alone or in combination with steroid can be useful in the management of both acute and chronic pain associated with diabetic truncal neuropathy. Thoracic epidural or intercostal nerve block with local anesthetic, steroid, or both may be beneficial. Occasionally, neuroaugmentation by spinal cord stimulation may provide significant pain relief in patients who have not been helped by more conservative measures. Neurodestructive procedures are rarely, if ever, indicated to treat the pain of diabetic truncal neuropathy; they often worsen the patient's pain and cause functional disability.

COMPLICATIONS AND PITFALLS

The major problem in the care of patients thought to be suffering from diabetic truncal neuropathy is failure to identify potentially serious disorders of the thorax or upper abdomen. Given the proximity of the pleural space, pneumothorax after intercostal nerve block is a distinct possibility. The incidence of the complication is less than 1%, but it occurs with greater frequency in patients with chronic obstructive pulmonary disease. Owing to the proximity to the intercostal nerve and artery, the clinician must carefully calculate the total dosage of local anesthetic administered, because vascular uptake by these vessels is high. Although uncommon, infection is always a possibility, especially in immunocompromised patients with cancer. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

BOX 65.2 Factors That Can Be Modified to Mitigate the Impact of Diabetes Mellitus on the Nervous System

- Decreasing glycemic variability with tight control of blood sugars
- Managing dyslipidemias, including hypertriglyceridemia
- Managing comorbidities, including hypertension
- Managing obesity
- Smoking cessation
- Improving diet
- Treat vitamin deficiencies

CLINICAL PEARLS

Diabetic truncal neuropathy is a commonly encountered cause of thoracic and subcostal pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious intrathoracic or intraabdominal disease. Pharmacologic agents generally provide adequate pain control, but a concurrent focus is on identifying factors that can be changed to mitigate the impact of diabetes (Box 65.2). If necessary, intercostal or epidural nerve block is a simple technique that can produce dramatic pain relief, but the proximity of the intercostal nerve to the pleural space makes careful attention to technique mandatory.

SUGGESTED READINGS

Janahi NM, Santos D, Blyth C, et al. Diabetic peripheral neuropathy, is it an autoimmune disease? *Immunol Lett.* 2015;168(1):73–79.

Otto-Buczkowska E, Dryzaøowski M. Neuropathy in young diabetic patients. *Pediatr Pol.* 2016;91(2):142–148.

Selvarajah D, Gandhi R, Tesfaye S. Cannabinoids and their effects on painful neuropathy. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:905–916.

Sloan G, Shillo P, Selvarajah D, et al. A new look at painful diabetic neuropathy. *Diabetes Res Clin Pract*. 2018;144:177–191.

Waldman SD. Intercostal nerve block. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:454–455.

Waldman SD. Thoracic epidural block: midline approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Saunders; 2021:328–338.

Tietze's Syndrome

ICD-10 CODE M94.0

THE CLINICAL SYNDROME

Tietze's syndrome is a frequent cause of chest wall pain. Distinct from the more common costosternal syndrome, Tietze's syndrome was first described in 1921 and is characterized by acute, painful swelling of the costal cartilage. In fact, painful swelling of the second and third costochondral joints is the sine qua non of Tietze's syndrome (Fig. 66.1); such swelling is absent in costosternal syndrome. Also distinguishing the two syndromes is the age of onset; costosternal syndrome usually occurs no earlier than the fourth decade of life, whereas Tietze's syndrome is a disease of the second and third decades. The onset is acute and is often associated with a concurrent viral infection of the respiratory tract. Investigators have postulated that microtrauma to the costosternal joints from severe coughing or heavy labor may be the cause of Tietze's syndrome.

SIGNS AND SYMPTOMS

Physical examination reveals that patients suffering from Tietze's syndrome vigorously attempt to splint the joints by keeping the shoulders stiffly in a neutral position. Pain is reproduced with active protraction or retraction of the shoulder, deep inspiration, and full elevation of the arm. Shrugging off the shoulder may also reproduce the pain. Coughing may be difficult, leading to inadequate pulmonary toilet in some patients. The costosternal joints, especially the second

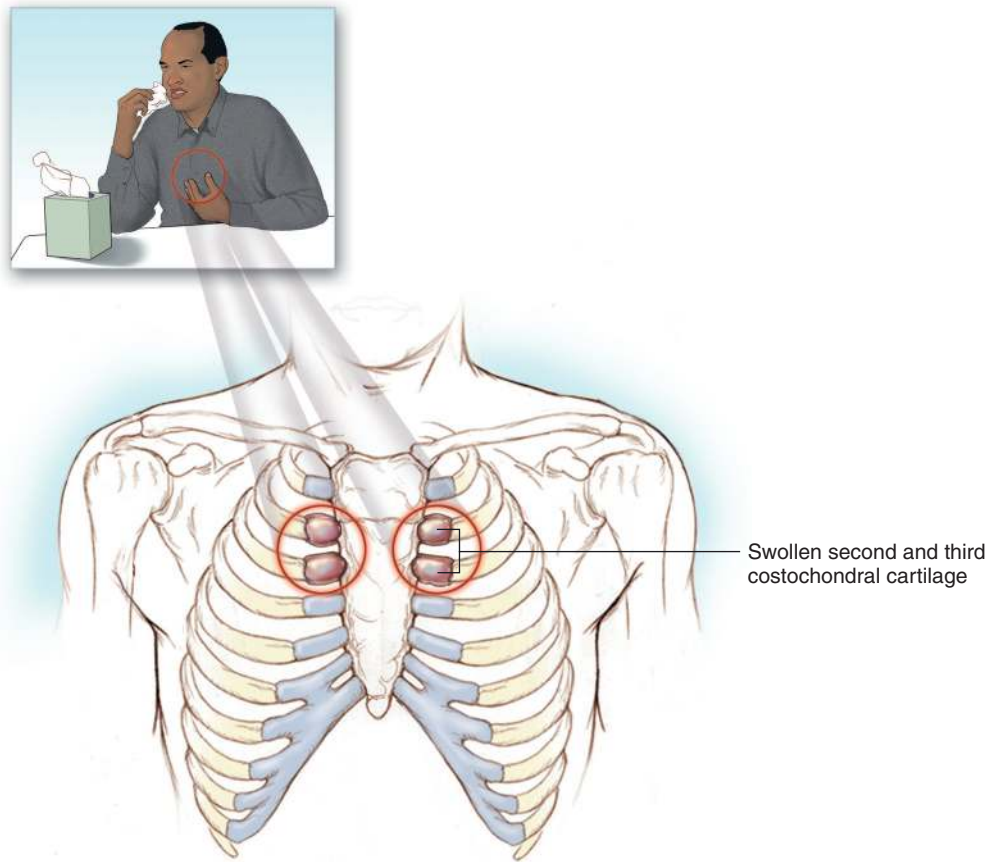

FIG 66.1 Swelling of the second and third costochondral joints is pathognomonic of Tietze's syndrome.

and third, are swollen and exquisitely tender to palpation (Fig. 66.2). This swollen costochondral joint sign is pathognomonic for Tietze's syndrome (Fig. 66.3). The adjacent intercostal muscles may also be tender to palpation. The patient may complain of a clicking sensation with movement of the joint.

TESTING

Plain radiographs are indicated for all patients who present with pain thought to be emanating from the costosternal joints, to rule out occult bony disorders, including tumor (Figs. 66.4 and 66.5). If trauma is present, radionuclide bone scanning should be considered to exclude occult fractures of the ribs or sternum. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Laboratory evaluation for collagen vascular disease is indicated in patients suffering from costosternal joint

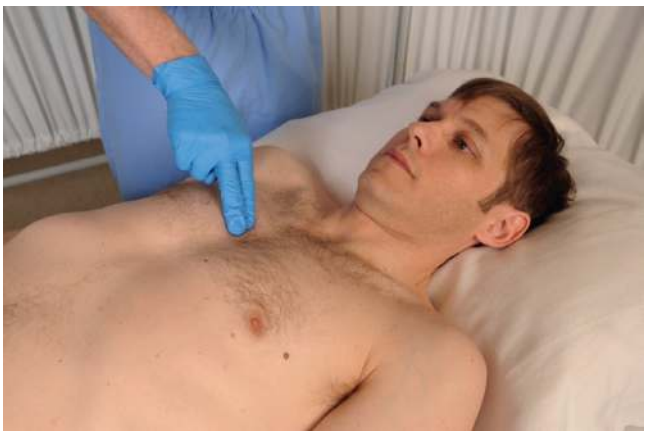

FIG 66.2 Palpation of the costosternal joint.

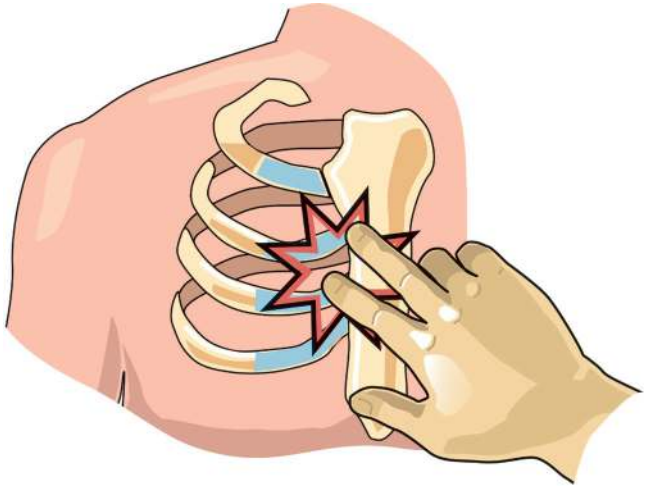

FIG 66.3 Inspection of the costosternal joint for swelling indicative of Tietze's syndrome. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:209.)

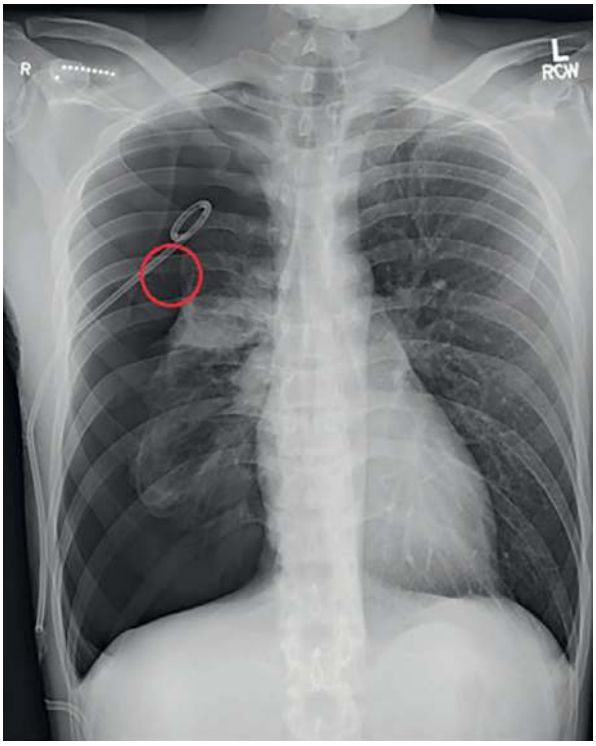

FIG 66.4 X-ray demonstrating right-sided pneumothorax (*red circle*). (From Segraves JM, Dulohery MM. Primary spontaneous pneumothorax due to high bleb burden. *Respir Med Case Rep*. 2016;19:109–111.)

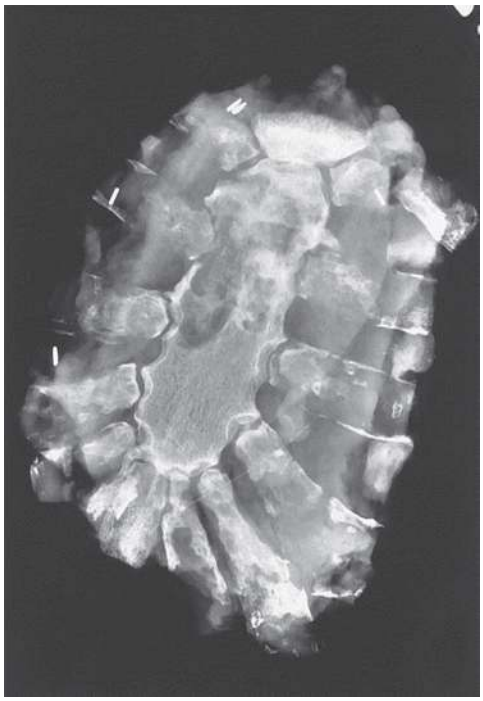

FIG 66.5 Radiograph of resected chest wall in 54-year-old man with low-grade chondrosarcoma of body of sternum. Resection included body, xiphoid process, and lower portion of manubrium. Tumor is clearly seen in the upper part of body of sternum. (From Martini N, Huvos AG, Burt ME, Heelan RT, Bains MS, McCormack PM, Rusch VW, Weber M, Downey RJ, Ginsberg RJ. Predictors of survival in malignant tumors of the sternum. *J Thorac Cardiovasc Surg*. 1996;111(1):96–106.)

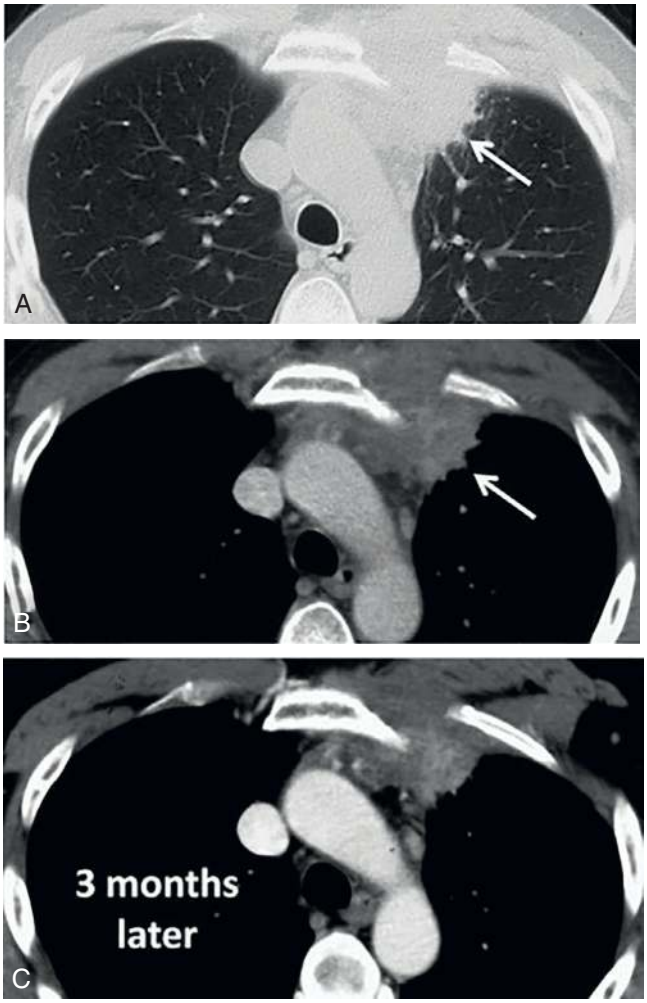

FIG 66.6 A–C, CT scan of a patient with anterior chest wall pain thought to be Tietze syndrome. CT scanning of anterior upper mediastinal mass after iodinated contrast administration. Lung window **(A)**. Mediastinum window **(B)** shows inhomogeneous contrast enhancement of the mass. Enhanced multidetector computed tomography (MDCT) follow-up after 3 months **(C)** shows decrease in volume of mediastinal mass. (From De Filippo M, Albini A, Castaldi V, Monaco D, Sverzellati N, Carbognani P, Rusca M, Rindi G, Zompatori M. MRI findings of Tietze's syndrome mimicking mediastinal malignancy on MDCT. *Eur J Radiol Extra*. 2008;65(1):33–35.)

pain if other joints are involved. Computerized tomographic scanning and/or magnetic resonance imaging of the joints is indicated if joint instability or occult mass is suspected or to confirm the diagnosis (Figs. 66.6 and 66.7). The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Many other painful conditions that affect the costosternal joints are much more common than Tietze's syndrome. For instance, the costosternal joints are susceptible to osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, and psoriatic arthritis. The joints are often traumatized

FIG 66.7 Proper needle placement for ultrasound-guided injection of the costosternal joint for Tieitze syndrome.

during acceleration–deceleration injuries and blunt trauma to the chest; with severe trauma, the joints may subluxate or dislocate. Overuse or misuse can result in acute inflammation of the costosternal joint, which can be quite debilitating. The joints are also subject to invasion by tumor from primary malignant tumors, including thymoma, or from metastatic disease.

TREATMENT

Initial treatment of the pain and functional disability associated with Tietze's syndrome includes nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors. The local application of heat and cold may also be beneficial. Use of an elastic rib belt may provide symptomatic relief and protect the costosternal joints from additional trauma. For patients who do not respond to these treatment modalities, injection using local anesthetic and steroid is a reasonable next step.

Injection for Tietze's syndrome is performed by placing the patient in the supine position. The skin overlying the affected costosternal joints is prepared with antiseptic solution. A sterile syringe containing 1-mL of 0.25% preservative-free bupivacaine for each joint to be injected and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The costosternal joints are identified; they should be easily palpable as a slight bulging at the point where the rib attaches to the sternum. The needle is carefully advanced through the skin and subcutaneous tissues medially, with a slight cephalad trajectory, in proximity to the joint. If bone is encountered, the needle is withdrawn from the periosteum. After the needle is in proximity to the joint, 1-mL of solution is gently injected. The clinician should feel limited resistance to injection. If significant resistance is encountered, the needle should be withdrawn slightly until the injection can proceed with only limited resistance. This procedure is repeated for each affected joint. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will decrease the incidence of needle-induced complications

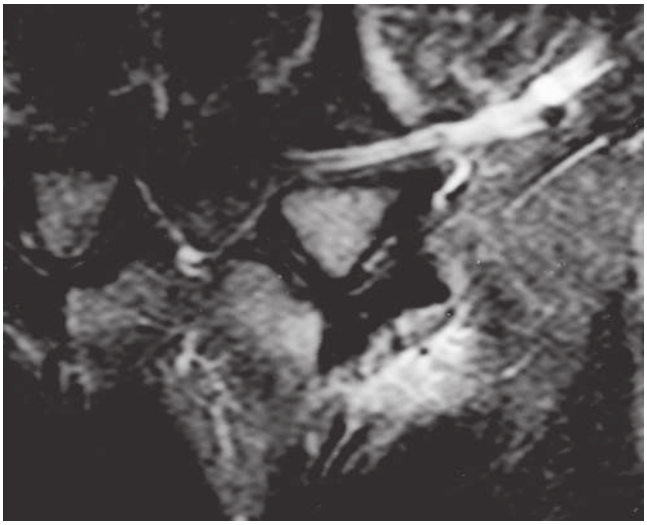

FIG 66.8 Tietze's syndrome. Coronal, short tau inversion recovery, magnetic resonance image of the thorax shows high signal intensity below the sternoclavicular joint at the costosternal junction in a 45-year-old man with pain and tenderness in this region. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:2605.)

including pneumothorax when performing injection of the costosternal joints (Fig. 66.8).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for Tietze's syndrome. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Because many pathologic processes can mimic the pain of Tietze's syndrome, the clinician must carefully rule out underlying cardiac disease and diseases of the lung and structures of the mediastinum. Failure to do so could lead to disastrous results. The major complication of the injection technique is pneumothorax if the needle is placed too laterally or deeply and invades the pleural space. Trauma to the contents of the mediastinum is also a possibility. This complication can be greatly decreased with strict attention to accurate needle placement. Infection, although rare, can occur if strict aseptic technique is not followed, and universal precautions should be used to minimize risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection.

CLINICAL PEARLS

Patients suffering from pain emanating from the costosternal joint often believe that they are having a heart attack. Reassurance is required, but clinicians should remember that musculoskeletal pain syndromes and coronary artery disease can coexist. Tietze's syndrome may be confused with the more common costosternal syndrome, although both respond to the aforementioned injection technique.

SUGGESTED READINGS

Gologorsky R, Hornik B, Velotta J. Surgical management of medically refractory Tietze syndrome. *Ann Thorac Surg*. 2017;104(6): e443–e445.

Hanak JA. Tietze syndrome. In: Frontera WR, Silver JK, Rizzo TD, eds. *Essentials of physical medicine and rehabilitation*. 4th ed. 2020: chap 117, 640–645.

Waldman SD. Arthritis and other abnormalities of the costosternal joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:513–518.

Waldman SD. The swollen costosternal joint sign for Tietze syndrome. In: *Physical diagnosis of pain. An atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:206–207.

Waldman SD. The swollen joint sign for Tietze syndrome. In: *Physical diagnosis of pain: An atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2021:194.

Waldman SD. Ultrasound guided injection technique for costosternal joint pain. In: *Waldman's comprehensive atlas of ultrasound guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:591–594.

Precordial Catch Syndrome

ICD-10 CODE R07.2

THE CLINICAL SYNDROME

Precordial catch syndrome, also known as Texidor's twinge, is a common cause of chest wall pain. Occurring most frequently in adolescents and young adults, precordial catch syndrome is the cause of anxiety among patients and clinicians alike, given the intensity of the pain and its frequent attribution to the heart. Precordial catch syndrome almost always occurs at rest, often while the patient is sitting in a slumped position on an old couch (Fig. 67.1).

Distinct from other causes of chest wall pain, precordial catch syndrome is characterized by sharp, stabbing, needle-like pain that is well localized in the precordial region. Symptoms begin without warning, only adding to the patient's anxiety, and they go away as suddenly as they came (Box 67.1). The pain lasts from 30 seconds to 3 minutes, and it is often made worse by deep inspiration. Patients suffering from precordial catch syndrome usually outgrow the syndrome by the third decade of life.

SIGNS AND SYMPTOMS

No physical findings (e.g., flushing, pallor, diaphoresis) are associated with the onset of pain, although some patients suffering from precordial catch syndrome may demonstrate tenderness to palpation in the anterior intercostal muscles overlying the painful area. Because the pain is made worse with deep inspiration, the patient may become lightheaded from prolonged shallow breathing.

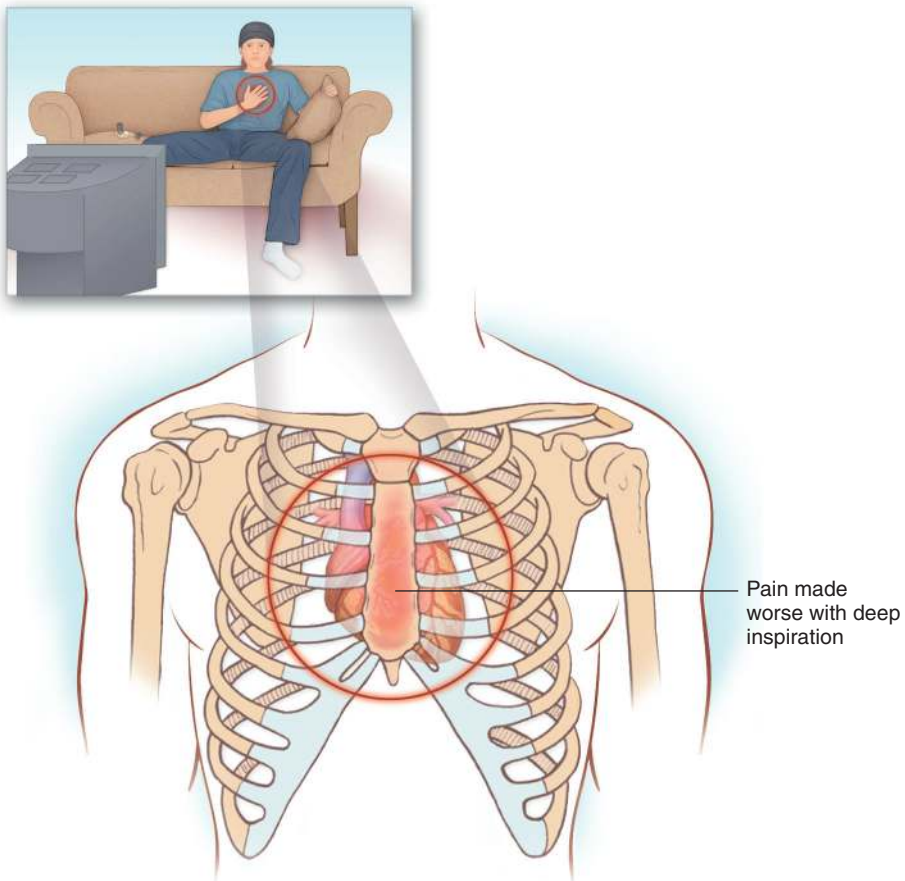

FIG 67.1 Precordial catch syndrome can be caused by prolonged sitting in a slumped position.

BOX 67.1 Anatomic Basis of Anterior Chest Wall Pain

Skeleton	Trauma
	Stress fractures (ribs or sternum)
	Chest wall deformities
	Chronic recurrent multifocal osteomyelitis
	SAPHO syndrome
	Precordial catch syndrome
	Hyperalgesia
Joints	Costochondritis
	Tietze syndrome
	Spondyloarthropathies (i.e., psoriatic arthritis)
	Hyperalgesia
Muscles	Muscle strains
	Viral myalgias
	Muscle contusions
	Hyperalgesia
Nerves	Slipping rib syndrome
	Intercostal neuralgia
	Hyperalgesia
Skin	Herpes zoster
	Hyperalgesia

Modified from Son MBF, Sundel RP, Musculoskeletal causes of pediatric chest pain. *Pediatr Clin N Am.* 2010;57(6):1385–1395.

TESTING

Plain radiographs and computerized tomography are indicated for all patients who present with pain thought to be emanating from the chest wall, to rule out occult bony disorders, including tumor (Fig. 67.2). If trauma is present, radionuclide bone scanning should be considered to exclude occult fractures of the ribs or sternum (Fig. 67.3). Given the location of the pain,

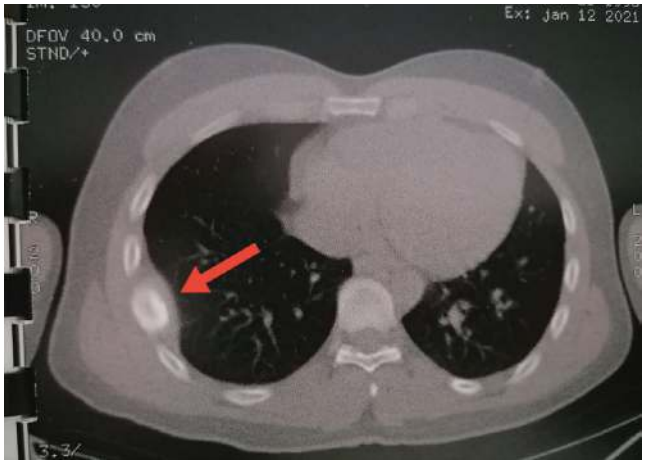

FIG 67.2 CT scan: osteocondensing lesion at the junction of the middle and posterior arches of the right sixth rib suggesting Ewing's sarcoma. (From El Haj NI, Hafidi S, Karam R, Boubia S, et al. Osteoid osteoma of the rib: a report of an extremely rare condition. *Int J Surg Case Rep.* 2022;94:107–139.)

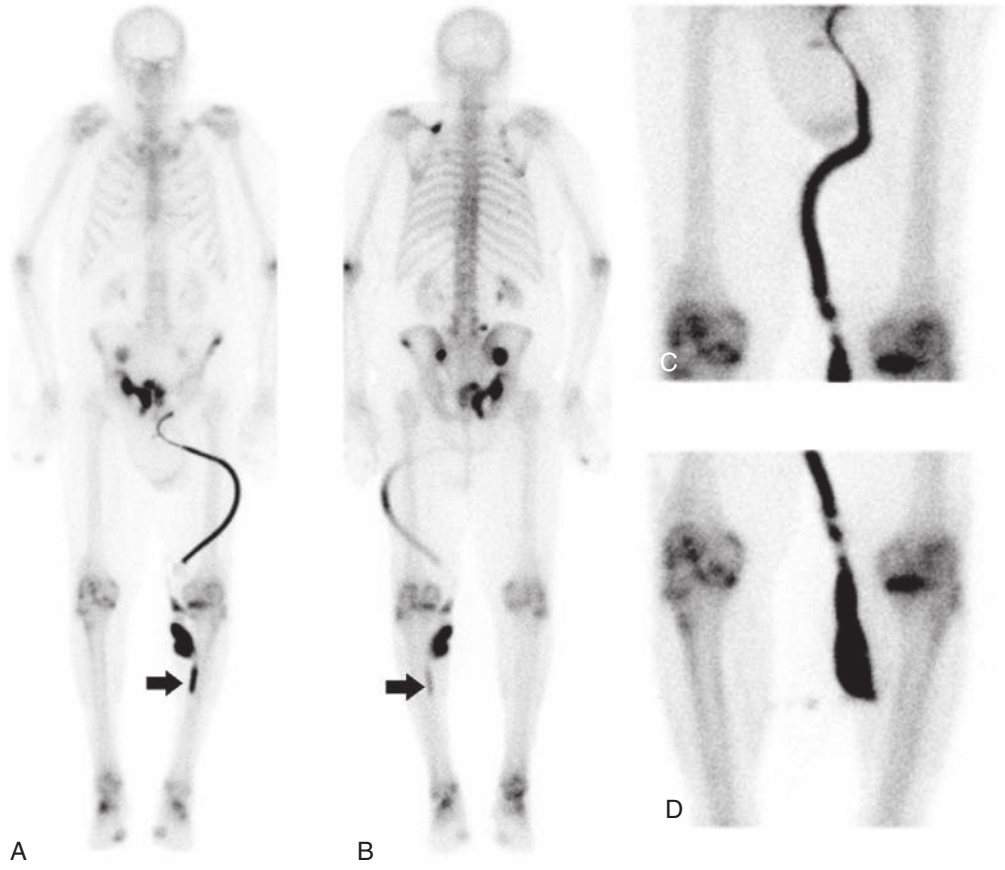

FIG 67.3 A, The bone scan study of a patient with prostate cancer showed multiple foci of abnormal tracer uptake in the left scapula, ribs, lower lumbar spine, and pelvic bones consistent with metastases. Note is made of urinary catheter with tracer in the urine. B, The tracer activity projected over the left tibia (*arrow*) is owing to tracer activity in the urinary catheter as shown in the separate static images (C, D) acquired after changing the position of the catheter. This can be misinterpreted as metastasis in the tibia and additional views may be needed. (From Agrawal K, Marafi F, Gnanasegaran G, et al., Pitfalls and limitations of radionuclide planar and hybrid bone imaging. *Semin Nucl Med.* 2015; 45(5):347–372.)

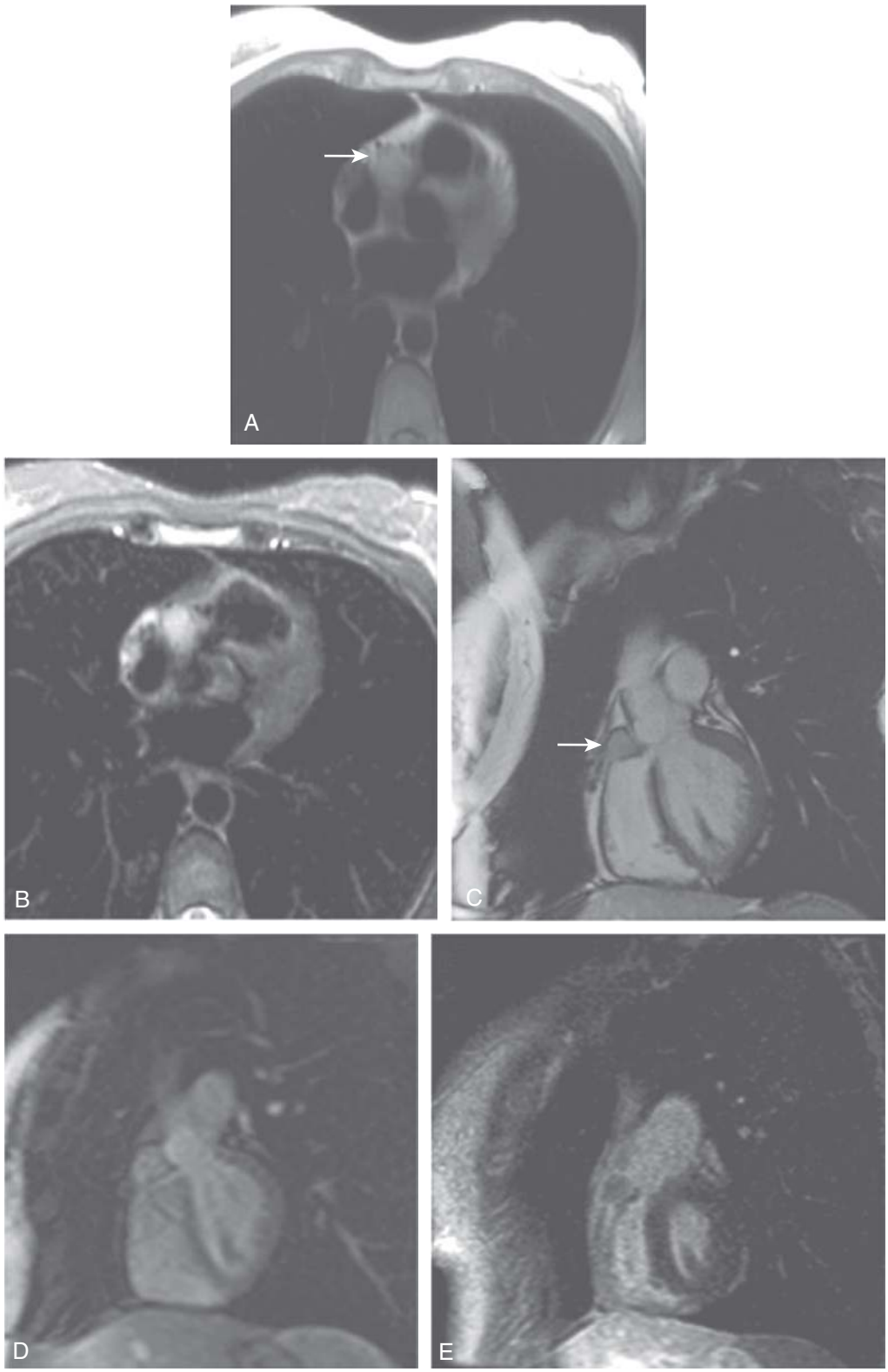

FIG 67.4 Magnetic resonance imaging demonstrating a paraganglioma in the right atrioventricular (AV) groove. **A,** Axial T1-weighted image demonstrates an isointense mass *(arrow)* in the right AV groove, immediately subjacent to the right coronary artery. Many of the imaging features of paragangliomas are a result of their high vascularity. **B,** This vascularity gives them a characteristic lightbulb bright appearance on T2-weighted images. **C,** Coronal steady-state free precession image shows a hyperintense mass in the right AV groove *(arrow)*, again a result of hypervascularity. **D,** Paragangliomas appear as hypervascular structures on first-pass perfusion imaging. **E,** Delayed enhancement (DE) inversion recovery image demonstrates mild contrast retention but no real DE. (From Syed IS, Feng D, Harris SR, et al. MR imaging of cardiac masses. *Magn Reson Imaging Clin N Am*. 2008;16(2):137–164.)

an electrocardiogram and an echocardiogram are indicated, but in patients with precordial catch syndrome, the results are expected to be normal. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the painful area are indicated if an occult mass is suspected or to clarify the diagnosis.

DIFFERENTIAL DIAGNOSIS

Many other painful conditions that affect the chest wall occur with much greater frequency than does precordial catch syndrome (Box 67.2). The costosternal joints are susceptible to osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, and psoriatic arthritis. The joints are often traumatized during acceleration-deceleration injuries and blunt trauma to the chest; with severe trauma, the joints may subluxate or dislocate. Overuse or misuse can result in acute inflammation of the costosternal joint, which can be quite debilitating. The joints are also subject to tumor invasion from primary malignant tumors, including thymoma, or from metastatic disease. Sharp pleuritic chest pain may be associated with devil's grip, pleurisy, pneumonia, or pulmonary embolus. Occult cardiac disease can also mimic the pain of precordial catch syndrome (Fig. 67.4).

BOX 67.2 Differential Diagnosis of Precordial Catch Syndrome

- Angina
- Cardiac ischemia
- Aortic stenosis
- Mitral valve prolapse
- Pericarditis
- Cardiomyopathy
- Pleuritis
- Devil's grip
- Pleurodynia
- Pulmonary embolus
- Pneumonia
- Rib fractures
- Rib tumors
- Costosternal syndrome
- Manubriosternal joint disorders

TREATMENT

Treatment of precordial catch syndrome consists of a combination of reassurance and instructing the patient to take a deep breath as soon as the pain begins, even though this produces a sharp, stabbing pain. Improving one's posture and changing position frequently while resting or watching television should also help decrease the frequency of attacks. Pharmacologic treatment is not indicated, given the rapid onset and offset of the pain.

COMPLICATIONS AND PITFALLS

Because many pathologic processes may mimic the pain of precordial catch syndrome, the clinician must carefully rule out underlying cardiac disease and diseases of the lung and structures of the mediastinum. Failure to do so could lead to disastrous results. The greatest risk in patients suffering from precordial catch syndrome is related to unnecessary testing (e.g., cardiac catheterization) to rule out cardiac disease.

CLINICAL PEARLS

Patients suffering from precordial catch syndrome often believe that they are having a heart attack. Reassurance is required, although the clinician should remember that musculoskeletal pain syndromes and coronary artery disease can coexist.

SUGGESTED READINGS

Ayloo A, Cvengros T, Marella S. Evaluation and treatment of musculoskeletal chest pain. *Prim Care*. 2013;40(4):863–887.

Hillen TJ, Wessell DE. Multidetector CT scan in the evaluation of chest pain of nontraumatic musculoskeletal origin. *Thorac Surg Clin*. 2010;20(1):167–173.

Son MBF, Sundel RP. Musculoskeletal causes of pediatric chest pain. *Pediatr Clin North Am*. 2010;57(6):1385–1395.

Stochkendahl MJ, Christensen HW. Chest pain in focal musculoskeletal disorders. *Med Clin North Am*. 2010;94(2):259–273.

Waldman SD. Precordial catch syndrome. In: *Atlas of uncommon pain syndromes*. 4th ed. Philadelphia: Elsevier; 2019:257–259.

Fractured Ribs

ICD-10 CODE S22.39xA

THE CLINICAL SYNDROME

Fractured ribs are among the most common causes of chest wall pain; they are usually associated with trauma to the chest wall (Fig. 68.1). In osteoporotic patients or in patients with primary tumors or metastatic disease involving the ribs, fractures may occur with coughing (tussive fractures) or spontaneously.

The pain and functional disability associated with fractured ribs are largely determined by the severity of the injury (e.g., number of ribs involved), the nature of the injury (e.g., partial or complete fracture, free-floating fragments), and the amount of damage to surrounding structures, including the intercostal nerves and pleura (Fig. 68.2). The pain associated with fractured ribs ranges from a dull, deep ache with partial osteoporotic fractures to severe, sharp, stabbing pain that may lead to inadequate pulmonary toilet. In the absence of significant trauma, the clinician should highly suspect the possibility of malignant lesions of the ribs (Fig. 68.3).

SIGNS AND SYMPTOMS

Rib fractures are aggravated by deep inspiration, coughing, and any movement of the chest wall. Palpation of the affected ribs may elicit pain and reflex spasm of the musculature of the chest wall. Ecchymosis overlying the fractures may be present. The clinician should be aware of the possibility of pneumothorax or hemopneumothorax. Fractures of the first rib may produce a Horner's syndrome (Fig. 68.4). Damage to the intercostal nerves may produce severe pain and result in reflex splinting of the chest wall that further compromises the patient's pulmonary status. Failure to treat this pain and splinting aggressively may result in a negative cycle of hypoventilation, atelectasis, and, ultimately, pneumonia.

TESTING

Plain radiographs or computed tomography (CT) scans of the ribs and chest are indicated for all patients who present with pain from fractured ribs, to rule out occult fractures and other bony disorders, including tumor, as well

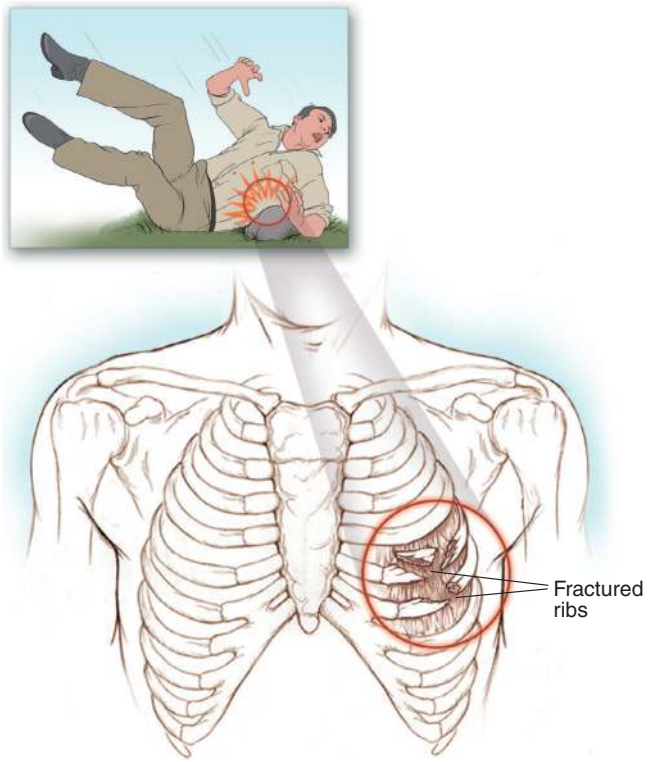

FIG 68.1 The pain of fractured ribs is amenable to intercostal nerve block with local anesthetic and steroid.

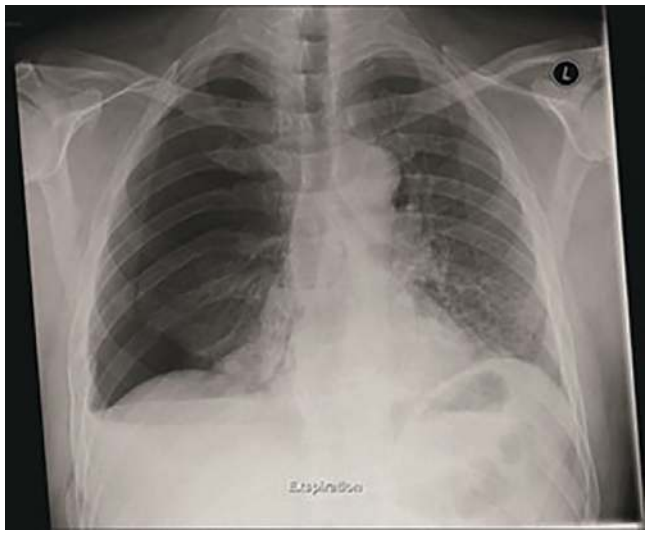

FIG 68.2 Chest X-ray revealing right-sided fractured ribs and a pneumothorax.

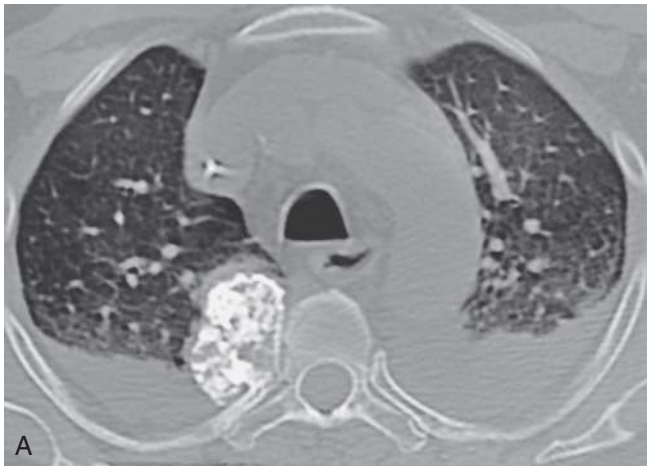

FIG 68.3 **A,** Noncontrast computed tomography scan reveals a heterogeneous, lobulated, calcified mass with a cartilaginous cap arising from the right third rib, a finding consistent with osteochondroma. **B,** Nonunion of rib fracture. Contrast-enhanced axial computed tomography scan shows expansile right rib lesion with questionable chondroid matrix (*arrow*). (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:1008–1009.)

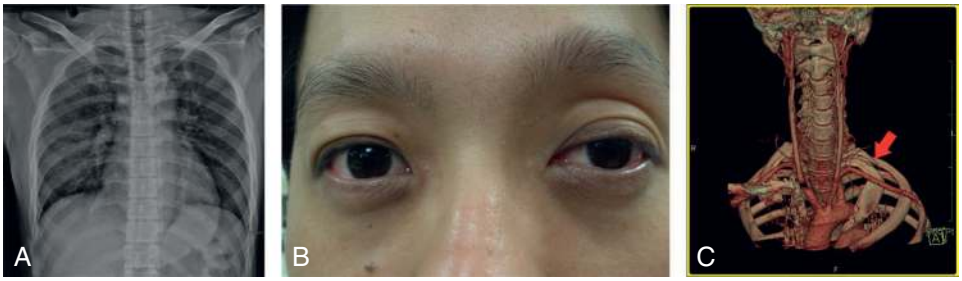

FIG 68.4 **A,** Chest radiography showing right chest contusion with fractures of the right second, third, and fourth ribs. **B,** The clinical picture with left eye ptosis. **C,** Three-dimensional reconstructed computed tomographic angiography reveals a rare transverse fracture in the left first rib (*arrow*) without left carotid artery dissection. (From Lin Y-C, Chuang M-T, Hsu C-H, Tailor A-RA, Lee J-S. First rib fracture resulting in Horner's syndrome. *J Emerg Med*. 2015;49(6):868–870.)

as pneumothorax and hemopneumothorax (Fig. 68.5). If trauma is present, radionuclide bone scanning may be useful to exclude occult fractures of the ribs or sternum. If no trauma is present, bone density testing to rule out osteoporosis is appropriate, as are serum protein electrophoresis and testing for hyperparathyroidism. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. CT and magnetic resonance imaging of the thoracic contents, soft tissues, and adjacent organs are indicated if an occult mass or significant trauma to the thoracic contents is suspected (Figs. 68.6–68.8). Positron emission tomography/computerized tomograph may help clarify the diagnosis in difficult cases (Fig. 68.9). Electrocardiography, to exclude cardiac contusion, is recommended for all patients with traumatic sternal fractures or significant anterior chest wall trauma. The injection technique described later should be used early to avoid pulmonary complications.

DIFFERENTIAL DIAGNOSIS

In the setting of trauma, the diagnosis of fractured ribs is usually straightforward. In the setting of spontaneous rib fracture secondary to osteoporosis or metastatic disease, the diagnosis may be less clear-cut. In this case, the pain of occult rib fracture is often mistaken for pain of cardiac or gallbladder origin, and it leads to visits to the emergency department and unnecessary cardiac and gastrointestinal workups. Tietze's syndrome, which is painful enlargement of the upper costochondral cartilage associated with viral infection, may be confused with fractured ribs, especially if the patient has been coughing.

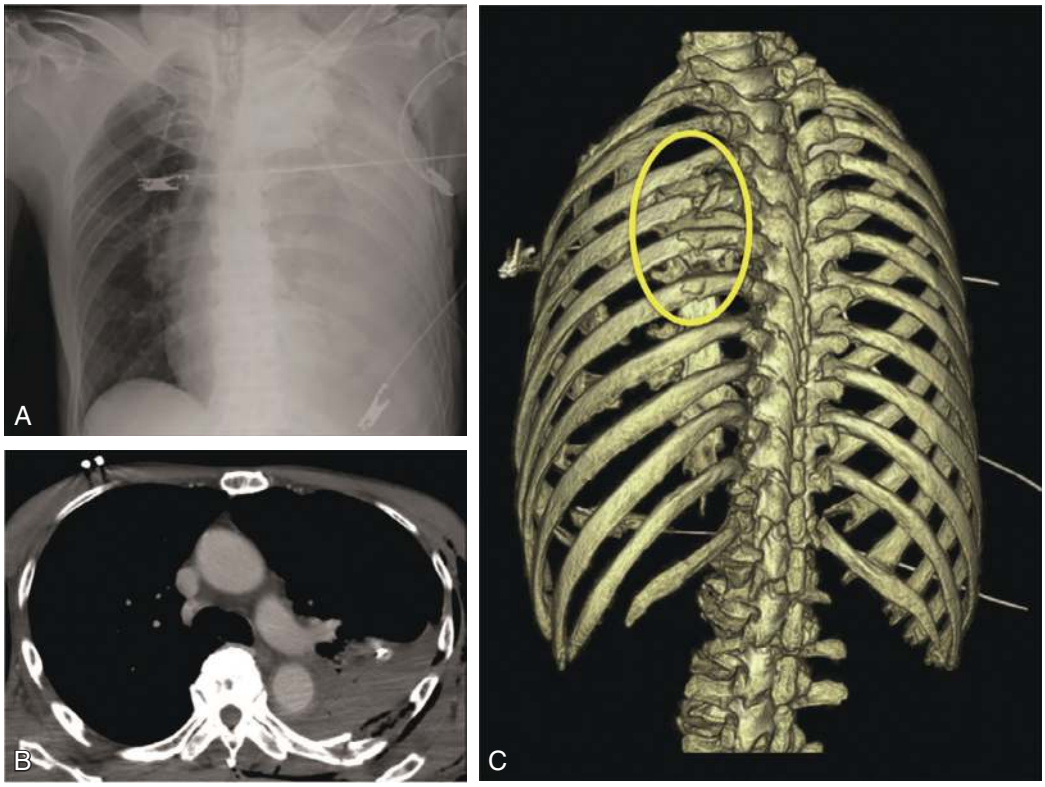

FIG 68.5 Chest radiography **(A)** and computed tomography (CT) **(B)** performed in a supine position on admission in the local hospital. Chest radiography and CT showed multiple left rib fractures and massive hemothorax (*circle*). The fractured fourth to seventh ribs are shown on three-dimensional chest CT scan **(C)**. (From Goda Y, Shoji T, Date N, et al. Hemothorax resulting from an initially masked aortic perforation caused by penetration of the sharp edge of a fractured rib: a case report. *Int J Surg Case Rep*. 2020;68:1821, Fig. 1. ISSN 2210-2612.)

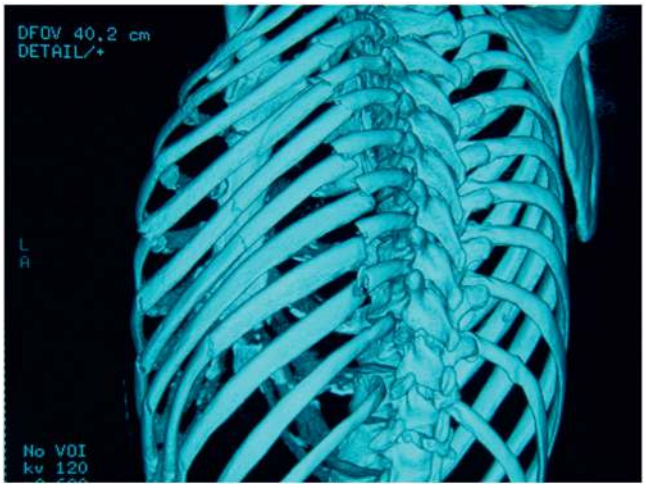

FIG 68.6 Multiple rib fractures. Three-dimensional computed tomography showing multiple displaced adjacent ribs fractured in multiple places, fracture of the left fourth through 10th ribs. (From Zhang Y, Tang X, Xie H, Wang RL. Comparison of surgical fixation and nonsurgical management of flail chest and pulmonary contusion. *Am J Emerg Med*. 2015;33(7):937–940.)

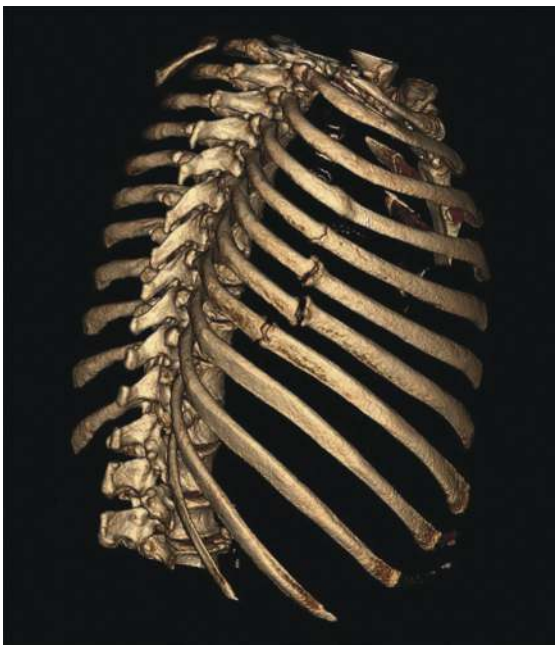

FIG 68.7 Computed tomography with three-dimensional reconstruction demonstrating nonunion fractures on right-sided ribs following traumatic injury. (From Buehler KE, Wilshire CL, Bograd AJ, et al. Rib plating offers favorable outcomes in patients with chronic nonunion of prior rib fractures. *Ann Thorac Surg*. 2020;110(3):993997, Fig. 1. ISSN 0003-4975.)

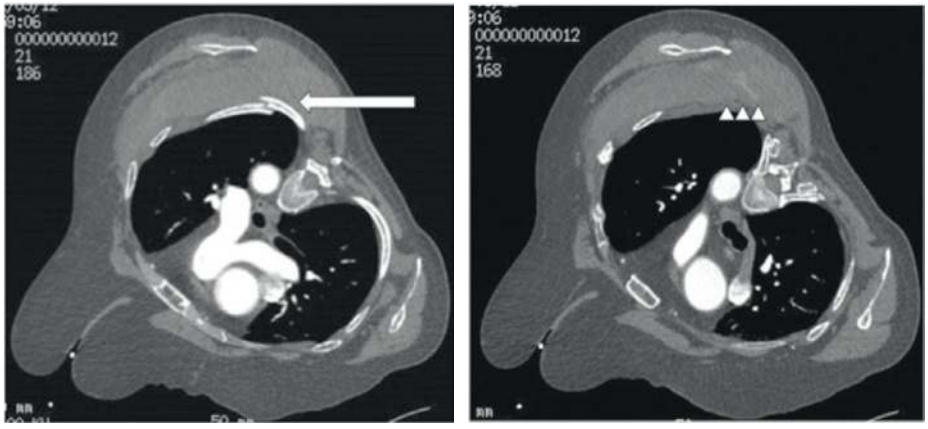

FIG 68.8 Computed tomography taken on day 26 with the patient in right lateral decubitus position because of severe left back pain. This scan reveals posterior fracture of the left sixth rib (*arrow*), and an enlargement of the chest wall hematoma with extravasation (*arrowhead*). (From Sato N, Sekiguchi H, Hirose Y, Yoshida S. Delayed chest wall hematoma caused by progressive displacement of rib fractures after blunt trauma. *Trauma Case Rep*. 2016;4:1–4.)

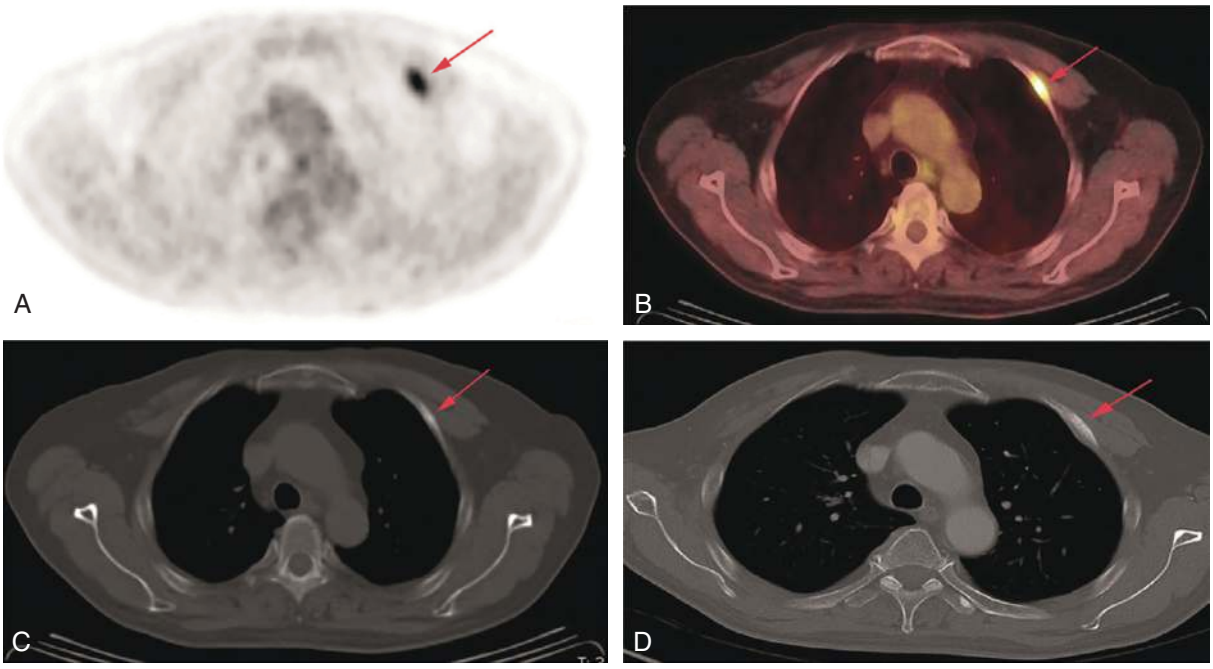

FIG 68.9 ^{18}F-fluorodeoxyglucose (FDG) positron emission tomography/computed tomography (PET/CT) scan of a 68-year-old male for restaging of nonsmall cell lung cancer after chemotherapy and radiation therapy. Discrete focal FDG uptake (SUVmax 5.2) was present in left second rib (*arrows*) on **(A)** PET and **(B)** PET/CT fusion axial images. Osteoblastic lesion was noted on **(C)** bone window setting CT image. After 9 months, osteoblastic lesion (*arrow*) showed progression even after chemotherapy on **(D)** chest CT images, and it was considered to be bony metastatic disease. (From Choi HS, Yoo IR, Park HL, et al. Role of ^{18}F-FDG PET/CT in differentiation of a benign lesion and metastasis on the ribs of cancer patients. *Clin Imag*. 2014;38(2):109114, Fig. 3. ISSN 0899-7071.)

TREATMENT

Initial treatment of rib fracture pain includes a combination of simple analgesics and nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors. If these medications do not adequately control the patient's symptoms, short-acting opioid analgesics such as hydrocodone are a reasonable next step. Because opioid analgesics have the potential to suppress the cough reflex and respiration, the patient must be closely monitored and instructed in adequate pulmonary toilet techniques. Transdermal lidocaine patches may also be used in conjunction with pharmacologic management of rib fracture pain.

The local application of heat and cold or the use of an elastic rib belt may also provide symptomatic relief. For patients who do not respond to these treatment modalities, injection using local anesthetic and steroid should be implemented to avoid pulmonary complications (Fig. 68.10).

The patient is placed in the prone position with the arms hanging loosely off the sides of the table. Alternatively, this injection technique can be performed with the patient in the sitting or lateral position. The rib to be blocked is identified by palpating its path at the posterior axillary line. The operator's index and middle fingers are placed on the rib, thus bracketing the site of needle insertion. The skin is prepared with antiseptic solution. A 1½-inch, 22-gauge needle is attached to a 12-mL syringe and is advanced perpendicular to the skin while aiming for the middle of the rib, between the operator's index and middle fingers. The needle should impinge on bone after being advanced approximately ¾ inch. Once bony contact is made, the needle is withdrawn into the subcutaneous tissues, and the skin and subcutaneous tissues are retracted with the palpating fingers inferiorly. This maneuver allows the needle to be walked off the inferior margin of the rib. As soon as bony contact is lost, the needle is slowly advanced approximately 2-mm deeper. This places the needle in proximity to the costal groove, which contains the intercostal nerve as well as the intercostal artery and vein. After careful aspiration reveals no blood or air, 3–5-mL of 1% preservative-free lidocaine is injected. If the pain has an inflammatory component, the local anesthetic is combined with 80-mg methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40-mg methylprednisolone for the initial 80-mg dose. Ultrasound needle guidance will decrease the incidence of needle-induced complications and improve the accuracy of needle placement.

Because of the overlapping innervation of the chest and upper abdominal wall, the intercostal nerves above and below the nerve suspected of causing the pain must also be blocked.

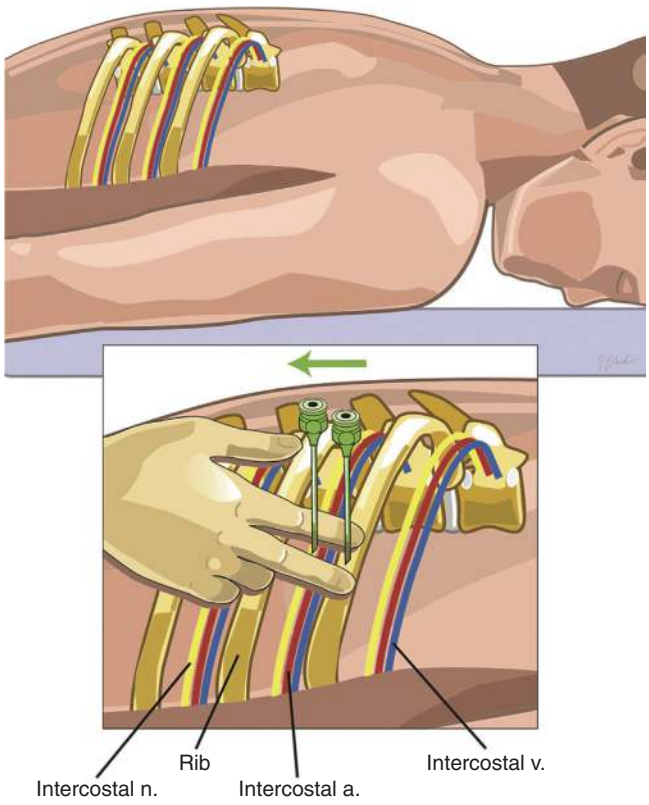

FIG 68.10 Injection technique for fractured ribs. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015.)

COMPLICATIONS AND PITFALLS

The major problem in the care of patients thought to be suffering from rib fracture is failure to identify potentially serious disorders of the thorax or upper abdomen, such as tumor, pneumothorax, or hemopneumothorax. Given the proximity of the pleural space, pneumothorax after intercostal nerve block is a distinct possibility. The incidence of this complication is less than 1%, but it occurs with greater frequency in patients with chronic obstructive pulmonary disease. Given the proximity to the intercostal nerve and artery, the clinician must carefully calculate the total dosage of local anesthetic administered, because vascular uptake by these vessels is high. Although uncommon, infection is always a possibility, especially in immunocompromised patients with cancer. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Rib fracture is a common cause of chest wall and thoracic pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious intrathoracic or intraabdominal disorders. Pharmacologic agents, including opioid analgesics, are usually adequate to control the pain of rib fracture. If necessary, intercostal nerve block is a simple technique that can produce dramatic pain relief. However, because of the proximity of the intercostal nerve to the pleural space, strict attention to technique is mandatory. The clinician should be aware of the direct correlation between the number of fractured ribs identified following trauma and mortality and morbidity.

SUGGESTED READINGS

Marco CA, Sorensen D, Hardman C, Bowers B, Holmes J, McCarthy MC. Risk factors for pneumonia following rib fractures. *Am J Emerg Med*. 2020;38(3):610–612.

Waldman SD. Abnormalities of the rib and intercostal space. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:535–544.

Waldman SD. Pneumothorax, pulmonary embolism, and other abnormalities of the lung. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:545–560.

Waldman SD. Fractured ribs. In: *Atlas of common pain syndromes*. 4th ed. Philadelphia: Elsevier; 2017:260–264.
Waldman SD. Intercostal nerve block. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:454–455.
Waldman SD. The intercostal nerves. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:113–114.
Waldman SD. Ultrasound-guided intercostal nerve block. In: *Waldman's comprehensive atlas of ultrasound-guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:653–661.
Waldman SD. Intercostal nerve block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:404–412.

Postthoracotomy Pain Syndrome

ICD-10 CODE R07.1

THE CLINICAL SYNDROME

Essentially all patients who undergo thoracotomy suffer from acute postoperative pain. This acute pain syndrome invariably responds to the rational use of systemic and spinal opioids, as well as intercostal nerve block. Unfortunately, in a few patients who undergo thoracotomy, the pain persists beyond the postoperative period and can be difficult to treat. The causes of postthoracotomy pain syndrome are listed in Box 69.1 and include direct surgical trauma, fractured ribs, compressive neuropathy, neuroma, and stretch injuries. When the syndrome is caused by fractured ribs, it produces local pain that is worse with deep inspiration, coughing, or movement of the affected ribs (Box 69.2). The other causes of the syndrome result in moderate to severe pain that is constant and follows the distribution of the affected intercostal nerves. The pain may be characterized as neuritic and may occasionally have a dysesthetic quality.

BOX 69.1 Causes of Postthoracotomy Pain Syndrome

- Direct surgical trauma to the intercostal nerves
- Fractured ribs resulting from the use of the rib spreader
- Compressive neuropathy of the intercostal nerves resulting from direct compression by retractors
- Stretch injuries to the intercostal nerves at the costovertebral junction

SIGNS AND SYMPTOMS

Physical examination generally reveals tenderness along the healed thoracotomy incision (Figs. 69.1 and 69.2). Occasionally, palpation of the scar elicits paresthesias, a finding suggestive of neuroma formation. Patients suffering from postthoracotomy syndrome may attempt to splint or protect the affected area. Careful sensory examination of the affected dermatomes may reveal decreased sensation or allodynia. With significant motor involvement of the subcostal nerve, patients may complain that the abdomen bulges outward. Occasionally, patients suffering from postthoracotomy syndrome develop reflex sympathetic dystrophy of the ipsilateral upper extremity that, if left untreated, may result in a frozen shoulder.

TESTING

For all patients who present with pain that is thought to be emanating from the intercostal nerve, plain radiographs are indicated to rule out occult bony disorders, including unsuspected fracture or tumor (Fig. 69.3). Radionuclide bone scanning may be useful to exclude occult fractures of the ribs or sternum. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Computed tomography scanning of the thoracic contents is indicated if an occult mass or pleural disease is suspected (Fig. 69.4). The injection technique described

BOX 69.2 Complications Following Thoracotomy

- Early postoperative complications
  - Pulmonary edema
  - Acute lung injury/acute respiratory distress syndrome
  - Pneumonia
  - Hemorrhage/hemothorax
  - Chylothorax
  - Dehiscence of bronchial stump, formation of bronchopleural fistula
  - Esophagopleural fistula
  - Empyema
  - Lobar torsion
  - Cardiac herniation
  - Gossypiboma
- Late postoperative complications
  - Pneumonia
  - Disease recurrence (tumor, infection)
  - Dehiscence of bronchial stump, formation of bronchopleural fistula
  - Esophagopleural fistula
  - Empyema
  - Stricture of bronchial anastomosis
  - Pulmonary artery stump thrombosis
  - Postpneumonectomy syndrome
  - Herniation of lung or chest wall soft tissues via thoracotomy defect
  - Gossypiboma

Modified from Alpert JB, Godoy MCB, De Groot PM, et al. Imaging of the post-thoracotomy patient: anatomic changes and postoperative complications. *Radiol Clin North Am.* 2014:52(1):85–103.

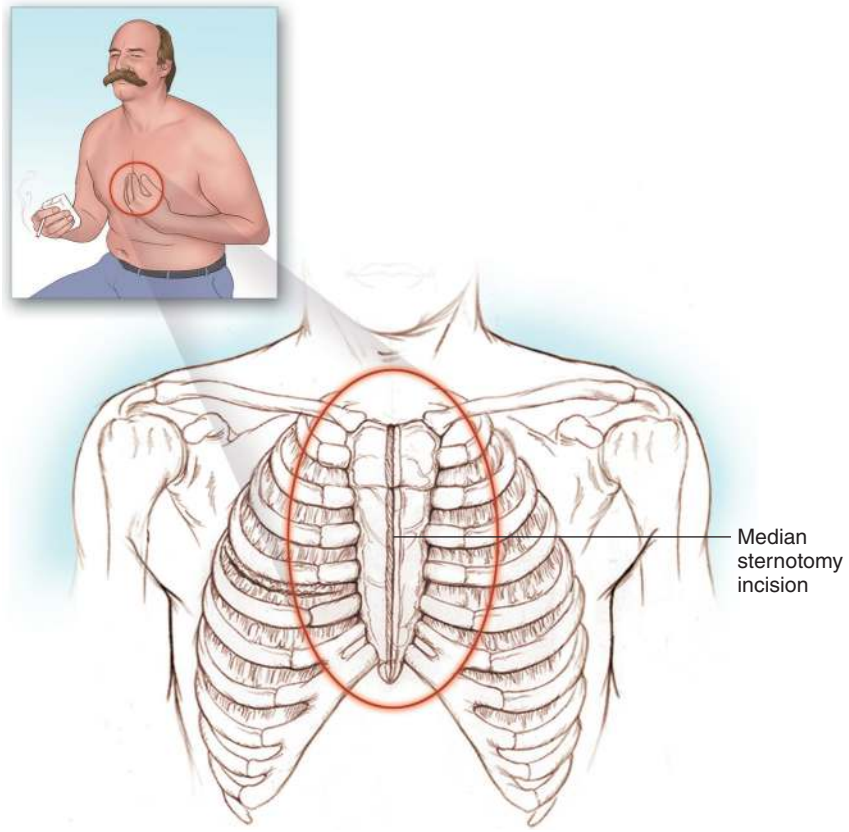

FIG 69.1 Patients with postthoracotomy syndrome exhibit tenderness to palpation of the scar.

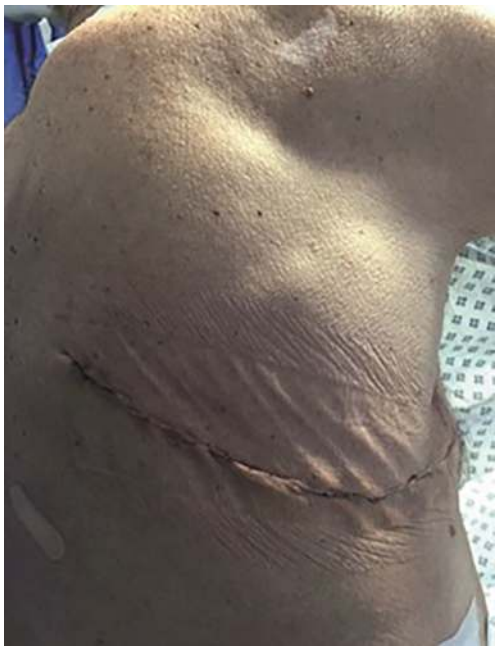

FIG 69.2 Healing thoracotomy incision. (From Raza A, Alzetani A. Principles of posterolateral thoracotomy and pneumonectomy. *Surgery (Oxford)*. 2014;32(5):266271, Fig. 1. ISSN 0263-9319. https://doi.org/10.1016/j.mpsur.2014.03.001)

later serves as both a diagnostic and a therapeutic maneuver. Electromyography is useful in distinguishing injury of the distal intercostal nerve from stretch injuries of the intercostal nerve at the costovertebral junction.

DIFFERENTIAL DIAGNOSIS

The pain of postthoracotomy syndrome may be mistaken for pain of cardiac or gallbladder origin, thus leading to visits to the emergency department and unnecessary cardiac and gastrointestinal workups. In the presence of trauma, postthoracotomy syndrome may coexist with fractured ribs or fractures of the sternum itself, which can be missed on plain radiographs and may require radionuclide bone scanning for proper identification. Tietze's syndrome, which is painful enlargement of the upper costochondral cartilage associated with viral infection, may be confused with postthoracotomy syndrome.

Neuropathic pain involving the chest wall may also be confused or coexist with postthoracotomy syndrome. Examples of such neuropathic pain syndromes include diabetic polyneuropathies and acute herpes zoster involving the thoracic nerves. Diseases of the structures of the mediastinum are possible and may be difficult to diagnose. Pathologic processes that inflame the pleura, such as pulmonary embolus,

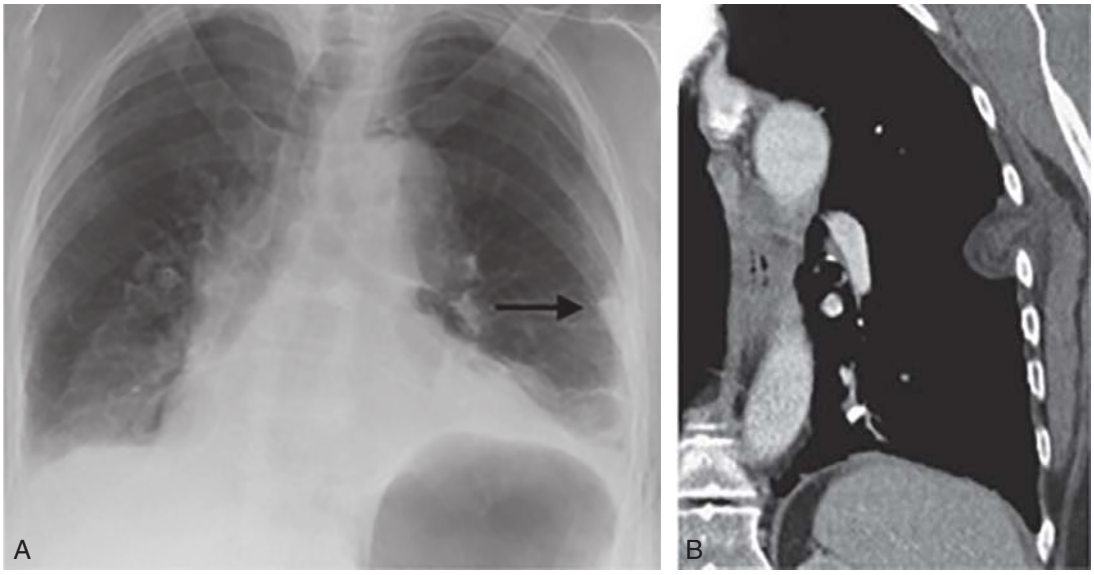

FIG 69.3 A, Frontal chest radiograph demonstrates a smoothly marginated opacity at the lateral margin of the left midlung (*arrow*), initially worrisome for a pleural mass such as metastasis. B, Corresponding coronal computed tomography image illustrates herniation of fat and soft tissue through the chest wall into the extrapleural space at a level corresponding to prior surgery. (From Alpert JB, Godoy MCB, DeGroot PM, et al. Imaging the post-thoracotomy patient: anatomic changes and postoperative complications. *Radiol Clin North Am.* 2014;52(1):85103, Fig. 15. ISSN 0033-8389, ISBN 9780323264105. https://doi.org/10.1016/j.rcl.2013.08.008)

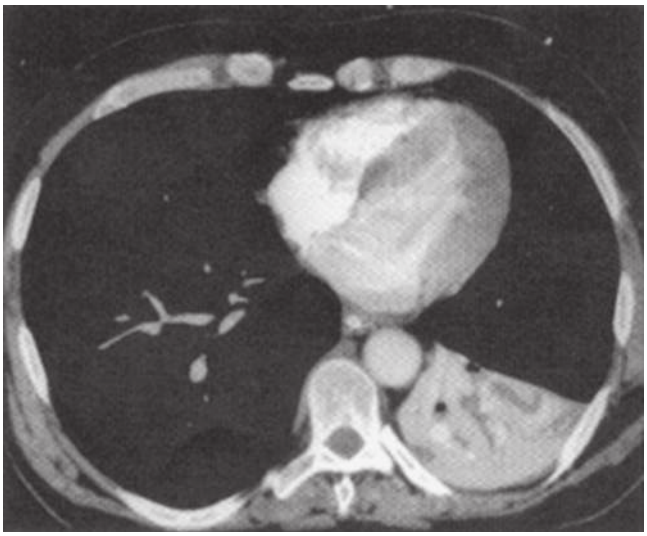

FIG 69.4 Computed tomography scan showing left lower lobe atelectasis. Some of the bronchi are open (air filled), and others are plugged (mucus filled). (From Grainger RG, Allison DJ, Adam A, Dixon AK. *Grainger & Allison's diagnostic radiology: a textbook of medical imaging*. 4th ed. Philadelphia: Churchill Livingstone; 2002.)

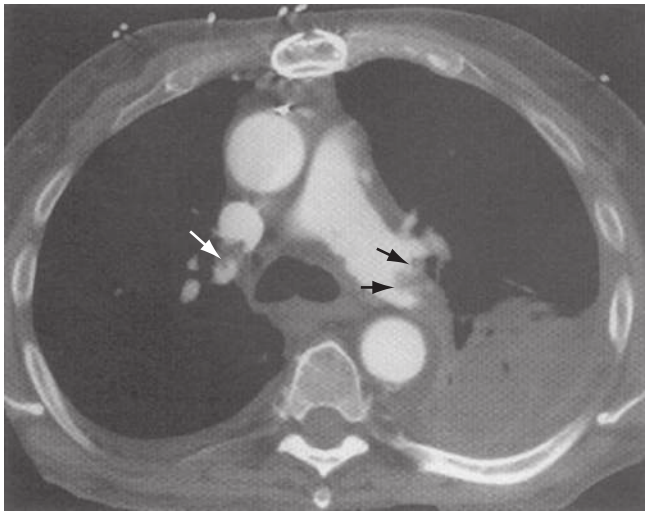

FIG 69.5 Computed tomography demonstrates bilateral pulmonary emboli in the presence of left lower lobe consolidation and bilateral pleural effusions. A large embolus is visible in the left main pulmonary artery (*black arrows*), and a small embolus is evident in the proximal right upper lobe pulmonary artery (*white arrow*). (From Grainger RG, Allison DJ, Adam A, Dixon AK. *Grainger & Allison's diagnostic radiology: a textbook of medical imaging*. 4th ed. Philadelphia: Churchill Livingstone; 2002.)

infection, and Bornholm disease, may also confuse the diagnosis and complicate treatment (Fig. 69.5).

TREATMENT

Initial treatment of postthoracotomy syndrome includes a combination of simple analgesics and nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors. If these medications do not adequately control the patient's symptoms, a tricyclic antidepressant or gabapentin should be added.

Traditionally, tricyclic antidepressants have been a mainstay in the palliation of pain caused by postthoracotomy

syndrome. Controlled studies have demonstrated the efficacy of amitriptyline, and nortriptyline and desipramine have also proved to be clinically useful. Unfortunately, this class of drugs is associated with significant anticholinergic side effects, including dry mouth, constipation, sedation, and urinary retention. These drugs should be used with caution in patients suffering from glaucoma, cardiac arrhythmia, and prostatism. To minimize side effects and encourage compliance, the physician should start amitriptyline or nortriptyline at a 10-mg dose at bedtime; the dose can then be titrated upward to 25 mg at bedtime, as side effects allow. Subsequently, upward titration in 25-mg increments can be carried out each week, as side effects allow. Even at lower doses, patients generally report a rapid reduction in sleep disturbance and begin to experience some pain relief in 10–14 days. If the patient does not show any reduction in pain as the dose is being titrated upward, the addition of gabapentin alone or in combination with nerve blocks is recommended (see later). The selective serotonin reuptake inhibitors, such as fluoxetine, have also been used to treat the pain of postthoracotomy syndrome, and although these drugs are better tolerated than are the tricyclic antidepressants, they appear to be less efficacious.

If the antidepressant compounds are ineffective or contraindicated, gabapentin is a reasonable alternative. Gabapentin is started at a 300-mg dose at bedtime for two nights. The patient should be cautioned about potential side effects, including dizziness, sedation, confusion, and rash. The drug is then increased in 300-mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a total dosage of 2400 mg/day is reached. At this point, if the patient has experienced partial pain relief, blood values are measured, and the drug is carefully titrated upward using 100-mg tablets. Rarely is a dose greater than 3600 mg/day required. Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function. Low-dose ketamine has also been recommended as an alternative to the above medications in the management of postthoracotomy pain.

The local application of heat and cold or the use of an elastic rib belt may also provide symptomatic relief. Concurrent use of a transdermal lidocaine patch may also offer additional pain relief in patients suffering from postthoracotomy pain. For patients who do not respond to these treatment modalities, injection using local anesthetic and steroid is a reasonable next step.

The patient is placed in the prone position with the arms hanging loosely off the sides of the table. Alternatively, this block can be done with the patient in the sitting or lateral position. The rib to be blocked is identified by palpating its path at the posterior axillary line. The operator's index and middle fingers are placed on the rib, thus bracketing the site of needle insertion. The skin is prepared with antiseptic solution. A 1½-inch, 22-gauge needle is attached to a 12-mL syringe and is advanced perpendicular to the skin while aiming for the middle of the rib between the operator's index and middle fingers. The needle should impinge on bone after being advanced approximately ¾ inch. Once bony contact is made, the needle is withdrawn into the subcutaneous tissues and the skin and subcutaneous tissues are retracted with the palpating fingers inferiorly. This maneuver allows the needle to be walked off the inferior margin of the rib. As soon as bony contact is lost, the needle is slowly advanced approximately 2-mm deeper. This places the needle in proximity to the costal groove, which contains the intercostal nerve as well as the intercostal artery and vein. After careful aspiration reveals no blood or air, 3–5 mL of 1% preservative-free lidocaine is injected. If the pain has an inflammatory component, the local anesthetic is combined with 80-mg methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40-mg methylprednisolone for the initial 80-mg dose. Because of the overlapping innervation of the chest and upper abdominal wall, the intercostal nerves above and below the nerve suspected of causing the pain must also be blocked. Ultrasound needle guidance will decrease the incidence of pneumothorax and other needle-related complications. Thoracic epidural block, paravertebral block in intrapleural analgesia, and wound infiltration may also provide relief of postthoracotomy pain. For persistent pain, cryoneurolysis of the intercostal nerves can be considered (Fig. 69.6).

If postthoracotomy pain syndrome is caused by stretch injury of the intercostal nerve (identified by electromyography), it may respond to thoracic epidural nerve block with steroid.

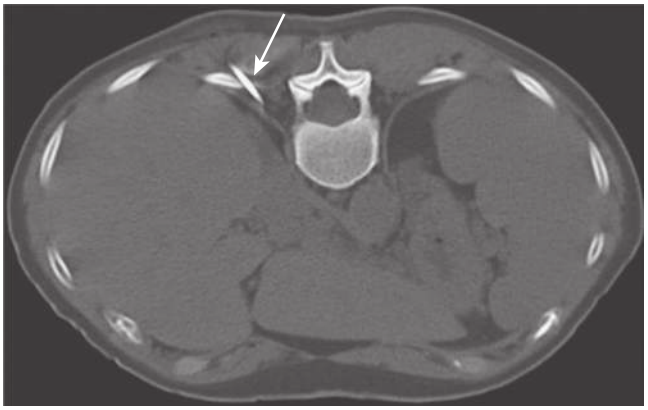

FIG 69.6 Axial computed tomographic image of a 45-year-old woman with history of postthoracotomy pain syndrome. The interprocedural computed tomographic image shows a cryoablation probe (*arrow*) placed just inferior to the 12th right posterior rib. (From Moore W, Kolnick D, Tan J, Yu HS. CT guided percutaneous cryoneurolysis for post-thoracotomy pain syndrome: early experience and effectiveness. *Acad Radiol.* 2010;17(5):603–606.)

COMPLICATIONS AND PITFALLS

The major problem in the care of patients thought to be suffering from postthoracotomy syndrome is failure to identify potentially serious disorders of the thorax or upper abdomen. Given the proximity of the pleural space, pneumothorax after intercostal nerve block is a distinct possibility. The incidence of this complication is less than 1%, but it occurs with greater frequency in patients with chronic obstructive pulmonary disease. Given the proximity to the intercostal nerve and artery, the clinician must carefully calculate the total dosage of local anesthetic administered, because vascular uptake by these vessels is high. Although uncommon, infection is always a possibility, especially in immunocompromised patients with cancer. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Postthoracotomy syndrome is a common cause of chest wall and thoracic pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious intrathoracic or intraabdominal disorders. Pharmacologic agents are usually adequate to control the pain of postthoracotomy syndrome. If necessary, intercostal nerve block is a simple technique that can produce dramatic pain relief. However, because of the proximity of the intercostal nerve to the pleural space, strict attention to technique is mandatory.

SUGGESTED READINGS

Moore W, Kolnick D, Tan J, et al. CT guided percutaneous cryoneurolysis for post-thoracotomy pain syndrome: early experience and effectiveness. *Acad Radiol*. 2010;17(5):603–606.

Niraj G, Kelkar A, Kaushik V, et al. Audit of postoperative pain management after open thoracotomy and the incidence of chronic postthoracotomy pain in more than 500 patients at a tertiary center. *J Clin Anesth*. 2017;36:174–177.

Romero A, Garcia JEL, Joshi GP. The state of the art in preventing postthoracotomy pain. *Semin Thorac Cardiovasc Surg*. 2013;25(2):116–124.

Waldman SD. Post-thoracotomy pain syndrome. In: *Pain review*. Philadelphia: Saunders; 2017:283–284.

Yoshimura N, Iida H, Takenaka M, et al. Effect of postoperative administration of pregabalin for post-thoracotomy pain: a randomized study. *J Cardiothorac Vasc Anesth*. 2015;29(6):1567–1572.

Pulmonary Embolus

ICD-10 CODE 126.9

THE CLINICAL SYNDROME

Pulmonary embolism is a serious and potentially life-threatening disease. Diagnosis of pulmonary embolism is challenging in that the identification of the classic triad of pulmonary embolism—sudden onset of pleuritic chest pain, shortness of breath, and hypoxia—is often difficult (Fig. 70.1). The fact that 40% of patients who die from pulmonary embolus have seen a physician in the weeks prior to their death hammers home this fact. Pulmonary embolism can be divided into thombotic (e.g., acute pulmonary thromboembolism) and nonthrombotic (e.g., cement or foreign body pulmonary embolization) (Box 70.1).

Risk factors that predispose the patient to pulmonary embolism include thrombophlebitis, immobilization, prolonged travel, venous stasis, hypercoagulable states, trauma, surgery, malignancy, oral contraceptives, and pregnancy (Fig. 70.2 and Box 70.2). Clinical-based scoring systems such as the modified Wells score and Geneva score combined with

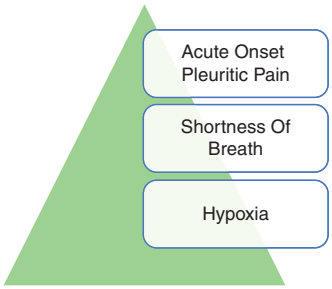

FIG 70.1 The classic presentation triad of pulmonary embolus.

BOX 70.1 Nonthrombotic Pulmonary Embolism

- Septic pulmonary embolism
- Macroscopic tumor pulmonary embolism
- Microscopic tumor pulmonary embolism
- Fat pulmonary embolism
- Foreign body pulmonary embolism
- Polymethylmethacrylate cement pulmonary embolism
- Air pulmonary embolism
- Talc pulmonary embolism
- Iodinated oil pulmonary embolism
- Hydatid pulmonary embolism
- Mercury pulmonary embolism

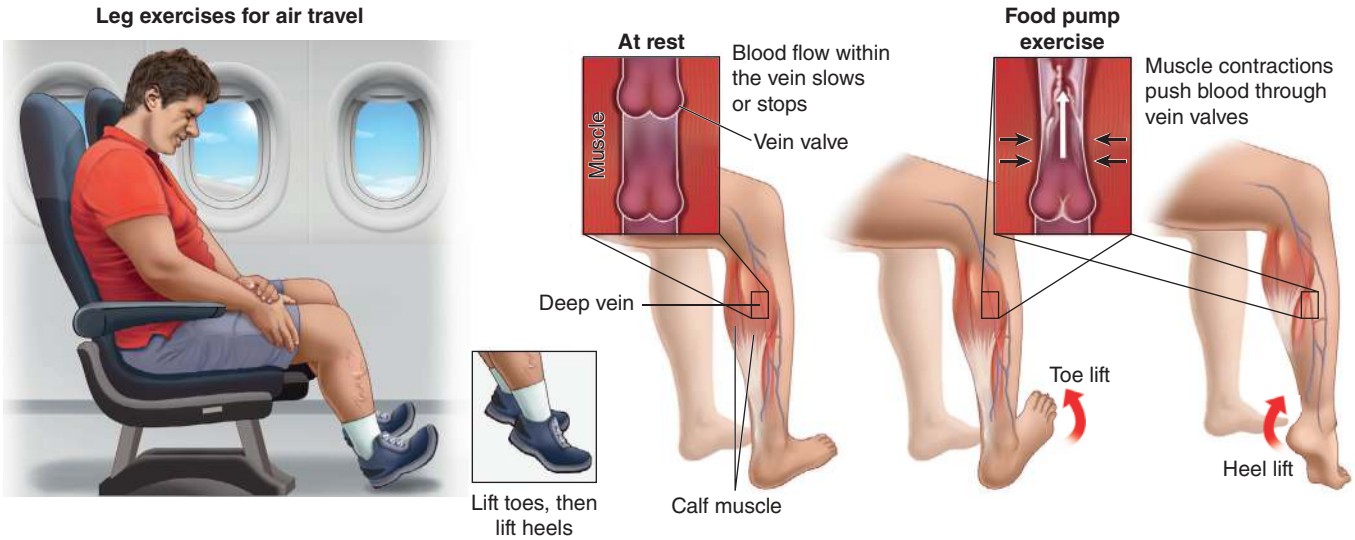

FIG 70.2 Risk factors that predispose the patient to pulmonary embolism include thrombophlebitis, immobilization, prolonged travel, venous stasis, hypercoagulable states, trauma, surgery, malignancy, oral contraceptives, and pregnancy.

BOX 70.2 Risk Factors for Pulmonary Embolism

- Thrombophlebitis
- Immobilization
- Travel of 4 hours or more
- Surgery within the last 3 months
- Venous stasis
- Hypercoagulable states
- Trauma, especially with long bone fractures
- Pregnancy
- Oral contraceptives
- Estrogen replacement
- Pregnancy
- Malignancy
- Varicose veins
- Venous pacemakers
- Hyperlipidemias
- Congenital diseases, including homocystinuria
- Thrombocytosis
- Inflammatory bowel disease
- IV drug abuse
- Drug-induced lupus anticoagulant
- Hemolytic anemias
- Heparin-associated thrombocytopenia

D-dimer testing will increase diagnostic accuracy when a patient is thought to be suffering from thrombotic pulmonary embolism (Table 70.1).

SIGNS AND SYMPTOMS

The physical findings associated with pulmonary embolism are quite variable and are driven by three variables: (1) the acuity of the embolus/emboli; (2) the magnitude of the embolus/emboli; and (3) the prior cardiopulmonary status of the patient (see Fig. 70.1). Although the patient presenting with the classic triad of signs and symptoms—sudden onset of pleuritic chest pain, shortness of breath, and hypoxia—is relatively straightforward, especially if risk factors are identified in the patient's history, it should be remembered that most patients with symptomatic deep venous thrombosis present with no pulmonary symptoms. With significant pulmonary embolism, symptoms may range from progressive dyspnea to catastrophic hemodynamic collapse. As mentioned above, prior cardiopulmonary disease may magnify the extent of the cardiopulmonary compromise. In some patients with pulmonary embolism, atypical symptoms, including syncope, hemoptysis, cough, seizures, alterations of consciousness, wheezing, or fever, may be the presenting feature of the patient's illness. The presence of tachypnea, tachycardia, râles, increased second heart sound, S3 or S4 gallop, fever, and cyanosis in the absence of an obvious explanation should heighten the clinician's index of suspicion for the diagnosis of pulmonary embolus, even in the absence of obvious risk factors.

TESTING

Although routine laboratory tests are not specific in the diagnosis of pulmonary embolism, they may aid in ruling in or ruling out diseases being considered in the differential diagnosis. Screening tests for hypercoagulable states including antithrombin III deficiency, connective tissue disorders, occult malignancy are indicated. D-Dimer testing measures the degradation product of plasmin-mediated proteases of cross-linked fibrin. High-sensitivity D-dimer testing such as the quantitative rapid assay (enzyme-linked immunosorbent assay) has a sensitivity or 0.95% and a negative likelihood in the presence of patients with a suspected pulmonary embolism ratio of 0.13 which is similar to that of testing on normal controls. This means that a negative high-sensitivity D-dimer test indicates a low probability of venous thromboembolism. When high-sensitivity D-dimer testing is combined with clinical-based scoring systems, the diagnostic accuracy is increased. It should be noted that because of the poor specificity of D-dimer testing, positive D-dimer results should only be used to heighten the clinician's suspicion of venous thromboembolic disease and that clinical judgement is of utmost important when considering the differential diagnosis. Ischemia-modified albumin levels are less readily available than D-dimer testing in some clinical settings but have a sensitivity of 93% and a specificity of 75% in the diagnosis of pulmonary embolism. Again, clinical judgement is crucial when interpreting the results of this test. A low arterial PO_2 in patients suspected of suffering from pulmonary embolus who do not have preexisting pulmonary or cardiac disease should further raise the index of suspicion. Elevated troponin and brain natriuretic peptide are also associated with pulmonary embolus but are nonspecific.

TABLE 70.1 The Modified Wells Criteria for the Diagnosis of Thrombotic Pulmonary Embolism

Features	Score (Points)
Clinical signs and symptoms of DVT	3.0
No alternative diagnosis	3.0
Heart rate >100 beats/min	1.5
Immobilization ≥3 days or surgery in the previous 4 wk	1.5
Previous DVT or PE	1.5
Hemoptysis	1.0
Malignancy with active treatment in the past 6 months or under palliative care	1.0
Pretest clinical probability	
PE unlikely	≤4.0
PE likely	>4.0

DVT, Deep vein thrombosis; *PE*, pulmonary embolism.

Venography has historically been the gold standard for the diagnosis of deep venous thrombosis, but improvements in ultrasonography and computed tomography (CT) angiography have decreased its use (Figs. 70.3 and 70.4). The diagnostic accuracy of angiography and CT angiography approaches 100%, although some investigators suggest that it has led to overdiagnosis of pulmonary embolus (Fig. 70.5). CT angiography may also be useful in the characterization as to the nature of the pulmonary embolus (e.g., thrombotic or nonthrombotic) (Figs. 70.6–70.9). Ventilation–perfusion scanning may be useful in the diagnosis of pulmonary embolus if there is a contraindication to the use of intravenous contrast material.

Chest X-rays are frequently positive in a patient with pulmonary embolus, but the findings are nonspecific; however, like routine laboratory testing, the results may provide the clinician with alternative diagnosis.

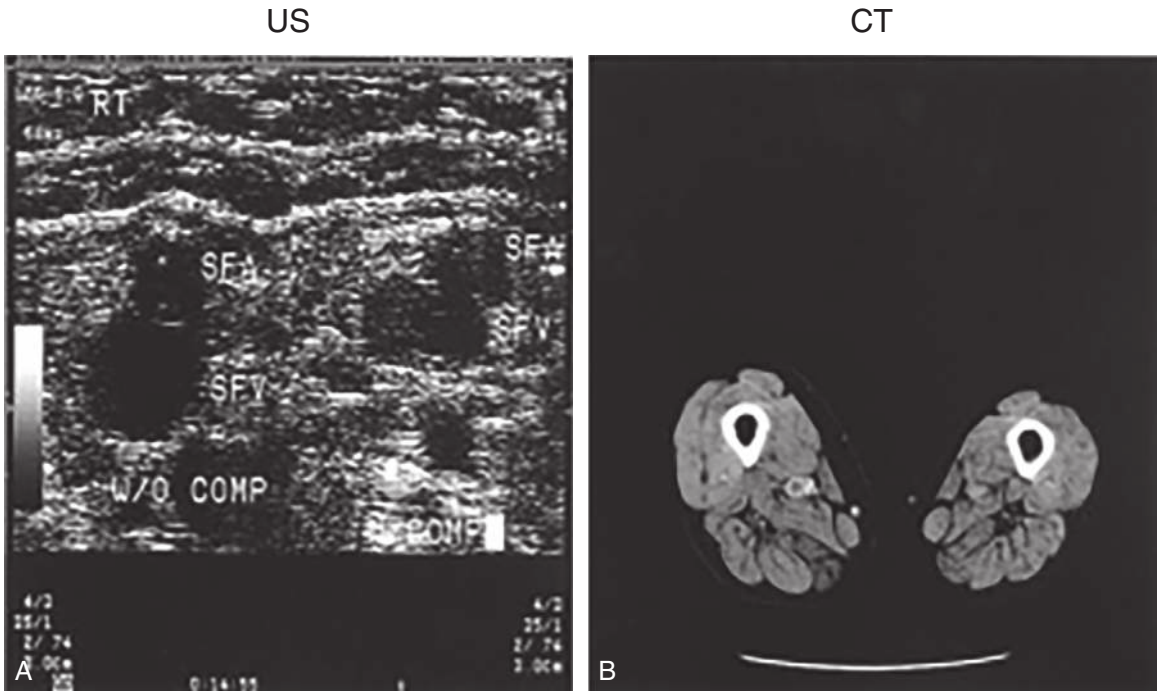

FIG 70.3 Concordant results: positive US and positive CTV (true positive). **A,** Noncompressible femoral vein shown by means of US. **B,** Filling defect in the femoral vein shown by means of CTV in the same patient. *CTV*, Computerized tomographic venography; *US*, ultrasound. (From Peterson DA, Kazerooni EA, Wakefield TW, et al. Computed tomographic venography is specific but not sensitive for diagnosis of acute lower-extremity deep venous thrombosis in patients with suspected pulmonary embolus. *J Vasc Surg*. 2001;34(5):798–804.)

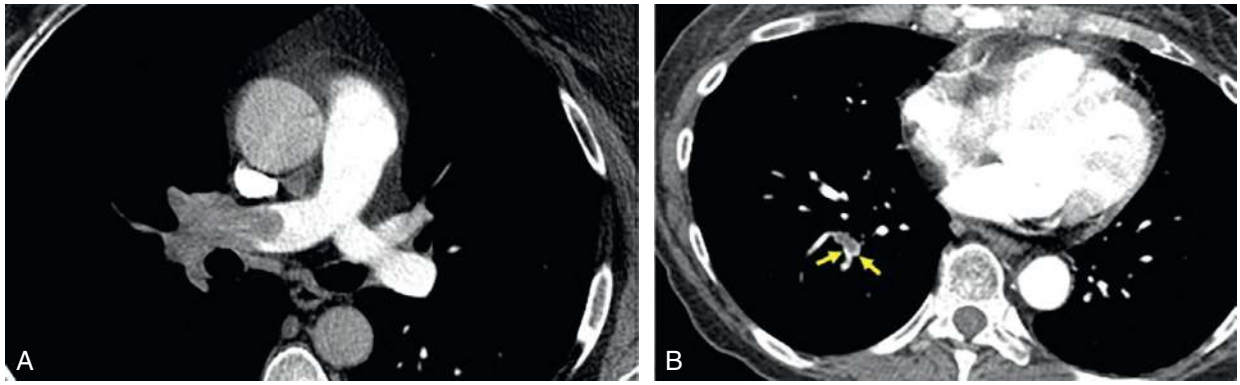

FIG 70.4 Axial computed tomography pulmonary angiography demonstrating classical pulmonary thromboembolism. **A,** Massive central pulmonary embolism with complete filling defect and obstruction of the main right and lobar pulmonary arteries. **B,** Segmental pulmonary embolism with partial central filling defect surrounded by a rim of contrast material (*arrows*) in the right lower lobe. (From Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J*. 2018;69(3):328–337.)

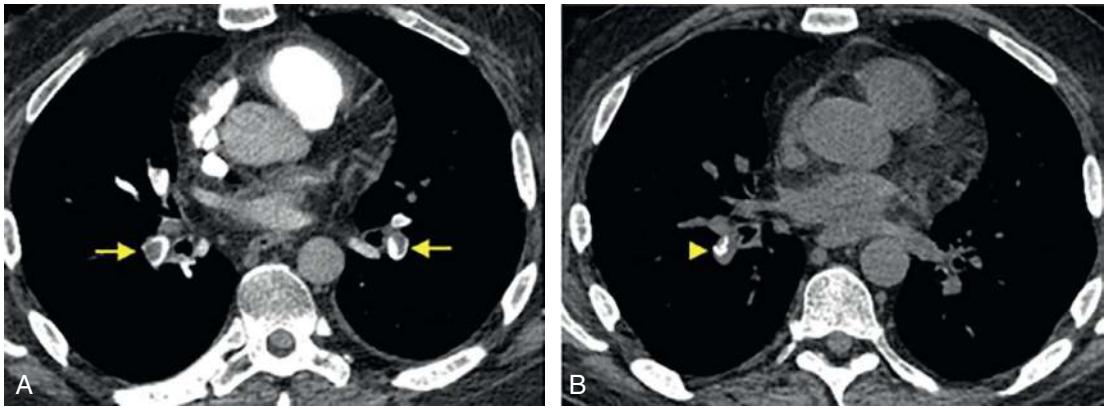

FIG 70.5 A 47-year-old man with recent diagnosis of acute lymphocytic leukemia. **A,** Initial computed tomography pulmonary angiography demonstrates acute pulmonary emboli (*arrows*). **B,** Follow-up nonenhanced chest computed tomography performed 9 months later demonstrates eccentric intraluminal calcification in the right interlobar pulmonary artery (*arrowhead*), consistent with chronic pulmonary embolus. (From Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J*. 2018;69(3):328–337.)

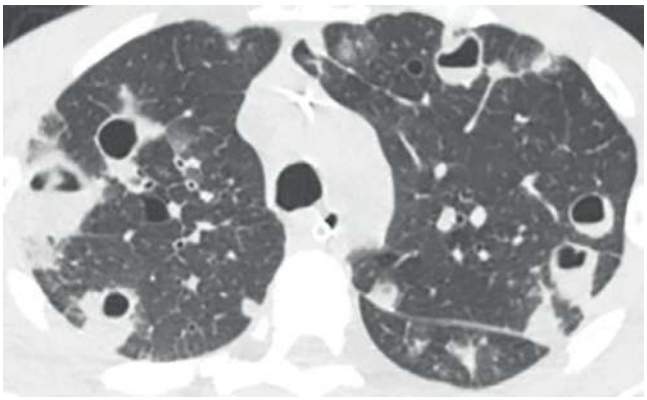

FIG 70.6 A 40-year-old man with recent diagnosis of bacterial endocarditis. Chest computed tomography demonstrates multiple bilateral irregular pulmonary nodules with cavitations, consistent with septic emboli. (From Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J*. 2018;69(3):328–337.)

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pulmonary embolus is extensive due to the variability in the clinical presentation of pulmonary embolism. Because it has been estimated that the diagnosis of pulmonary embolus is missed in over 400,000 patients per year in the United States alone, the imperative for correct diagnosis looms large. The clinician should avoid attributing symptoms to the disorders listed in Box 70.3 without first confirming the diagnosis and ruling out the possibility of pulmonary embolism.

TREATMENT

The choice of treatment for pulmonary embolus is driven by the type of pulmonary embolus (e.g., thrombotic or nonthrombotic), the presence of comorbidities, and the level of expertise available. Therapeutic considerations include thrombolytic therapy, anticoagulation, embolectomy, removal of foreign bodies, vena cava filters, and supportive care. Thrombolytic therapy is indicated in those patients with acute pulmonary embolic hypotension trending toward cardiogenic shock. In patients who are hemodynamically stable, anticoagulation is with intravenous unfractionated, low-molecular-weight heparin, or fondaparinux. Unfractionated heparin is preferred by many clinicians when medical or surgical procedures (e.g., vena cava filters) are being considered due the drug's shorter half life and ability to reverse its effects. Direct thrombin inhibitors including dabigatran and factor Xa inhibitors including apixaban, edoxaban, and rivaroxaban may also be used to prevent recurrent pulmonary embolism. Embolectomy may be considered in patients with massive pulmonary embolism or in those patients in whom fibrinolysis is contraindicated. Vena caval filters have limited indications in the setting of acute pulmonary embolism. These indications include patients with an absolute contraindication to anticoagulation (e.g., recent hemorrhagic stroke, surgery) and in patients with massive pulmonary embolus who will not survive additional emboli.

COMPLICATIONS AND PITFALLS

Massive pulmonary embolus is a life-threatening diagnosis that is often fatal in spite of best efforts to treat. The prognosis for patients with systemic hypotension, hypoxia, under-perfused extremities, and oliguria is extremely poor. Approximately 10 of patients suffering from a peripheral occlusion of the pulmonary artery will suffer acute pulmonary infarction which is often misdiagnosed as acute myocardial infarction.

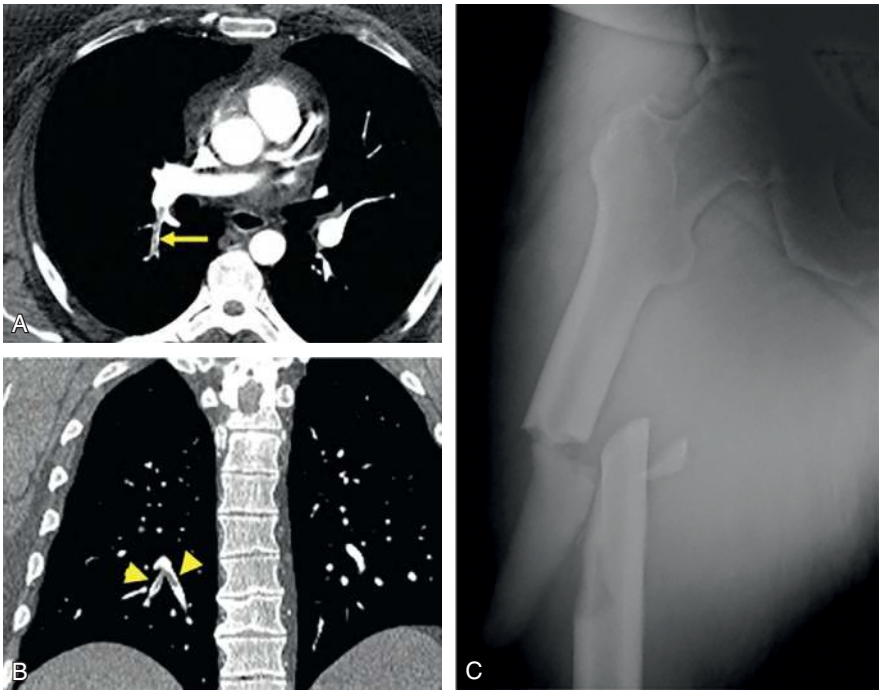

FIG 70.7 A 30-year-old man with fat pulmonary embolus after high velocity trauma. **A,** Axial image of computed tomography pulmonary angiography shows a long filling defect in the right lower lobe pulmonary artery (*arrow*). **B,** Coronal reconstruction of the computed tomography pulmonary angiography shows extension of filling defect in the segmental pulmonary arteries (*arrowheads*). **C,** Radiograph of the femur shows a comminuted, displaced fracture of the right femoral diaphysis. (From Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J*. 2018;69(3):328–337.)

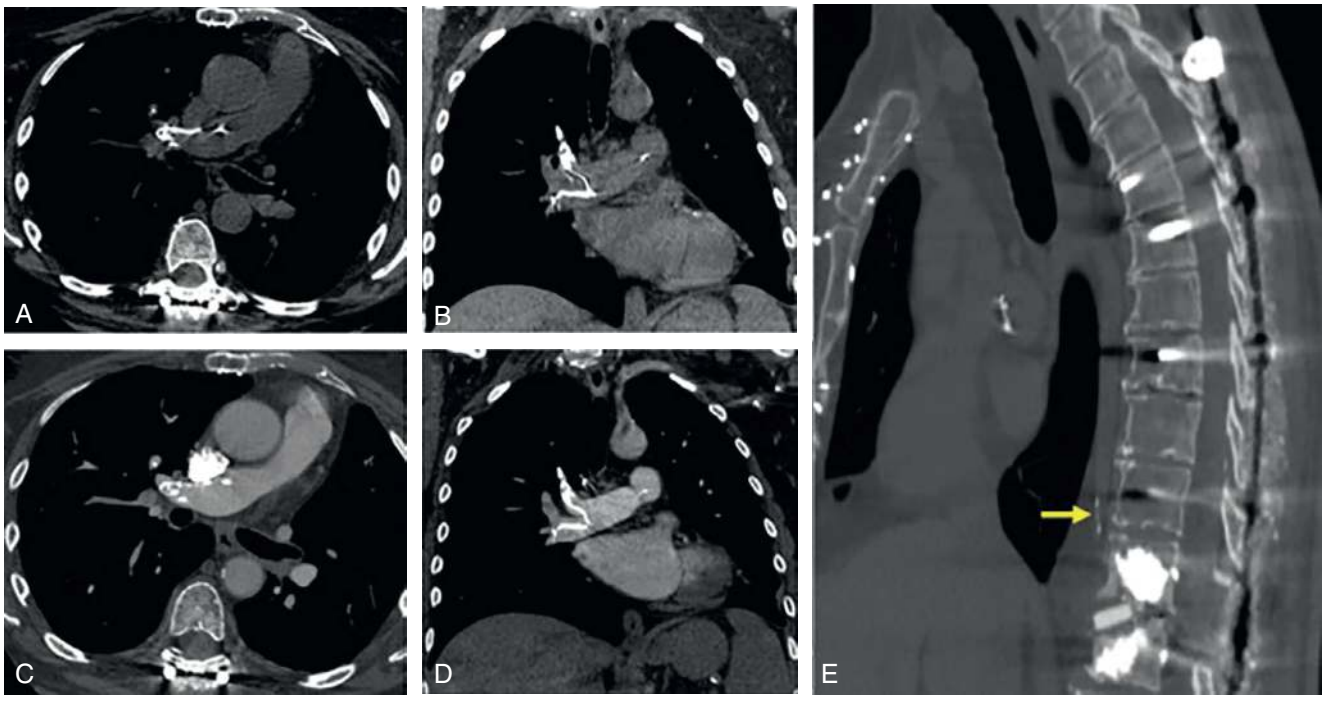

FIG 70.8 A 71-year-old woman status postextensive scoliosis corrective surgery. Axial and coronal images of nonenhanced computed tomography **(A, B)** and computed tomography pulmonary angiography **(C, D)** demonstrate linear, branching hyperdense filling defects in the central pulmonary arteries, extending into several right lobar pulmonary arteries, in keeping with vertebroplasty cement pulmonary embolization. **E,** Sagittal nonenhanced computed tomography image shows sequela of extensive spinal surgery and vertebroplasties of two lower dorsal vertebrae; the thin line of cement is tracking from the T11 vertebral body through a small vein (*arrow*) into the azygos vein. (From Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J*. 2018;69(3):328–337.)

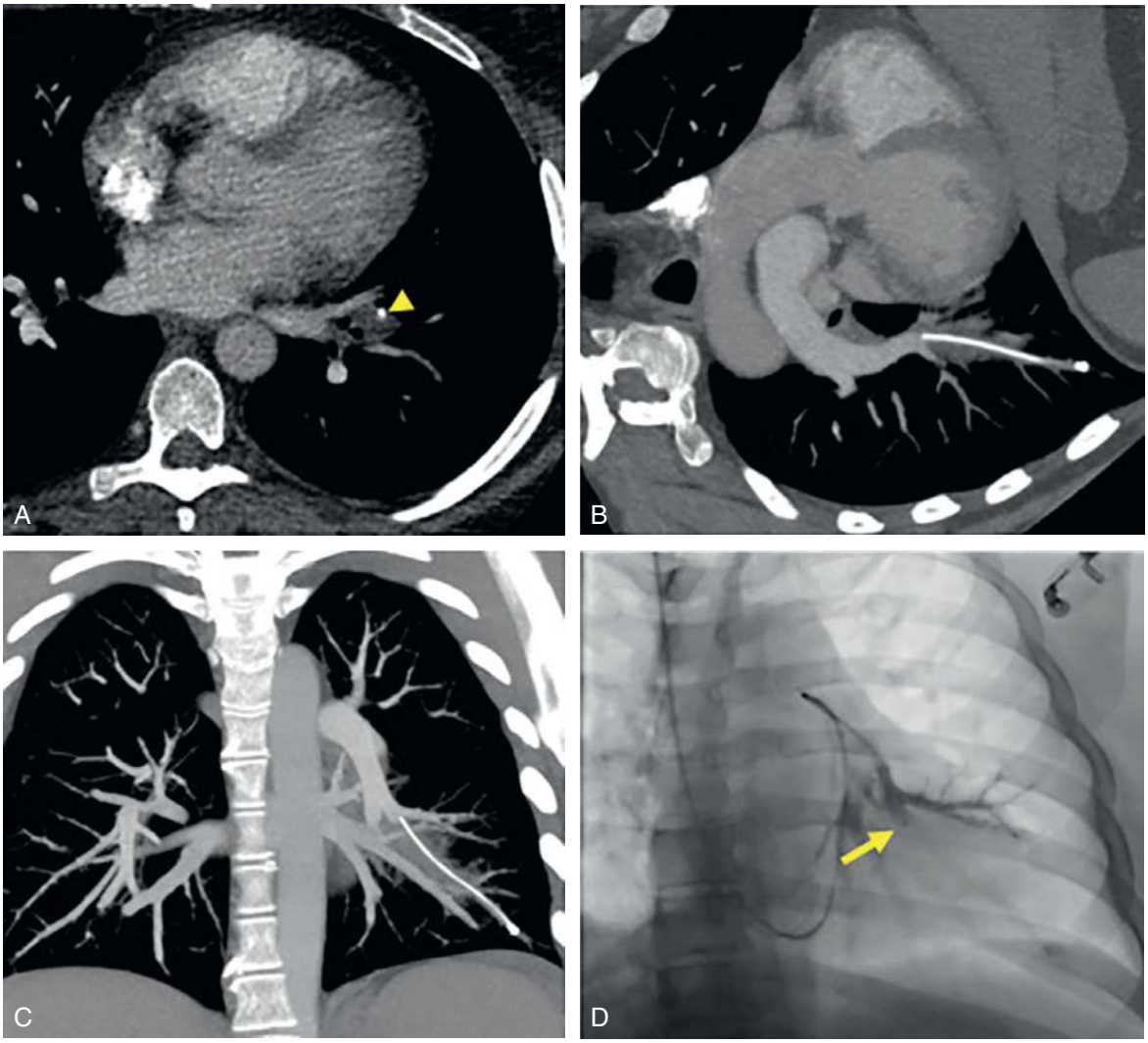

FIG 70.9 A 43-year-old woman with distal embolization of a fragment of a broken port-a-cath. **A,** Axial image of computed tomography pulmonary angiography shows thrombosis of the left lower lobe basal anteromedial segmental pulmonary artery, which also contains a central hyperdensity (*arrowhead*). The **(B)** axial oblique and **(C)** coronal oblique maximal intensity projection reformations show the entire length of the endovascular foreign body surrounded by a thrombus. **D,** Single fluoroscopic image obtained during an unsuccessful retrieval attempt of the fractured catheter confirms a complete occlusion of the left lower lobe anteromedial segmental pulmonary artery (*arrow*). (From Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J.* 2018;69(3):328–337.)

BOX 70.3 Differential Diagnosis of Pulmonary Embolus

- Acute respiratory distress syndrome
- Pneumonia
- Acute myocardial infarction
- Acute pericarditis
- Panic attacks
- Sudden cardiac death
- Syncope
- Anxiety disorders
- Aortic stenosis
- Atypical angina
- Atrial fibrillation
- Septic shock
- Pneumothorax
- Restrictive cardiomyopathy
- Cor pulmonale
- Cardiac failure
- Emphysema
- Mitral stenosis
- Idiopathic pulmonary arterial hypertension
- Pulmonary arteriovenous malformation (PAVM)
- Pulmonary arterial hypertension

CLINICAL PEARLS

To improve the clinical outcome for patients suffering from pulmonary embolus, the clinician must: (1) have a high index of suspicion of the potential for the diagnosis; (2) take a targeted history designed to identify risk factors for pulmonary embolus; and (3) use rapid risk stratification to drive decision making regarding testing and therapeutic interventions.

SUGGESTED READINGS

Farag A, Fielding J, Catanzano T. Role of dual-energy computed tomography in diagnosis of acute pulmonary emboli, a review. *Semin Ultrasound CT MRI*. 2022;43(4):333–343. https://doi.org/10.1053/j.sult.2022.04.003.

Guo H, Jinglan Li, Yanhuai Ma, et al. Long-term outcomes of peripheral pulmonary cement embolism in patients with polymethylmethacrylate augmentation: a case series with a minimum follow-up of five years. *World Neurosurg*. 2021;155:e315–e322. https://doi.org/10.1016/j.wneu.2021.08.044.

Hennessey A, Setyono DA, Lau WB, Fields JM. A patient with a large pulmonary saddle embolus eluding both clinical gestalt and validated decision rules. *Ann Emerg Med*. 2012;59(6):521–523. https://doi.org/10.1016/j.annemergmed.2011.11.016.

Roto D, Lachant NA, White RJ, Lachant DJ. Resting heart rate as a surrogate for improvement in intermediate risk pulmonary embolus patients? *Respir Med*. 2021;187:106578. https://doi.org/10.1016/j.rmed.2021.106578.

Sun H-B, Jing X-S, Shan J-L, et al. Risk factors for pulmonary cement embolism associated with percutaneous vertebral augmentation: a systematic review and meta-analysis. *Int J Surg*. 2022;101:106632. https://doi.org/10.1016/j.ijsu.2022.106632.

Thomas-Gittens J, Semionov A, Pressacco J. Imaging of pulmonary embolus: thrombotic, nonthrombotic, and mimickers. *Can Assoc Radiol J*. 2018;69(3):328–337. https://doi.org/10.1016/j.carj.2018.03.005.

Pneumothorax

ICD 10 J93.9

THE CLINICAL SYNDROME

Pneumothorax occurs when air or anesthetic gases enter the interpleural space between the visceral and parietal pleura of the lung (Fig. 71.1). Air can enter the interpleural space via a communication through the chest wall and/or mediastinum or via the lung parenchyma across the visceral pleura (Fig. 71.2).

The impact of pneumothorax on the patient can range from completely asymptomatic to life-threatening respiratory compromise. The severity of symptoms is related

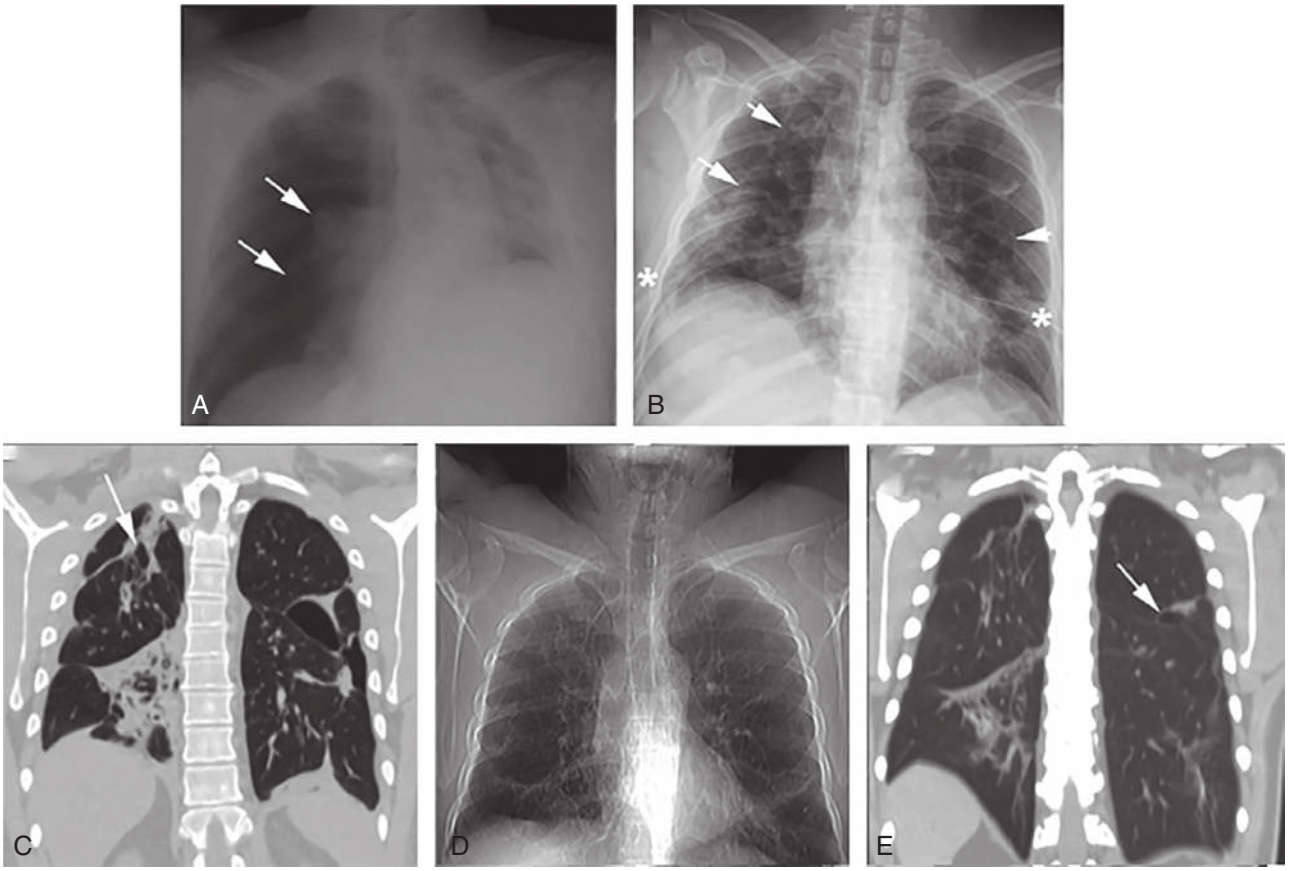

FIG 71.1 Findings of bilateral pneumothorax progression. **A,** Simple anteroposterior thorax radiograph that shows the complete collapse of the right lung (*arrows*) and displacement of the mediastinal structures to the contralateral side is observed. **B,** Simple posteroanterior thorax radiograph that shows the right chest drain accessing the fifth intercostal space and left chest drain in the sixth intercostal space (*). *White arrows* show the pulmonary parenchyma's line with evidence of pneumothorax on both sides. **C,** Simple tomography of thorax with irregular interlobular septal thickening mainly in the right lung, the left lung with an intraparenchymal bulla in the posterior apical region around 6 × 4 cm (*white arrow*). **D,** Posteroanterior radiograph showing a pneumothorax resolution after removing both chest drains. **E,** Simple thorax tomography a month later, there are fibrotic scars in right pulmonary parenchyma, a bulla in the posterior apical segment of left lung decreasing to roughly 3 × 2 cm (*white arrow*). (From Plata-Corona MA, López-Aguilar MA, Ibarra-Hernández JM, et al. A case report, bilateral spontaneous pneumothorax as a late complication for SARS CoV-2 infection. *Radiol Case Rep*. 2022;17(6):2265–2268. https://doi.org/10.1016/j.radcr.2022.03.009.)

to multiple factors including the extent of the pneumothorax, whether the pneumothorax is unilateral or bilateral, the level of impairment on oxygenation, the impact on hemodynamic status, and the presence of preexisting cardiopulmonary disease. Pneumothorax can be classified as primary, secondary, catamenial, traumatic, and iatrogenic and can be unilateral or bilateral (Box 71.1). Primary pneumothorax occurs in patients without a history of pulmonary disease and in the absence of trauma (Fig. 71.3). Secondary pneumothorax occurs in people with preexisting pulmonary disease (Figs. 71.4 and 71.5). Catamenial or menstrual pneumothorax is pneumothorax that is associated with menstruation (Fig. 71.6). Traumatic pneumothorax occurs as the result of blunt or penetrating trauma that disrupts the parietal or visceral pleura (Fig. 71.7). Iatrogenic pneumothorax is a traumatic pneumothorax as the result of diagnostic or therapeutic procedure (Figs. 71.8 and 71.9). Risk factors for the development of pneumothorax can be further classified as operator-related, procedure-related, and patient-related (Box 71.2).

BOX 71.1 Classification of Pneumothorax

- Spontaneous pneumothorax
  - Primary spontaneous pneumothorax
  - Secondary spontaneous pneumothorax
- Traumatic pneumothorax
  - Iatrogenic pneumothorax
  - Noniatrogenic pneumothorax
- Tension pneumothorax
- Catamenial (menstrual) pneumothorax

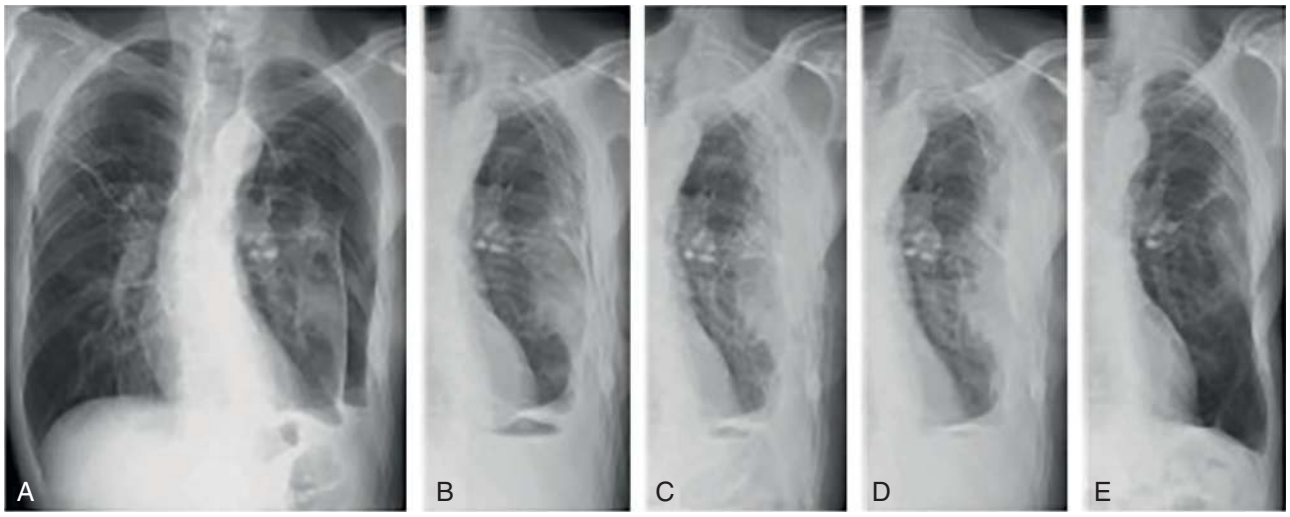

FIG 71.2 Chest radiographic findings over time. **A,** Immediately after hospitalization, a large left pneumothorax was visible. **B,** After three pleurodeses courses, the intrathoracic air space narrowed. **C,** Postsurgery, the air space was completely eliminated. **D,** After the drain removal, the pleural space was completely obliterated, and fluid was observed in the extrapleural space. **E,** At 7 months postsurgery, pleural effusion in the extrapleural space completely disappeared, with satisfactory expansion of the left lung. (From Matsumiya H, Mori M, Kanayama M, et al. Thickened parietal pleural covering in intractable pneumothorax: a case report. *Ann Med Surg*. 2022;78:103792. https://doi.org/10.1016/j.amsu.2022.103792.)

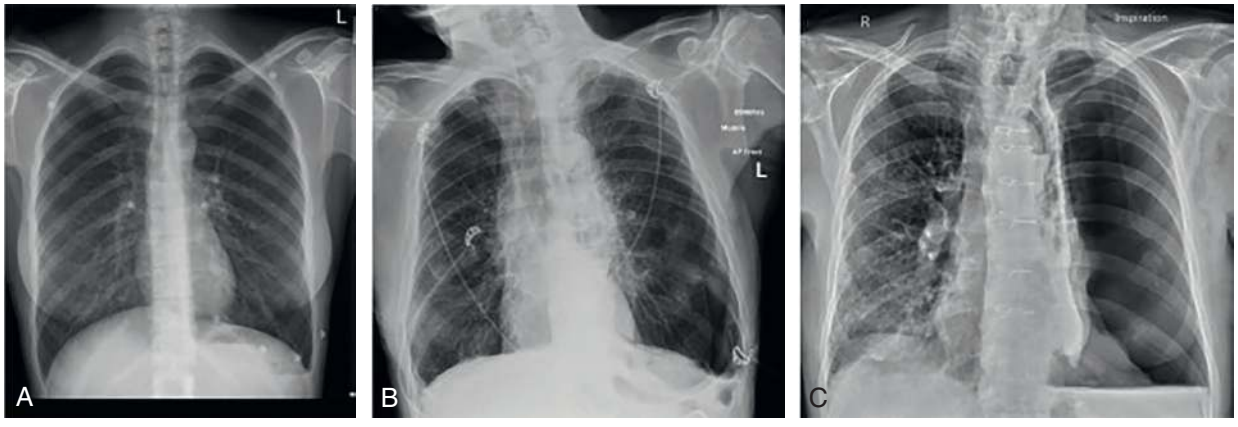

FIG 71.3 Left partial left pneumothorax **(A)**. Left incomplete pneumothorax **(B)**. Left complete pneumothorax **(C)**. (From Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

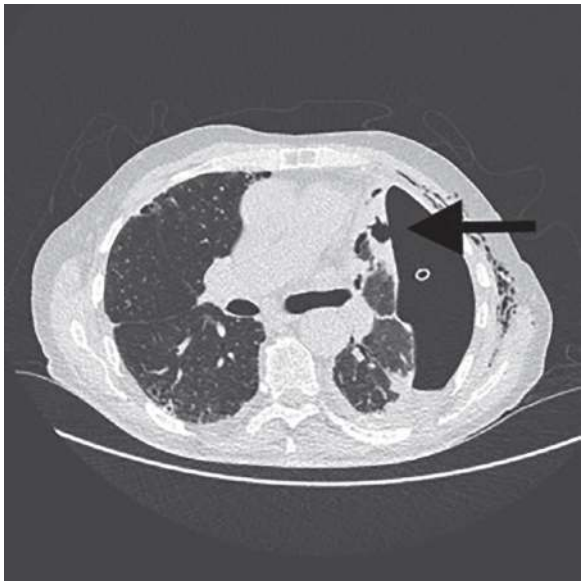

FIG 71.4 Secondary spontaneous pneumothorax. CT chest (axial view) shows necrotizing lung abscess with bronchopleural fistula (*arrow*) in the left upper lobe causing spontaneous pneumothorax. *CT*, Computed tomography. (From Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

SIGNS AND SYMPTOMS

The clinical presentation of the patient with pneumothorax is extremely variable with symptoms ranging from completely asymptomatic to life-threatening respiratory compromise. Respiratory symptoms may include:

- Dyspnea
- Tachypnea
- Distant or absent lung sounds
- Asymmetrical lung expansion
- Ipsilateral adventitious lung sounds (unless pneumothorax is bilateral)
- Decreased tactile fremitus

Cardiovascular symptoms may include:

- Tachycardia
- Hypotension
- Jugular venous distension
- Pulsus paradoxus

TESTING

The key to the accurate diagnosis of pneumothorax is history and physical examination with confirmatory chest X-ray, ultrasonography, and computerized tomography. On chest X-ray, characteristic findings of pneumothorax include linear shadow of visceral pleura without lung markings and/or an ipsilateral lung edge parallel to the chest wall (Fig. 71.10).

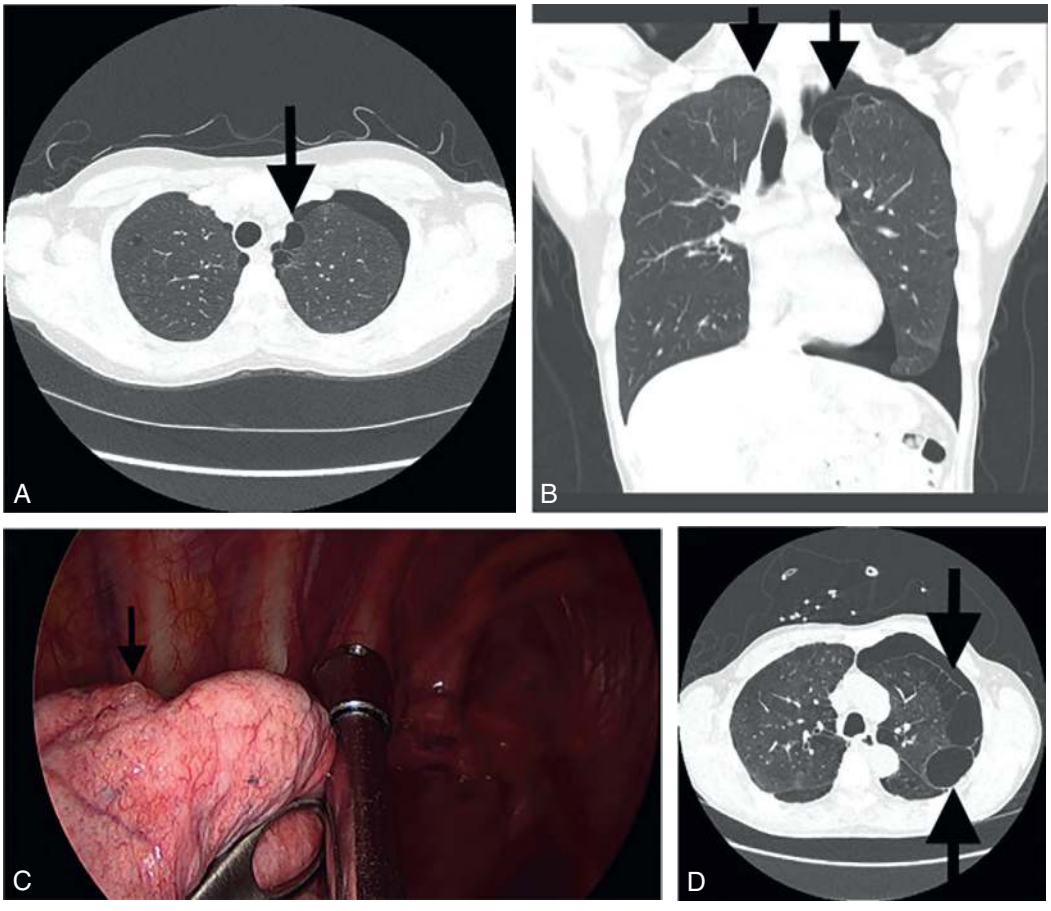

FIG 71.5 Secondary pneumothorax due to apical blebs. CT chest axial **(A)** and coronal **(B)** views showing bilateral apical blebs (*arrows*). An apical bleb is seen on direct visualization during video-assisted thoracoscopic surgery **(C)**. CT chest (axial view) shows multiple large left upper lobe bullae (*arrows*) and spontaneous secondary pneumothorax **(D)**. *CT*, Computed tomography. (From Huan N-C, Sidhu C, Thomas R. Pneu-mothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

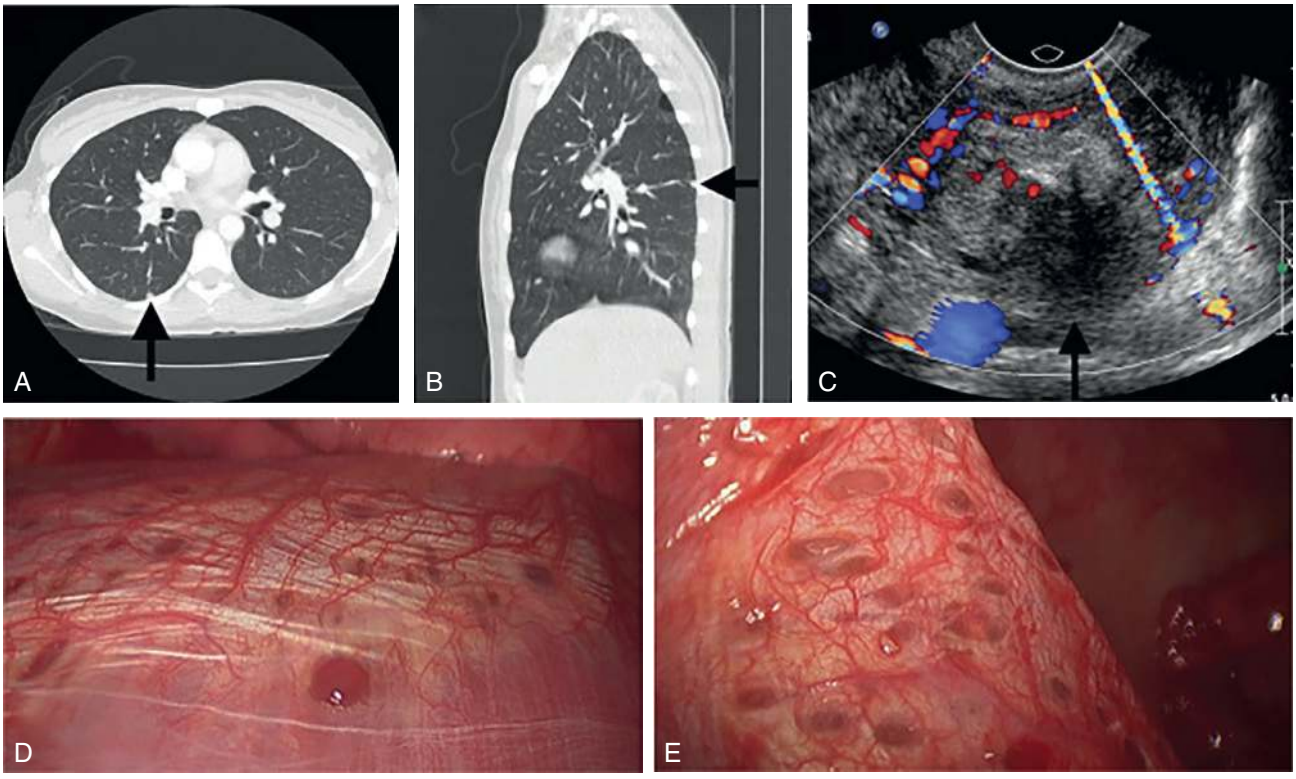

FIG 71.6 Catamenial pneumothorax. CT chest axial **(A)** and sagittal **(B)** views showing subpleural nodules (*arrows*) representing thoracic endometriosis in a patient with catamenial pneumothorax. Ultrasound confirmed the presence of pelvic endometriosis **(C)**. Multiple reddish-brown endometrial nodules were seen on the diaphragmatic pleura during video-assisted thoracoscopic surgery **(D, E)**. *CT*, Computed tomography. (From Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

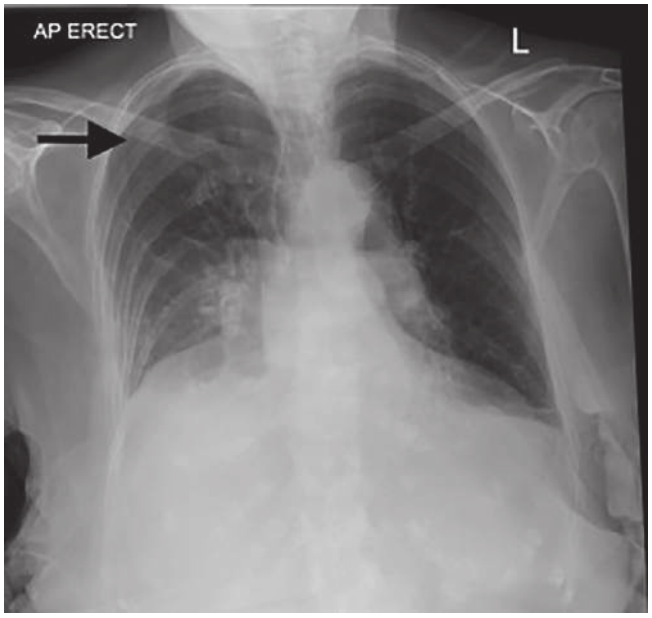

FIG 71.7 Traumatic right-sided pneumothorax in an elderly woman with displaced rib fractures (*arrow*) following a fall. (From Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

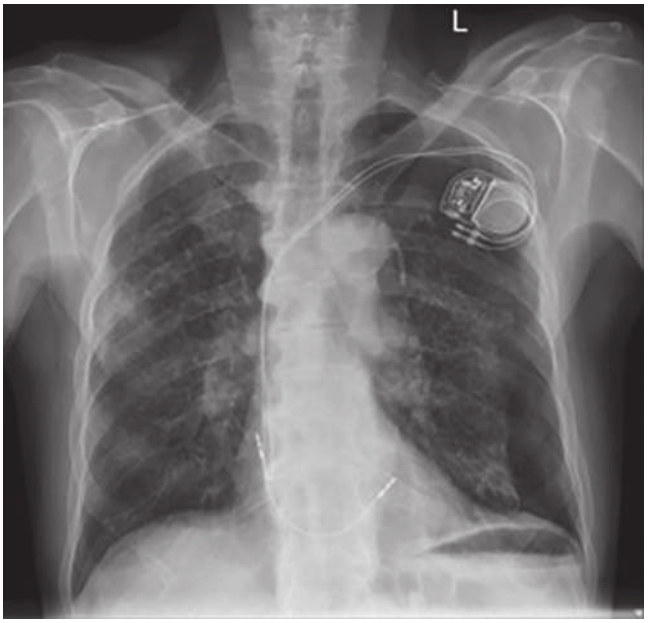

FIG 71.8 Iatrogenic pneumothorax. Chest radiograph showing left-sided iatrogenic pneumothorax that developed 2 hours following permanent pacemaker insertion. (From Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

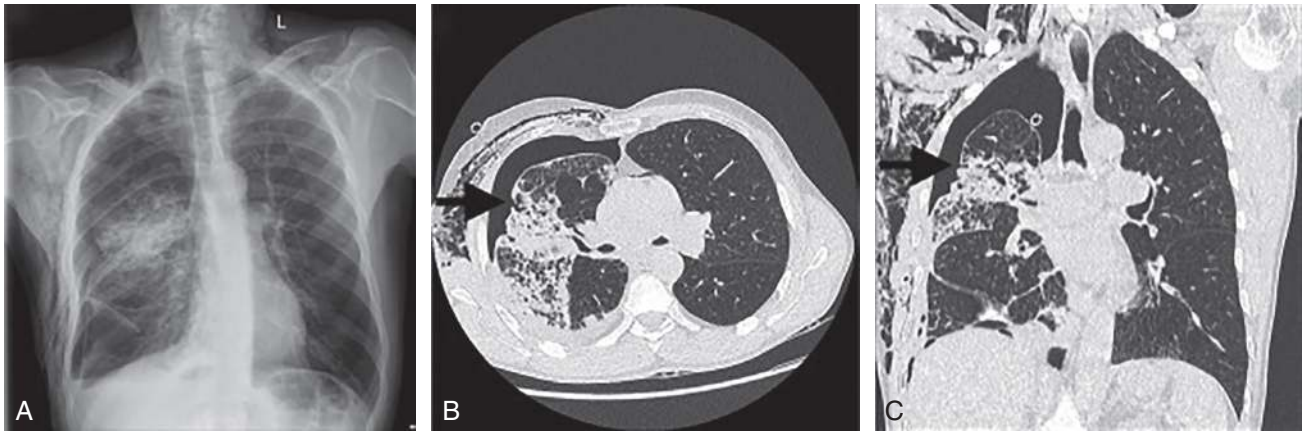

FIG 71.9 Iatrogenic pneumothorax. Chest radiograph showing iatrogenic right-sided pneumothorax following radiofrequency ablation (RFA) of right upper lobe tumor **(A)**. CT scan of chest (axial and coronal views) shows a bronchopleural fistula and breach of visceral pleura (*arrows*) caused by RFA leading to iatrogenic pneumothorax **(B, C)**. (From Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.)

BOX 71.2 Factors That Increase the Risk of Iatrogenic Pneumothorax

- Operator-related risk factors
  - Inexperience
  - Lack of skill
  - Fatigue
  - Multiple attempts at procedure
  - Failure to use image guidance
- Patient-related risk factors
  - Improper patient positioning
  - Uncooperative patient
  - High BMI
  - Very low BMI
  - Abnormal anatomy
  - Preexisting lung disease
  - Severe emphysema
  - Bullous lung disease
  - Smoking history
  - Scarring at site of procedure
  - Previous radiation to site of procedure
- Procedure-related risk factors
  - Failure to use image guidance
  - Lung versus nonlung procedures
  - Needle or trocar size
  - Number of biopsies, catheter/electrode placements, etc.
  - Elective versus emergency procedures

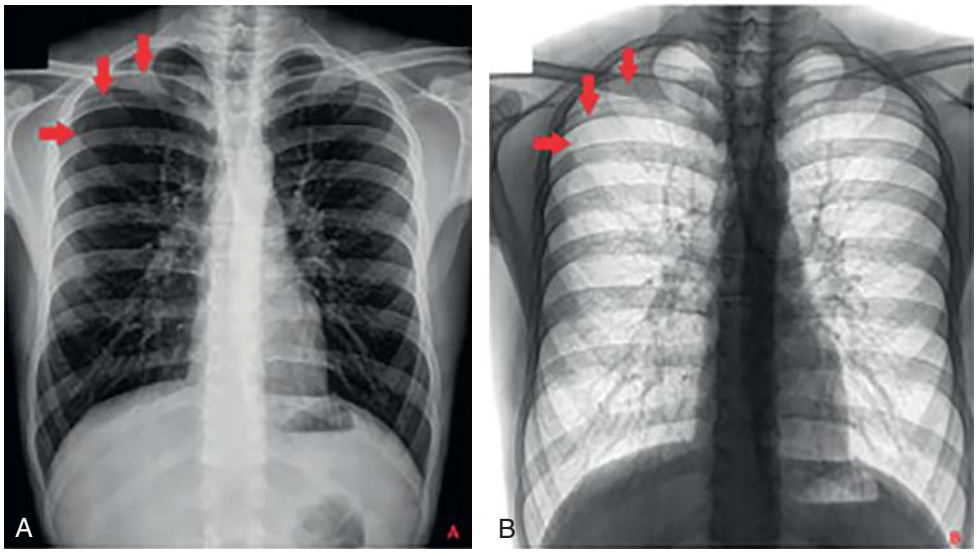

FIG 71.10 On digital–conventional **(A)** and inverted gray-scale **(B)** PACXs of a 19-year-old male patient, visceral pleura line (*red arrows*) is seen. (From Musalar E, Ekinci S, Ünek O, et al. Conventional vs invert-grayscale X-ray for diagnosis of pneumothorax in the emergency setting. *Am J Emerg Med*. 2017;35(9):1217–1221. https://doi.org/10.1016/j.ajem.2017.03.031.)

In supine patients, a deep sulcus sign characterized as an exceptionally dark, deep costophrenic angle is highly suggestive of pneumothorax (Fig. 71.11). Other chest X-ray findings include small pleural effusions and a mediastinal shift to the contralateral lung. Because the volume of the pneumothorax is constant, it will be easier to see on expiratory images. Point of care ultrasonography is being increasingly used to diagnose pneumothorax and may in fact be more accurate than chest X-ray (Fig. 71.12).

Computerized tomography of the chest is the most accurate imaging modality in the diagnosis and quantification as to the extent of pneumothorax, especially in the presence of preexisting pulmonary disease (see Fig. 71.5).

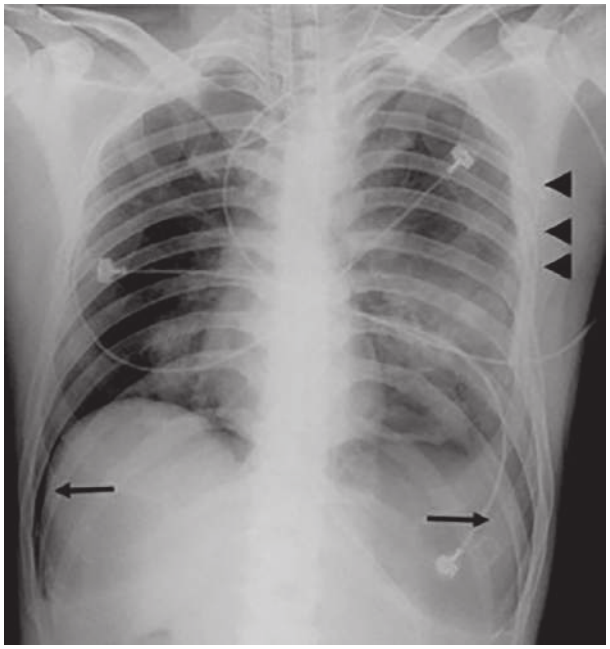

FIG 71.11 Supine chest radiography shows bilateral deep sulcus signs (*arrows*) and multiple rib fractures (*arrowheads*). (From Hung C-M, Tsai I-T, Hsu C-W. Bilateral traumatic pneumothoraces with bilateral deep sulcus signs. *Tzu Chi Med J.* 2015;27(4):188–189. https://doi.org/10.1016/j.tcmj.2015.09.001.)

DIFFERENTIAL DIAGNOSIS

Because of the highly variable clinical presentation of pneumothorax, the number of diseases that mimic pneumothorax is large (Box 71.3). In particular, diseases that cause acute respiratory symptoms such as pneumonia, bronchospasm, pulmonary

BOX 71.3 Differential Diagnosis of Pneumothorax

- Pneumonia
- Pulmonary embolus
- Bronchospastic disease
- Pleurodynia
- Myocardial infarction
- Pericarditis
- Myocarditis
- Mediastinitis
- Esophageal spasm
- Esophageal tear
- Tracheal or intrabronchial foreign bodies
- Chest wall pain
- Rib fracture

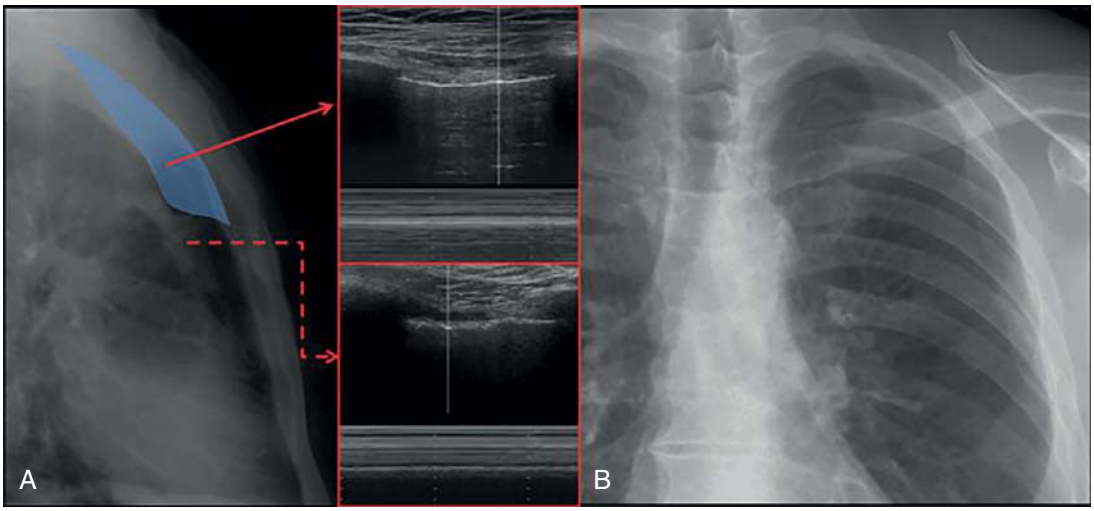

FIG 71.12 Ultrasound imaging identified an apical pneumothorax in a patient who underwent a left upper lobectomy. **(A)** Lateral view roentgenogram (not performed as part of this study) shows anterior pneumothorax (highlighted in blue). Ultrasound image shows the stratosphere sign at the second intercostal space (*red arrow*) and the seashore sign at the third intercostal space (*dotted arrow*). **(B)** Posteroanterior roentgenogram shows no pneumothorax. (From Patella M, Saporito A, Puligheddu C, et al. Lung ultrasound to detect residual pneumothorax after chest drain removal in lung resections. *Ann Thorac Surg.* 2018;105(5):1537–1542. https://doi.org/10.1016/j.athoracsur.2017.12.008.)

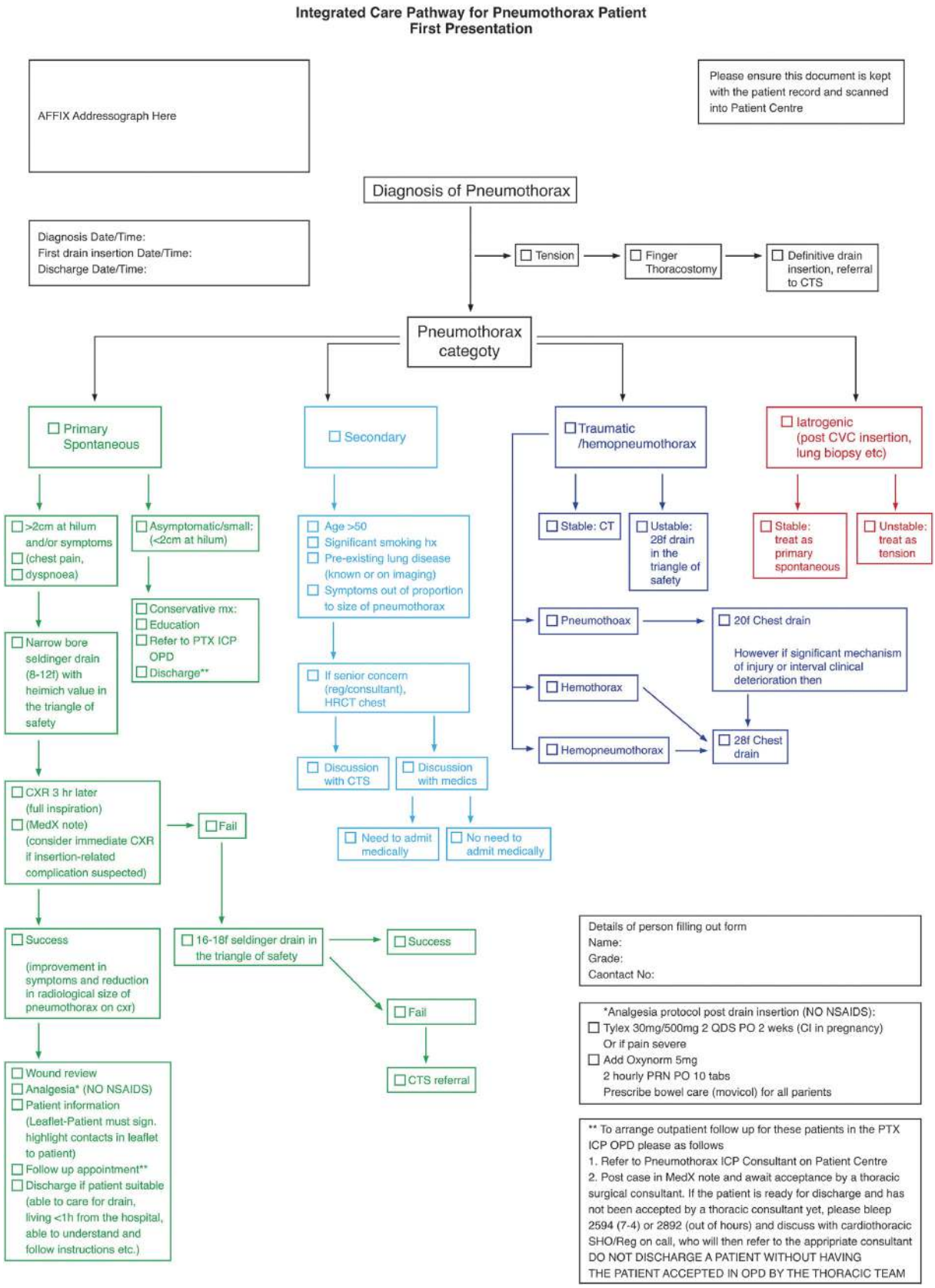

FIG 71.13 Pathway of care for the patient presenting with a first-time pneumothorax. (From Shanahan B, O'Keeffe F, Breslin T, et al. To develop, implement and evaluate an integrated care pathway for patients presenting with pneumothorax. *Surgeon*. 2022. https://doi.org/10.1016/j.surge.2022.03.004.)

embolus, pleurodynia, and a variety of chest wall pain must be at the top of the list. In the setting of significant pneumothorax or in the rare instance of tension pneumothorax, acute cardiovascular events such as myocardial infarction, acute pericarditis, acute aortic dissection need to be considered.

TREATMENT

The treatment of pneumothorax is driven primarily by the clinical impact of the pneumothorax on the patient. The skilled clinician uses the findings on physical examination as well as an evaluation of confirmatory testing to drive treatment decision making (Fig. 71.13; see Box 71.2). In patients with small asymptomatic pneumothorax that is likely to resolve, careful watchful waiting is often appropriate. For those patients suffering from a larger pneumothorax which is less likely to resolve, more aggressive interventions, namely tube thoracostomy is indicated. For those patients with life-threatening pneumothorax including significant tension pneumothorax, emergent treatment is critical to avoid disaster. Comfort care measures including the inhalation of high concentration oxygen, simple analgesics, and patient positioning are always reasonable first steps. For moderate-sized pneumothorax, some clinicians will perform simple aspiration of the interpleural air followed by chest tube placement should the pneumothorax quickly recur. Should the collapsed lung fail to re-expand within 5 days of tube thoracostomy placement, medical procedures including pleurodesis and surgical treatments including video-assisted thoracoscopic surgery or thoracotomy may be indicated.

COMPLICATIONS AND PITFALLS

Misdiagnosis of pneumothorax can result in unnecessary interventions, such as needle aspiration, chest tube placement, and surgical interventions that result in iatrogenic injury. Damage the intercostal neurovascular bundle during tube thoracostomy or trocar placement for thoracoscopic surgery can also result in significant patient morbidity. Failure to diagnose and immediately treat tension pneumothorax can lead to patient death. In patients with massive pneumothorax, re-expansion pulmonary edema can occur and may complicate an already precarious clinical situation.

CLINICAL PEARLS

Risk stratification and careful patient evaluation will best guide the treatment decision making for the patient suffering from pneumothorax. The risk of iatrogenic injury when choosing to perform needle aspiration, the placement of tube thoracostomy, or surgery must always be considered. A failure to promptly diagnose and immediately treat tension pneumothorax can lead to disastrous results.

SUGGESTED READINGS

Huan N-C, Sidhu C, Thomas R. Pneumothorax: classification and etiology. *Clin Chest Med*. 2021;42(4):711–727. https://doi.org/10.1016/j.ccm.2021.08.007.

Malik S, Kaushik C, Heidelman E, et al. Characteristics and factors associated with mortality in patients with coronavirus disease 2019 and pneumothorax. *Mayo Clin Proc Innov Qual Outcomes*. 2022;6(3):257–268. https://doi.org/10.1016/j.mayocpiqo.2022.04.003.

Musalar E, Ekinci Salih, Ünek Orkun, et al. Conventional vs invert-grayscale X-ray for diagnosis of pneumothorax in the emergency setting. *Am J Emerg Med*. 2017;35(9):1217–1221. https://doi.org/10.1016/j.ajem.2017.03.031.

Plata-Corona MA, López-Aguilar MA, Ibarra-Hernández JM, et al. A case report, bilateral spontaneous pneumothorax as a late complication for SARS CoV-2 infection. *Radiol Case Rep*. 2022;17(6):2265–2268. https://doi.org/10.1016/j.radcr.2022.03.009.

Shanahan B, O'Keeffe F, Breslin T, et al. To develop, implement and evaluate an integrated care pathway for patients presenting with pneumothorax. *Surgeon*. 2022;21(1):40–47. https://doi.org/10.1016/j.surge.2022.03.004.

Vahidirad A, Jangjoo A, Ghelichli M, et al. Tension pneumothorax in patient with COVID-19 infection. *Radiol Case Rep*. 2021;16(2):358–360. https://doi.org/10.1016/j.radcr.2020.11.044.

72

Acute Herpes Zoster of the Thoracic Dermatomes

ICD-10 CODE B02.9

THE CLINICAL SYNDROME

Herpes zoster is an infectious disease caused by the varicella-zoster virus (VZV). Primary infection with VZV in a non-immune host manifests clinically as the childhood disease chickenpox. Investigators have postulated that during the course of the primary infection, the virus migrates to the dorsal root of the thoracic nerves, where it remains dormant in the ganglia and produces no clinically evident disease. In some individuals, the virus reactivates and travels along the sensory pathways of the thoracic nerves, to produce the pain and skin lesions characteristic of herpes zoster, or shingles. Although the thoracic nerve roots are the most common site for the development of acute herpes zoster, the first division of the trigeminal nerve may also be affected.

Why reactivation occurs in some individuals but not in others is not fully understood. Investigators have theorized, however, that a decrease in cell-mediated immunity may play an important role in the evolution of this disease by allowing the virus to multiply in the ganglia and spread to the corresponding sensory nerves, thus producing clinical disease. Patients who are suffering from malignant (particularly lymphoma) or chronic disease and those receiving immunosuppressive therapy (chemotherapy, steroids, radiation) are generally debilitated and thus are much more likely than the healthy population to develop acute herpes zoster. These patients all have in common a decreased cell-mediated immune response, which may also explain why the incidence of shingles increases dramatically in patients older than 60 years and is relatively uncommon in those younger than 20 years. Recent anecdotal reports suggest an increased incidence of acute herpes zoster in patients receiving COVID-19 vaccinations.

SIGNS AND SYMPTOMS

As viral reactivation occurs, ganglionitis and peripheral neuritis cause pain that may be accompanied by flu-like symptoms. The pain generally progresses from a dull, aching sensation to dysesthetic or neuritic pain in the distribution of the thoracic nerve roots (Fig. 72.1). In most patients, the pain of acute herpes zoster precedes the eruption of rash by

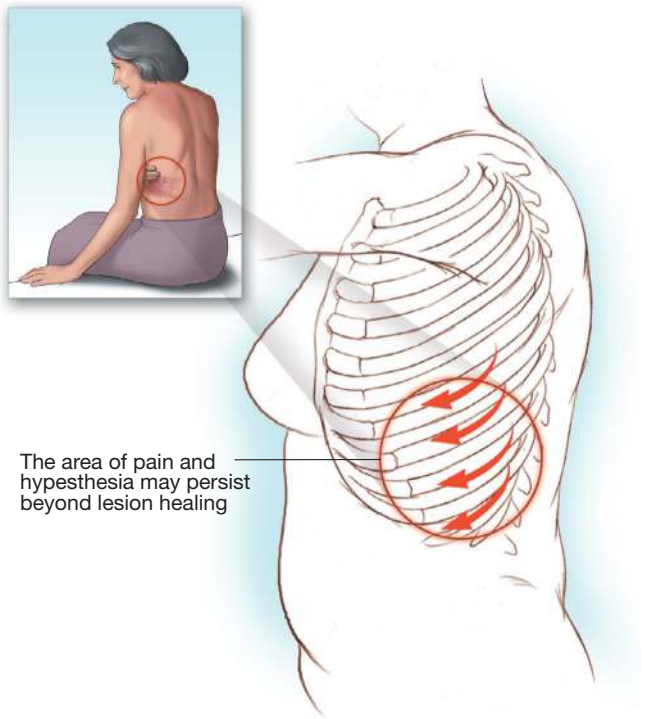

FIG 72.1 Acute herpes zoster occurs most commonly in the thoracic dermatomes.

3–7 days, and this delay often leads to an erroneous diagnosis (see "Differential Diagnosis"). However, in most patients, the clinical diagnosis of shingles is readily made when the characteristic rash appears. As in chickenpox, the rash of herpes zoster appears in crops of macular lesions, which rapidly progress to papules and then to vesicles (Fig. 72.2). Eventually, the vesicles coalesce, and crusting occurs. The affected area can be extremely painful, and the pain tends to be exacerbated by any movement or contact (e.g., with clothing or sheets). As the lesions heal, the crust falls away, leaving pink scars that gradually become hypopigmented and atrophic.

In most patients, the hyperesthesia and pain resolve as the skin lesions heal. In some patients, however, pain persists beyond lesion healing. This common and feared complication

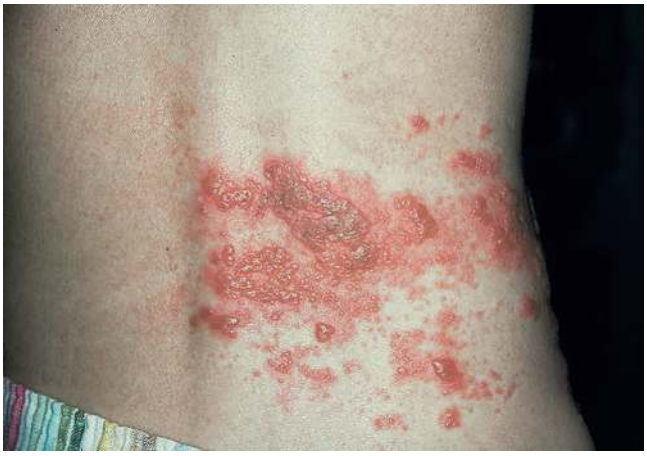

FIG 72.2 Herpes zoster involving the lumbar dermatome. (From Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*. 7th ed. Philadelphia: Churchill Livingstone; 2010:1965.)

of acute herpes zoster is called postherpetic neuralgia, and older patients are affected at a higher rate than is the general population suffering from acute herpes zoster. The symptoms of postherpetic neuralgia can vary from a mild, self-limited condition to a debilitating, constantly burning pain that is exacerbated by light touch, movement, anxiety, or temperature change. This unremitting pain may be so severe that it completely devastates the patient's life; ultimately, it can lead to suicide. To avoid this disastrous sequela to a usually benign, self-limited disease, the clinician must use all possible therapeutic efforts in patients with acute herpes zoster of the thoracic nerve roots.

TESTING

Although in most instances the diagnosis of acute herpes zoster involving the thoracic nerve roots is easily made on clinical grounds, confirmatory testing is occasionally required. Such testing may be desirable in patients with other skin lesions that confuse the clinical picture, such as patients with human immunodeficiency virus infection who are suffering from Kaposi's sarcoma. In such patients, the diagnosis of acute herpes zoster may be confirmed by obtaining a Tzanck smear from the base of a fresh vesicle that reveals multinucleated giant cells and eosinophilic inclusions. To differentiate acute herpes zoster from localized herpes simplex infection, the clinician can obtain fluid from a fresh vesicle and submit it for immunofluorescent testing.

DIFFERENTIAL DIAGNOSIS

A careful initial evaluation, including a thorough history and physical examination, is indicated in all patients suffering from acute herpes zoster involving the thoracic nerve roots. The goal is to rule out occult malignant or systemic disease that may be responsible for the patient's immunocompromised state. A prompt diagnosis allows early recognition of changes in clinical status that may presage the development of complications, including myelitis or dissemination of the disease. Other causes of pain in the distribution of the thoracic nerve roots include thoracic radiculopathy and peripheral neuropathy. Intrathoracic and intraabdominal disorders may also mimic the pain of acute herpes zoster involving the thoracic dermatomes.

TREATMENT

The therapeutic challenge in patients presenting with acute herpes zoster involving the thoracic nerve roots is twofold: (1) the immediate relief of acute pain and other symptoms and (2) the prevention of complications, including postherpetic neuralgia. Most pain specialists agree that the earlier treatment is initiated, the less likely postherpetic neuralgia will be to develop. Furthermore, because older individuals are at highest risk for developing postherpetic neuralgia, early and aggressive treatment of this group of patients is mandatory.

Nerve Block

Thoracic epidural nerve block with local anesthetic and steroid is the treatment of choice to relieve the symptoms of acute herpes zoster involving the thoracic nerve roots, as well as to prevent the occurrence of postherpetic neuralgia. As vesicular crusting occurs, the steroid may also reduce neural scarring. Neural blockade is thought to achieve these goals by blocking the profound sympathetic stimulation that results from viral inflammation of the nerve and dorsal root ganglion. If untreated, this sympathetic hyperactivity can cause ischemia secondary to decreased blood flow to the intraneural capillary bed. If this ischemia is allowed to persist, endoneural edema will form. Endoneural edema increases endoneural pressure and further reduces endoneural blood flow, with resulting irreversible nerve damage.

These sympathetic blocks should be continued aggressively until the patient is pain free and should be reimplemented if the pain returns. Failure to use sympathetic neural blockade immediately and aggressively, especially in older patients, may sentence the patient to a lifetime of suffering from postherpetic neuralgia. Occasionally, some patients with acute herpes zoster of the thoracic nerve roots do not experience pain relief from thoracic epidural nerve block but may respond to blockade of the thoracic sympathetic nerves.

Opioid Analgesics

Opioid analgesics can be useful to relieve the aching pain that is common during the acute stages of herpes zoster while sympathetic nerve blocks are being implemented. Opioids are less effective in relieving neuritic pain, which is also common. Careful administration of potent, long-acting opioid analgesics (e.g., oral morphine elixir, methadone) on a time-contingent rather than an as-needed basis may be a beneficial adjunct to the pain relief provided by sympathetic neural blockade. Because many patients suffering from acute herpes zoster are older or have severe multisystem disease,

close monitoring for the potential side effects of potent opioid analgesics (e.g., confusion or dizziness, which may cause a patient to fall) is warranted. Daily dietary fiber supplementation and milk of magnesia should be started along with opioid analgesics to prevent constipation.

Adjuvant Analgesics

The anticonvulsant gabapentin represents a first-line treatment for the neuritic pain of acute herpes zoster of the thoracic nerve roots. Studies suggest that gabapentin may also help prevent postherpetic neuralgia. Treatment with gabapentin should begin early in the course of the disease; this drug may be used concurrently with neural blockade, opioid analgesics, and other adjuvant analgesics, including antidepressants, if care is taken to avoid central nervous system side effects. Gabapentin is started at a bedtime dose of 300 mg and is titrated upward in 300-mg increments to a maximum of 3600 mg/day given in divided doses, as side effects allow.

Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine should be considered in patients suffering from severe neuritic pain who fail to respond to nerve blocks and gabapentin. If this drug is used, strict monitoring of hematologic parameters is indicated, especially in patients receiving chemotherapy or radiation therapy. Phenytoin may also be beneficial to treat neuritic pain, but it should not be used in patients with lymphoma; the drug may induce a pseudolymphoma-like state that is difficult to distinguish from the actual lymphoma.

Antidepressants

Antidepressants may also be useful adjuncts in the initial treatment of patients suffering from acute herpes zoster. On a short-term basis, these drugs help alleviate the significant sleep disturbance that is commonly seen. In addition, antidepressants may be valuable in ameliorating the neuritic component of the pain, which is treated less effectively with opioid analgesics. After several weeks of treatment, antidepressants may exert a mood-elevating effect, which may be desirable in some patients. Care must be taken to observe closely for central nervous system side effects in this patient population. In addition, these drugs may cause urinary retention and constipation, which may mistakenly be attributed to herpes zoster myelitis.

Antiviral Agents

A limited number of antiviral agents, including famciclovir, valacyclovir, and acyclovir, can shorten the course of acute herpes zoster and may even help prevent its development. These drugs are probably useful in attenuating the disease in immunosuppressed patients. These antiviral agents can be used in conjunction with the aforementioned treatment modalities. Careful monitoring for side effects is mandatory.

Adjunctive Treatments

The application of ice packs to the lesions of acute herpes zoster may provide relief in some patients. Application of heat increases pain in most patients, presumably because of the increased conduction of small fibers; however, it is beneficial in an occasional patient and may be worth trying if the application of cold is ineffective. Transcutaneous electrical nerve stimulation and vibration may also be effective in a limited number of patients. The use of transdermal lidocaine may be effective, but its utility is limited on nonintact skin. The favorable risk-to-benefit ratio of these modalities makes them reasonable alternatives for patients who cannot or will not undergo sympathetic neural blockade or who cannot tolerate pharmacologic interventions.

Topical application of aluminum sulfate as a tepid soak provides excellent drying of the crusting and weeping lesions of acute herpes zoster, and most patients find these soaks soothing. Zinc oxide ointment may also be used as a protective agent, especially during the healing phase, when temperature sensitivity is a problem. Disposable diapers can be used as absorbent padding to protect healing lesions from contact with clothing and sheets.

COMPLICATIONS AND PITFALLS

In most patients, acute herpes zoster involving the thoracic nerve roots is a self-limited disease. In older and immunosuppressed patients, however, complications may occur. Cutaneous and visceral dissemination may range from a mild rash resembling chickenpox to an overwhelming, life-threatening infection in patients already suffering from severe multisystem disease. Myelitis may cause bowel, bladder, and lower extremity paresis.

CLINICAL PEARLS

Because the pain of herpes zoster usually precedes the eruption of skin lesions by 3–7 days, some other painful condition (e.g., thoracic radiculopathy, cholecystitis) may erroneously be diagnosed. In this setting, an astute clinician should advise the patient to call immediately if a rash appears, because acute herpes zoster is a possibility. Some pain specialists believe that in a few immunocompetent patients, when reactivation of VZV occurs, a rapid immune response attenuates the natural course of the disease and the characteristic rash of acute herpes zoster may not appear. In this case, pain in the distribution of the thoracic nerve roots without an associated rash is called zoster sine herpete and is, by necessity, a diagnosis of exclusion. Therefore other causes of thoracic and subcostal pain must be ruled out before this diagnosis is invoked. VZV vaccine has been effective in preventing and modifying acute herpes zoster, and the widespread use of this immunization in older adults should decrease the incidence of this disease.

SUGGESTED READINGS

Beydoun N, Brunner P, De La Torre Y, et al. Effectiveness of CT-guided epidural infiltration of steroids and local anesthetics for acute and chronic herpes zoster neuralgia. *Diagn Interv Imaging*. 2021;102(9):525–530. https://doi.org/10.1016/j.diii.2021.02.007.

Malec-Milewska M, Horosz B, Sękowska A, et al. 5% lidocaine medicated plasters vs. sympathetic nerve blocks as a part of multimodal treatment strategy for the management of postherpetic neuralgia: a retrospective, consecutive, case-series study. *Neurol Neurochir Pol*. 2015;49(1):24–28.

Santiago-Pérez S, Nevado-Estévez R, Pérez-Conde MC. Herpes zoster-induced abdominal wall paresis: neurophysiological examination in this unusual complication. *J Neurol Sci*. 2012;312(1–2):177–179.

Schmader K. Herpes zoster. *Clin Geriatr Med*. 2016;32(3):539–553.

Shors AR. Herpes zoster and severe acute herpetic neuralgia as a complication of COVID-19 infection. *JAAD Case Rep*. 2020;6(7):656–657. https://doi.org/10.1016/j.jdcr.2020.05.012.

Tekin E, Ahiskalioglu A, Aydin ME, et al. High-thoracic ultrasound-guided erector spinae plane block for acute herpes zoster pain management in emergency department. *Am J Emerg Med*. 2019;37(2):375.e1–375.e3. https://doi.org/10.1016/j.ajem.2018.10.028.

Vallianou NG, Tsilingiris D, Karampela I, et al. Herpes zoster following COVID-19 vaccination in an immunocompetent and vaccinated for herpes zoster adult: a two-vaccine related event? *Metab Open*. 2022;13:100171. https://doi.org/10.1016/j.metop.2022.100171.

Waldman SD. Acute herpes zoster of the thoracic dermatomes. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:27–273.

Waldman SD. Postherpetic neuralgia. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:273–274.

Costovertebral Joint Syndrome

ICD-10 CODE M25.50

THE CLINICAL SYNDROME

The costovertebral joint is a true joint; it is susceptible to osteoarthritis, rheumatoid arthritis, psoriatic arthritis, Reiter's syndrome, and, in particular, ankylosing spondylitis (Figs. 73.1 and 73.2). The joint is often traumatized during acceleration–deceleration injuries and blunt trauma to the chest; with severe trauma, the joint may subluxate or dislocate. Overuse or misuse can result in acute inflammation of the costovertebral joint that can be quite debilitating. The joint is also subject to invasion by tumor from primary malignant disease, including lung cancer, or from metastatic disease. Pain emanating from the costovertebral joint can mimic pain of pulmonary or cardiac origin.

SIGNS AND SYMPTOMS

On physical examination, patients attempt to splint the affected joint or joints by avoiding flexion, extension, and lateral bending of the spine; they may also retract the scapulae in an effort to relieve the pain. The costovertebral joint may be tender to palpation and feel hot and swollen if it is acutely inflamed. Patients may also complain of a "clicking" sensation with movement of the joint. Because ankylosing spondylitis commonly affects both the costovertebral joint and the sacroiliac joint, many patients assume a stooped posture, which should alert the clinician to the possibility of this disease as the cause of costovertebral joint pain.

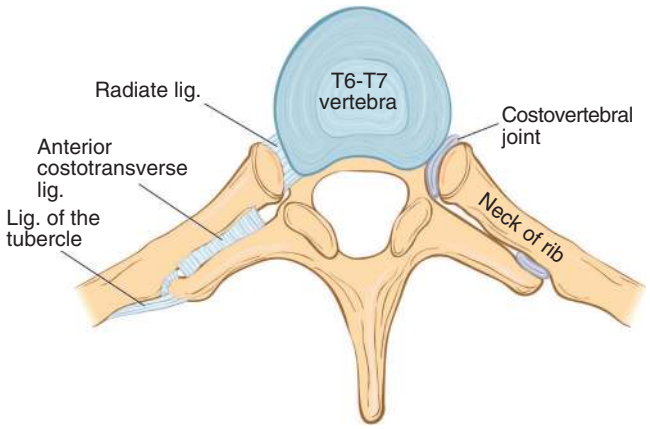

FIG 73.1 Costovertebral joint. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007.)

TESTING

Plain radiographs or computed tomography scans are indicated for all patients who present with pain that is thought to be emanating from the costovertebral joint, to rule out occult bony disorders, including tumor (Fig. 73.3). If trauma is present, radionuclide bone scanning may be useful to exclude occult fractures of the ribs or sternum. Laboratory evaluation for collagen vascular disease and other joint diseases, including ankylosing spondylitis, is indicated for patients with costovertebral joint pain, especially if other joints are involved. Because of the high incidence of costovertebral joint abnormalities in patients with ankylosing spondylitis, human leukocyte antigen-B27 testing should also be considered. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and/or ultrasound imaging of the joints is indicated if joint instability or occult mass is suspected or to elucidate the cause of the pain further (Figs. 73.4 and 74.5).

DIFFERENTIAL DIAGNOSIS

As mentioned earlier, the pain of costovertebral joint syndrome is often mistaken for pain of pulmonary or cardiac origin, and it leads to visits to the emergency department and unnecessary pulmonary or cardiac workups. If trauma has occurred, costovertebral joint syndrome may coexist with fractured ribs or fractures of the spine or sternum itself, injuries that can be missed on plain radiographs and may require radionuclide bone scanning for proper identification.

Neuropathic pain involving the chest wall may also be confused or coexist with costovertebral joint syndrome. Examples of such neuropathic pain include diabetic polyneuropathies and acute herpes zoster involving the thoracic nerves. Diseases of the structures of the mediastinum are possible and can be difficult to diagnose. Pathologic processes that inflame the pleura, such as pulmonary embolus, infection, and Bornholm disease, may also confuse the diagnosis and complicate treatment.

TREATMENT

Initial treatment of the pain and functional disability associated with costovertebral joint syndrome is with nonsteroidal

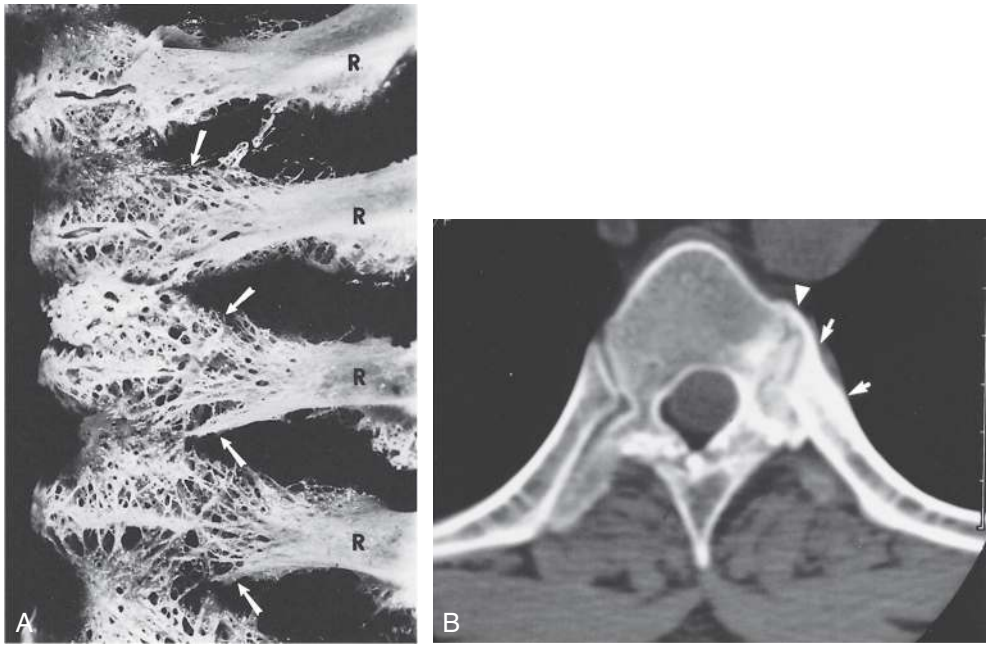

FIG 73.2 Costovertebral joint ankylosis. **A,** Photograph of the lateral aspect of the macerated thoracic spine of a spondylitic cadaver demonstrates extensive bony ankylosis (*arrows*) of the head of the ribs (*R*) and vertebral bodies. Disk ossification is also seen. **B,** Transaxial computed tomography scan of a thoracic vertebra in a patient with ankylosing spondylitis reveals bone erosions and partial ankylosis (*arrowhead*) of the costovertebral joints on one side. Note the involvement of the ipsilateral rib with cortical thickening (*arrows*). (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:1045.)

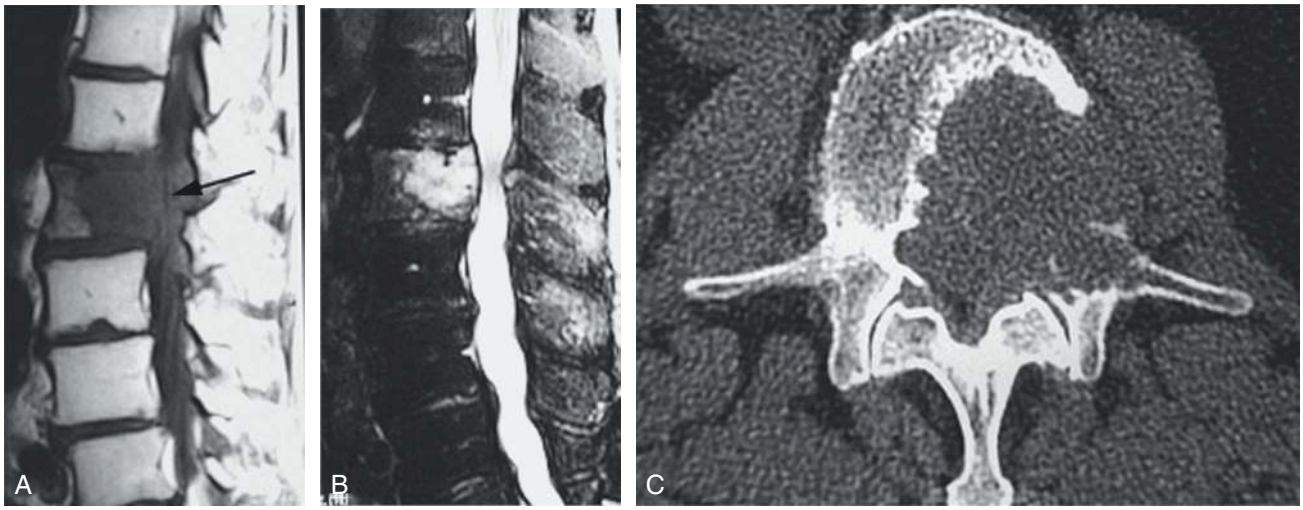

FIG 73.3 Metastatic renal carcinoma. Sagittal T1-weighted **(A)** and short tau inversion recovery **(B)** magnetic resonance images show an expansile, destructive mass replacing a lumbar vertebra, with remodeling of the posterior margin and an epidural component. Axial computed tomography scan **(C)** shows the lesion involving the pedicle and transverse process, with a significant intraspinal component. (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2324.)

antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors. The local application of heat and cold may also be beneficial. The use of an elastic rib belt may provide symptomatic relief and protect the costovertebral joints from additional trauma. For patients who do not respond to these treatment modalities, injection of the costovertebral joint with local anesthetic and steroid is a reasonable next step (Fig. 73.6). Spinal opioids may also provide palliation of pain and improve respiratory function in selected patients. Physical modalities, including local heat and gentle range-of-motion

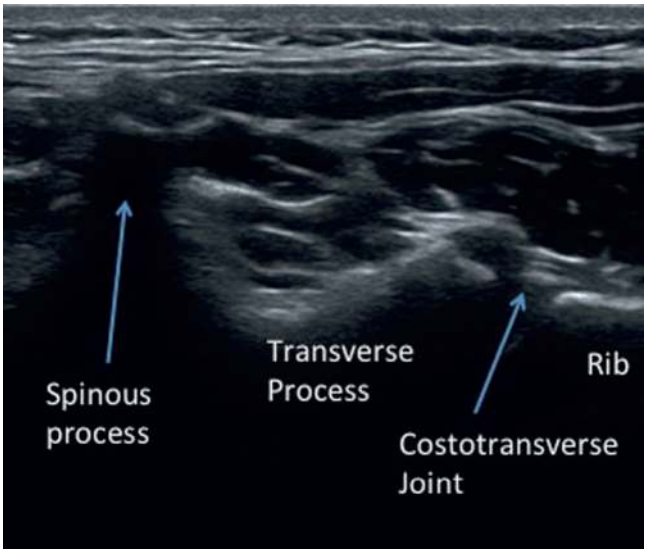

FIG 73.4 Transverse ultrasound image of the costotransverse joint.

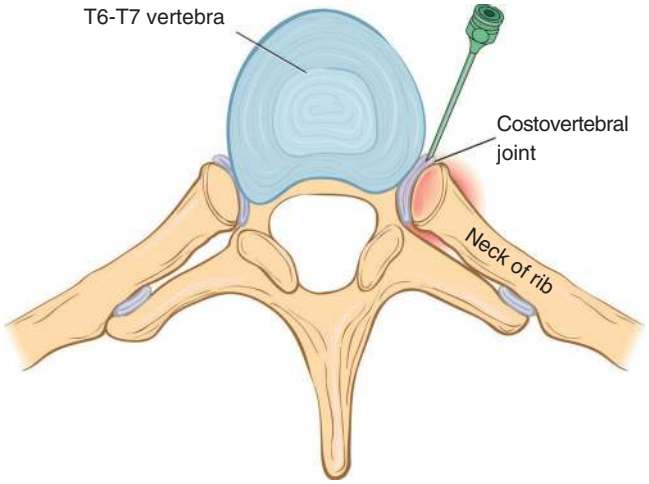

FIG 73.6 Needle placement for costovertebral joint injection. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007.)

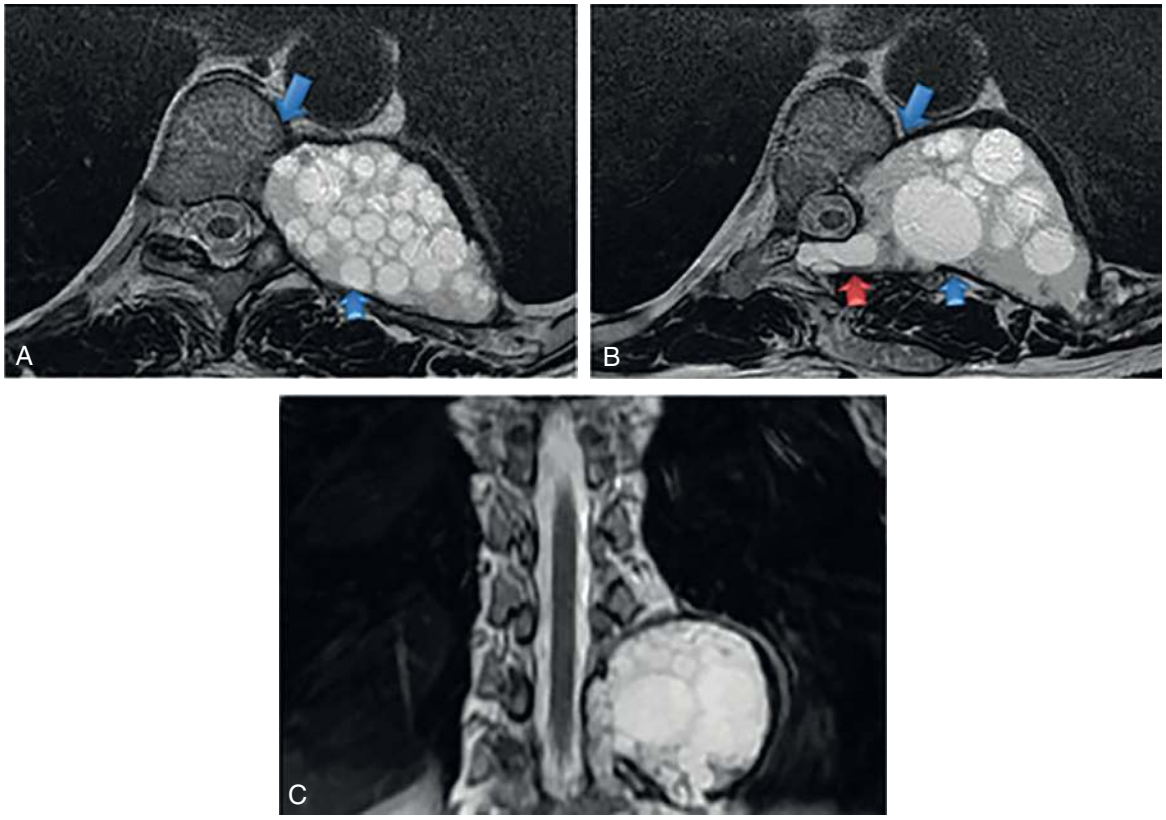

FIG 73.5 Multivesicular costovertebral hydatidosis. Preoperative MRI T2 weighted images (A, B) osteolysis of the eighth right rib, transverse process, and vertebral body (*blue arrow*) with the presence of hydatid vesicles in the intervertebral foramen (*red arrow*). C, Localization of lesions in the spine. *MRI*, Magnetic resonance imaging. (From Kassimi M, Rami A, Habi J, et al. Recurrent costovertebral hydatidosis with epidural extension. *Radiol Case Rep*. 2021;16(7):1712–1714.)

exercises, should be introduced several days after injection for costovertebral joint pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with the injection technique.

COMPLICATIONS AND PITFALLS

Because many pathologic processes can mimic the pain of costovertebral joint syndrome, the clinician must carefully rule out underlying diseases of the lung, heart, and structures

of the spine and mediastinum. Failure to do so could lead to disastrous results.

The major complication of the injection technique is pneumothorax if the needle is placed too laterally or deeply and invades the pleural space. Infection, although rare, can occur if strict aseptic technique is not followed. Trauma to the contents of the mediastinum is also a possibility. This complication can be greatly reduced with strict attention to accurate needle placement.

CLINICAL PEARLS

Patients with pain emanating from the costovertebral joint may believe that they are suffering from pneumonia or having a heart attack. Reassurance is required. In the absence of trauma, the clinician should have a high index of suspicion for the presence of an occult mass or infection.

SUGGESTED READINGS

Arslan G, Cevikol C, Karaali K, et al. Single rib sclerosis as a sequel of compression fracture of adjacent vertebra and costovertebral joint ankylosis. *Eur J Radiol Extra*. 2004;51(1):43–46.

Bogduk N. Functional anatomy of the spine. In: Masdeu JC, González RG, eds. *Handbook of clinical neurology*. Elsevier; 2016;(136):675–688.

Duprey S, Subit D, Guillemot H, et al. Biomechanical properties of the costovertebral joint. *Med Eng Phys*. 2010;32(2):222–227.

Illiasch H, Likar R, Stanton-Hicks M. CT use in pain management. *Tech Reg Anesth Pain Manag*. 2007;11(2):103–112.

Waldman SD. Arthritis and other abnormalities of the costotransverse and costovertebral joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:530–534.

Waldman SD. Costovertebral joint pain. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:252–254.

Wendling D, Aubry S, Verhoeven F, Prati C. Costovertebral arthritis revealing spondyloarthritis. *Jt Bone Spine*. 2019;86(1):107–108. https://doi.org/10.1016/j.jbspin.2018.06.005.

Postherpetic Neuralgia

ICD-10 CODE B02.23

THE CLINICAL SYNDROME

Postherpetic neuralgia is one of the most difficult pain syndromes to treat. It affects 10% of patients with acute herpes zoster. Although the reason that this painful condition occurs in some patients but not in others is unknown, postherpetic neuralgia is more common in older individuals and appears to occur more frequently after acute herpes zoster involving the trigeminal nerve, as opposed to the thoracic dermatomes. Conditions that cause vulnerable nerve syndrome, such as diabetes, may also predispose patients to develop postherpetic neuralgia. Recent neuroimaging studies have shown that patients suffering from postherpetic neuralgia have abnormal central pain processes. Peripheral nerve pain specialists agree that aggressive treatment of acute herpes zoster can help prevent postherpetic neuralgia.

SIGNS AND SYMPTOMS

As the lesions of acute herpes zoster heal, the crust falls away, leaving pink scars that gradually become hypopigmented and atrophic. The affected cutaneous areas are often allodynic, although hypesthesia and, rarely, anesthesia may occur. In most patients, the sensory abnormalities and pain resolve as the skin lesions heal. In some patients, however, pain persists beyond lesion healing.

The pain of postherpetic neuralgia is characterized as a constant dysesthetic pain that may be exacerbated by movement or stimulation of the affected cutaneous regions (Fig. 74.1). Sharp, shooting neuritic pain may be superimposed on the constant dysesthetic pain. Some patients suffering from postherpetic neuralgia also note a burning component, reminiscent of reflex sympathetic dystrophy.

TESTING

In most cases, the diagnosis of postherpetic neuralgia is made on clinical grounds. Testing is generally used to evaluate other treatable coexisting conditions, such as vertebral compression fractures, or to identify any underlying disease responsible for the patient's immunocompromised state. Such testing includes basic laboratory screening, rectal

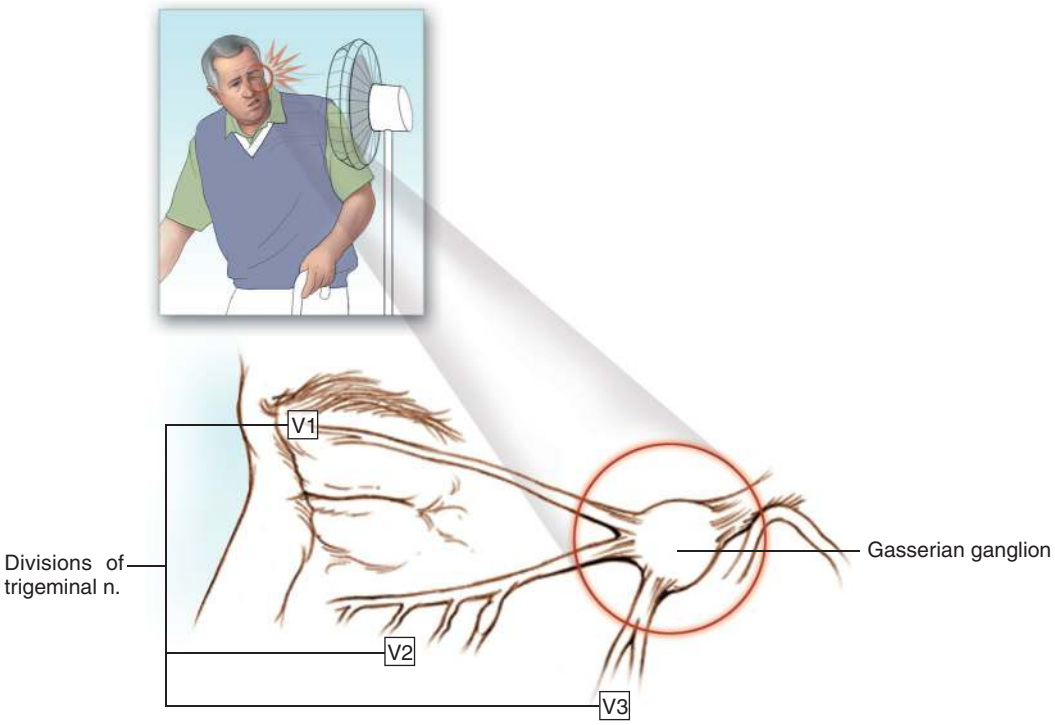

FIG 74.1 Allodynia and dysesthesia are characteristic of postherpetic neuralgia.

examination, mammography, and testing for collagen vascular diseases and human immunodeficiency virus infection. Skin biopsy may confirm the presence of previous infection with herpes zoster if the history is in question.

DIFFERENTIAL DIAGNOSIS

A careful initial evaluation, including a thorough history and physical examination, is indicated for all patients suffering from postherpetic neuralgia, to rule out an occult malignant or systemic disease that may be responsible for the patient's immunocompromised state. This evaluation also allows early recognition of changes in clinical status that may presage the development of complications, including myelitis or dissemination of the disease. Other causes of pain in the distribution of the thoracic nerve roots include thoracic radiculopathy and peripheral neuropathy. Intrathoracic and intraabdominal disorders may also mimic the pain of acute herpes zoster involving the thoracic dermatomes. For pain in the distribution of the first division of the trigeminal nerve, the clinician must exclude diseases of the eye, ear, nose, and throat, as well as intracranial disorders.

TREATMENT

Ideally, rapid and aggressive treatment of acute herpes zoster is instituted in every patient, because most pain specialists believe that the earlier treatment is initiated, the less likely postherpetic neuralgia will be to develop. This approach is especially important in older individuals, who are at the greatest risk for postherpetic neuralgia. If, despite everyone's best efforts, postherpetic neuralgia occurs, the following treatment regimens are appropriate.

Analgesics, Anticonvulsants, and Cannabinoids

The anticonvulsant gabapentin is the first-line treatment for the pain of postherpetic neuralgia. Treatment with gabapentin should begin early in the course of the disease, and this drug may be used concurrently with neural blockade, opioid analgesics, and other analgesics, including antidepressants, if care is taken to avoid central nervous system side effects. Gabapentin is started at a dose of 300-mg at bedtime and is titrated upward in 300-mg increments to a maximum of 3600-mg/day in divided doses, as side effects allow.

Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50-mg three times a day and may be titrated upward to 100-mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine should be considered in patients suffering from severe neuritic pain in whom nerve blocks and gabapentin fail to provide relief. If this drug is used, rigid monitoring of hematologic parameters is indicated, especially in patients receiving chemotherapy or radiation therapy. Phenytoin may also be beneficial to treat neuritic pain, but it should not be used in patients with lymphoma; the drug may induce a pseudolymphoma-like state that is difficult to distinguish from actual lymphoma.

Recent clinical reports suggest that cannabinoids may be useful in a variety of neuropathic pain syndromes including postherpetic neuralgia. Controlled studies are underway evaluating the relative risk-to-benefit ratio and efficacy of this class of drugs.

Antidepressants

Antidepressants may be useful adjuncts in the initial treatment of postherpetic neuralgia. On a short-term basis, these drugs help alleviate the significant sleep disturbance that is common in this setting. In addition, antidepressants may be valuable in ameliorating the neuritic component of the pain, which is treated less effectively with opioid analgesics. After several weeks of treatment, antidepressants may exert a mood-elevating effect that is desirable in some patients. Care must be taken to observe closely for central nervous system side effects in this patient population. These drugs may cause urinary retention and constipation that may mistakenly be attributed to herpes zoster myelitis.

Nerve Block and Neuroaugmentation

Neural blockade with local anesthetic and steroid through either epidural nerve block or blockade of the sympathetic nerves subserving the painful area is a reasonable next step if the aforementioned pharmacologic modalities fail to control the pain of postherpetic neuralgia. Although the exact mechanism of pain relief is unknown, it may be related to modulation of pain transmission at the spinal cord level. Spinal cord stimulation is a reasonable next step in the treatment of postherpetic neuralgia in patients who have failed to respond to pharmacologic, adjuvant, and nerve block modalities. In general, neurodestructive procedures have a very low success rate and should be used only after all other treatments have failed, if at all.

Opioid Analgesics

Opioid analgesics have a limited role in the management of postherpetic neuralgia and may do more harm than good. Careful administration of potent, long-acting opioid analgesics (e.g., oral morphine elixir, methadone) on a time-contingent rather than an as-needed basis may be a beneficial adjunct to the pain relief provided by sympathetic neural blockade. Because many patients suffering from postherpetic neuralgia are older or have severe multisystem disease, close monitoring for the potential side effects of opioid analgesics (e.g., confusion or dizziness, which may cause a patient to fall) is warranted. Daily dietary fiber supplementation and milk of magnesia should be started along with opioid analgesics to prevent constipation.

Adjunctive Treatments

The application of ice packs to the affected area may provide relief in some patients. The application of heat increases pain in most patients, presumably because of increased conduction

of small fibers; however, it is beneficial in an occasional patient and may be worth trying if the application of cold is ineffective. Transcutaneous electrical nerve stimulation, acupuncture, and vibration may also be effective in a limited number of patients. The favorable risk-to-benefit ratio of all these modalities makes them reasonable alternatives for patients who cannot or will not undergo sympathetic neural blockade or who cannot tolerate pharmacologic interventions. The topical application of capsaicin may be beneficial in some patients suffering from postherpetic neuralgia; however, this substance tends to burn when applied, thus limiting its usefulness. Recent clinical reports suggest that low level pulsed laser may also help prevent postherpetic neuralgia.

COMPLICATIONS AND PITFALLS

Although no specific complications are associated with postherpetic neuralgia itself, the consequences of the unremitting pain can be devastating. Failure to treat the pain of postherpetic neuralgia and the associated symptoms of sleep disturbance and depression aggressively can result in suicide.

CLINICAL PEARLS

Because the pain of postherpetic neuralgia is so severe, the clinician must make every effort to avoid its occurrence by rapidly and aggressively treating acute herpes zoster. If postherpetic neuralgia develops, the aggressive treatment outlined here, with special attention to the insidious onset of severe depression, should be undertaken. If serious depression occurs, hospitalization with suicide precautions is mandatory. The widespread use of the varicella zoster live virus vaccine will hopefully decrease the incidence, or at least modify the course, of acute herpes zoster, which should ultimately decrease the incidence of postherpetic neuralgia.

SUGGESTED READINGS

Bandaranayake T, Shaw AC. Host resistance and immune aging. *Clin Geriatr Med*. 2016;32(3):415–432.

Ghanavatian S, Wie CS, Low RS, et al. Premedication with gabapentin significantly reduces the risk of postherpetic neuralgia in patients with neuropathy. *Mayo Clin Proc*. 2019;94(3):484–489. https://doi.org/10.1016/j.mayocp.2018.11.004.

Kawai K, Rampakakis E, Tsai TF, et al. Predictors of postherpetic neuralgia in patients with herpes zoster: a pooled analysis of prospective cohort studies from North and Latin America and Asia. *Int J Infect Dis*. 2015;34:126–131.

Klein NP, Bartlett J, Fireman B, Marks MA, Hansen J, Lewis E, Aukes L, Saddier P. Long-term effectiveness of zoster vaccine live for postherpetic neuralgia prevention. *Vaccine*. 2019;37(36):5422–5427. https://doi.org/10.1016/j.vaccine.2019.07.004.

Muñoz-Quiles C, López-Lacort M, Orrico-Sánchez A, Díez-Domingo J. Impact of postherpetic neuralgia: a six year population-based analysis on people aged 50 years or older. *J Infect*. 2018;77(2):131–136. https://doi.org/10.1016/j.jinf.2018.04.004.

Schmader K. Herpes zoster. *Clin Geriatr Med*. 2016;32(3):539–553.

Tang Y, Wang Maohua, Zheng Ting, et al. Structural and functional brain abnormalities in postherpetic neuralgia: a systematic review of neuroimaging studies. *Brain Res*. 2021;1752:147219. https://doi.org/10.1016/j.brainres.2020.147219.

Waldman SD. Postherpetic neuralgia. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:2273–2274.

Wan C-F, Song T. Comparison of two different pulsed radiofrequency modes for prevention of postherpetic neuralgia in elderly patients with acute/subacute trigeminal herpes zoster. *Neuromodulation*. 2022 Dec;25(8):1364–1371. https://doi.org/10.1111/ner.13457.

75

Nephrolithiasis

ICD-10 CODE N20.0

THE CLINICAL SYNDROME

Nephrolithiasis, which is also known as renal calculi and kidney stones, is a stone-like deposit of acid salts and minerals that form within the kidneys when these substances exist in concentrations above the saturation point within the urine. This disease occurs more commonly in males and peaks between the ages of 30 and 50 years. Nephrolithiasis occurs more commonly in Caucasians than in Hispanics and is much less common in Blacks. There is a family clustering of nephrolithiasis; men have a family history of kidney stones with two to three times greater probability of suffering from this disease.

Variables that encourage the formation of renal calculi include the presence of red blood cells, urinary casts, low calcium diets, diets high in high fructose corn syrup and sodium, and other crystals that can form as nucleating nidus that may promote stone formation. Ambient temperature may also correlate with the increased formation of stones with a seasonal predilection for stone formation in the warmer southeast United States, during the summer months, and in occupations exposed to high ambient temperatures (e.g., military deployments to hot desert climates). Urinary tract abnormalities such as horseshoe kidney may also increase the risk of nephrolithiasis. Some investigators believe that the obesity–metabolic syndrome–diabetes spectrum is also a risk factor for nephrolithiasis. The solubility of stone-forming solutes can also be inhibited by the presence of citrate, glycoproteins, and magnesium. The pH of the urine can increase or decrease the incidence of renal calculi, depending on which type of kidney stone is being formed, with acidic pH encouraging the calcium-based stone formation and discouraging the formation of uric acid stones.

Renal calculi are most commonly calcium-based, with calcium oxalate-containing stones accounting for approximately 60%–70% of stones (Fig. 75.1). Calcium oxalate stones are seen in patients suffering from hyperparathyroidism, malabsorption postbariatric surgery, hypervitaminosis D, diets high in high oxalate foods such as nuts and chocolate, and in patients with chronic pancreatitis. Calcium phosphate stones are associated with hypercalciuria and urinary alkalization secondary to renal tubular acidosis or the use of topiramate and carbonic anhydrase inhibitors such as acetazolamide. Much less common are uric acid stones whose formation is thought to be associated with excessive protein intake, gout, low urine output, and acidic urine. Ammonium acid urate stones and struvite stones are also less common than calcium-containing stones. Ammonium acid stones are associated with inflammatory bowel disease, laxative abuse, and ileostomy. Struvite stones are most commonly associated with urinary tract infections with urease-positive bacteria that convert urea to ammonium. Disorders of cystine transport can also cause kidney stones.

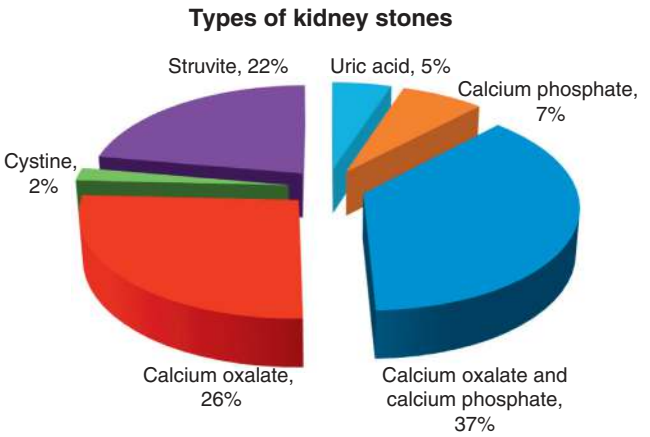

FIG 75.1 The composition of kidney stones. (Redrawn from Monk RD, Bushinsky DA. Nephrolithiasis and nephrocalcinosis. In: Frehally J, Floege J, Johnson RJ, eds. *Comprehensive clinical nephrology*. London: Mosby; 2007:641–655. Courtesy Dr. Patrick Fleet, University of Washington, Seattle, Washington, DC, USA.)

SIGNS AND SYMPTOMS

Calculi can form in the intraparenchymal space, the calyx, and the pelvis of the kidney, as well as the ureter and the bladder. Variables, including the size of the calculus, its location, and the patient's anatomy, will affect its clinical impact and symptomatology. The symptoms of nephrolithiasis are primarily the result of increased intraurinary tract pressure that stretches and stimulates nociceptive nerve endings in the urothelium. These pain impulses are carried via the afferent sympathetic and somatic nerves at the T11 to L1 levels.

FIG 75.2 The pain location often reflects the anatomic site at which the stone is obstructing the urinary system.

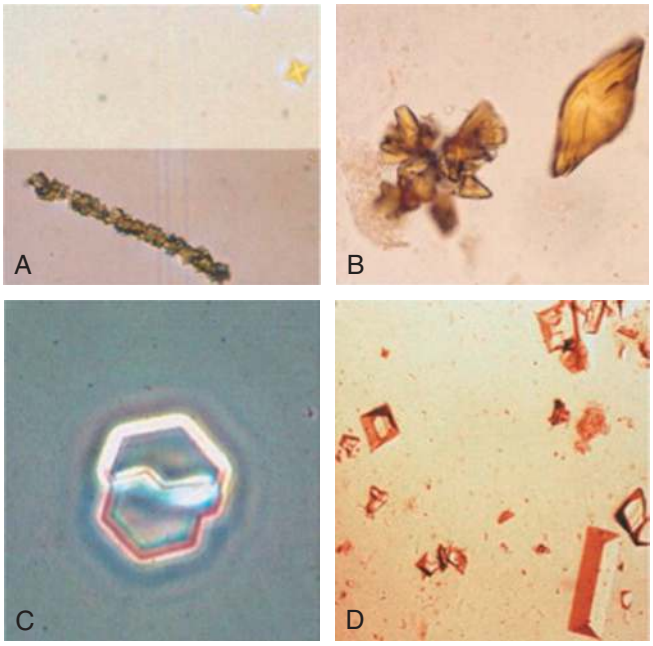

FIG 75.3 Crystals seen in the urine of stone formers. **A,** Calcium oxalate; **B,** urate; **C,** cystine; **D,** struvite. (From Monk RD, Bushinsky DA. Nephrolithiasis and nephrocalcinosis. In: Frehally J, Floege J, Johnson RJ, eds. *Comprehensive clinical nephrology*. London: Mosby; 2007:641–655. Courtesy Dr. Patrick Fleet, University of Washington, Seattle, Washington, DC, USA.)

The pain of nephrolithiasis tends to wax and wane and is often colicky in nature with spasm of the ureters and the bladder occurring as stones pass distally. If the urinary obstruction is incomplete or intermittent, the pain will tend to wax and wane, with complete obstruction causing constant severe pain. Pain may be referred to the flank, groin, testicle, or labia with the location of the pain often reflecting the anatomic location at which the stone is obstructing the urinary system (Fig. 75.2). Nausea and vomiting are frequently present as is hematuria. Urinary urgency, frequency, dysuria, and meatal pain are also common. The patient suffering from acute kidney stones may find it difficult to find a comfortable position and may pace the floor. Fever, rigors, and chills in patients with signs and symptoms thought to be caused by kidney stones are serious findings, and immediate culture of urine and any retrieved calculi should be obtained and appropriate antibiotic therapy instituted.

Findings on physical examination of the patient suffering from the acute pain of nephrolithiasis include diaphoresis, tachycardia, and hypertension. Costovertebral angle tenderness is invariably present as is the absence of abdominal and genital findings. A commonly used diagnostic rubric to increase the specificity of the diagnosis of renal calculi is the STONE score. STONE is an acronym that allows easy scoring to determine the likelihood that a patient is suffering from renal and/or ureteral calculi. A score greater than 13 provides diagnostic accuracy approaching 90%.

TESTING

Unless there is significant dehydration or compromise of renal function secondary to obstruction, the serum creatinine and serum chemistry will be within normal limits, although careful attention to serum calcium levels is mandatory to help identify patients suffering from hyperparathyroidism. Leukocytosis with a left shift secondary to the stress of the pain may also be present. On urinalysis, microscopic hematuria is common, with some patients experiencing gross hematuria. Crystalluria may be observed on microscopic evaluation (Fig. 75.3). Leukouria and the presence of nitrates and leukocyte esterase in the urine are highly suggestive of a urinary tract infection. Strained urine may reveal renal calculi (Fig. 75.4).

Noncontrast, low-dose computerized tomographic (CT) scans of the urinary tract have replaced intravenous pyelography as the first step in the diagnosis of nephrolithiasis. The CT scan not only provides important information as to the location and shape of the stone and the nature of

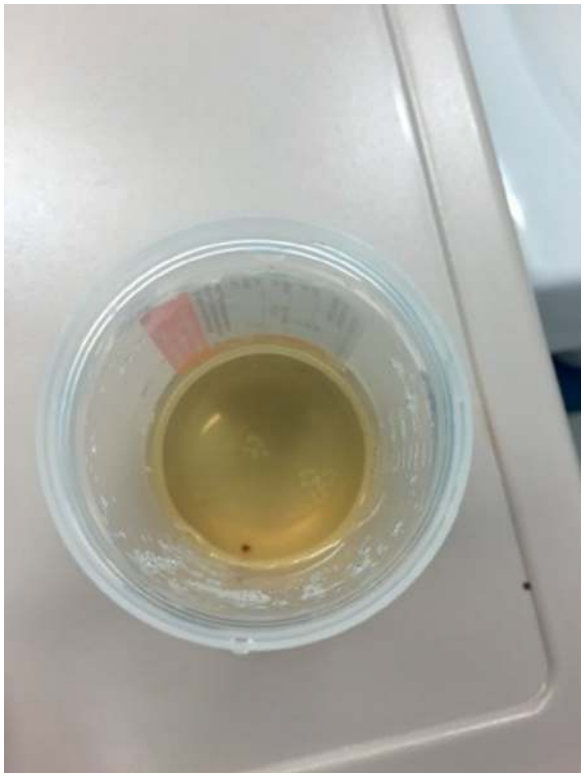

FIG 75.4 A small renal calculus in the strained urine. (From Isenberg D, Jacobs D. I just passed something in my urine. *Vis J Emerg Med*. 2016;5:31.)

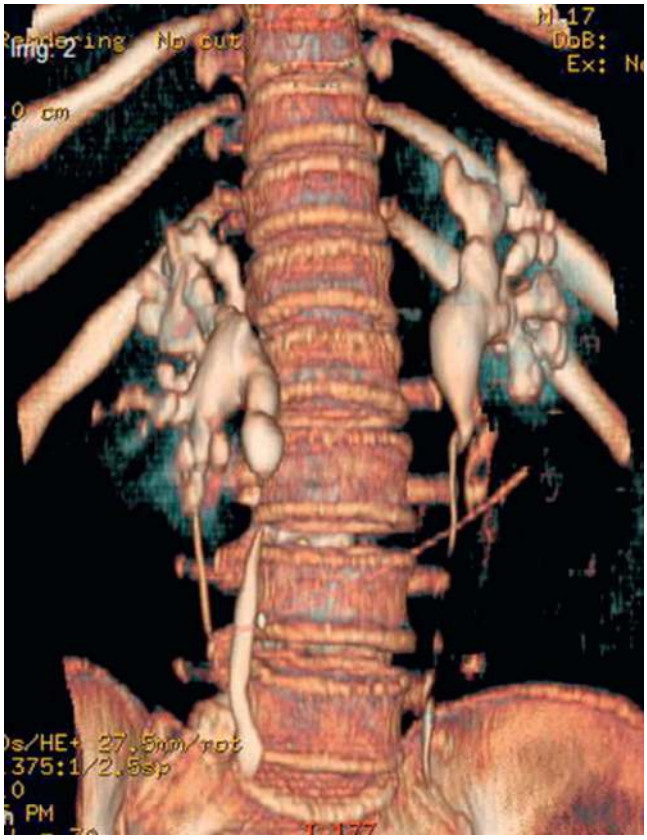

FIG 75.5 Renal three-dimensional computed tomography (CT) reconstruction demonstrating bilateral staghorn calculi. (From Szczepańska M, Zachurzok-Buczyńska A, Adamczyk P, et al. Pelvico-calyceal system rupture due to staghorn calculus with urinoma formation in a boy with neurofibromatosis type 1 and quadriplegia. *Pediatr Polska*. 2014;89(4):302–306.)

obstruction but can also identify anatomic abnormalities of the urinary tract that may complicate surgical interventions (Figs. 75.5 and 75.6). Retroperitoneal ultrasound scans represent an alternative to CT scans, albeit with less sensitivity and specificity. The KUB (kidney, ureter, and bladder) plain radiography can also be used to follow up on the position of radiopaque stones.

DIFFERENTIAL DIAGNOSIS

In most patients, the diagnosis of symptomatic renal calculi is straightforward; however, basically any disorder that affects the organs that are subserved by the celiac plexus and spinal nerves T11 to L2 may mimic the clinical presentation of kidney stones. These disorders include appendicitis, biliary colic, cholecystitis, obstructive cholelithiasis, cystitis, ileus, bowel obstruction, pyelonephritis, ovarian cyst rupture, incarcerated hernia, testicular torsion, orchitis, and viral gastroenteritis.

TREATMENT

As mentioned above, variables including the size of the calculus, its location, and the patient's anatomy will affect its clinical impact and symptomatology. In general, the smaller the stone, the more likely that conservative medical treatment will be successful. With small symptomatic stones, increased fluids given orally or intravenously to increase the amount of urine may help the stone to pass and relieve the obstruction. The addition of alpha and calcium channel blockers to inhibit the contraction and peristalsis of the smooth muscle of the ureter may also help decrease symptomatology and speed stone passage.

For larger stones or in clinical situations where smaller stones fail to pass, temporizing drainage (e.g., percutaneous nephrostomy tubes), shock wave lithotripsy to fracture calculi into smaller pieces, percutaneous lithotomy, and uroendoscopic and open stone removal may be required (Fig. 75.7). Again, the size and location of the offending stone or stones will dictate the best interventional therapy.

Palliation of symptoms with the use of nonopioid analgesics should be considered. Morphine and morphine-like

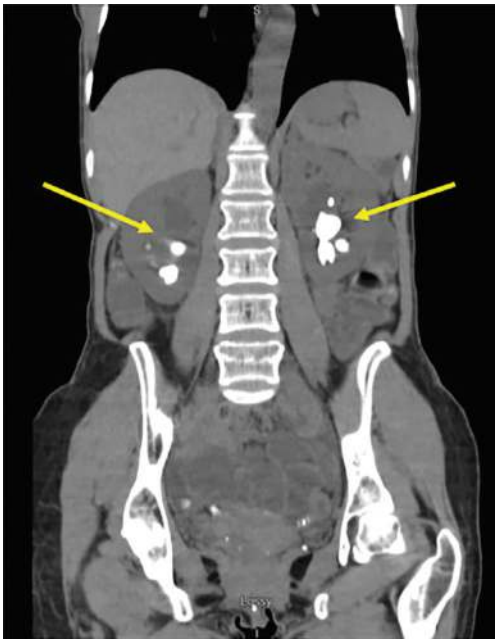

FIG 75.6 CT demonstrating bilateral staghorn calculi. *CT*, Computed tomography. (From Khooblall P, Morcos D, Mahmood F, Ricchiuti VS. Staged treatment for substantial bilateral calcium carbonate nephrolithiasis in vegan patient, *Urol Case Rep*. 2021;39:101831. https://doi.org/10.1016/j.eucr.2021.101831.)

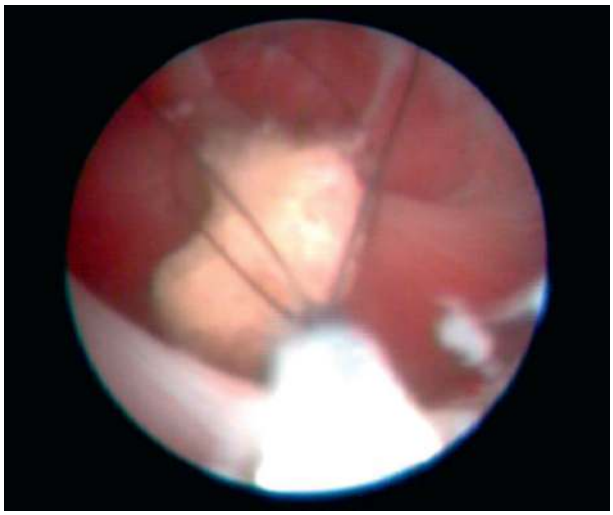

FIG 75.7 Endoscopic retrieval of renal calculus. (From Azili MN, Ozcan F, Tiryaki T. Retrograde intrarenal surgery for the treatment of renal stones in children: factors influencing stone clearance and complications. *J Pediatr Surg*. 2014;49(7):1161–1165.)

drugs may increase intraurethral pressure and should be avoided. Intravenous lidocaine infusions may provide palliation of the acute pain of renal and ureteral calculi. Local heat to the painful flank may also provide symptomatic relief. Thiazide diuretics may help prevent the recurrence of calcium stones as will potassium citrate.

COMPLICATIONS AND PITFALLS

The major problem in the care of patients thought to be suffering from nephrolithiasis is related to incorrect diagnosis where the signs and symptoms of a life-threatening condition (e.g., dissecting aortic aneurysm) are attributed to kidney stones. A failure to promptly diagnose urosepsis associated with nephrolithiasis can cause significant mortality and morbidity. A failure to identify the underlying cause of calculi formation can result in recurrent episodes of pain and risks compromise of renal function over time.

CLINICAL PEARLS

Nephrolithiasis is a common cause of flank, abdominal, groin, and genital pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious retroperitoneal, intraabdominal, or intrapelvic disorders. Pharmacologic agents, including nonsteroidal antiinflammatory agents, will help palliate the acute pain associated with kidney stones as will the infusion of intravenous lidocaine. In general, opioids are to be avoided as they may worsen the patient's symptoms. Dietary restriction of foods and drinks that may contribute to calculus formation (e.g., sweetened sodas, calcium, nuts, and chocolate) may help prevent recurrent stone formation in selected patients.

SUGGESTED READINGS

Bishop K, Momah T, Ricks J. Nephrolithiasis. *Prim Care*. 2020;47(4):661–671. https://doi.org/10.1016/j.pop.2020.08.005.

Chen TT, Wang C, Ferrandino MN, et al. Radiation exposure during the evaluation and management of nephrolithiasis. *J Urol*. 2015;194(4):878–885.

Edgar L, Ashenburg N. Nephrolithiasis diagnosed by point of care ultrasound. *Vis J Emerg Med*. 2020;21:100804. https://doi.org/10.1016/j.visj.2020.100804.

Elia M, Monga M, De S. Increased nephrolithiasis prevalence in people with disabilities: a National Health and Nutrition Survey analysis. *Urology*. 2022;163:185–189. https://doi.org/10.1016/j.urology.2021.07.047.

Feldman HA. Rolling stones: the evaluation, prevention, and medical management of nephrolithiasis. *Physician Assist Clin*. 2016;1(1):127–147.

Ingimarsson JP, Krambeck AE, Pais VM Jr. Diagnosis and management of nephrolithiasis. *Surg Clin North Am*. 2016;96(3):517–532.

Lui MS, Jason CF, Underwood HJ, Patel KN, Ogilvie JB. Stones left unturned: missed opportunities to diagnose primary hyperparathyroidism in patients with nephrolithiasis. *Surgery*. 2022;171(1):23–28. https://doi.org/10.1016/j.surg.2021.03.073.

Pfau A, Knauf F. Update on nephrolithiasis: core curriculum 2016. *Am J Kidney Dis*. 2016;68(6):973–985.

Ruggenenti P, Caruso MR, Cortinovis M, et al. Fresh lemon juice supplementation for the prevention of recurrent stones in calcium oxalate nephrolithiasis: a pragmatic, prospective, randomised, open, blinded endpoint (PROBE) trial. *eClinicalMedicine*. 2022;43:101227. https://doi.org/10.1016/j.eclinm.2021.101227.

Vestergaard P. Primary hyperparathyroidism and nephrolithiasis. *Ann Endocrinol (Paris)*. 2015;76(2):116–119.

Virapongse A. Nephrolithiasis. *Hosp Med Clin*. 2016;5(1):43–57.

Yogo A, Yamamoto S, Sumiyoshi S, et al. Two cases of pyelonephritis with bacteremia by *Staphylococcus epidermidis* in male patients with nephrolithiasis: case reports and a literature review. *J Infect Chemother*. 2022. https://doi.org/10.1016/j.jiac.2022.04.030.

Thoracic Vertebral Compression Fracture

ICD-10 CODES S22.009A

THE CLINICAL SYNDROME

Thoracic vertebral compression fracture is one of the most common causes of dorsal spine pain. Vertebral compression fractures are most often the result of osteoporosis (Fig. 76.1), but they may also occur with trauma to the dorsal spine caused by acceleration–deceleration injuries. In osteoporotic patients or in those with primary tumors or metastatic disease involving the thoracic vertebrae, fracture may occur with coughing (tussive fractures) or spontaneously.

The pain and functional disability associated with fractured vertebrae are determined largely by the severity of the injury (e.g., number of vertebrae involved) and the nature of the injury (e.g., whether the fracture causes impingement on the spinal nerves or spinal cord). The pain associated with thoracic vertebral compression fracture may range from a dull, deep ache (with minimal compression of the vertebrae and no nerve impingement) to severe, sharp, stabbing pain that limits the patient's ability to ambulate and cough.

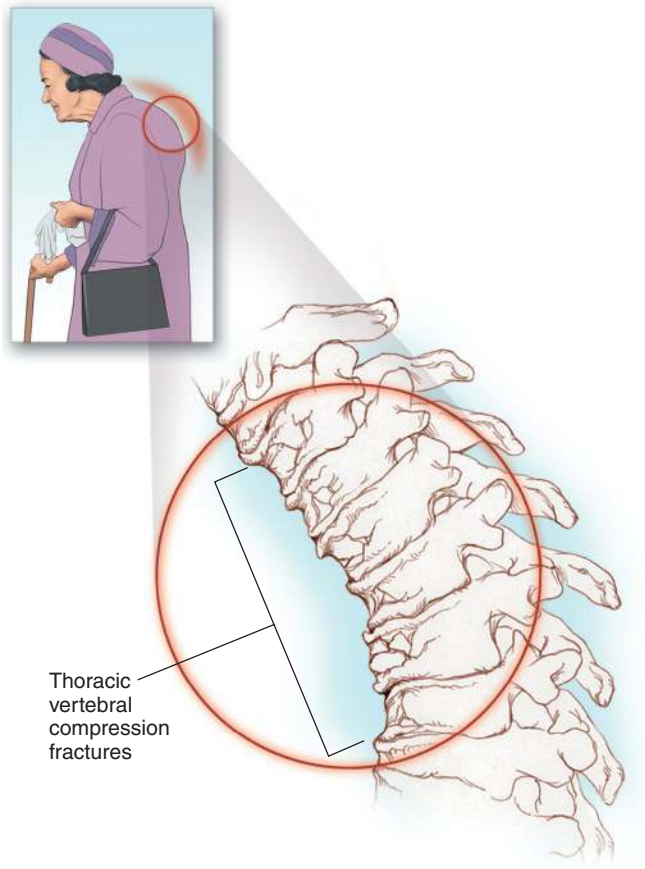

FIG 76.1 Osteoporosis is a common cause of thoracic vertebral fractures.

SIGNS AND SYMPTOMS

Compression fractures of the thoracic vertebrae are aggravated by deep inspiration, coughing, and any movement of the dorsal spine. Palpation of the affected vertebra may elicit pain and reflex spasm of the paraspinous musculature of the dorsal spine. If trauma has occurred, hematoma and ecchymosis may be present overlying the fracture site and the clinician should be aware of the possibility of damage to the bony thorax and the intraabdominal and intrathoracic contents. Damage to the spinal nerves may produce abdominal ileus and severe pain, resulting in splinting of the paraspinous muscles, and further compromise to the patient's pulmonary status and ability to ambulate. Failure to treat this pain and splinting aggressively may result in a negative cycle of hypoventilation, atelectasis, and, ultimately, pneumonia.

TESTING

Plain radiographs of the vertebrae are indicated to rule out other occult fractures and other bony disorders, including tumor (Fig. 76.2). Magnetic resonance imaging, radionucleotide bone scanning, and positron emission tomography may help characterize the nature of the fracture and distinguish benign from malignant causes of pain (Figs. 76.2 and 76.3). If trauma is present, radionuclide bone scanning may be useful to exclude occult fractures of the vertebrae or sternum. If no trauma is present, bone density testing to evaluate for osteoporosis is appropriate (Fig. 76.4), as are serum protein electrophoresis and testing for hyperparathyroidism. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Computed tomography of the thoracic contents is indicated if an occult mass or significant trauma is suspected. Electrocardiography to rule out cardiac contusion is indicated in all patients with traumatic sternal fractures or significant anterior dorsal spine trauma. The injection technique described later should be used early to avoid pulmonary complications.

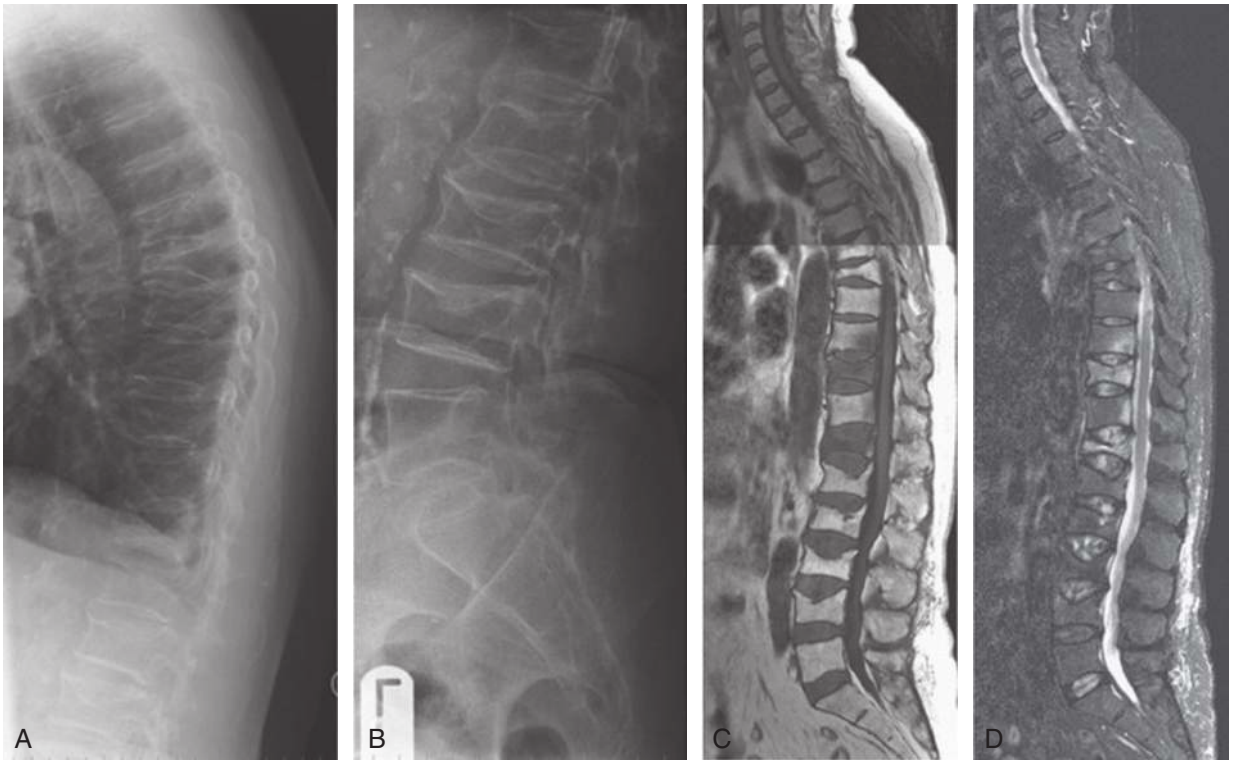

FIG 76.2 Lateral radiographs of the thoracic **(A)** and lumbar **(B)** spine demonstrate multilevel anterior vertebral body fractures. It is not possible to distinguish between acute and chronic fractures. The sagittal T1W **(C)** and STIR **(D)** magnetic resonance (MR) images, however, visualize multiple anterior wedge fractures. The recent acute fractures have marrow edema, which are low SI on the T1W MR image and high SI on the STIR image. The chronic fractures have normal fatty marrow SI. *STIR*, Short tau inversion recovery.

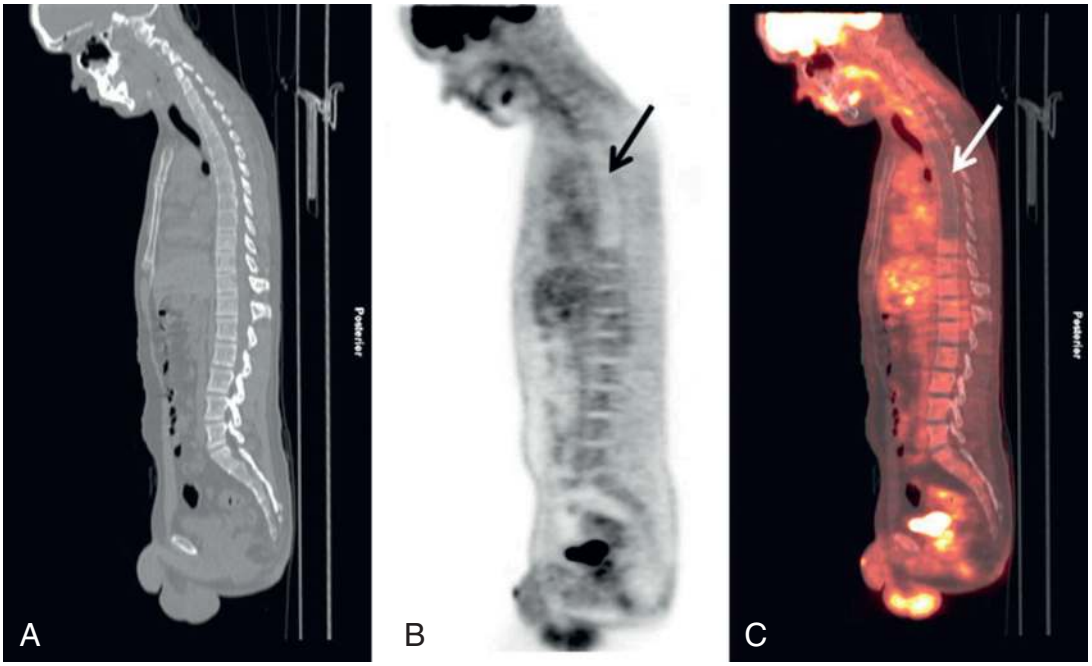

FIG 76.3 **A–C,** Sagittal computed tomography (CT), FDG-PET, and fused PET/CT images showing decreased FDG marrow uptake in thoracic vertebrae following external beam radiation for mediastinal malignancy (*arrows* in **B, C**). *FDG-PET*, Fluorodeoxyglucose-positron emission tomography. (From Wachsmann JW, Gerbaudo VH. Thorax: normal and benign pathologic patterns in FDG-PET/CT imaging. *PET Clin.* 2014;9(2):147–168.)

Body Composition Results

Region	Fat Mass (g)	Lean + BMC (g)	Total Mass (g)	% Fat	% Fat YN	Percentile AM
L Arm	1998	1954	3952	50.6		94
R Arm	1751	2078	3829	45.7		87
Trunk	14687	16207	30894	47.5		97
L Leg	5535	6271	11806	46.9		88
R Leg	5804	6293	12097	48.0		90
Subtotal	29776	32802	62578	47.6		96
Head	1194	3272	4466	26.7		
Total	**30970**	**36074**	**67044**	**46.2**		**95**
Android (A)	2210	2178	4387	50.4		
Gynoid (G)	4948	5468	10416	47.5		

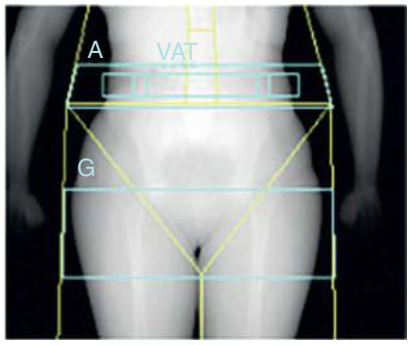

FIG 76.4 DXA uses the attenuation of x-ray beams to assess body composition. Fat, muscle, and bone differ in density, so they attenuate the x-ray beams in varying amounts. **A,** Shows that fat, muscle, and bone of a whole body image in an obese female, and accompanying body composition analysis results for each subregion of the body. **B,** Shows the outline of the android region (*A*) in the mid-trunk area, the gynoid region (*G*) in the hip area, and visceral adipose tissue region (*VAT*) in the center of the body. *DXA*, Dual-energy x-ray absorptiometry. (From Zemel BS. Body composition during growth and development. In: Cameron N, Schell LM, eds. *Human growth and development*. 3rd ed. Academic Press; 2022:chap 19, 517–545, https://doi.org/10.1016/B978-0-12-822652-0.00018-3.)

BOX 76.1 Differential Diagnosis of Vertebral Compression Fracture

- Pathologic fractures secondary to bone metastases from cancer
- Leukemia
- Lymphoma
- Metastatic disease
- Multiple myeloma
- Hyperparathyroidism
- Paget disease
- Scurvy
- Renal osteodystrophy
- Sickle cell anemia
- Homocystinuria/homocysteinemia

DIFFERENTIAL DIAGNOSIS

In the setting of trauma, the diagnosis of thoracic vertebral compression fracture is usually straightforward. In the setting of spontaneous vertebral fracture secondary to osteoporosis or metastatic disease, the diagnosis may be less clear-cut (Box 76.1). In this case, the pain of occult vertebral compression fracture is often mistaken for pain of cardiac or gallbladder origin, and it leads to visits to the emergency department and unnecessary cardiac and gastrointestinal workups. Acute sprain of the thoracic paraspinous muscles may be confused with thoracic vertebral compression fracture, especially if the patient has been coughing. Because the pain of acute herpes zoster may precede the rash by 3–7 days, it may erroneously be attributed to vertebral compression fracture (Fig. 76.5).

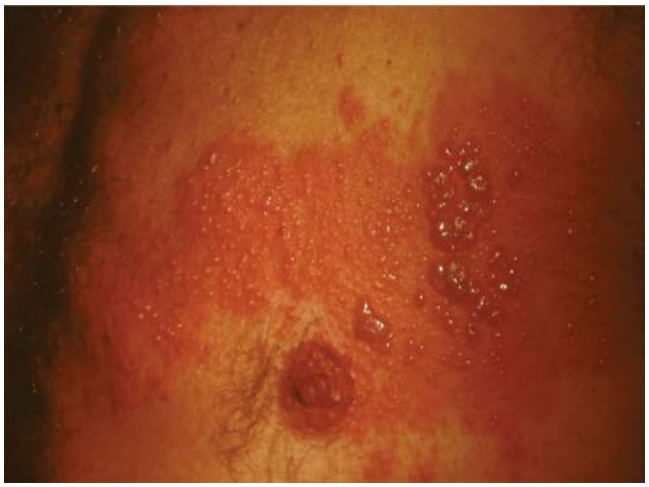

FIG 76.5 The pain of acute herpes zoster may mimic the pain of vertebral compression fracture. It is important to remember that the pain of acute herpes zoster may begin a few days before the vesicular rash appears.

TREATMENT

The initial treatment of pain secondary to compression fracture of the thoracic spine includes a combination of simple analgesics and nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors (Box 76.2). If these medications do not adequately control the patient's symptoms, a short-acting opioid analgesic such as hydrocodone is a reasonable next step. Because the opioid analgesics have the potential to suppress the cough reflex and respiration, the patient must be closely monitored and instructed in adequate pulmonary toilet techniques.

The local application of heat and cold or the use of an orthotic device (e.g., the Cash brace) may provide symptomatic relief. For patients who do not respond to these treatment modalities, thoracic epidural block with local anesthetic and steroid is a reasonable next step. Kyphoplasty with cement fixation of the fracture is also a good option if pain and decreased mobility become a problem (Figs. 76.6 and 76.7).

COMPLICATIONS AND PITFALLS

The major problem in the care of patients thought to be suffering from compression fractures of the thoracic vertebrae is failure to identify compression of the spinal cord or to recognize that the fracture is caused by metastatic disease. In patients with thoracic vertebral compression fractures resulting from osteoporosis, rapid pain control and early ambulation are mandatory to avoid complications such as pneumonia and thrombophlebitis.

BOX 76.2 Pharmacologic Treatments for Osteoporosis

Biophosphonates
- Ibandronate (Boniva)
- Alendronate (Binosto, Fosamax)
- Risedronate (Actonel, Atelvia)
- Zoledronic acid (Reclast, Zometa)

Monoclonal Antibody Medications
- Denosumab (Prolia, Xgeva)

Hormone-related Therapy
- Estrogen
- Raloxifene (Evista)
- Testosterone (in men)

Bone-building Medications
- Romosozumab (Evenity)
- Teriparatide (Forteo)
- Abaloparatide (Tymlos)

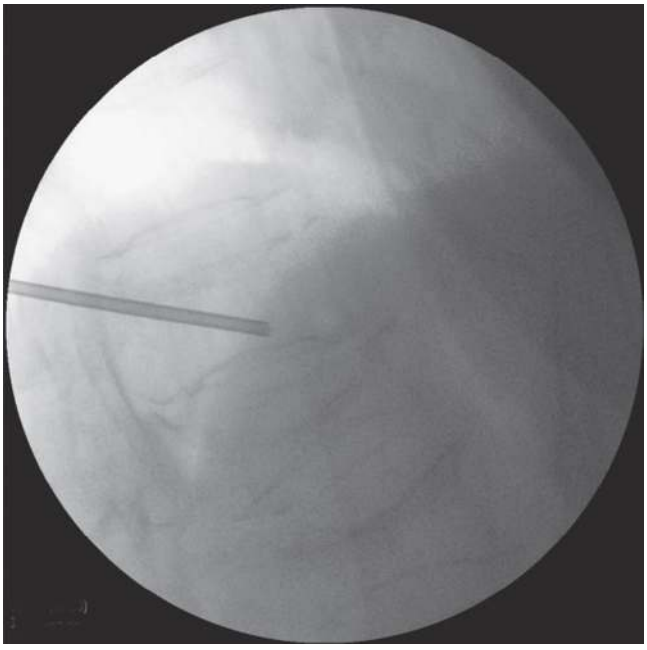

FIG 76.6 Trocar in the proper position to inject polymethyl methacrylate. Note the relative degree of demineralization of the vertebral body.

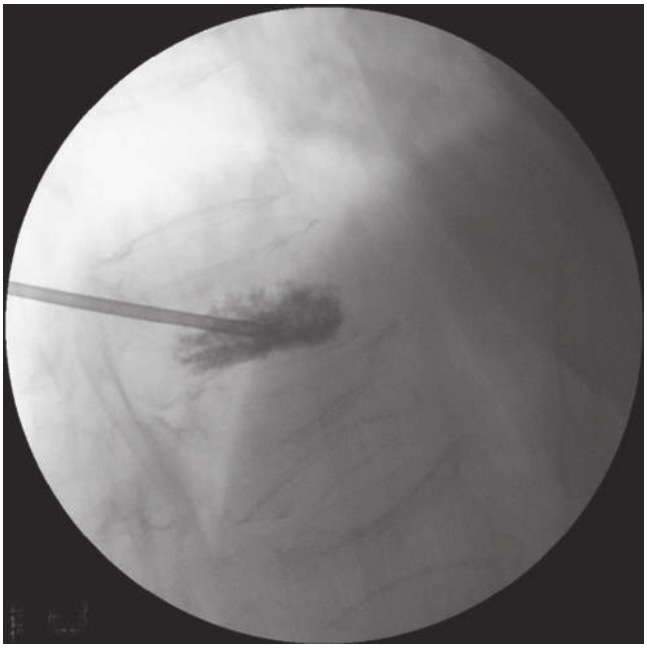

FIG 76.7 Fluoroscopy image showing satisfactory injection of polymethyl methacrylate into the vertebral body. Note the relative degree of demineralization of the vertebral body.

CLINICAL PEARLS

Compression fractures of the thoracic vertebrae are a common cause of dorsal spine pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious intrathoracic or upper intraabdominal disorders. Pharmacologic agents usually provide adequate pain control. If necessary, thoracic epidural block is a simple technique that can produce dramatic pain relief.

SUGGESTED READINGS

Goz V, Errico TJ, Weinreb JH, et al. Vertebroplasty and kyphoplasty: national outcomes and trends in utilization from 2005 through 2010. *Spine J*. 2015;15(5):959–965.

Kendler L, Bauer DC, Davison KS, et al. Vertebral fractures: clinical importance and management. *Am J Med*. 2016;129(2): 221.e1–221.e10.

Miller PD. Clinical management of vertebral compression fractures. *J Clin Densitom*. 2016;19(1):97–101.

Schousboe JT. Epidemiology of vertebral fractures. *J Clin Densitom*. 2016;19(1):8–22.

Waldman SD. Percutaneous kyphoplasty. In: *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2016:866–876.

Waldman SD. Percutaneous vertebroplasty. In: *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2016:874–878.

Waldman SD. Thoracic radiculopathy. In: *Pain review*. 2nd ed. Philadelphia: Saunders; 2017:228–229.

Waldman SD. Functional anatomy of the thoracic spine. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 4th ed. Philadelphia: Elsevier; 2020.

Waldman SD. Thoacic epidural nerve block: the translaminar approach. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2020.

77

Gastroesophageal Reflux Disease

ICD-10 CODE K21.9

THE CLINICAL SYNDROME

Gastroesophageal reflux disease (GERD) is caused by the refluxing of stomach contents into the esophagus. In most patients, symptoms are limited to heartburn, dysphagia, and regurgitation of stomach contents (Box 77.1). When the endogenous physiologic defense mechanisms designed to protect the esophagus and prevent reflux are defective or if there is excess acid production within the stomach, complications can occur. These complications include esophagitis, esophageal stricture, Barrett esophagus, laryngeal damage, lung damage, bleeding, and otitis media in infants. GERD occurs more commonly in females, and obesity, overeating, certain foods, caffeine, alcohol, antihistamine, alpha blockers, nitrates, and antidepressant use may increase the incidence of GERD (Box 77.2). There is an increased incidence of GERD during pregnancy.

SIGNS AND SYMPTOMS

The diagnosis of GERD is most often made on the basis of the presence of the classic triad of GERD: (1) heartburn; (2) dysphagia; and (3) regurgitation (Figs. 77.1 and 77.2). Atypical symptoms including cough, wheezing, and chest pain are common as are halitosis and a sour taste in the mouth. Heartburn is felt by the patient as a retrosternal burning sensation or feeling of pressure that occurs after eating or when bending over or lying in the supine position. Heartburn may be accompanied by regurgitation which is the effortless reflux of the gastric and or esophageal contents

BOX 77.1 Symptoms Associated With GERD

- Heartburn
- Dysphagia
- Regurgitation
  - Pyosis
  - Brash water
  - Sour taste in the mouth
- Coughing
- Aspiration
- Laryngitis
- Hoarseness
- Bronchospasm
- Pneumonia
- Pneumonitis
- Dysphagia

BOX 77.2 Foods Associated With GERD

- Fried or fatty foods
- Citrus
- Chocolate
- Caffeine-containing beverages
- Carbonated beverages
- Alcohol

FIG 77.1 GERD is a common gastrointestinal disorder associated with heartburn, dysphagia, and regurgitation of stomach content. *GERD*, gastroesophageal reflux disease.

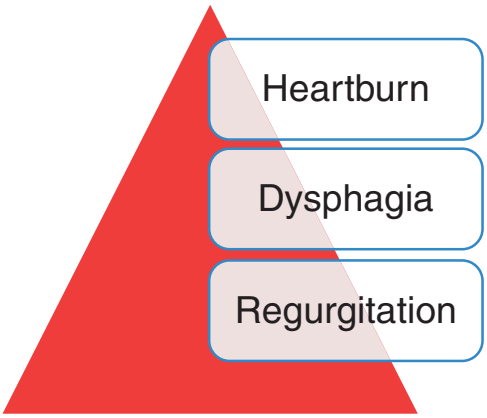

FIG 77.2 Triad of GERD. *GERD*, gastroesophageal reflux disease.

into the hypopharynx, pharynx, or mouth. If GERD remains untreated, difficulty in swallowing often occurs. This dysphagia is the result of abnormal esophageal motility, esophageal stricture, or both (Fig. 77.3). Hoarseness may be present, and wheezing may be identified on auscultation as a result of aspiration of gastric and esophageal contents into the tracheobronchial tree or from increased vagal tone (Fig. 77.4).

TESTING

Most clinicians include esophagogastroduodenoscopy, esophageal manometry, and ambulatory 24-hour pH monitoring in the initial workup of the patient presenting with GERD that has not responded to lifestyle modifications and a short course of H2 receptor antagonists or proton pump inhibitors and

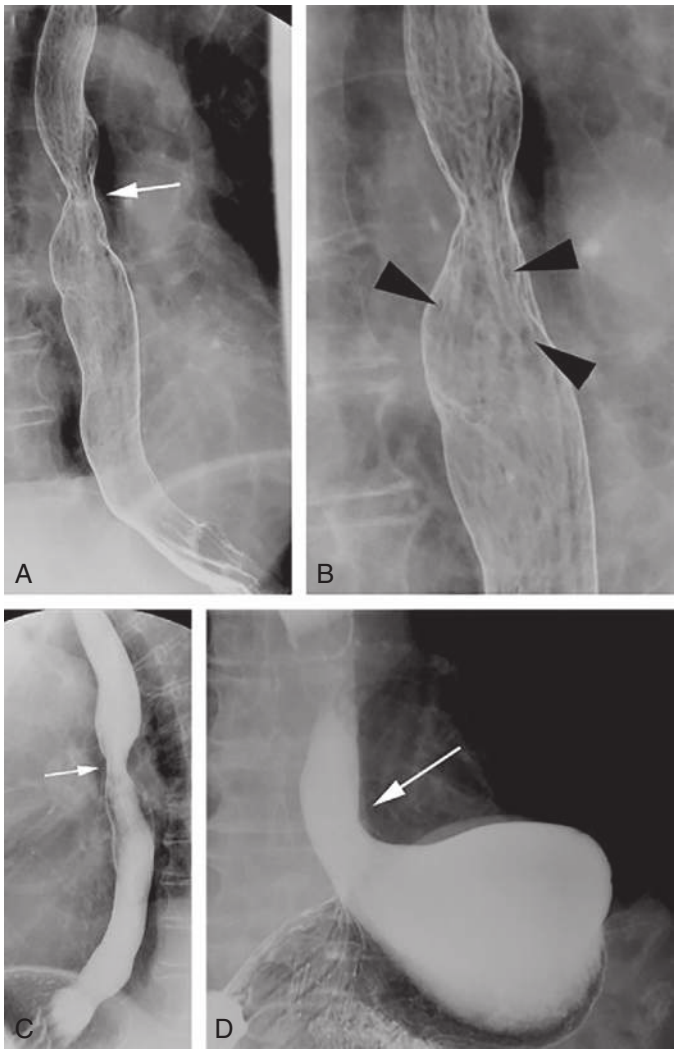

FIG 77.3 Barrett stricture in a patient with long-standing GERD and solid-food dysphagia. **A,** Smooth, tapered narrowing (*white arrow*) at the level of the left pulmonary artery (mid-esophagus) on the air-contrast portion of the examination. **B,** A smaller field-of-view spot film of the air-contrast portion showing nodular folds (*black arrowheads*). **C,** Persistent, smooth narrowing (*white arrow*) on the semiprone, full-column portion of the examination. **D,** Spontaneous and continuous reflux (*white arrow*) in the supine portion of the examination. This continuous reflux was present to the level of the cervical esophagus and never cleared. *GERD*, gastroesophageal reflux disease. (From Baker ME, Einstein DM. Barium esophagram: does it have a role in gastroesophageal reflux disease? *Gastroenterol Clin North Am.* 2014;43(1):47–68.)

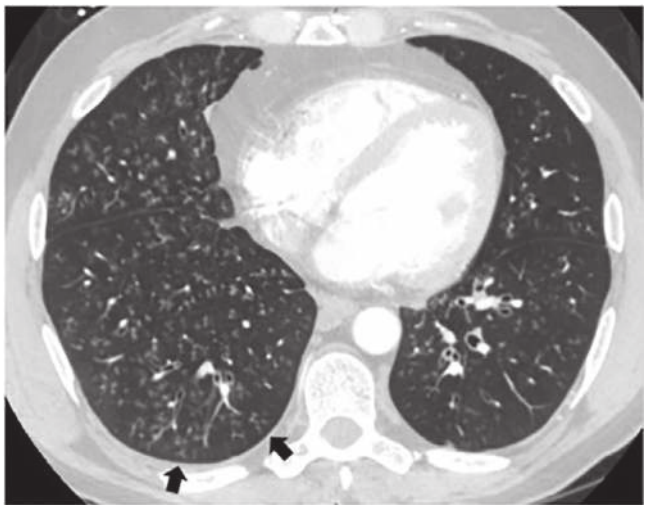

FIG 77.4 High-resolution CT (HRCT) scan of diffuse aspiration bronchiolitis. An HRCT scan of the chest shows micronodular opacities throughout both lungs, more numerous in the right lung. "Tree-in-bud" branching opacities (*arrows*) are seen in peripheral lung zones. *CT*, computed tomography. (From Hu X, Lee JS, Pianosi PT, Ryu JH. Aspiration-related pulmonary syndromes. *Chest*. 2015;147(3):815–823.)

antacids (Figs. 77.5 and 77.6). Upper gastrointestinal barium contrast-enhanced studies are also indicated early in the evaluation process to provide diagnostic information regarding the esophagus, the presence of hiatal hernia, the physiology of swallowing, and if there is reflux into the tracheobronchial tree (see Figs. 77.4 and 77.7). There has been recent interest in the use of scintigraphy as a diagnostic tool to identify the anatomic basis for reflux symptoms (Fig. 77.8).

DIFFERENTIAL DIAGNOSIS

Although the diagnosis of GERD is generally straightforward, the presence of atypical symptoms may lead to diagnostic misadventures. The clinician must remember that GERD may be the result of secondary gastroesophageal reflux and simply the presenting symptom of more serious undiagnosed pathology such as gastric or mediastinal tumors which may alter the normal gastroesophageal anatomy. Common diseases that may be misdiagnosed as GERD are listed in Box 77.3.

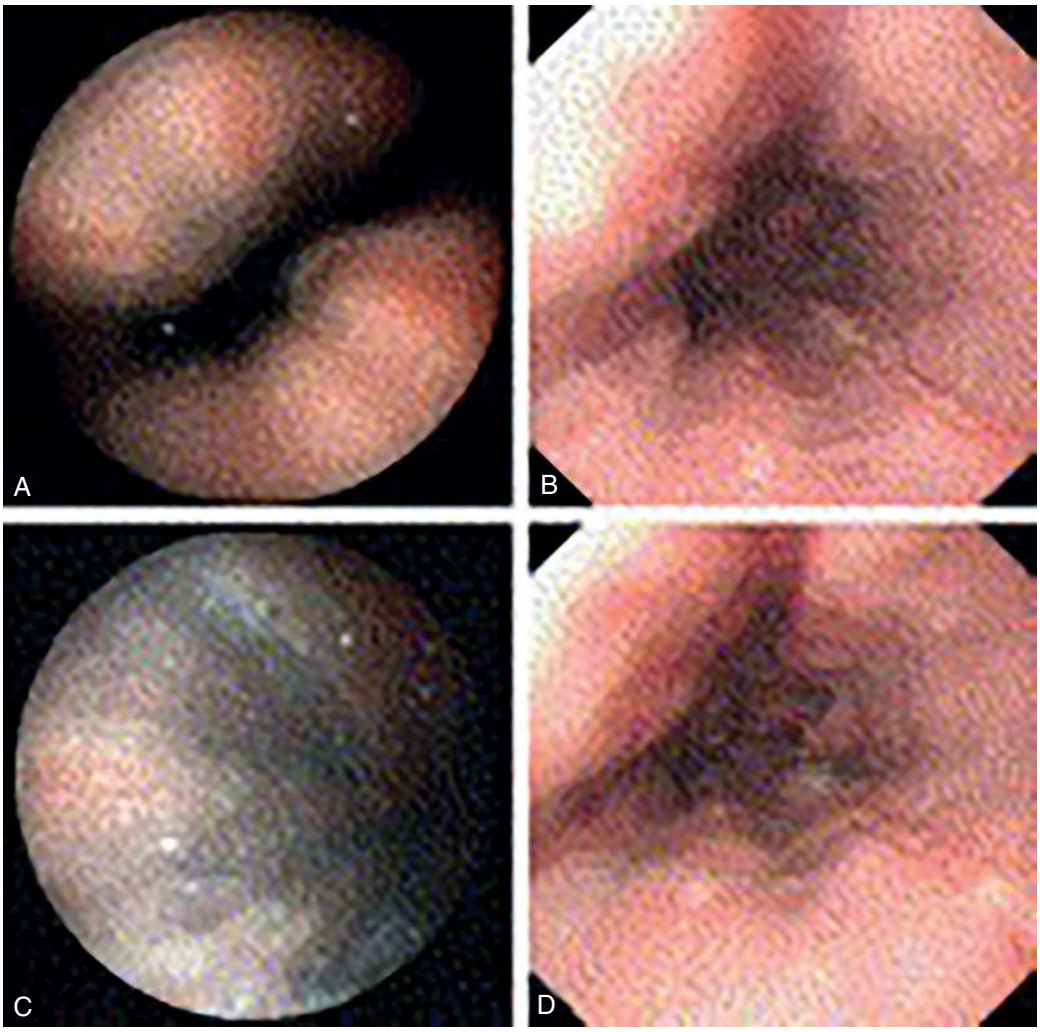

FIG 77.5 Endoscopic images of gastroesophageal reflux disease. (From Z. Liao, R. Gao, C. Xu, D.F. Xu, Z.S. Li, Sleeve string capsule endoscopy for real-time viewing of the esophagus: a pilot study (with video). *Gastrointest Endosc*, 70 (2009), pp. 201–209.)

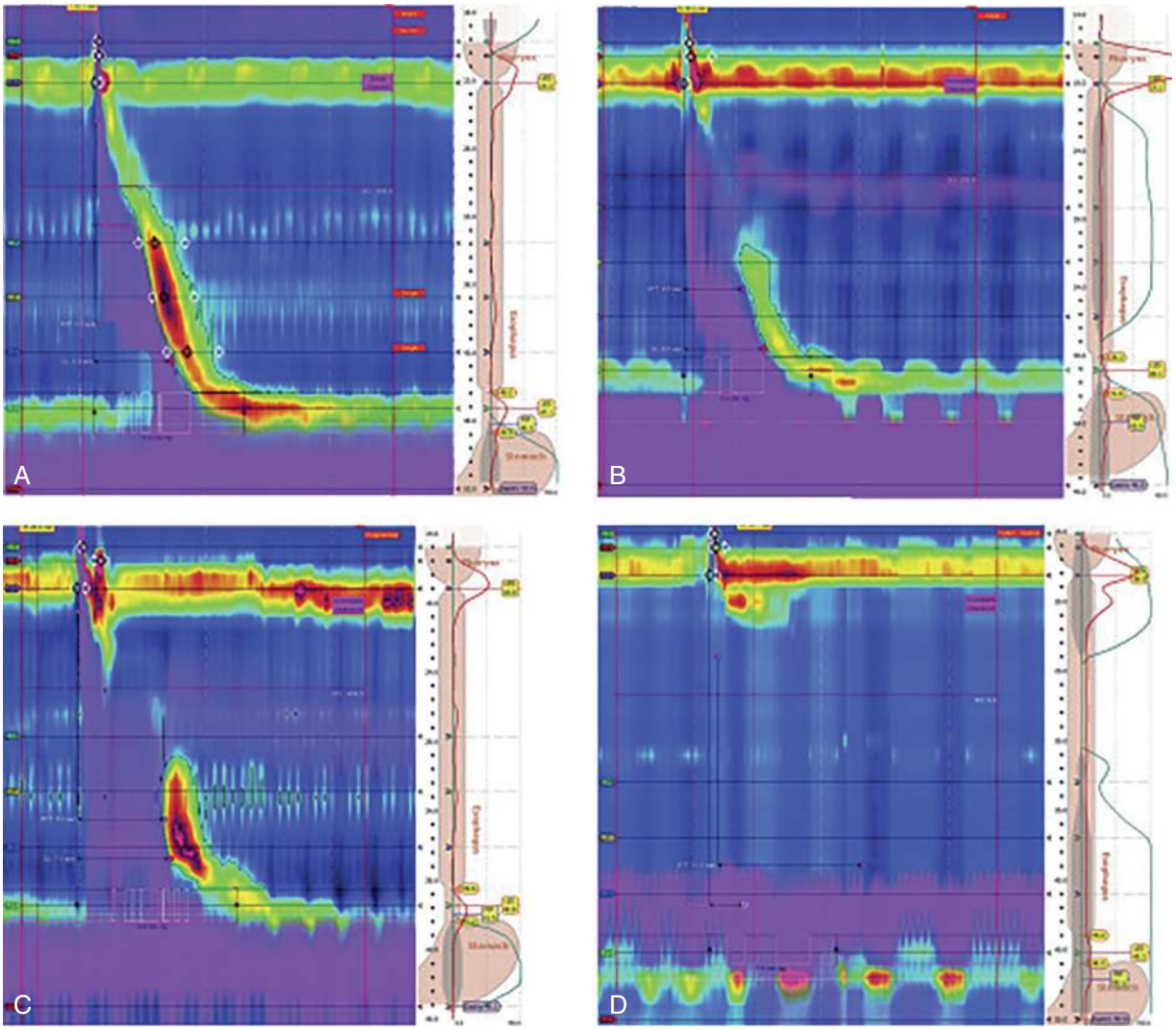

FIG 77.6 High-resolution esophogeal manometry. **A,** Normal esophageal motility. **B,** Ineffective esophogeal motility. **C,** Fragmented peristalsis. **D,** Failed peristalsis. *BTT,* bolus transit time; *DL,* distal latency; *PIP,* pressure inversion point; *SC,* secondary contraction; *UES,* upper esophageal sphincter. (From Bakhos CT, Abbas AE, Petrov RV. Tailoring endoscopic and surgical treatments for gastroesophageal reflux disease. *Gastroenterol Clin North Am.* 2020;49(3):467–480.)

TREATMENT

The goal of treatment of GERD is to provide prompt symptom relief, heal esophagitis, and to prevent the development of long-term complications related to recurrent or persistent esophagitis and damage to the lungs and teeth. The foundation to achieve these treatment goals center on lifestyle modification and pharmacotherapy. Lifestyle modifications include avoiding alcohol, chocolate, citrus, and tomato-based products as well as coffee and other caffeine-containing products. Changing eating habits by eating smaller more frequent meals and refraining from eating for 3 hours before lying down may also provide an immediate improvement of symptoms. Using bed blocks to raise the head of the bed at least 8 inches will provide relief of symptoms, but may interfere with sleep. For overweight patients, weight loss will also help (Box 77.4). Pharmacotherapy for the management of GERD should include the use of H2 receptor antagonists such as cimetidine, famotidine, nizatidine, and proton pump inhibitors such as omeprazole, lansoprazole, rabeprazole, esomeprazole, and pantoprazole. Short-term use of prokinetic agents such as metoclopramide and the limited use of antacids are also indicated (Box 77.5).

The use of surgical treatment of GERD is limited to those patients whose symptoms cannot be controlled with more conservative lifestyle modification and pharmacotherapy (Fig. 77.9). The presence of Barrett esophagus as well as the persistence of extraesophageal manifestations of GERD including dental, ear, nose and throat, and pulmonary symptoms may also suggest that surgical treatment is a reasonable next step. Surgical options consist of fundoplication procedures, magnetic esophageal closure devices, radiofrequency lesioning, muscosal ablation, and more extensive procedures such as roux en y surgery (Figs. 77.10–77.13).

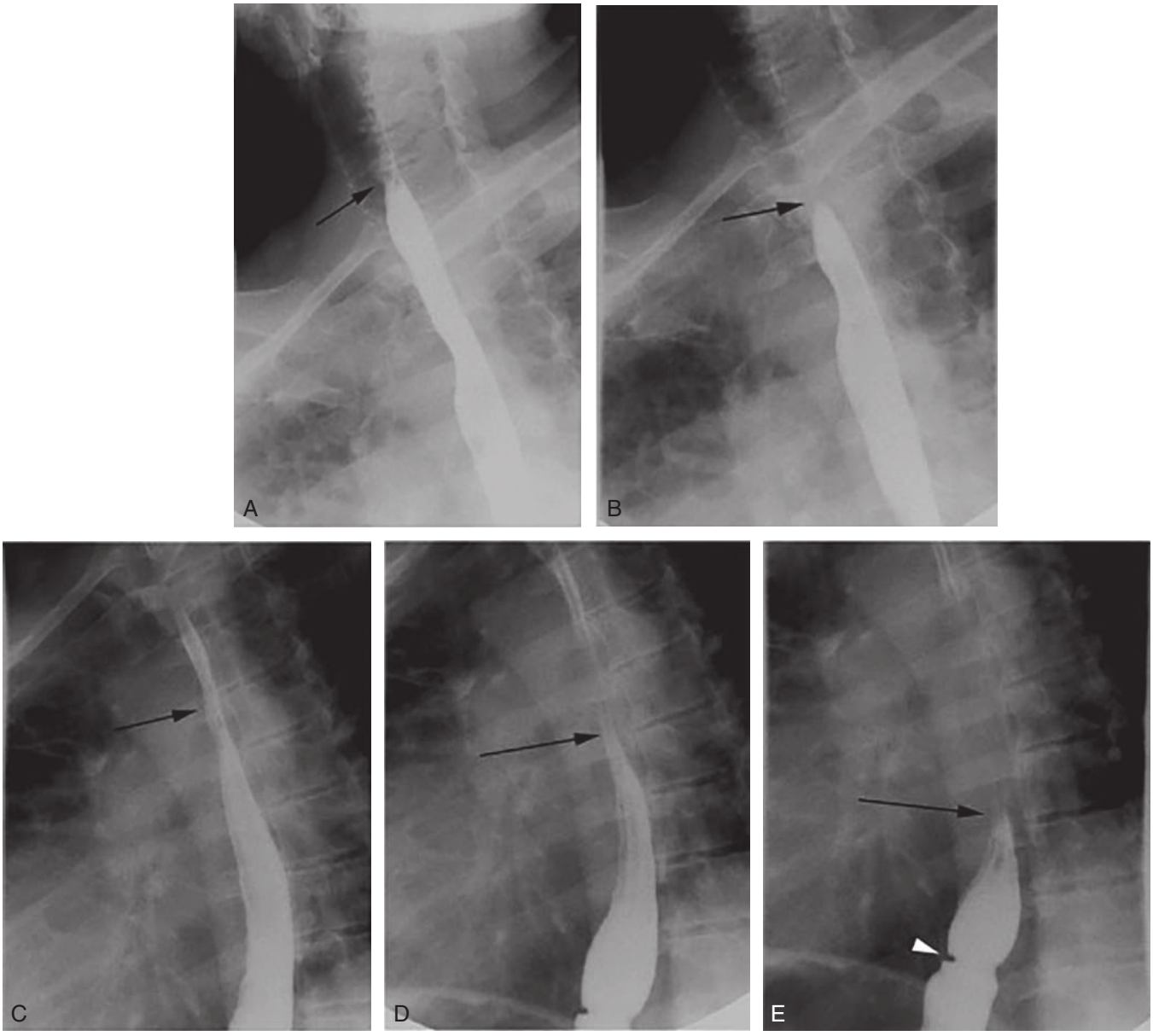

FIG 77.7 Esophageal motility. **A–E,** Freeze frames from a video esophagram demonstrating aboral transmission of the pressure wave distally from the cervical esophagus to the epiphrenic ampulla. The inverted V (*black arrow*) corresponds to the upstroke of the pressure wave. There is some retrograde escape of barium above the inverted V at the juncture of the proximal and middle third of the esophagus (at the juncture of the skeletal and smooth muscle) **(C, D)**. This finding is generally not clinically significant unless a large amount escapes above the pressure wave. There is also a distal mucosal ring (*white arrowhead* in **E**). (From Baker ME, Einstein DM. Barium esophagram: does it have a role in gastroesophageal reflux disease? *Gastroenterol Clin North Am.* 2014;43(1):47–68.)

COMPLICATIONS AND PITFALLS

Diagnostic misadventures when evaluating the patient suspected of suffering from GERD usually involve the failure to identify occult pathology that is responsible for the patient's symptoms. Gastrointestinal, mediastinal, and intrathoracic tumors, aneurysms, and infections are common culprits. Careful evaluation of those patients suffering from GERD who have failed to respond to conservative therapy is of paramount importance to avoid missing potentially serious diagnoses.

CLINICAL PEARLS

The combination of lifestyle modification and targeted pharmacotherapy will provide success symptom relief and management for most patients suffering from GERD. The use of antacids can result in rebound symptoms that can be difficult to manage so their use should be limited to rescue situations. It should be remembered that sometimes over the counter H2 antagonists and proton pump inhibitors are significantly more expensive than their prescription counterparts.

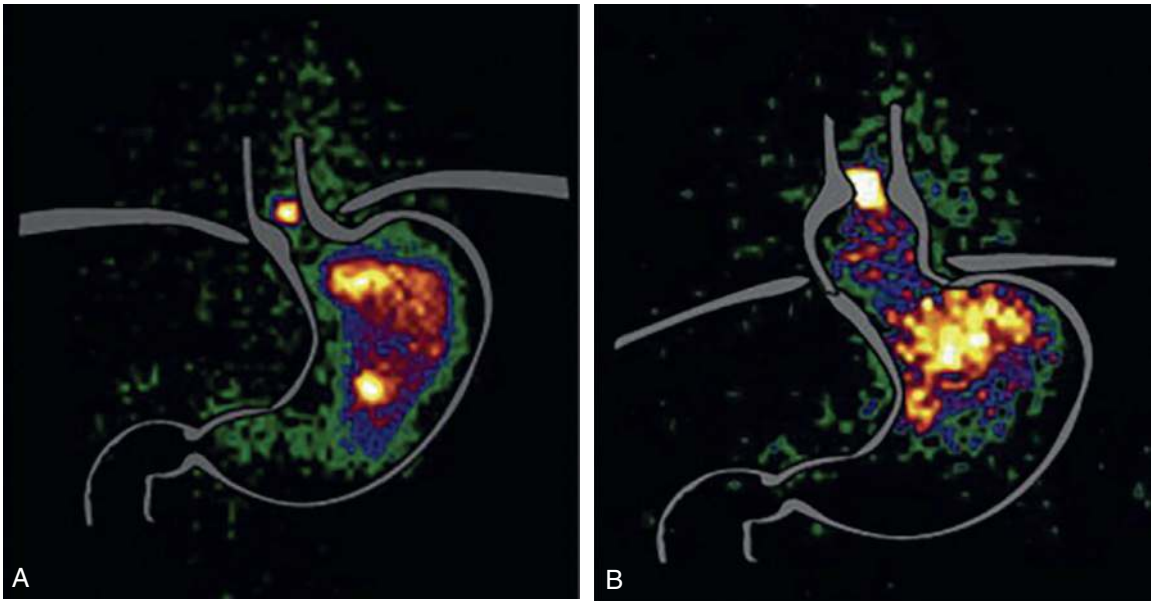

FIG 77.8 Acid pocket. **A, B,** Scintigraphic images of the acid pocket are demonstrated. Postprandially, the acid pocket is formed and located in the proximal stomach floating on top of the ingested food. In healthy volunteers and most patients without a hiatal hernia, the acid pocket is located below the crural diaphragm **(A)**. If the acid pocket is located below the diaphragm, the risk for acid reflux is low (10%–20%). In contrast, if the acid pocket is located in the hiatal sac and, thus, above the crural diaphragm **(B),** the risk for acidic reflux is very high (90%–95%). (From Boeckxstaens GE, Rohof WO. Pathophysiology of gastroesophageal reflux disease. *Gastroenterol Clin North Am.* 2014;43(1):15–25.)

BOX 77.3 Differential Diagnosis of GERD

- Hiatal hernia
- Esophagitis
- Esophageal cancer
- Gastritis
- Angina
- Esophageal spasm
- Peptic ulcer disease
- Helicobacter pylori infection
- Cholelithiasis
- Achalasia
- Esophageal motility disorders
- Irritable bowel disorders
- Functional bowel disorders
- Anxiety disorders
- Intestinal motility disorders

BOX 77.4 Lifestyle Modification for the Management of GERD

Foods to Avoid
Alcohol
Coffee and other caffeine-containing products
Chocolate
Citrus
Tomato-based products
Onions
Peppermint

Changes in Eating Habits
Avoiding large meals
Eating frequent small meals
No food or drink for 3 hours before lying down

Other Effective Lifestyle Modifications
Using bed blocks to raise the head of the bed at least 8 inches
Weight loss if overweight

BOX 77.5 Pharmacotherapy for Management of GERD

H2 Receptor Antagonists
Cimetidine
Famotidine
Nizatidine

Proton Pump Inhibitors
Omeprazole
Lansoprazole
Rabeprazole
Esomeprazole
Pantoprazole

Prokinetic Agents
Metoclopramide (short-term use only)
Antacids
Aluminum hydroxide
Magnesium hydroxide

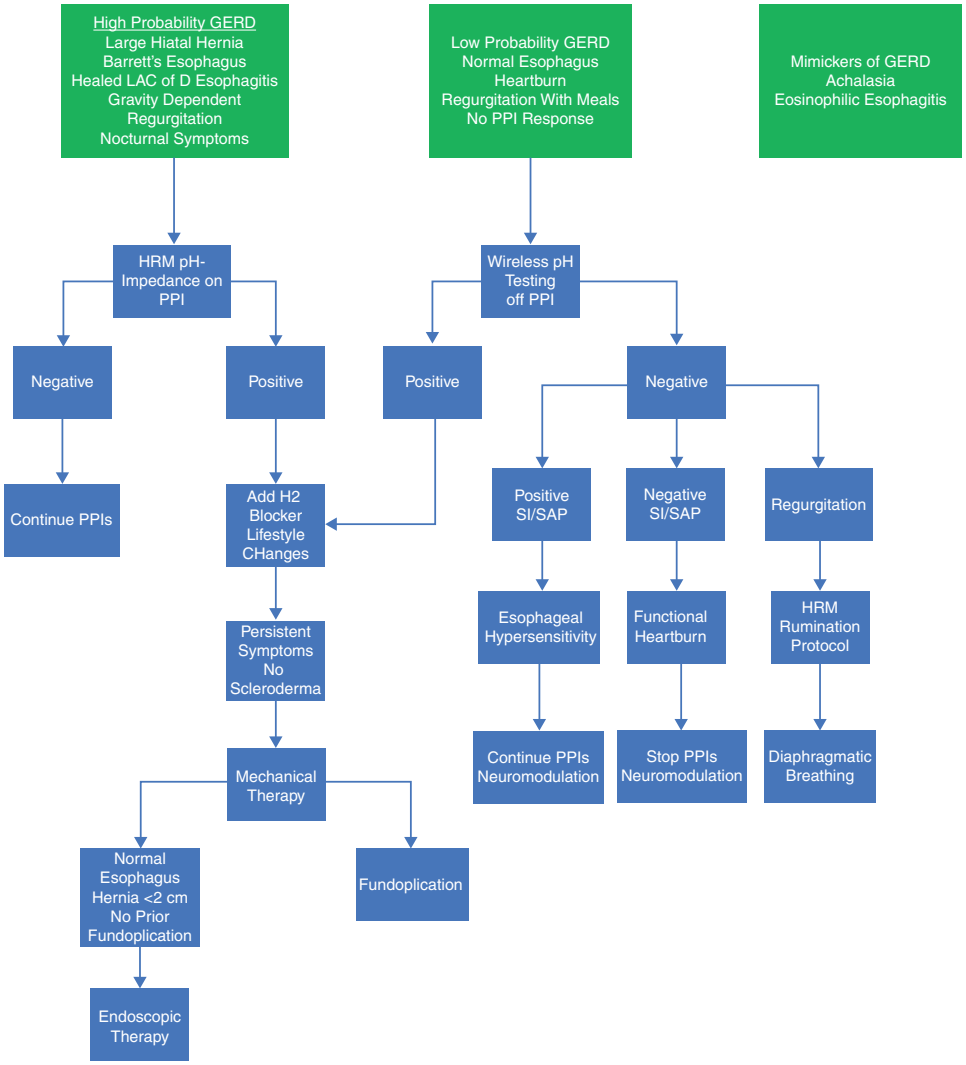

FIG 77.9 Approach to heartburn and regurgitation that do not respond to proton pump inhibitors. (From Snyder DL, Katzka DA. Complex gastroesophageal reflux disease. *Gastro Hep Adv.* 2022;1(3):420–430.)

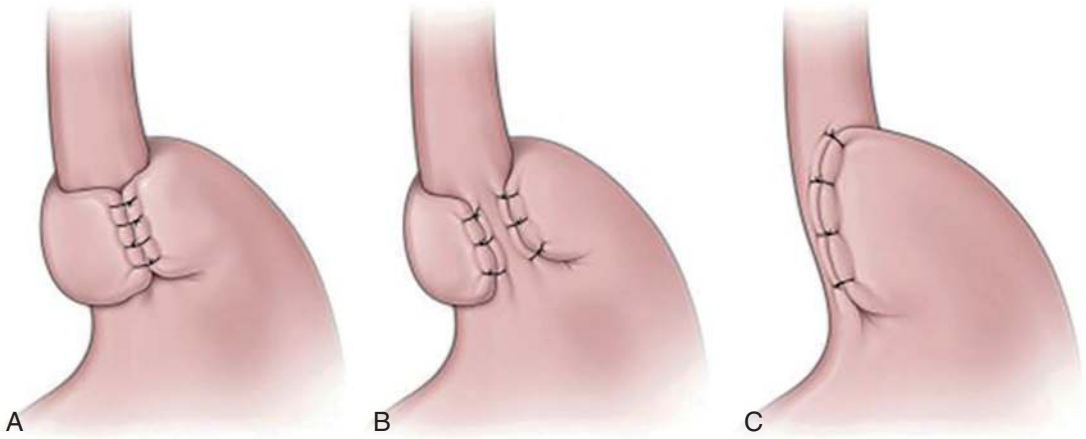

FIG 77.10 Different fundic wraps used in the management of GERD. **A,** Nissen fundoplication (360 degree posterior). **B,** Toupet fundoplication (270 degree posterior). **C,** Dor fundoplication (180 degree anterior). *GERD*, gastroesophageal reflux disease. (From Bakhos CT, Abbas AE, Petrov RV. Tailoring endoscopic and surgical treatments for gastroesophageal reflux disease. *Gastroenterol Clin North Am.* 2020;49(3):467–480.)

FIG 77.11 Magnetic sphincter augmentation. Intraoperative photo of the LINX device in place about the esophagus. *, Esophagus; ‡, right crus; S, spleen; †, stomach. (From Zimmermann CJ, Lidor A. Endoscopic and surgical management of gastroesophageal reflux disease. *Gastroenterol Clin North Am.* 2021;50(4):809–823.)

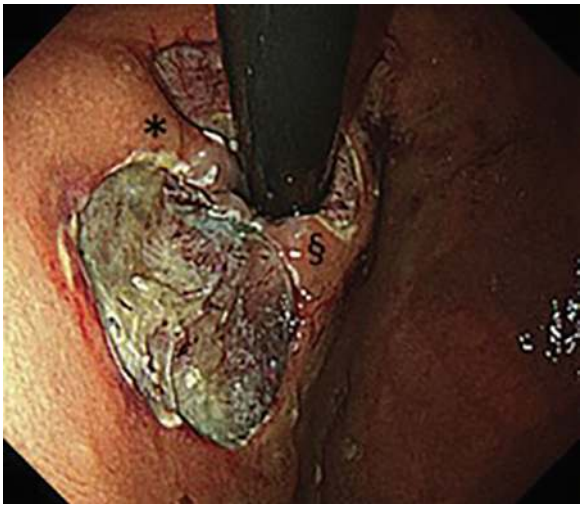

FIG 77.13 Antireflux mucosectomy. An endoscopic photograph with the endoscope in retroflexion showing a completed antireflux mucosectomy. Note the "butterfly" shape created about the gastroesophageal junction on the gastric side. * marks the lesser curvature of the stomach; § marks the greater curvature. (From Zimmermann CJ, Lidor A. Endoscopic and surgical management of gastroesophageal reflux disease. *Gastroenterol Clin North Am.* 2021;50(4):809–823.)

FIG 77.12 Radiofrequency ablation therapy for GERD. Procedure steps. **A,** Ablation catheter was introduced over the guidewire and positioned by using endoscopic visualization. **B,** Ablation catheter was inflated and energy was delivered to the tissue. **C,** After a second, more distal ablation, entire segment of BE was treated. (From Virender K. Sharma, Kenneth K. Wang, Bergein F. Overholt, Charles J. Lightdale, M. Brian Fennerty, Patrick J. Dean, Douglas K. Pleskow, Ram Chuttani, Alvaro Reymunde, Nilda Santiago, Kenneth J. Chang, Michael B. Kimmey, David E. Fleischer, Balloon-based, circumferential, endoscopic radiofrequency ablation of Barrett's esophagus: 1-year follow-up of 100 patients (with video), *Gastrointestinal Endoscopy*, 2007;65(2):185–195.)

SUGGESTED READINGS

Dunn C, Bildzukewicz N, Lipham J. Magnetic sphincter augmentation for gastroesophageal reflux disease. *Gastrointest. Endosc. Clin. North Am.* 2020;30(2):325–342. https://doi.org/10.1016/j.giec.2019.12.010.

Hastings J. Refractory gastroesophageal reflux disease: a closer look. *Physician Assist Clin.* 2021;6(4):613–623. https://doi.org/10.1016/j.cpha.2021.05.006.

Holmberg D, Santoni G, von Euler-Chelpin MC, et al. Incidence and mortality in upper gastrointestinal cancer after negative endoscopy for gastroesophageal reflux disease. *Gastroenterology.* 2022;162(2):431–438. e4. https://doi.org/10.1053/j.gastro.2021.10.003.

Kanemitsu Y, Kurokawa R, Takeda N, et al. Clinical impact of gastroesophageal reflux disease in patients with subacute/chronic cough. *Allergol Int.* 2019;68(4):478–485. https://doi.org/10.1016/j.alit.2019.04.011.

Milani DC, Borba M, Farré R, et al. Gastroesophageal reflux disease and dental erosion: the role of bile acids. *Arch Oral Biol.* 2022;139:105429. https://doi.org/10.1016/j.archoralbio.2022.105429.

Otaki F, Iyer PG. Gastroesophageal reflux disease and Barrett esophagus in the elderly. *Clin Geriatr Med.* 2021;37(1):17–29. https://doi.org/10.1016/j.cger.2020.08.003.

Saleh S, Trujillo S, Ghoneim S, et al. Effect of hormonal replacement therapy on gastroesophageal reflux disease and its complications in postmenopausal women. *Clin Gastroenterol Hepatol.* 2022. https://doi.org/10.1016/j.cgh.2022.01.058.

Snyder DL, Katzka DA. Complex gastroesophageal reflux disease. *Gastro Hep Adv.* 2022;1(3):420–430. https://doi.org/10.1016/j.gastha.2022.02.014.

78

Acute Appendicitis

ICD-130 CODE K35.80

THE CLINICAL SYNDROME

Acute appendicitis is one of the most common causes of abdominal pain, with an incidence of approximately 8.5% in males and 6.7% in females; the mortality rate is approximately 0.5%. Although acute appendicitis can occur at any age, it most commonly occurs in the second or third decades. Conventional wisdom holds that acute appendicitis is the result of obstruction of the appendicular lumen with subsequent impairment of the wall leading to perforation and phlegmon formation. More recent thinking posits that mild uncomplicated appendicitis and severe complicated appendicitis are caused by different pathologic processes and are in fact two completely separate diseases requiring very different treatments.

The diagnosis is made on clinical grounds in many countries, and appendectomy has remained the standard of care in the treatment of acute appendicitis for the last century. This is despite that approximately 15% of appendectomies yield a pathologically normal appendix and that appendectomy is not without morbidity and, rarely, mortality. The routine use of imaging, including ultrasound and computerized tomography (CT) as an adjunct to the clinical diagnosis of acute appendicitis, has decreased the number of "normal result" appendectomies to approximately 10%. Recent interest in the nonsurgical management of mild uncomplicated acute appendicitis is also impacting this statistic.

Abdominal pain is a common feature of acute appendicitis (Fig. 78.1). Although the clinical presentation of the pain of acute appendicitis can be variable, its classic clinical presentation begins as mild periumbilical pain that becomes more severe and then migrates to the right lower quadrant at a point that is one-third the distance from the anterior superior iliac spine and the umbilicus known as McBurney's point (Figs. 78.2 and 78.3). The pain becomes more localized and constant with associated anorexia, nausea, vomiting, and fever. Constipation and diarrhea, as well as urinary tract symptoms, may also occur. Symptoms are usually present for less than 48 hours before the patient seeks medical attention.

FIG 78.1 The patient suffering from acute appendicitis will have pain localized to the right lower quadrant at McBurney's point and associated anorexia, nausea, and vomiting. Low-grade fever is invariably present. (From Waldman SD, Acute appendicitis, Waldman SD, ed. *Atlas of common pain syndromes*. 4th ed. New York: Elsevier; 2019: chap 79, 306–310.)

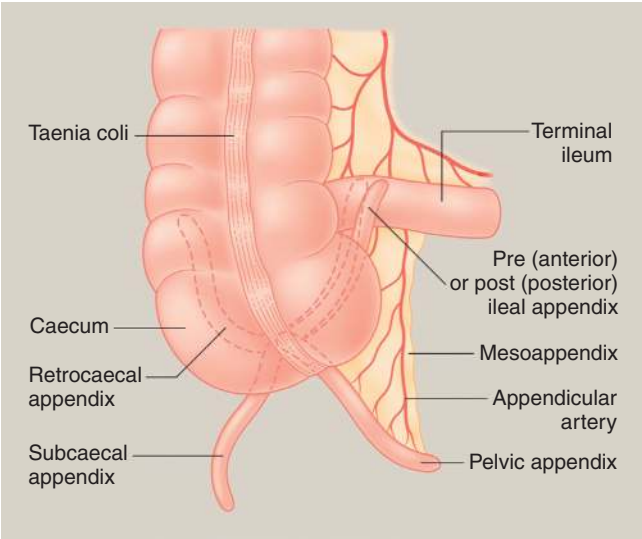

FIG 78.2 The anatomy of the appendix. Note the mesoappendix containing the appendicular artery in its lateral edge. The *dotted lines* depict a number of orientations the appendix is found within the population (pelvic, subcecal, retrocecal, pre, and postileal). (From Sellars H, Boorman P. Acute appendicitis. *Surgery (Oxford)*. 2017;35(8):432–438.)

SIGNS AND SYMPTOMS

Patients with acute appendicitis appear ill and anxious. A low-grade fever is often present. Patients will often flex their hips and draw up their knees in an effort to splint the abdomen and decrease the pain. Early in the course of the

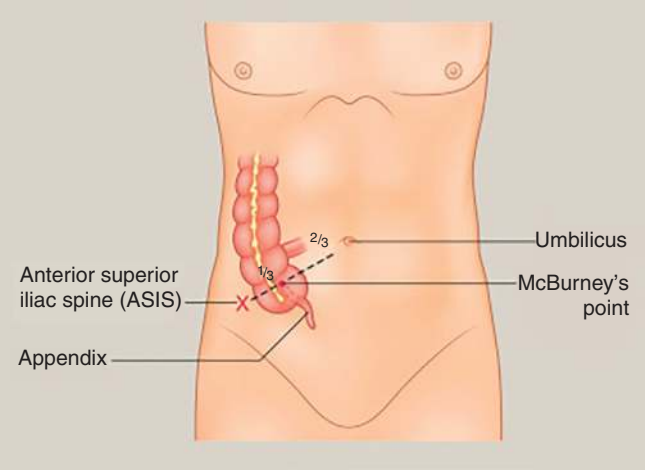

FIG 78.3 McBurney's point, the surface anatomy representing the base of the appendix. (From Sellars H, Boorman P. Acute appendicitis. *Surgery (Oxford)*. 2017;35(8):432–438.)

disease, there is diffuse periumbilical tenderness and nonspecific findings including decreased bowel sounds on physical examination. As the pain localizes to the right lower quadrant at McBurney's point, peritoneal signs including abdominal guarding, pain on percussion, and rebound tenderness become prominent. In addition to these physical findings, a number of physical examination tests can increase the diagnostic specificity of the physical examination (Table 78.1 and Fig. 78.4). All exploit the consistent finding of increased pain at the point of peritoneal or structural irritation when the test is performed in patients suffering from acute appendicitis. Although nonspecific, increased right-sided pain on rectal and vaginal examination may support the diagnosis of acute appendicitis, but more importantly helps rule out other pathologic processes that may mimic the disease.

TESTING

Currently, there is no specific laboratory test for acute appendicitis, but the finding of leukocytosis with a left shift and elevated C-reactive protein levels increase the likelihood of acute appendicitis by a factor of 5 when history and physical findings support that clinical diagnosis. Urinalysis may reveal mild pyuria that is thought to be caused by inflammation of the ureter secondary to the proximity of the inflamed appendix to the right ureter. Recent studies suggest that levels of urinary 5-hydroxyindoleacetic acid (5-HIAA) may be elevated in the early stages of acute appendicitis secondary to the inflammation of serotonin-containing cells within the appendix. A downward trajectory of urinary 5-HIAA levels after initial elevations is thought to correlate with disease progression. It should be noted that pregnancy testing is mandatory in all female patients of childbearing age presenting with abdominal pain.

Because of the desire to avoid unnecessary appendectomy, a scoring tool to improve the accuracy of diagnosis of acute appendicitis has been developed. The Alvarado score provides a consistent and reproducible tool to help diagnose acute appendicitis. The score is based on the scoring of symptoms, and physical and laboratory findings (Table 78.2). A score of 9–10 suggests that a diagnosis of appendicitis is highly probable, a score of 7–8 suggests that the diagnosis is probable, and a score of 5–6 is compatible with the diagnosis of acute appendicitis. Experience with use of the Alvarado score suggests that appendectomy should be considered in those patients with clinical findings suggesting acute appendicitis who have an Alvarado score of 7 or greater. Other scoring systems designed to improve the accuracy of acute appendicitis have been proposed. The Andersson Inflammatory Response Score expands the parameters of the

TABLE 78.1 Useful Physical Examination Tests When Diagnosing Acute Appendicitis

Test	Maneuver	Basis of Physical Findings
McBurney's point	Palpation at a point that is one-third the distance from the anterior superior iliac spine and the umbilicus yields maximal pain	Suggests peritoneal irritation at point where appendix attaches to cecum
Rovsing's sign	Palpation of left lower quadrant yields pain at McBurney's point	Suggests peritoneal irritation at McBurney's point
Dunphy sign	Having patient cough elicits sharp pain at McBurney's point	Suggests peritoneal irritation at McBurney's point
Markle sign	When the standing patient drops from standing on toes to the heels with a jarring landing, the pain increases at McBurney's point	Suggests peritoneal irritation at McBurney's point
Obturator sign	Internal and external rotation of the flexed right hip yields pain at McBurney's point	Suggests peritoneal irritation at McBurney's point and may point to a retrocecal appendix
Psoas sign	Extension of the right hip or with flexion of the right hip against resistance yields pain at McBurney's point	Suggests that appendix may lie against the psoas muscle
Blumberg sign	The abdomen is palpated and the pressure is suddenly released eliciting pain	Suggests peritoneal irritation

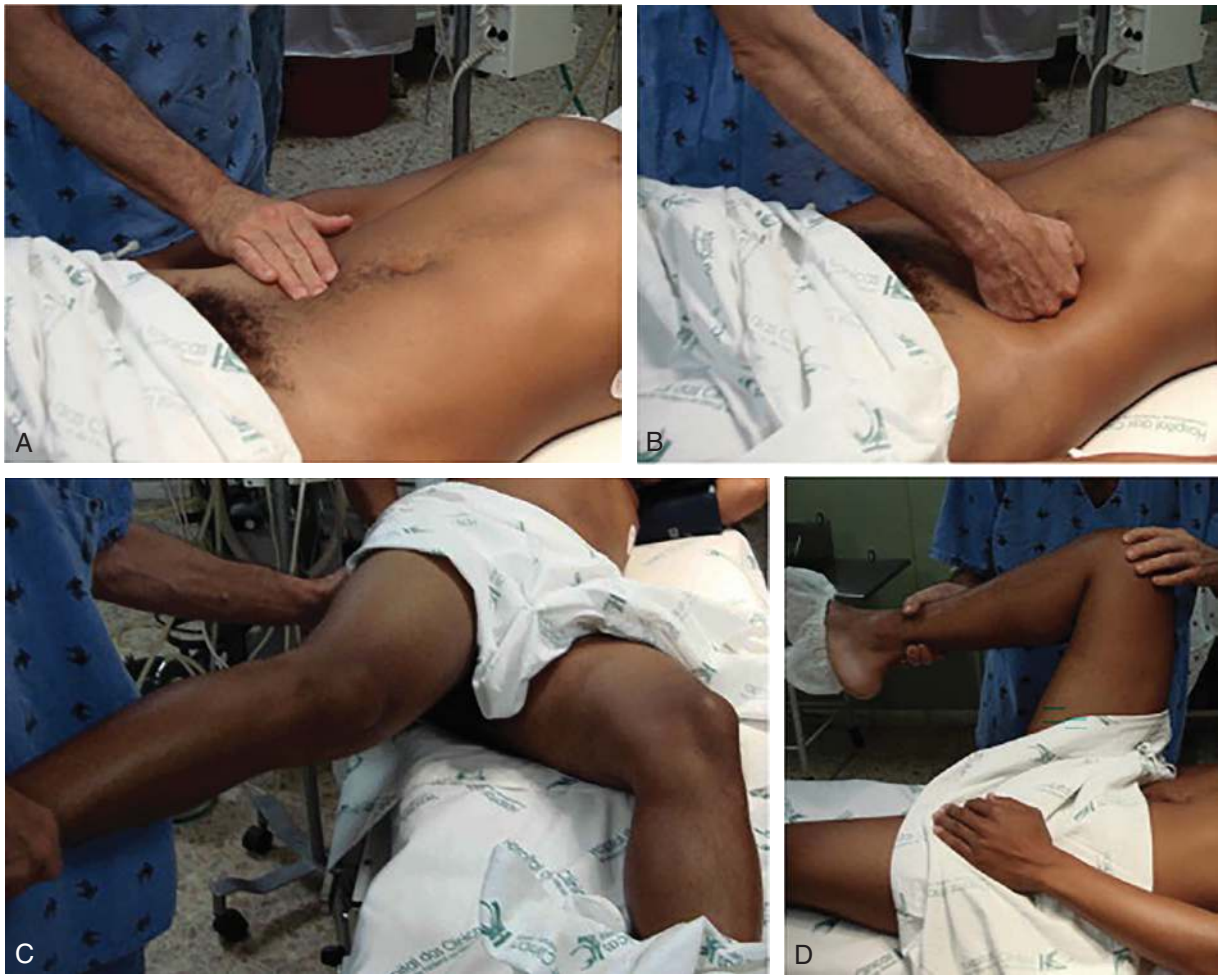

FIG 78.4 Physical exam of a patient with right abdominal pain. **A,** Blumberg's sign. **B,** Rovsing's sign. **C,** Psoas sign. **D,** Obturator sign. (From Petroianu A. Diagnosis of acute appendicitis. *Int J Surg*. 2012;10(3):115–119.)

TABLE 78.2 The Alvarado Scoring System for Acute Appendicitis

Symptoms	Migratory right iliac fossa pain	1
	Nausea/vomiting	1
	Anorexia	1
Signs	Right iliac fossa tenderness	2
	Elevation of temperature	1
	Rebound tenderness right iliac fossa	1
Laboratory	Leukocytosis	2
	Left shift	1
Total Score		1–10
Sum	0–4	Not likely appendicitis
	5–6	Equivocal
	7–8	Probably appendicitis
	9–10	Highly likely appendicitis

Alvarado scoring system by adding gradations of physical findings and laboratory parameters, including the C-reactive protein, and deducting points for temperatures above 38°C (Table 78.3).

The use of point-of-care ultrasound imaging has become a mainstay in the diagnosis of acute appendicitis. In health, appendicitis is not easily identifiable on ultrasound imaging (Fig. 78.5). As the appendix becomes inflamed, it is more easily identifiable as a noncompressible tubular structure of 7–9-mm in diameter that is surrounded by fluid and that lacks peristalsis. The presence of an appendicolith, phlegmon, and free air may also be identified on ultrasound imaging of the right lower quadrant. Ultrasound imaging may also provide information regarding surrounding structures or provide alternative causes for the patient's abdominal pain especially in females of childbearing age. Limitations on the use of ultrasound to diagnose acute appendicitis include operator experience, equipment quality, the presence of large quantities of intestinal gas, patient obesity, and abnormal positioning of the appendix (e.g., retrocecal; see Fig. 78.5).

TABLE 78.3 The Andersson Scoring System for Acute Appendicitis

		Andersson Inflammatory Response Score
Vomiting		1
Pain in right inferior fossa		1
Rebound tenderness or muscular defense	Light	1
	Medium	2
	Strong	3
Body temperature >38°C		0
		1
White blood cell count	10.0–78.9 × 109/L	1
	15.0 × 109/L	2
Polymorphonuclear leukocytes	70%–84%	1
	>85%	2
C-Reactive protein concentration	10–49 g/L	1
	>50 g/L	2
Total Score		**0–12**
If the sum is:		
0–4	Low probability. Outpatient follow-up if unaltered general condition.	
5–8	Indeterminate group. In hospital active observation with re-scoring/imaging or diagnostic laparoscopy according to local traditions.	
9–12	High probability. Surgical exploration is proposed.	

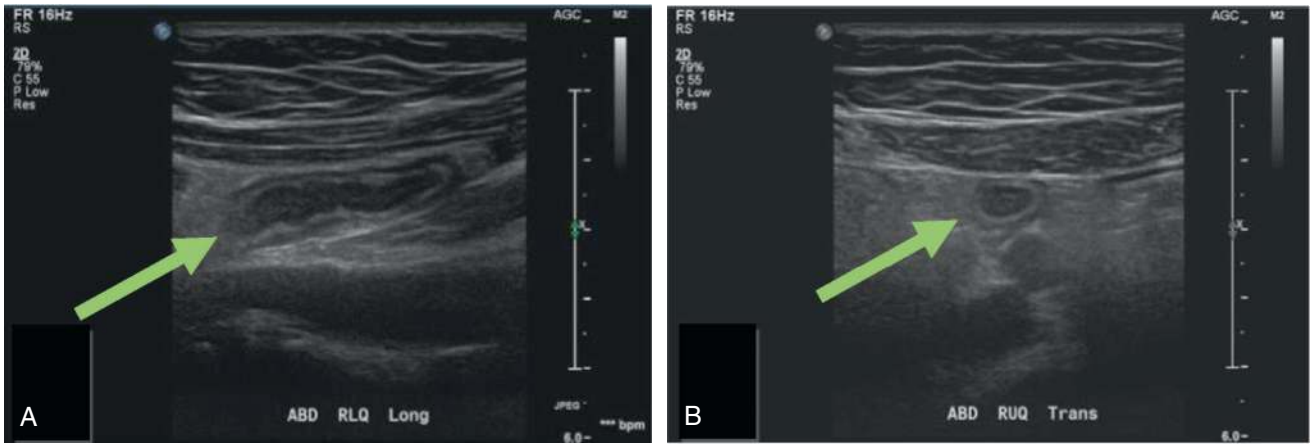

FIG 78.5 Ultrasound demonstrating acute appendicitis. A, Longitudinal view. B, Transverse view. Ultrasound of right lower quadrant. Patient presented with a 72-hour history of abdominal pain that now localizes to the right lower quadrant. Ultrasound demonstrated acute wall thickening and dilation up to 9.1-mm. Note fluid surround the appendix. (From Murphy EEK, Berman L. Clinical evaluation of acute appendicitis. *Clin Pediatr Emerg Med*. 2014;15(3):223–230.)

CT has an even higher degree of specificity and sensitivity when used in the diagnosis of acute appendicitis with a positive predictive value approaching 98%. CT can provide all of the diagnostic information obtainable on ultrasound imaging of the appendix and can accurately identify circumferential appendiceal wall thickening, periappendicular fat stranding, and adjacent adenopathy that strengthens the diagnosis of acute appendicitis (Fig. 78.6). Limitations on the use of CT to diagnose acute appendicitis include availability, cost, and radiation exposure, especially in pregnant women and children.

Although plain radiography of the abdomen and barium enemas were commonly used when attempting to diagnose acute appendicitis, their use has been supplanted by ultrasound and CT (Fig. 78.7). Radionucleotide scanning with technetium Tc99 labeled white cells can accurately identify acute appendicitis, but the prolonged scan times of over 4 hours and cost limit clinical utility in this setting (Fig. 78.8). Magnetic resonance imaging has recently gained acceptance as an alternative to CT in selected patient populations, specifically children and pregnant women present with abdominal pain thought to be compatible with acute appendicitis and in patients in who ultrasound findings are nondiagnostic and when the patient has increased risk factors that weigh against exploratory laparotomy/laparoscopy (e.g., anticoagulation, recent myocardial infarction; see Fig. 78.8).

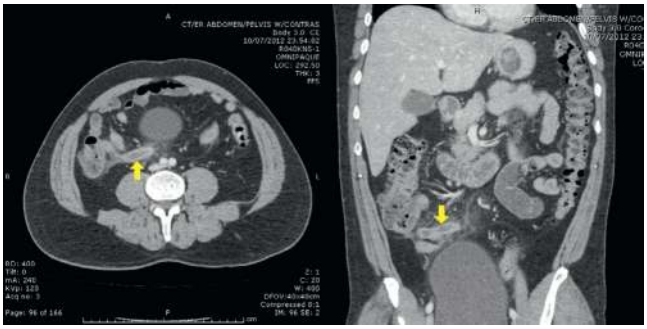

FIG 78.6 Computed tomography (CT) scan images (axial and coronal sections) demonstrating a dilated appendix, with adjacent fat stranding, suggestive of mild acute appendicitis. *Arrows*, dilated appendix. (From Teixeira PGR. Demetriades D. Appendicitis: changing perspectives. *Adv Surg*. 2013;47(1): 119–140.)

DIFFERENTIAL DIAGNOSIS

Most causes of acute abdominal pain can mimic the acute appendicitis. Most commonly, acute gastroenteritis, inflammatory bowel disease, right-sided diverticulitis, irritable bowel syndrome, ectopic pregnancy, ischemic colitis, psoas abscess, and mesenteric artery ischemia are misdiagnosed as diverticulitis. Black widow spider envenomation has also been misdiagnosed as acute appendicitis. Because the pain of acute herpes zoster may precede the rash by 3–5 days, it may erroneously be attributed to acute appendicitis.

TREATMENT

Any discussion regarding the treatment of acute appendicitis is that appendectomy remains the only 100% curative treatment

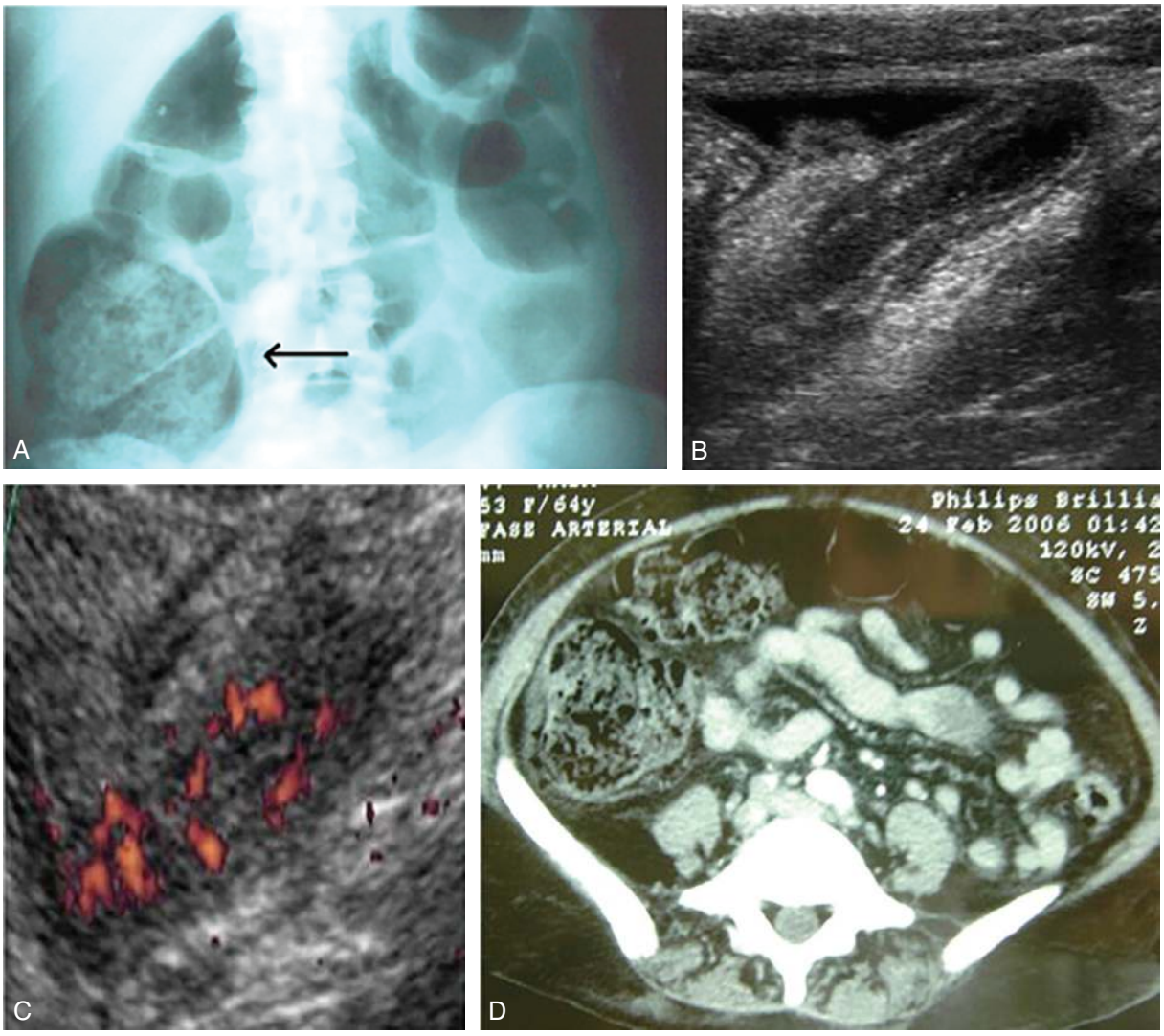

FIG 78.7 Abdominal images of appendicitis. **A,** Abdominal plain radiography showing distension of the cecum with fecal loading image. **B,** Abdominal ultrasound showing an enlarged appendix with a thick wall. **C,** Doppler ultrasound showing an inflamed appendix. **D,** Computed tomography of a patient with appendicitis. Observe the fecal loading in the cecum. (From Petroianu A. Diagnosis of acute appendicitis. *Int J Surg*. 2012;10(3):115–119.)

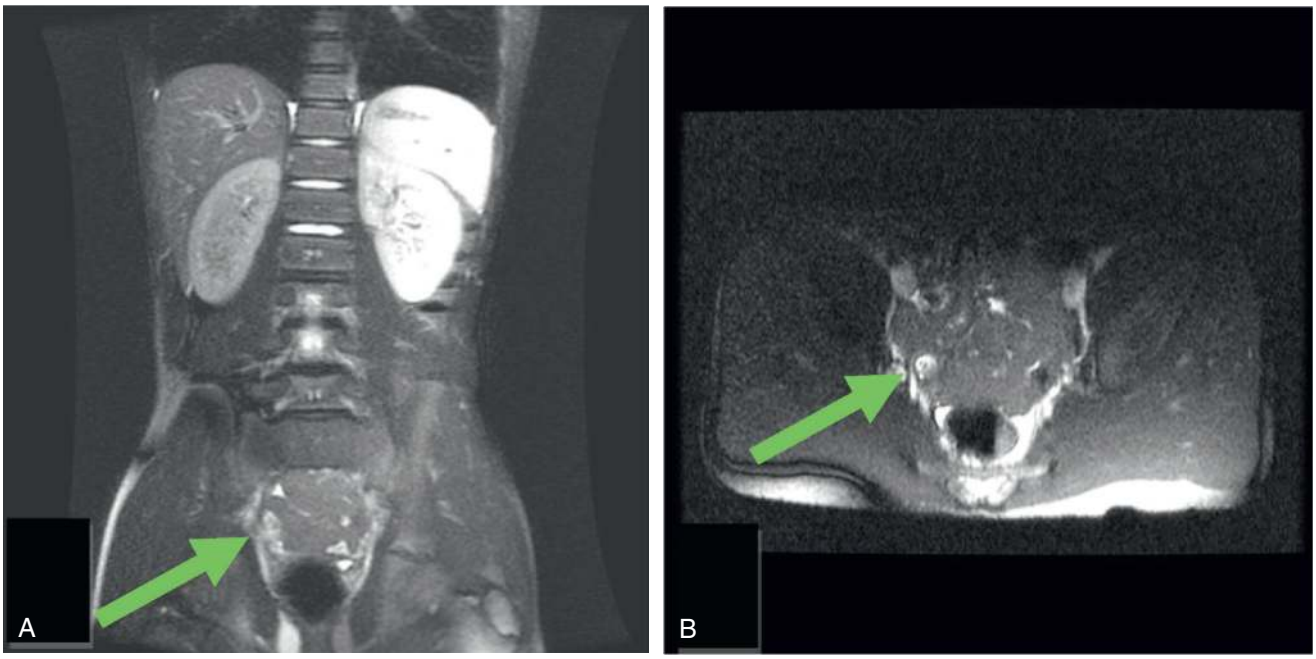

FIG 78.8 Magnetic resonance imaging abdomen demonstrating acute, nonperforated appendicitis. **A,** Magnetic resonance imaging of abdomen of an 11-year-old boy with a 78-hour history of right lower quadrant abdominal pain. **B,** MRI demonstrated an 8-mm appendix with periappendiceal fluid. (From Murphy EEK, Berman L. Clinical evaluation of acute appendicitis. *Clin Pediatr Emerg Med*. 2014;15(3):223–230.)

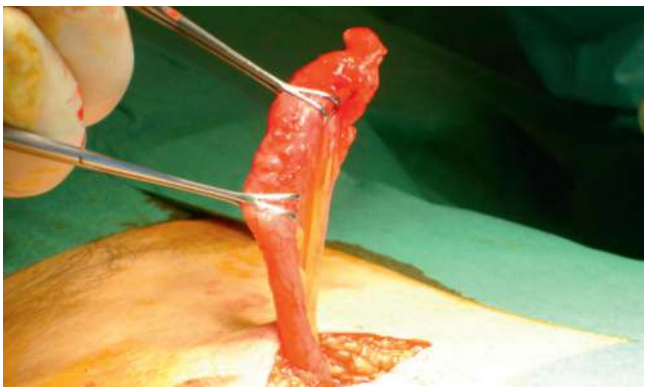

FIG 78.9 Acute appendicitis as seen at surgery. (From Sugrue C, Hogan A, Robertson I, et al. Incisional hernia appendicitis: a report of two unique cases and literature review. *Int J Surg Case Rep*. 2013;4(3):256–258.)

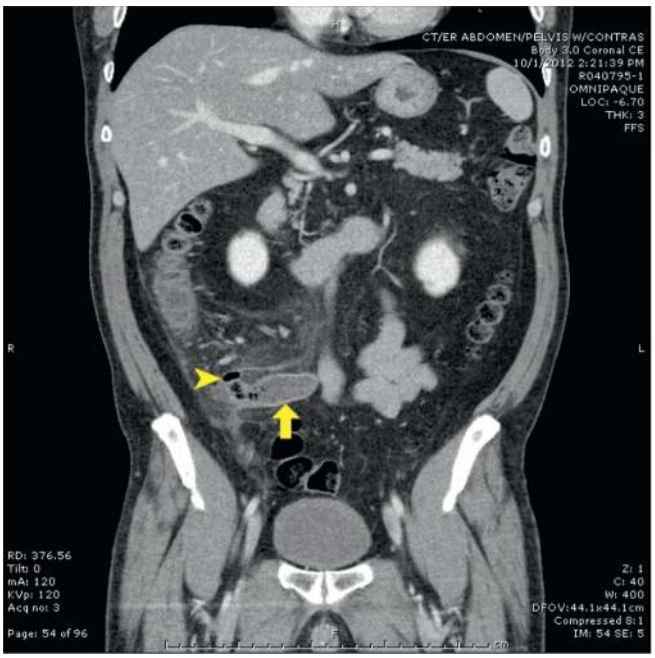

FIG 78.10 Computed tomography (CT) scan: coronal section image demonstrating a dilated appendix, with periappendicular fat stranding and extraluminal gas, suggestive of perforated appendicitis. *Arrow*, dilated appendix. *Arrowhead*, extraluminal gas. (From Teixeira PGR. Demetriades D. Appendicitis: changing perspectives. *Adv Surg*. 2013;47(1):119–140.)

(Fig. 78.9). That being said, recent experience suggests that many treatment decisions regarding patients suffering from acute appendicitis are based on traditions, many of which find their origins in the preantibiotic era, rather than evidence-based medicine. These traditions are reinforced by the belief held by both the lay public and medical professionals that appendectomy is a benign procedure. Current clinical thinking suggests that the best outcomes for patients with acute appendicitis can be obtained by subsetting the individual patient into one of three groups: (1) patients with mild acute appendicitis with a small phlegmon or abscess; (2) patients with more severe acute appendicitis with well-defined abscess that is anatomically amenable to percutaneous draining; and (3) patients with severe systemic symptoms and larger multicompartmental or multiple abscesses not amenable to percutaneous drainage (Figs. 78.10 and 78.11). This subsetting allows a more rational timing of appendectomy that has the potential to significantly reduce the mortality and morbidity associated with both the

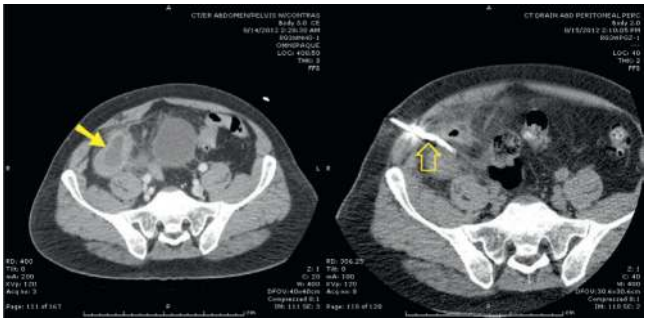

FIG 78.11 Computed tomography (CT) scan axial images demonstrating perforated appendicitis with abscess *(left)* and percutaneous CT-guided drainage of the abscess *(right). Arrow*, abscess. *Empty arrow*, percutaneous catheter insertion. (From Teixeira PGR. Demetriades D. Appendicitis: changing perspectives. *Adv Surg*. 2013;47(1):119–140.)

disease and the surgery. Patients in all groups are treated with antibiotics that cover both aerobic and anaerobic microbes. Patients in group 1 are allowed to recover from their acute illness and an interval appendectomy can be performed under ideal anesthetic and operative conditions (e.g., empty stomach, normal hydration, blood sugar control, discontinuation of anticoagulants, and antiplatelet medications). Group 2 can be treated with emergent percutaneous drainage of well-defined abscess and interval appendectomy can be performed after fistula closure. Group 3 requires urgent surgical intervention in all circumstances. It should be noted that there are some additional advantages to appendectomy including the opportunity for the surgeon to evaluate the entire abdomen at the time of surgery to correct the working diagnosis and to identify coexistent occult pathology and to remove appendiceal tissue containing malignant cells (e.g., adenocarcinoma and carcinoid which have an incidence of 0.7% and 0.07%, respectively).

COMPLICATIONS AND PITFALLS

The major pitfall in the evaluation and treatment of acute appendicitis is misdiagnosis coupled with failure to recognize the severity of the patient's condition. Comorbidities can greatly increase the risk of perioperative complications and must be accurately identified and treated.

CLINICAL PEARLS

Acute appendicitis is a common cause of abdominal pain. Correct diagnosis is necessary to properly treat this painful condition and to avoid overlooking other serious diseases that may mimic this disease.

Variations in the position of the appendix, age of the patient, and the extent of the disease make the clinical presentation of acute appendicitis challenging. In spite of this being a very common disease, misdiagnosis occurs by even the most experienced clinicians. The use of scoring schema, confirmatory testing, and the subsetting of patients into subgroups will improve outcomes.

SUGGESTED READINGS

Al-Faouri AF, Ajarma KY, Al-Abbadi AM, et al. The Alvarado score vs computed tomography in the diagnosis of acute appendicitis: a prospective study. *Med J Armed Forces India*. 2016;72(4):332–337.

Debnath J, Sharma V, Ravikumar R, et al. Clinical mimics of acute appendicitis: is there any role of imaging? *Med J Armed Forces India*. 2016;72(3):285–292.

Lourenco P, Brown J, Leipsic J, et al. The current utility of ultrasound in the diagnosis of acute appendicitis. *Clin Imaging*. 2016;40(5):944–948.

Pisano M, Capponi MG, Ansaloni L. Acute appendicitis: an open issue. Current trends in diagnostic and therapeutic options. In: Kon K, Rai M, eds., *Microbiology for surgical infections*. Amsterdam: Academic Press; 2014:97–110.

Romero J, Valencia S, Guerrero A. Acute appendicitis during coronavirus disease 2019 (COVID-19): changes in clinical presentation and CT findings. *J Am Coll Radiol*. 2020;17(8):1011–1013.

Peptic Ulcer Disease

ICD-10 CODE K279

THE CLINICAL SYNDROME

Peptic ulcer disease causes significant morbidity and mortality worldwide with a prevalence in the United States of approximately 8.4%. Patients suffering from peptic ulcer disease most commonly present with the complaint of unexplained epigastric pain (Fig. 79.1). This pain is often associated with nausea, dyspepsia, bloating, a sensation of abdominal fullness, and early satiety. Many patients with gastic ulcers experience a gnawing sensation shortly after meals. Patients suffering from duodenal ulcer experience this same phenomenon 2–3 hours after eating. Antacids tend to relieve the pain of duodenal ulcers but have less impact on the pain associated with gastric ulcers. Pain tends to worsen at night. Hematemesis or melena may occur with hematochezia occurring with a rapidly bleeding ulcer.

The majority of peptic ulcer disease is now thought to be associated with infection with *Helicobacter pylori* and the use of nonsteroidal antiinflammatory drugs (NSAIDs). Less common causes of drug-induced peptic ulcer disease include corticosteroid and bisphosphonate use, selective serotonin reuptake inhibitors, 5-fluorouricil, and sirolimus (Box 79.1). Zollinger–Ellison syndrome (gastrinoma with associated symptoms), gastric adenocarcinoma, and carcinoid syndrome are also associated with an increased risk of peptic ulcer disease (Fig. 79.2).

BOX 79.1 Drugs Associated With an Increased Risk of Peptic Ulcer Disease

- Nonsteroidal antiinflammatory drugs
- Aspirin
- Corticosteroids
- Bisphosphonates
- Selective serotonin reuptake inhibitors
- 5-Fluorouracil
- Sirolimus

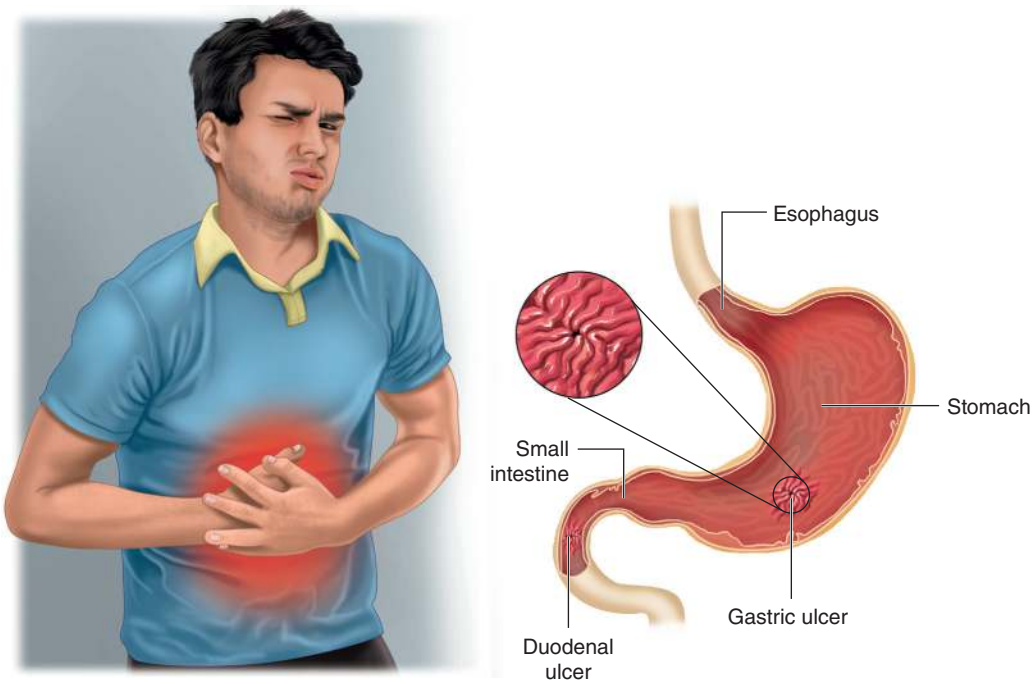

FIG 79.1 Patients suffering from peptic ulcer disease most commonly present with the complaint of unexplained epigastric pain.

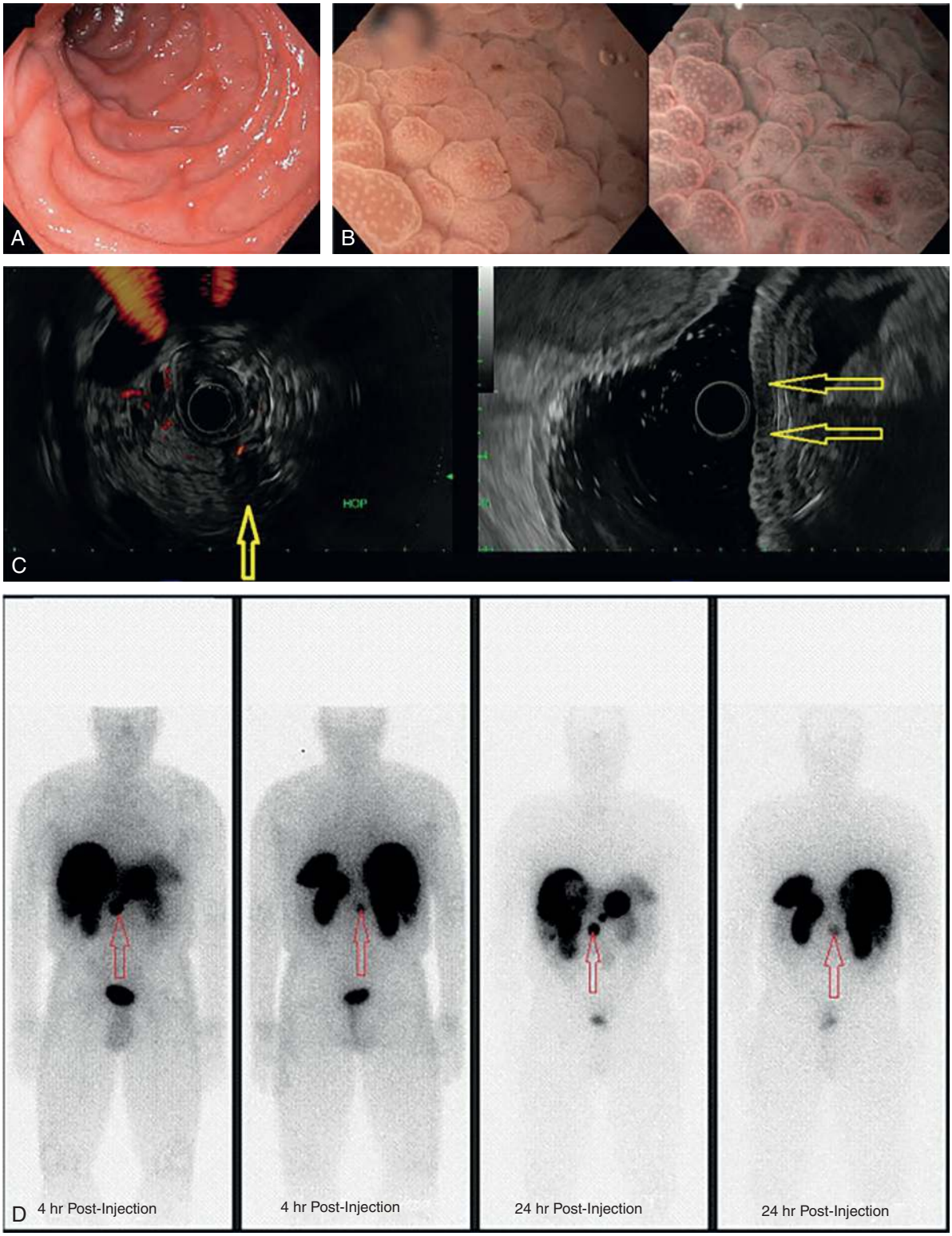

FIG 79.2 Zollinger-Ellison syndrome **(A)**. A closer endoscopic look under water magnification showed the hypertrophied parietal cells **(B)**. Examination of duodenal biopsy specimens showed findings consistent with chronic peptic duodenitis with no evidence of dysplasia or malignancy. endosonography (EUS) was ordered to look for pancreatic gastrinomas after completion of the CT-guided liver biopsy. EUS demonstrated a single, well-defined 2×2 cm, round, hypoechoic, heterogeneous solid mass in the head of the pancreas (**C,** left, *yellow arrow*). The outer margin of the mass was slightly irregular. EUS also showed gastric wall thickening (**C,** right, *yellow arrows*). Beside the physiologic uptake in the kidneys and the spleen, the pentetreotide scan demonstrated intense focal uptake in a 2.3-cm soft tissue area related to the ventral surface of the pancreatic head (**D,** *red arrows*), consistent with malignancy. It also showed multiple radiotracer-avid liver metastases. *CT*, Computed tomography. (From Alshati A, Kachaamy T. Classical features of Zollinger-Ellison syndrome, in images. *Gastrointest Endosc*. 2019;89(6):1255–1257.)

SIGNS AND SYMPTOMS

In most patients with uncomplicated peptic ulcer disease, physical findings are very nonspecific. Common findings on physical examination include epigastric tenderness, guaiac-positive stool from occult blood loss, vague right upper quadrant tenderness to deep palpation, and succussion splash as a result of partial or complete gastric outlet obstruction (Fig. 79.3). Patients suffering from perforation of a gastric or peptic ulcer will present with the sudden onset of severe epigastric pain (Fig. 79.4). The extent and severity of peritoneal findings are influenced by the size of the perforation, the amount of gastric or duodenal contents spilled into the peritoneal cavity, the amount of bacterial contamination, and if the perforation spontaneously seals. If the perforation persists, peritoneal findings will become more pronounced and the patient may manifest signs of septic shock including cardiac, pulmonary, and renal compromise. Immunocompromised patients including the elderly may not immediately present with a full-blown peritonitis.

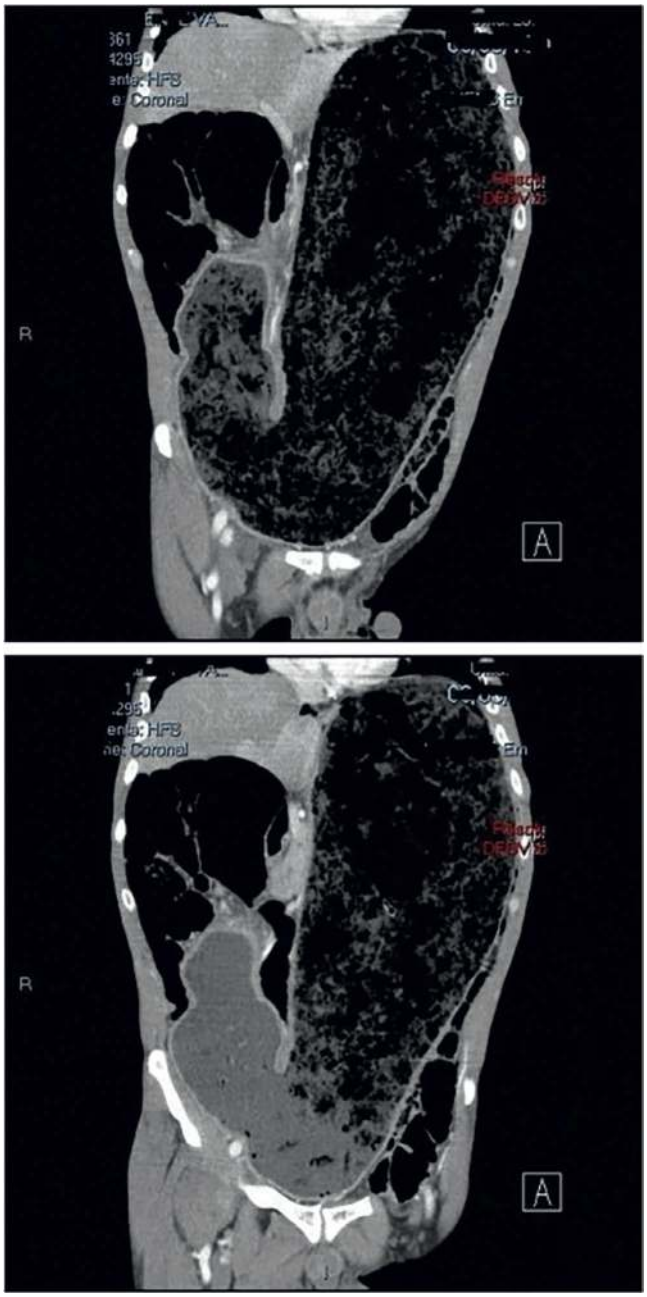

FIG 79.3 CT of massive gastric dilation due to pyloric stenosis. *CT*, Computed tomography. (From Costa CS, Pratas N, Capote H. Massive gastric dilation caused by gastric outlet obstruction in the setting of peptic ulcer disease—a case report. *Int J Surg Case Rep*. 2020;70:64–67.)

TESTING

Testing for *H. pylori* is indicated in all patients thought to be suffering from peptic ulcer disease. A complete blood count is essential to identify both acute and chronic anemia as is serum amylase and lipase determinations and complete blood chemistries including liver function testing. *H. pylori* testing can be done using endoscopic or invasive testing with the rapid urease substrate testing the current test of choice in this setting. Breath tests may also be utilized and identify active *H. pylori* infection by testing for the presence of enzymatic activity of bacterial urease produced by *H. pylori*. An upright chest X-ray is indicated to identify the presence of free air below the diaphragm and to identify occult pulmonary or upper subdiaphragmatic findings such as abscess (Fig. 79.5). Serum gastrin testing is indicated in any patient presenting with ulcers refractory to medical therapy, patients with multiple ulcers, ulcers associated with diarrhea, steatorrhea, and/or weight loss, and in all patients with a strong family history of peptic ulcer disease. The secretin stimulation

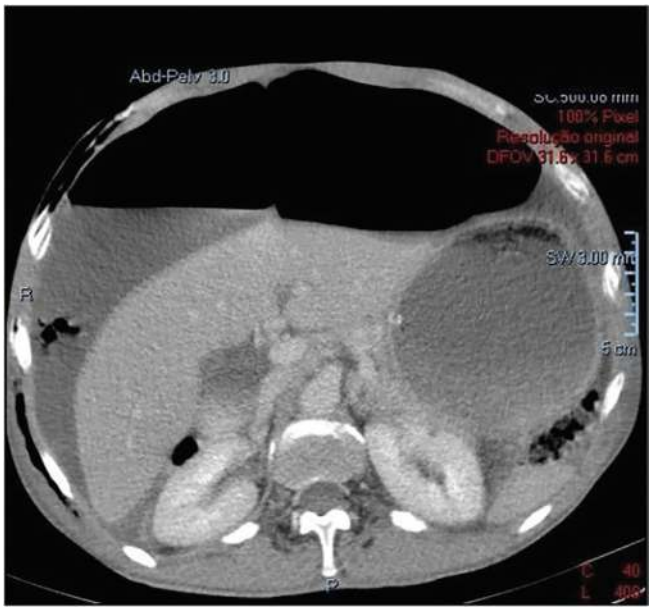

FIG 79.4 CT of pneumoperitoneum after perforation of peptic ulcer disease. *CT*, Computed tomography. (From Costa CS, Pratas N, Capote H. Massive gastric dilation caused by gastric outlet obstruction in the setting of peptic ulcer disease—a case report. *Int J Surg Case Rep*. 2020;70:64–67.)

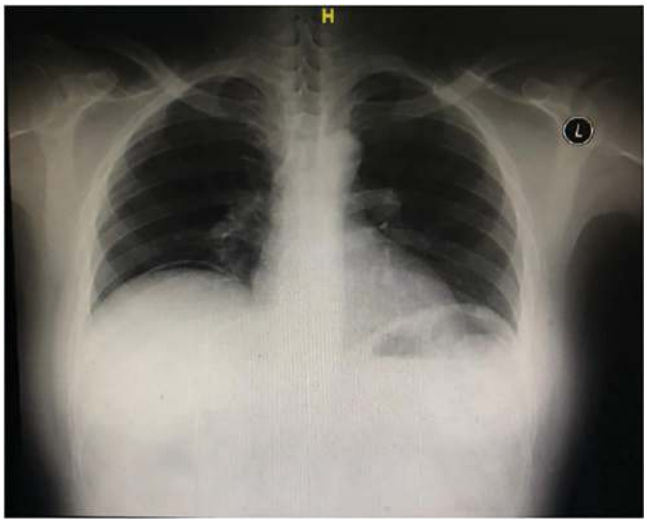

FIG 79.5 Upright chest X-ray demonstrating free air under the diaphragm. (From AlKhayat A, Qadhi I. Pneumoperitoneum in perforated appendicitis in the COVID pandemic: a case report. *Int J Surg Open*. 2022;42:100469.)

test combined with serum gastrin testing will help identify the presence of Zollinger–Ellison syndrome (see Fig. 79.2).

Upper gastrointestinal endoscopy is currently the gold standard for the diagnosis of peptic ulcer disease because: (1) it is highly sensitive in the identification of gastric and duodenal peptic ulcer disease; (2) it can establish the severity of the disease and identify the presence of bleeding; (3) it can characterize the nature of the lesions as potentially benign or malignant; and (4) it simplifies the ability to biopsy and obtain cytologic brushings from suspected lesions (Fig. 79.6). If upper gastrointestinal endoscopy is not available, upper gastrointestinal double-contrast radiography is an acceptable alternative (Fig. 79.7). In the setting of massive gastrointestinal bleeding, angiography may be useful in helping identify the bleeding source (Fig. 79.8).

DIFFERENTIAL DIAGNOSIS

Most diseases affecting the gastrointestinal tract may mimic the presentation of peptic ulcer disease (Box 79.2). More common mimics include gastroesophageal reflux disease, acute

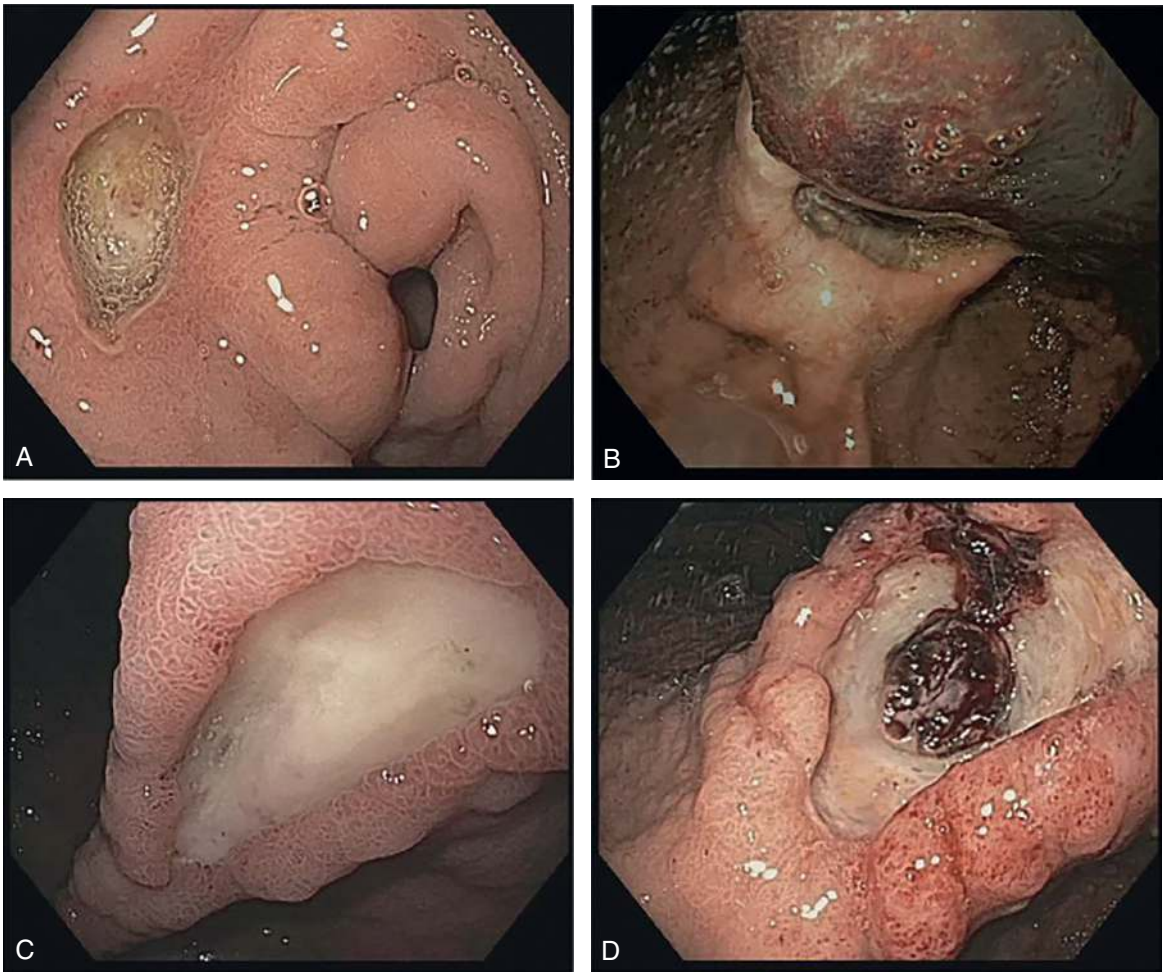

FIG 79.6 Endoscopic images of peptic ulcer disease. **A,** Clean-based ulcer of gastric antrum. **B,** Ulcer of incisura with adherent clot. **C,** Large ulcer of incisura. **D,** Gastric ulcer with adherent clot.

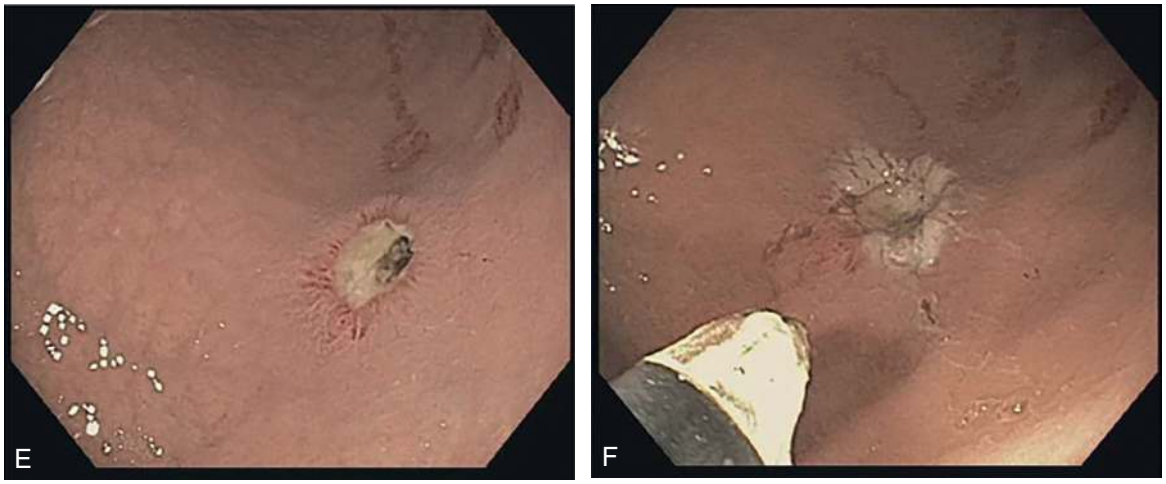

FIG 79.6 cont'd E, Gastric ulcer with nonbleeding visible vessel. F, Gastric ulcer with nonbleeding visible vessel after thermal coagulation therapy. (From Kavitt RT, Lipowska AM, Anyane-Yeboa A, Gralnek IM. Diagnosis and treatment of peptic ulcer disease. *Am J Med*. 2019;132(4):447–456.)

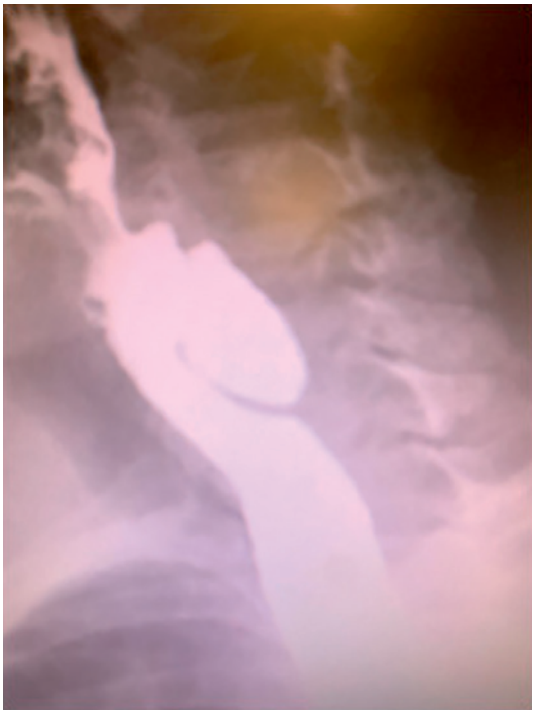

FIG 79.7 Esophogram showing posterior cervical outpouching consistent with Zenker's diverticulum. (From Sharma NR: Top tips for endoscopic diverticulotomy for Zenker's diverticula (with video). Gastrointestinal Endoscopy, 2023-02-01, Volume 97, Issue 2, Pages 365-368.)

gastritis, nonulcer dyspepsia, acute cholecystitis, esophagitis, and diverticulitis. The possibility of gastrointestinal malignancies including Zollinger–Ellison syndrome remains ever present, and the clinician must maintain a high index of suspicion to avoid delayed diagnosis.

BOX 79.2 Differential Diagnosis of Peptic Ulcer Disease

- Gastroesophageal reflux disease
- Nonulcer dyspepsia
- Acute cholecystitis
- Acute coronary syndrome
- Acute gastritis
- Diverticulitis
- Gastroenteritis
- Esophageal rupture and tears
- Esophagitis
- Gallstones (cholelithiasis)
- Acute cholangitis
- Inflammatory bowel disease
- Zollinger–Ellison syndrome

TREATMENT

The treatment decision making for patients suffering from peptic ulcer disease is driven by the severity of the symptoms and the presence of acute and/or chronic bleeding. In uncomplicated cases, some clinicians will opt for empiric treatment including acid suppression with histamine-2 receptor antagonists or proton pump inhibitors combined with triple therapy including bismuth, tetracycline, and a nitroimidazole to treat presumptive *H. pylori* infection. NSAIDs and aspirin are always discontinued. Given the safety of upper gastrointestinal endoscopy, the threshold for proceeding with the diagnostic and potentially therapeutic maneuver should be low, especially in the older population. Urgent surgery is reserved for those patients with active hemorrhage who fail attempts at endoscopic hemostasis as an active hemorrhage in this setting has a high incidence of rebleeding.

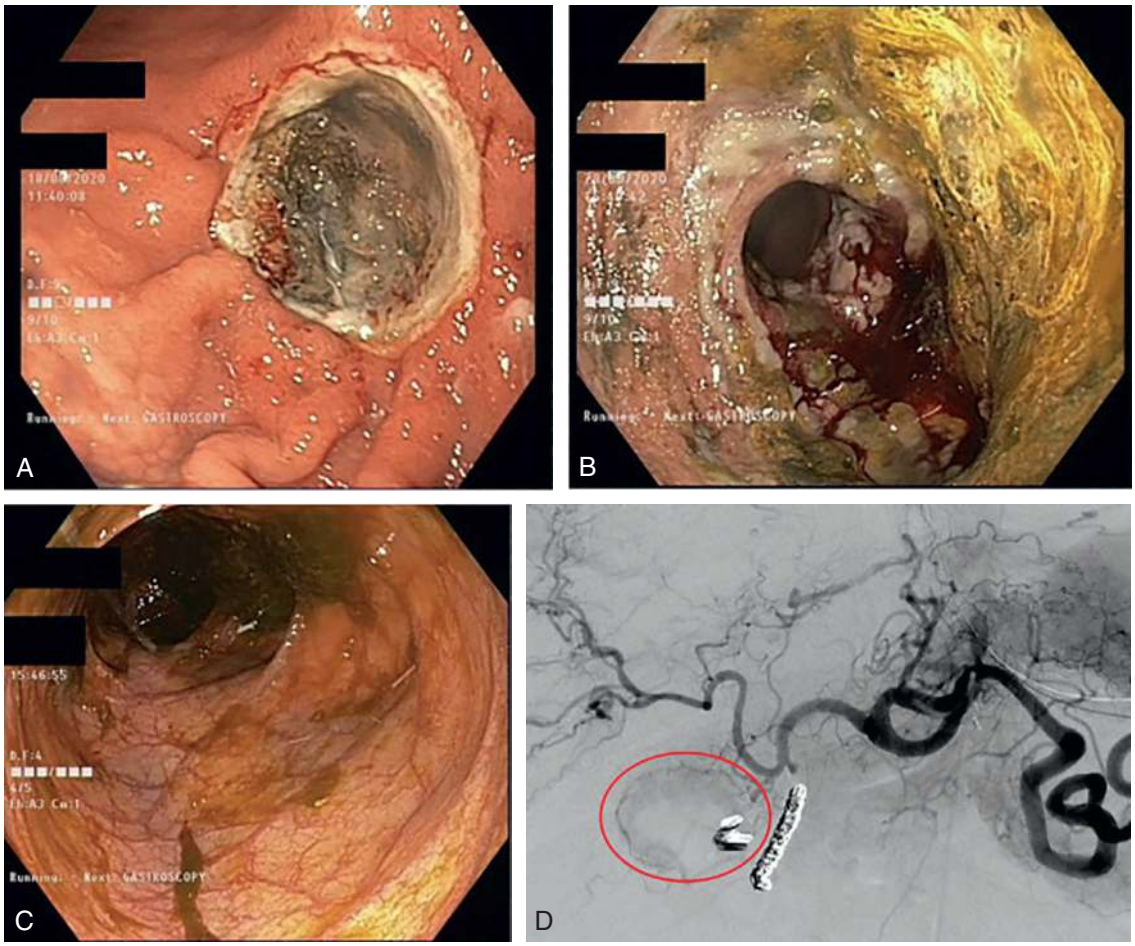

FIG 79.8 (A–D) Digital subtraction angiography (DSA) of the celiac trunk showed the region of interest involving the proximal duodenum (*circle*), with endoscopically placed clips in the vicinity and previously coil-embolized GDA. *GDA*, Gastroduodenal artery. (From Han Q, Qian C, Gabriel G, Krohmer S, Raissi D. Gastrointestinal bleeding from supraduodenal artery with aberrant origin. *Radiol Case Rep*. 2017;12(3):526–528.)

COMPLICATIONS AND PITFALLS

Warning signs in patients thought to be suffering from peptic ulcer disease include anemia, unexplained weight loss, early satiety, odynphagia, dysphagia, recurrent emesis, and a family history of gastrointestinal malignancy. A high index of suspicion as to the possibility of underlying gastrointestinal malignancy is critical to avoid missed diagnosis. The delay in surgical treatment in patients experiencing bleeding uncontrolled by endoscopic hemostasis can lead to significant mortality and morbidity. It should be remembered that peptic ulcers may penetrate into adjacent structures resulting in confusing clinical presentations (Fig. 79.9).

CLINICAL PEARLS

In most instances, peptic ulcer disease is highly treatable. However, in some patients, refractory peptic ulcers persist in spite of the clinician's best efforts. In this setting, a careful search of the cause is indicated, in particular, a careful evaluation for malignancy including Zollinger–Ellison syndrome that might have been missed during the initial evaluation. Gastric ulcers and chronic *H. pylori* infection may predispose the patient to gastric malignancy. The threshold for repeat endoscopy and re-biopsy should be low in any patient with persistent or recurrent symptoms thought to be related to peptic ulcer disease.

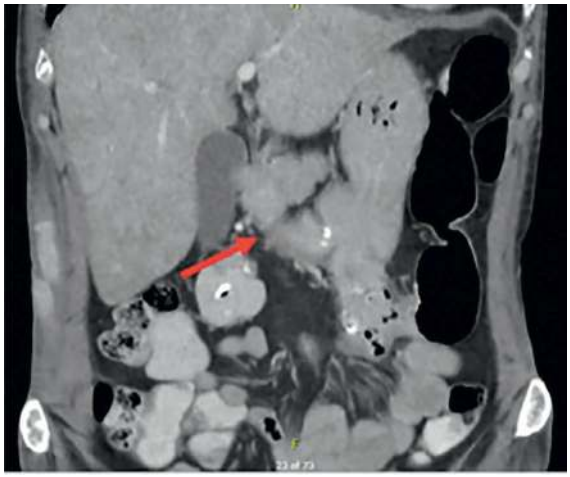

FIG 79.9 Gastrocolic fistula as a complication of peptic ulcer disease. (From Sharma S, Bhatia R, Vasudevan A. Abdominal pain and diarrhea in peptic ulcer disease. *Gastroenterology*. 2021;161(2):e48–e49.)

SUGGESTED READINGS

Costa CS, Pratas N, Capote H. Massive gastric dilation caused by gastric outlet obstruction in the setting of peptic ulcer disease—a case report. *Int J Surg Case Rep*. 2020;70:64–67. https://doi.org/10.1016/j.ijscr.2020.04.015.

Hung KW, Knotts RM, Faye AS, et al. Factors associated with adherence to *Helicobacter pylori* testing during hospitalization for bleeding peptic ulcer disease. *Clin Gastroenterol Hepatol*. 2020;18(5):1091–1098. e1. https://doi.org/10.1016/j.cgh.2019.07.037.

Kavitt RT, Lipowska AM, Anyane-Yeboa A, Gralnek IM. Diagnosis and treatment of peptic ulcer disease. *Am J Med*. 2019;132(4):447–456. https://doi.org/10.1016/j.amjmed.2018.12.009.

Nelms DW, Carlos AP. The acute upper gastrointestinal bleed. *Surg Clin North Am*. 2018;98(5):1047–1057. https://doi.org/10.1016/j.suc.2018.05.004.

Sharma S, Bhatia R, Vasudevan A. Abdominal pain and diarrhea in peptic ulcer disease. *Gastroenterology*. 2021;161(2):e48–e49. https://doi.org/10.1053/j.gastro.2021.01.003.

Shell EJ. Pathophysiology of peptic ulcer disease. *Physician Assist Clin*. 2021;6(4):603–611. https://doi.org/10.1016/j.cpha.2021.05.005.

Wang A, Yerxa J, Agarwal S, et al. Surgical management of peptic ulcer disease. *Curr Probl Surg*. 2020;57(2):100728. https://doi.org/10.1016/j.cpsurg.2019.100728.

80

Acute Pancreatitis

ICD-10 CODE R85.9

THE CLINICAL SYNDROME

Acute pancreatitis is one of the most common causes of abdominal pain, with an incidence of approximately 0.5% among the general population. The mortality rate is 1%–1.5%. In the United States, acute pancreatitis is most commonly caused by excessive alcohol consumption (Fig. 80.1); gallstones are the most frequent cause in most European countries. Acute pancreatitis has many other causes, however, including viral infection, hypertriglyceridemia, tumor, and medications (Boxes 80.1 and 80.2). Less common causes of acute pancreatitis include scorpion venom (Fig. 80.2), cardiac bypass-induced ischemia, pregnancy, cystic fibrosis, and infection with Chinese liver fluke.

Abdominal pain is a common feature of acute pancreatitis. It may range from mild to severe and is characterized by steady, boring epigastric pain that radiates to the flanks and chest. The pain is worse in the supine position, and patients with acute pancreatitis often prefer to sit with the dorsal spine flexed and the knees drawn up to the abdomen. Nausea, vomiting, and anorexia are other common features.

SIGNS AND SYMPTOMS

Patients with acute pancreatitis appear ill and anxious. Tachycardia and hypotension resulting from hypovolemia are common as is low-grade fever. Saponification of subcutaneous fat is seen in approximately 15% of patients

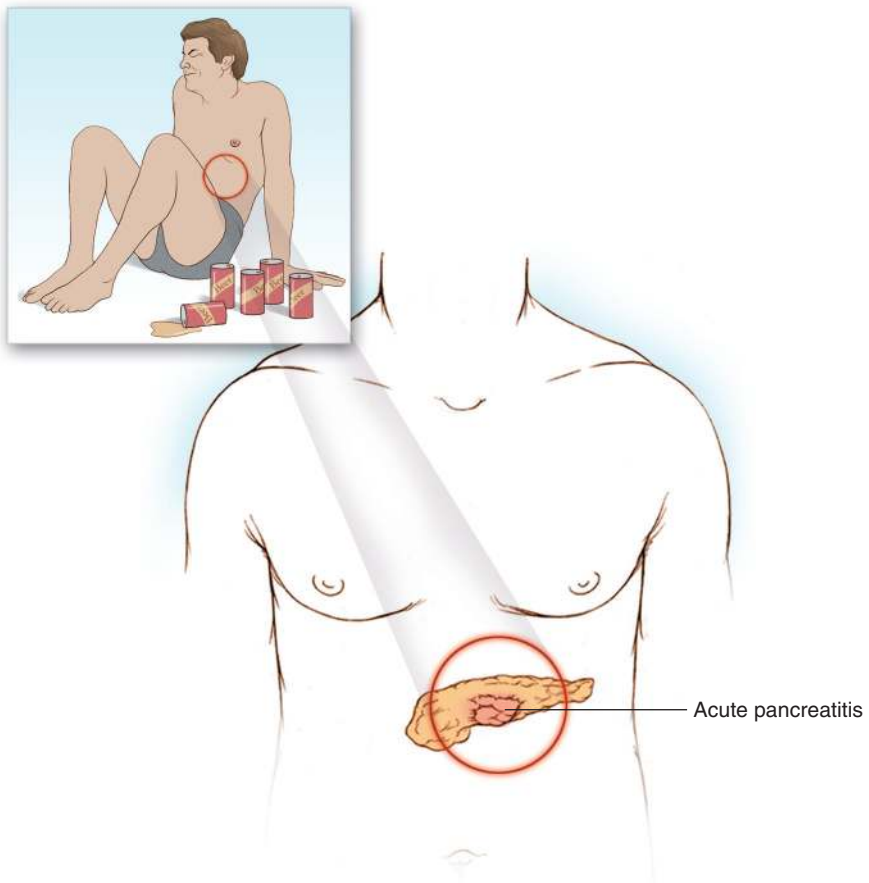

FIG 80.1 Excessive consumption of alcohol is one of the causes of acute pancreatitis.

BOX 80.1 Common Causes of Acute Pancreatitis

- Alcohol
- Gallstones
- Abdominal trauma
- Infections
  - Mumps
  - Viral hepatitis, cytomegalovirus
- Coxsackie B virus
- Ascaris
- Mycoplasma pneumonia
- Medications
  - Thiazide diuretics
  - Furosemide
  - Gliptins
  - Tetracycline
  - Sulfonamides
  - Steroids
  - Estrogens
  - Azathioprine
  - Pentamidine
- Metabolic causes
  - Hypertriglyceridemia
  - Hypercalcemia
  - Malnutrition
- Perforating ulcers
- Carcinoma of the head of the pancreas
- Tumor obstructing the ampulla of Vater
- Structural abnormalities pancreas divisum
  - Choledochocele
- Connective tissue diseases
- Postendoscopic retrograde cholangiopancreatography
- Radiation induced
- Hereditary causes
  - Scorpion sting

BOX 80.2 Drugs Associated With Acute Pancreatitis

Sulindac
Tetracycline
Furosemide
Thiazide diuretics
Chlorthalidone
Metronidazole
Nitrofurantoin
Phenformin
Procainamide
Cancer chemotherapy drug combinations containing asparaginase
Cisplatin
Cimetidine
Azathioprine
Steroids
Estrogens
Piroxicam
Valproic acid
Pentamidine
Methyldopa
Octreotide
Didanosine
6-Mercaptopurine
5-Aminosalicylic acid compounds

FIG 80.2 The venom of the Trinidad scorpion (*Tityus trinitatis*) is particularly potent, as is common with small-bodied large-tailed scorpions, and can cause acute pancreatitis.(From the Centers for Disease Control and Prevention Public Health Image Library (PHIL): https://phil.cdc.gov/Details.aspx?pid=6295.

suffering from acute pancreatitis; a similar percentage of patients experience pulmonary complications, including pleural effusion and pleuritic pain that may compromise respiration. Diffuse abdominal tenderness with peritoneal signs is invariably present. A pancreatic mass or pseudocyst secondary to pancreatic edema may be palpable. If hemorrhage occurs, periumbilical ecchymosis (Cullen's sign) and flank ecchymosis (Turner's sign) may be present. Both these findings suggest severe necrotizing pancreatitis and indicate a poor prognosis (Fig. 80.3). If the patient has hypocalcemia, Chvostek's or Trousseau's sign may be present (Fig. 80.4).

TESTING

Elevation of serum amylase levels is the sine qua non of acute pancreatitis. Levels tend to peak at 48–80 hours and then begin to drift toward normal. Serum lipase remains elevated and may correlate better with actual disease severity. Because serum amylase levels may be elevated in other diseases, such as parotitis, amylase isozymes may be necessary to confirm a pancreatic basis for this finding. Plain radiographs of the chest and abdomen are indicated for all patients who present with acute pancreatitis, to identify pulmonary complications, including pleural effusion (Figs. 80.5 and 80.6). Given the extrapancreatic manifestations (e.g., acute renal or hepatic failure), serial complete blood counts, serum calcium and

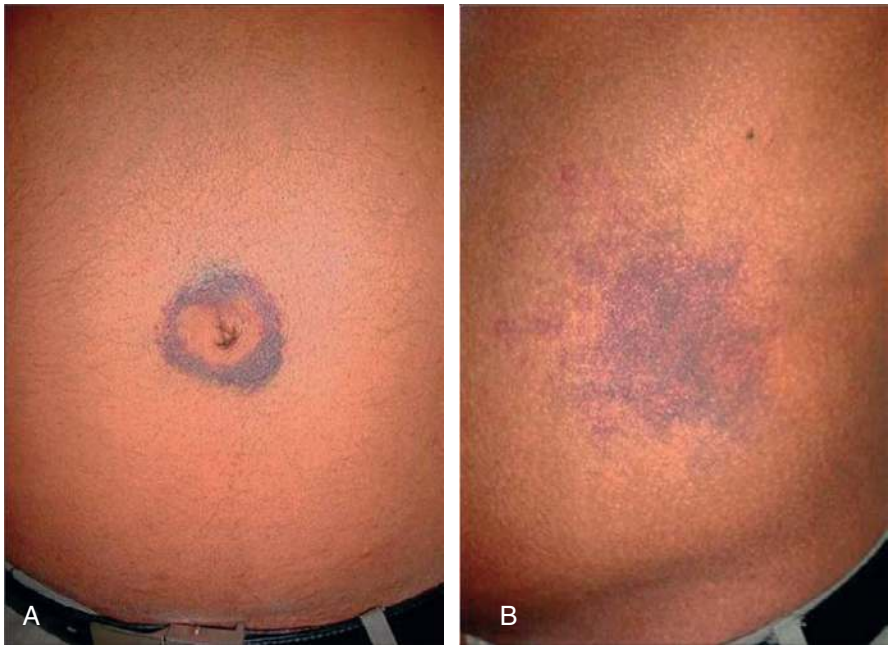

FIG 80.3 Cullen (A) and Turner (B) signs. A result of hemorrhagic pancreatitis, periumbilical ecchymosis (Cullen's sign), and flank ecchymosis (Turner's sign) may occur. (Reprinted with permission from Elsevier, Sandeep Chauhan, Manisha Gupta, Atul Sachdev, Sanjay D'Cruz, Ikjot Kaur, Cullen's and Turner's sign associated with portal hypertension. *Lancet*. 2008;372(9632):54.)

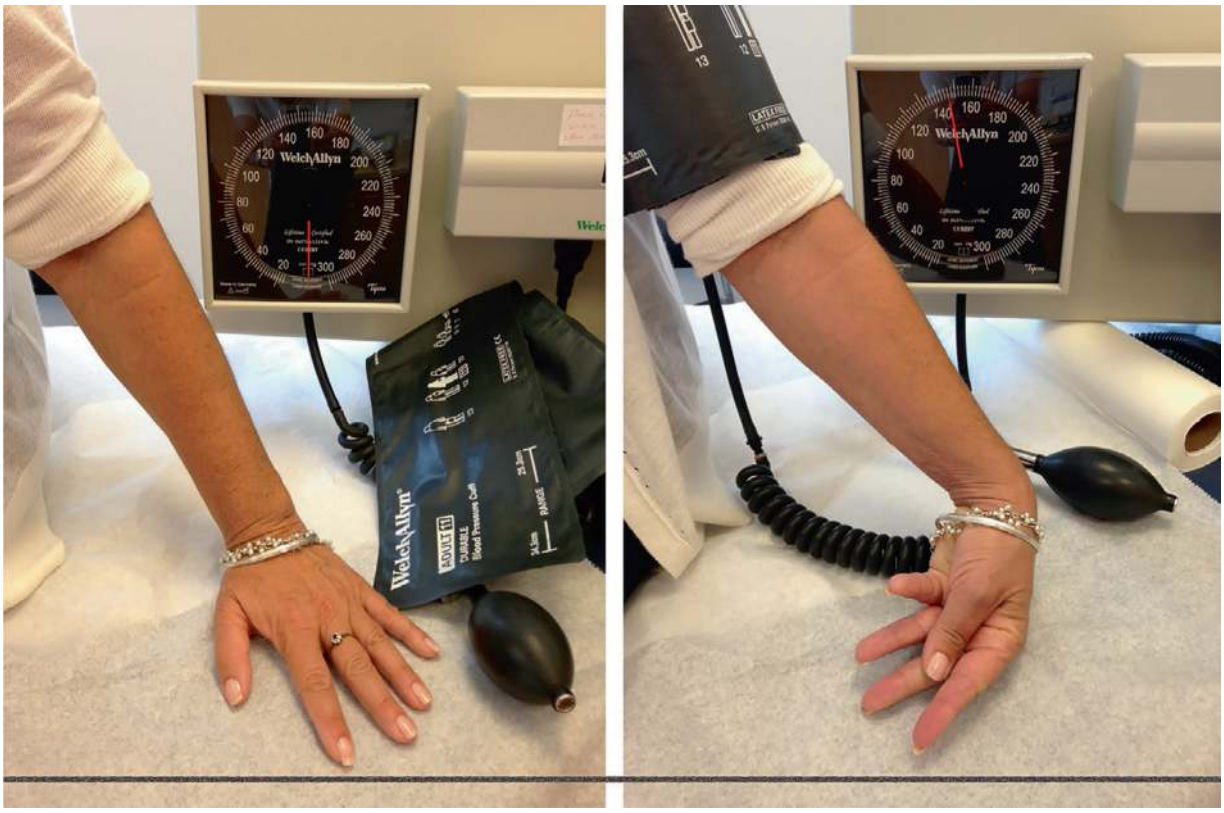

FIG 80.4 Trousseau sign. Three minutes after inflation of the blood pressure cuff above systolic blood pressure, the patient's and (*left panel*) shows muscular contraction (*right panel*) with flexion of the wrist, metacarpophalangeal joints, and thumb, and hyperextension of the fingers, described as "main d'accoucheur" (hand of the obstetrician). (From Bilezikian, J. *The parathyroids*. 3rd ed. San Diego: Academic Press; 2015.)

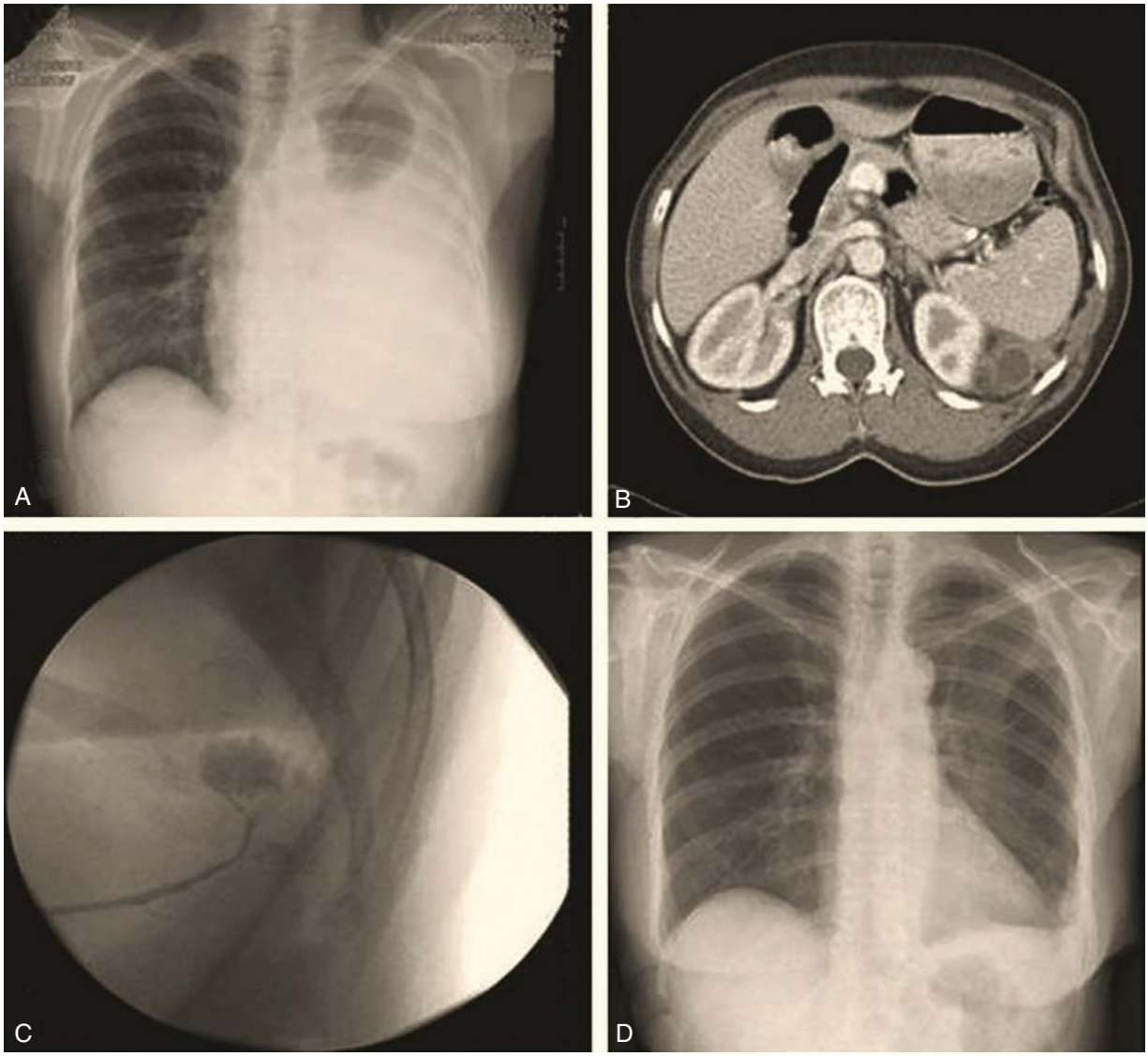

FIG 80.5 A–D, Radiographic imaging of acute pancreatitis. Chest x-ray of a patient with acute pancreatitis demonstrating a small pleural effusion on the left as evidenced by blunting of the left costophrenic angle.

glucose levels, liver function tests, and electrolytes are indicated in all patients with acute pancreatitis. There is a correlation between the degree of anion gap and the severity of acute pancreatitis. Computed tomography (CT), ultrasound imaging, or magnetic resonance imaging of the abdomen can identify pancreatic pseudocyst and edema and may help the clinician gauge the severity and progress of the disease (Figs. 80.7–80.9). Gallbladder evaluation with radionuclides is indicated if gallstones may be the cause of acute pancreatitis. Arterial blood gas analysis can identify respiratory failure and metabolic acidosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes perforated peptic ulcer, acute cholecystitis, bowel obstruction, renal calculi, myocardial infarction, mesenteric infarction, diabetic ketoacidosis, and pneumonia. Rarely, the collagen vascular diseases, including systemic lupus erythematosus and polyarteritis nodosa, may mimic pancreatitis. Because the pain of acute herpes zoster may precede the rash by 3–5 days, it may erroneously be attributed to acute pancreatitis.

TREATMENT

Most cases of acute pancreatitis are self-limited and resolve within 5–7 days. Initial treatment is aimed primarily at allowing the pancreas to rest, which is accomplished by giving the patient nothing by mouth to decrease serum gastrin secretion and, if ileus is present, by instituting nasogastric suction. Short-acting opioid analgesics such as hydrocodone are a reasonable next step if conservative measures do not control the patient's pain. If ileus is present, a parenteral opioid such as meperidine is a good alternative. Because the

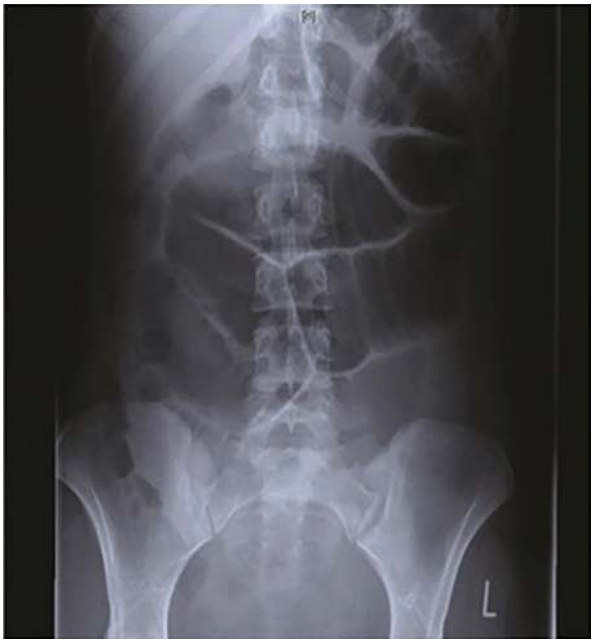

FIG 80.6 Abdominal x-ray of a patient with acute pancreatitis demonstrating dilated loops of small bowel in the upper and mid-abdomen. No free air is noted.

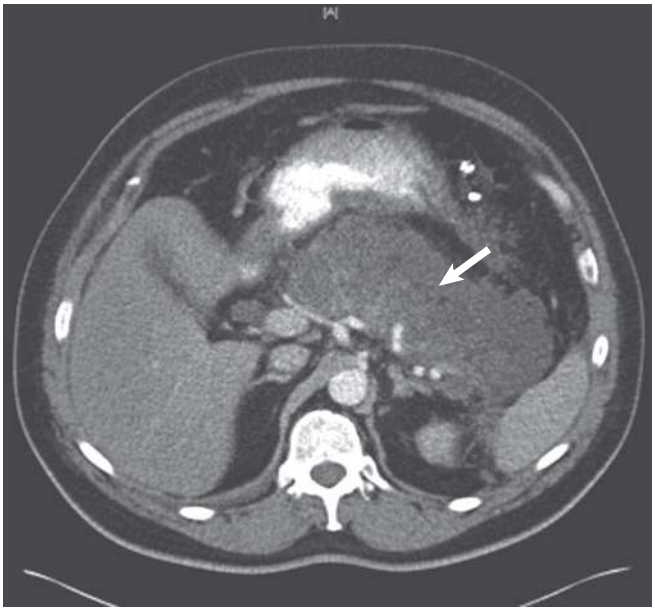

FIG 80.8 Contrast-enhanced computed tomography scan from the same patient as in Fig. 80.6 on hospital day 5. The scan demonstrates extensive pancreatic necrosis *(arrow)*. (From Wu BU, Conwell DL. Acute pancreatitis. Part I. Approach to early management. *Clin Gastroenterol Hepatol*. 2010;8(5): 410–416.)

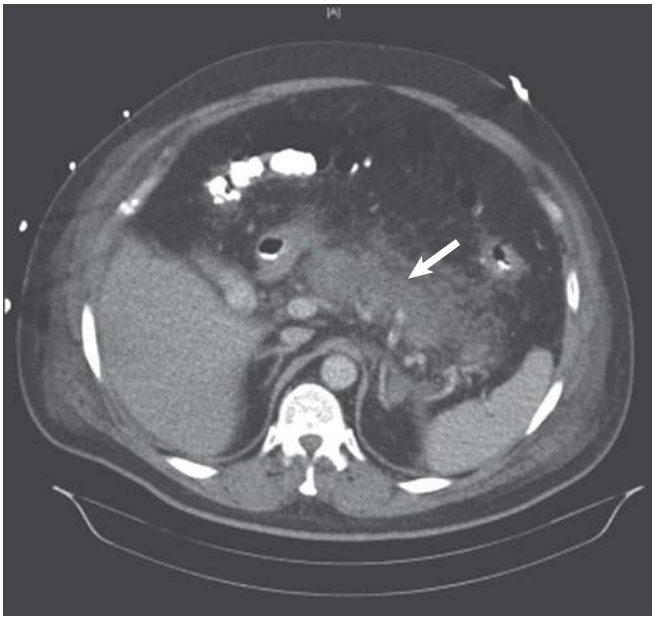

FIG 80.7 Contrast-enhanced computed tomography scan on hospital day 1 that demonstrates interstitial pancreatitis *(arrow)*. (From Wu BU, Conwell DL. Acute pancreatitis. Part I: approach to early management. *Clin Gastroenterol Hepatol*. 2010;8(5):410–416.)

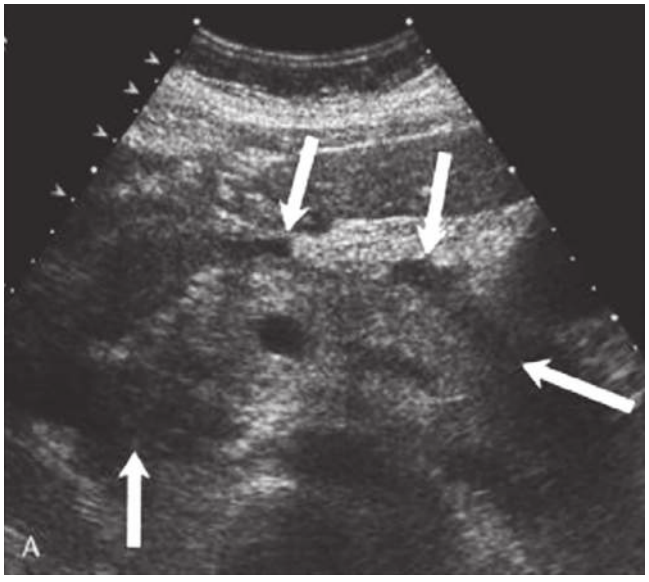

FIG 80.9 Ultrasound of a patient with acute pancreatitis demonstrating peripancreatic inflammation as evidenced by hypoechoic inflammation ventral to the body and head and dorsal to the head *(arrows)*. (From Tchelepi H, Ralls PW. Ultrasound of acute pancreatitis. *Ultrasound Clin*. 2007;2(3): 415–422.)

opioid analgesics have the potential to suppress the cough reflex and respiration, the patient must be closely monitored and instructed in pulmonary toilet techniques. If symptoms persist, CT-guided celiac plexus block with local anesthetic and steroid is indicated and may decrease the mortality and morbidity associated with the disease (Figs. 80.10 and 80.11). Alternatively, continuous thoracic epidural block with local anesthetic, opioid, or both may provide adequate pain control and allow the patient to avoid the respiratory depression associated with systemic opioid analgesics.

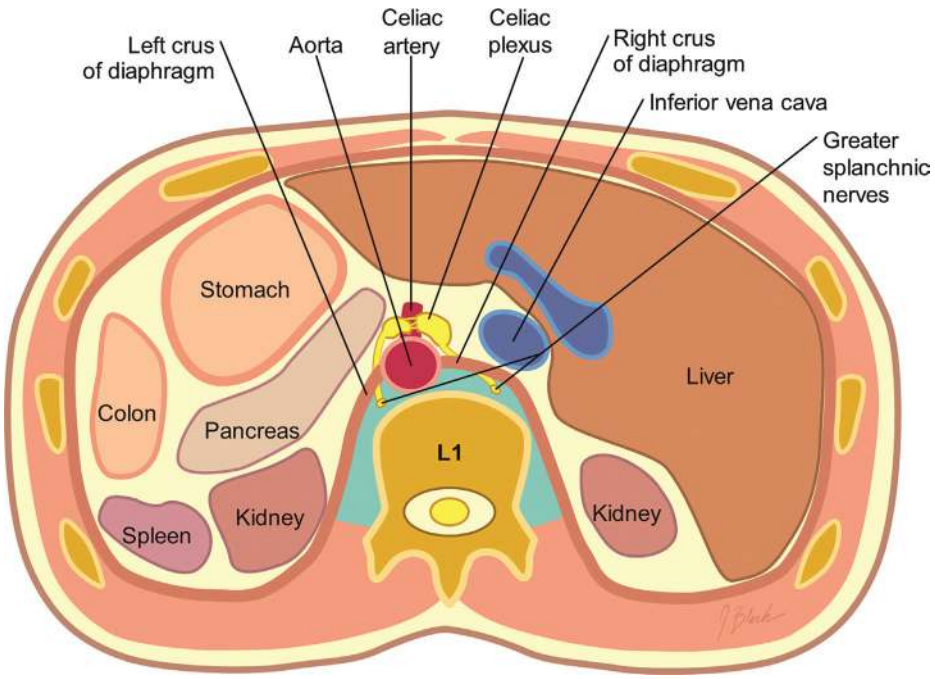

FIG 80.10 Cross-sectional anatomy of the celiac plexus. (From Waldman S. *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021.)

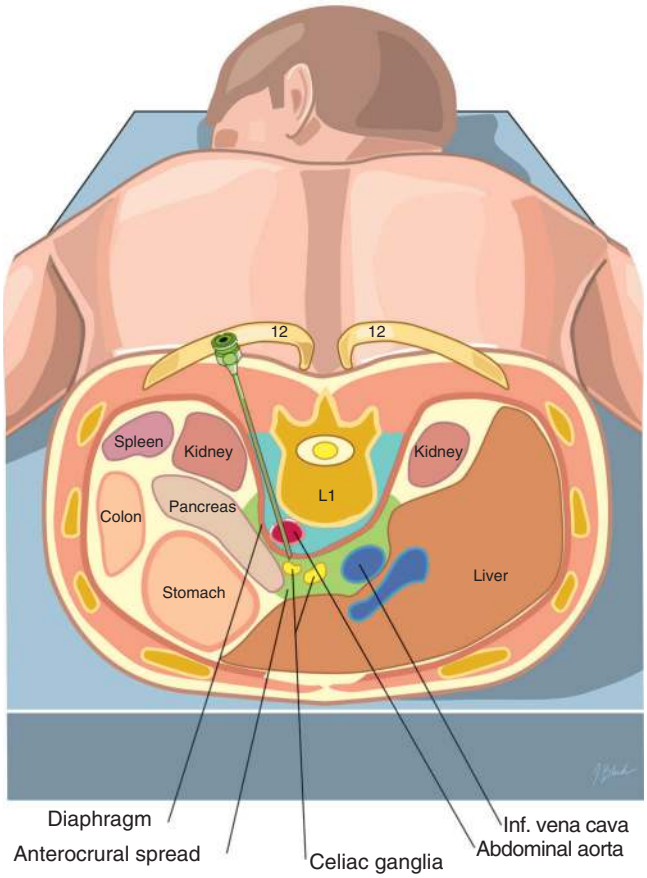

FIG 80.11 Celiac plexus block using the single-needle transaortic approach. (From Waldman SD. *Atlas of interventional pain management*. 2nd ed. Philadelphia: Saunders; 2004:286.)

Hypovolemia following celiac plexus block should be treated aggressively with crystalloid and colloid infusions. For prolonged cases of acute pancreatitis, parenteral nutrition is indicated to avoid malnutrition. Surgical drainage and removal of necrotic tissue may be required if severe necrotizing pancreatitis fails to respond to these treatment modalities.

COMPLICATIONS AND PITFALLS

The major pitfall is failure to recognize the severity of the patient's condition and to identify and aggressively treat the extrapancreatic manifestations of acute pancreatitis (Fig. 80.12). Hypovolemia, hypocalcemia, and renal and respiratory failures occur with sufficient frequency that the clinician must actively seek these potentially fatal complications and treat them aggressively.

CLINICAL PEARLS

Acute pancreatitis is a common cause of abdominal pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious extrapancreatic complications associated with this disease. Opioid analgesics generally provide adequate pain control. If necessary, celiac plexus block and thoracic epidural block are straightforward techniques that can produce dramatic pain relief.

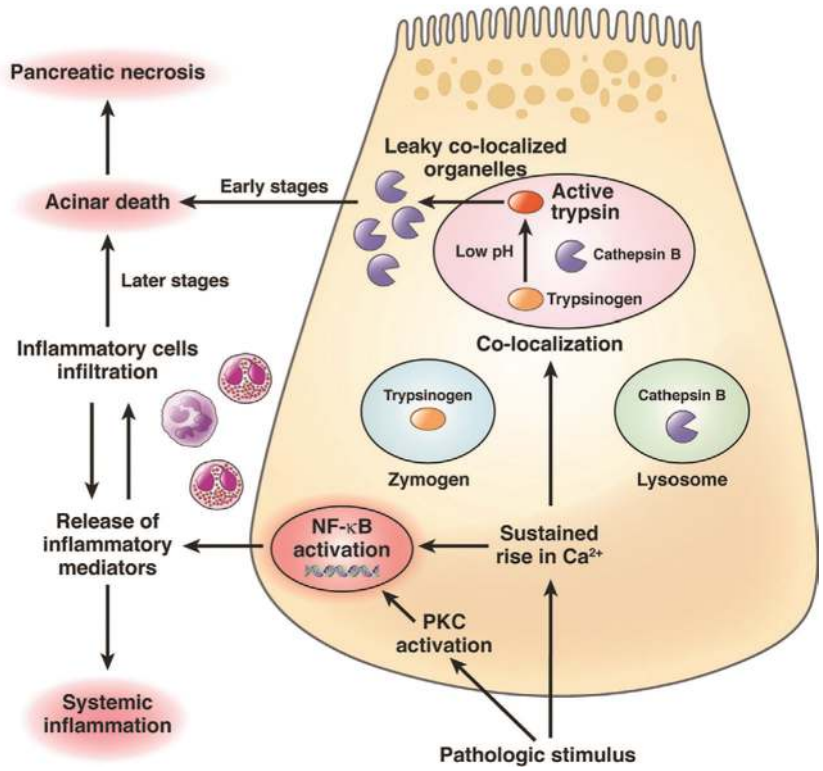

FIG 80.12 Pathogenesis of acute pancreatitis. This paradigm demonstrates pathogenic events in acute pancreatitis. Activation of trypsinogen in the organelles formed from colocalization of lysosomal and zymogen compartments leads to leakage of cathepsin B in the cytosol, resulting in acinar death in the early stages of pancreatitis. Activation of NFκB occurs early, independent of trypsinogen activation, and leads to release of inflammatory mediators and recruitment of inflammatory cells, which causes acinar cell death in later stages of pancreatitis and drives the systemic inflammatory response seen in pancreatitis. See text for details. *PKC*, Protein kinase C. (From Saluja A, Dudeja V, Dawra R, et al. Early intraacinar events in pathogenesis of pancreatitis. *Gastroenterology*. 2019;156(7):1979–1993.)

SUGGESTED READINGS

Carr RA, Rejowski BJ, Cote GA, et al. Systematic review of hypertriglyceridemia-induced acute pancreatitis: a more virulent etiology? *Pancreatology*. 2016;16(4):469–476.

Koutroumpakis E, Slivka A, Furlan A, et al. Management and outcomes of acute pancreatitis patients over the last decade: a United States tertiary-center experience. *Pancreatology*. 2017;17(1):32–40.

Mikolasevic I, Milic S, Orlic L, et al. Metabolic syndrome and acute pancreatitis. *Eur J Intern Med*. 2016;32:79–83.

Rashid N, Sharma PP, Scott RD, et al. Severe hypertriglyceridemia and factors associated with acute pancreatitis in an integrated health care system. *J Clin Lipidol*. 2016;10(4):880–890.

Waldman SD. Acute pancreatitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:278–279.

Waldman SD. Celiac plexus block. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:473–476.

Waldman SD. Celiac plexus block: single needle transaortic approach. In: *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2016:401–407.

Waldman SD. The celiac plexus. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:117–118.

Chronic Pancreatitis

ICD-10 CODE K86.1

THE CLINICAL SYNDROME

Chronic pancreatitis may manifest as recurrent episodes of acute inflammation of the pancreas superimposed on chronic pancreatic dysfunction, or it may be a more constant problem. As the exocrine function of the pancreas deteriorates, malabsorption with steatorrhea and azotorrhea develops. In the United States, chronic pancreatitis is most commonly caused by alcohol consumption, followed by cystic fibrosis and malignant pancreatic tumors (Box 81.1). Hereditary causes such as alpha$_1$-antitrypsin deficiency are also common. In developing countries, the most common cause of chronic pancreatitis is severe protein-calorie malnutrition. Chronic pancreatitis can also result from acute pancreatitis.

Abdominal pain is a common feature of chronic pancreatitis, and it mimics the pain of acute pancreatitis; it ranges from mild to severe and is characterized by steady, boring epigastric pain that radiates to the flanks and chest. The pain is worse after the consumption of alcohol and fatty meals. Nausea, vomiting, and anorexia are also common features. With chronic pancreatitis, the clinical symptoms are often subject to periods of exacerbation and remission.

BOX 81.1 Causes of Chronic Pancreatitis

Calcifying Pancreatitis
- Alcohol
- Tobacco
- Idiopathic
  - Tropical
  - Juvenile
  - Senile

Obstructive Pancreatitis
- Tumor
  - Adenocarcinoma
  - Islet cell tumor
  - Cystadenoma
  - Intraductal papillary tumor

Steroid-Responsive Pancreatitis
- Autoimmune pancreatitis
  - Type 1
  - Type 2

SIGNS AND SYMPTOMS

Patients with chronic pancreatitis present similar to those with acute pancreatitis but may appear more chronically ill than acutely ill (Fig. 81.1). Tachycardia and hypotension resulting from hypovolemia are much less common in chronic pancreatitis and are ominous prognostic indicators, or they may suggest the presence of another pathologic process, such as perforated peptic ulcer. Diffuse abdominal tenderness with peritoneal signs may be noted if the patient has acute inflammation. A pancreatic mass or pseudocyst secondary to pancreatic edema may be palpable.

TESTING

Although serum amylase levels are always elevated in acute pancreatitis, they may be only mildly elevated or even within normal limits in chronic pancreatitis. Serum lipase levels are also attenuated in chronic, compared with acute, pancreatitis, although lipase may remain elevated longer than amylase in

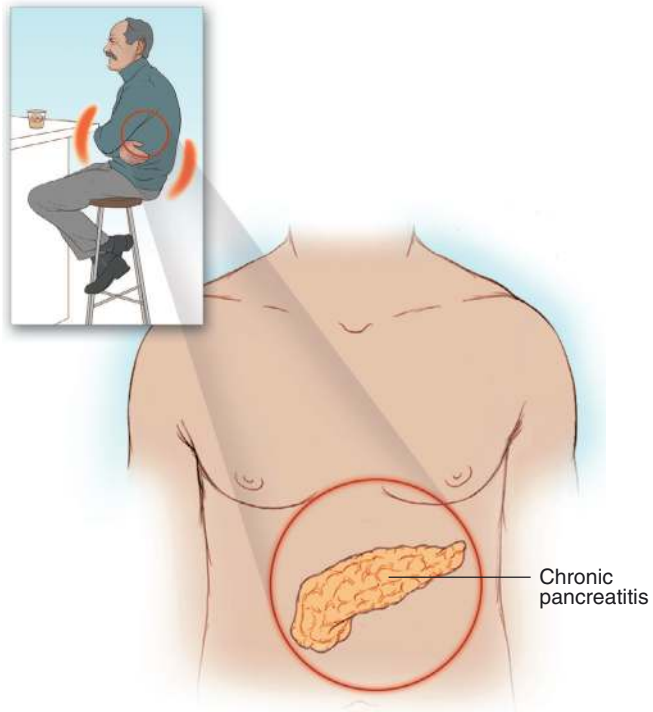

FIG 81.1 Chronic pancreatitis may present similarly to acute pancreatitis, but it can be more challenging to treat.

this setting and be more indicative of actual disease severity. Because serum amylase may be elevated in other diseases, such as parotitis, amylase isozymes may be necessary to confirm a pancreatic basis for this finding. Plain radiographs of the chest are indicated in all patients with chronic pancreatitis to identify pulmonary complications, including pleural effusion. Given its extrapancreatic manifestations (e.g., acute renal or hepatic failure), serial complete blood counts, serum calcium and glucose levels, liver function tests, and electrolytes are indicated in all patients with chronic pancreatitis. Computed tomography (CT) and ultrasound imaging of the abdomen can identify pancreatic pseudocyst or pancreatic tumor that may have been overlooked, and it may help the clinician gauge the severity and progress of the disease (Figs. 81.2 and 81.3). Gallbladder evaluation with radionuclides is indicated if gallstones are a possible cause of chronic

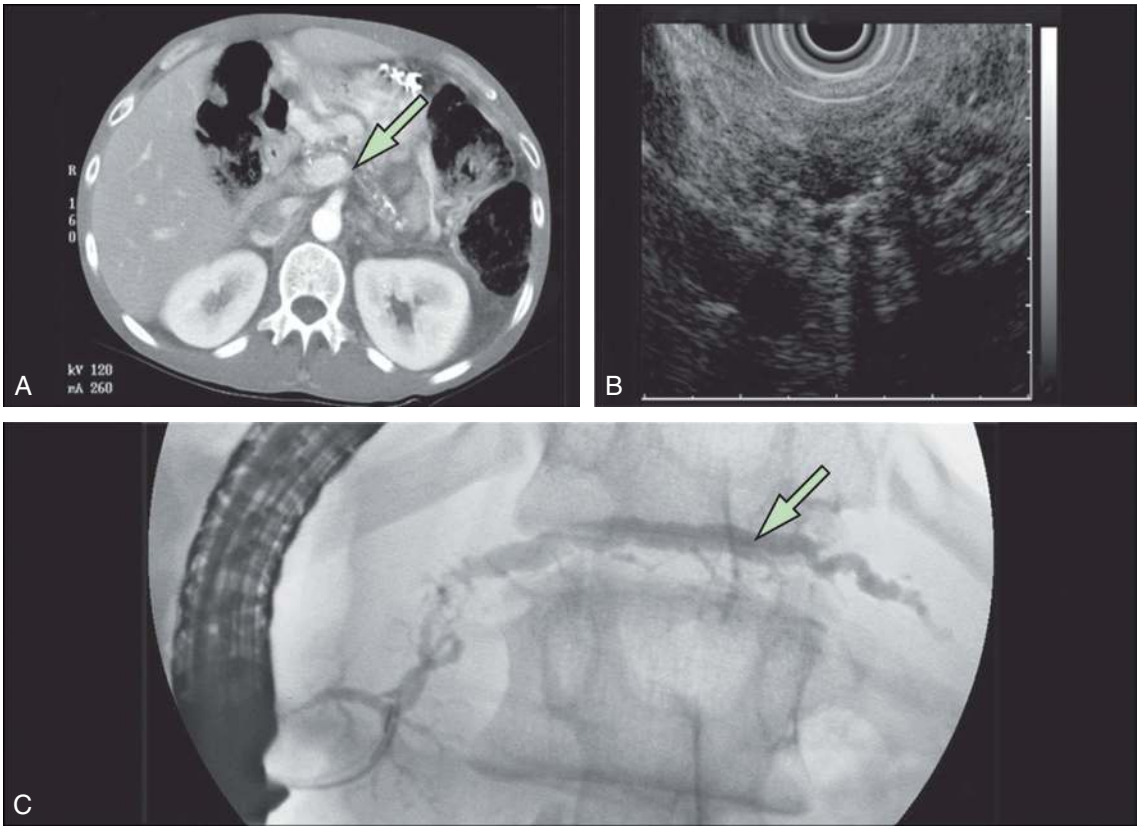

FIG 81.2 Radiographic and endoscopic images for the diagnosis of chronic pancreatitis. **A,** Computed tomography (CT) scan of the abdomen showing pancreatic ductal dilation (*arrow*) and parenchymal calcification in an atrophic pancreas. **B,** Image from endoscopic ultrasound showing hyperechoic foci with shadowing. **C,** Endoscopic retrograde pancreatogram showing an irregular pancreatic duct (*arrow*). (From Majumder S, Chari ST. Chronic pancreatitis. *Lancet*. 2016;387(10031):1957–1966.)

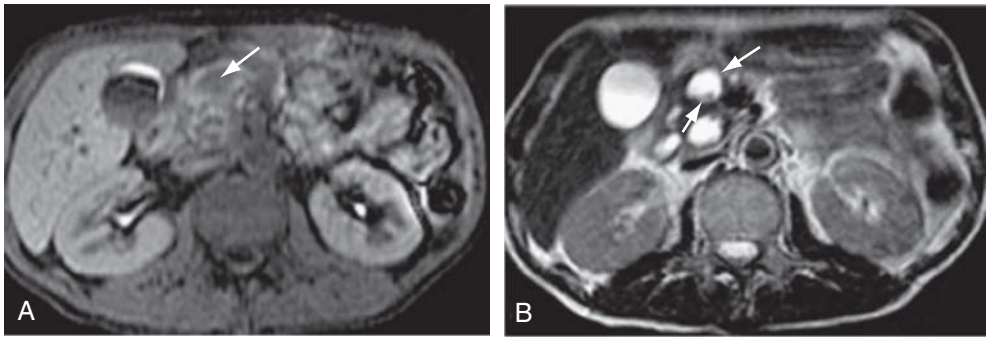

FIG 81.3 Chronic pancreatic pseudocyst. **A,** Axial T1-weighted postgadolinium magnetic resonance imaging (MRI) shows a low-signal collection anterior to the pancreas (*arrow*). **B,** Axial T2-weighted MRI shows a high-signal collection (*long arrow*). In a patient with a prior history of pancreatitis, this finding is consistent with a pseudocyst. Debris is noted within the pseudocyst (*short arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2666.)

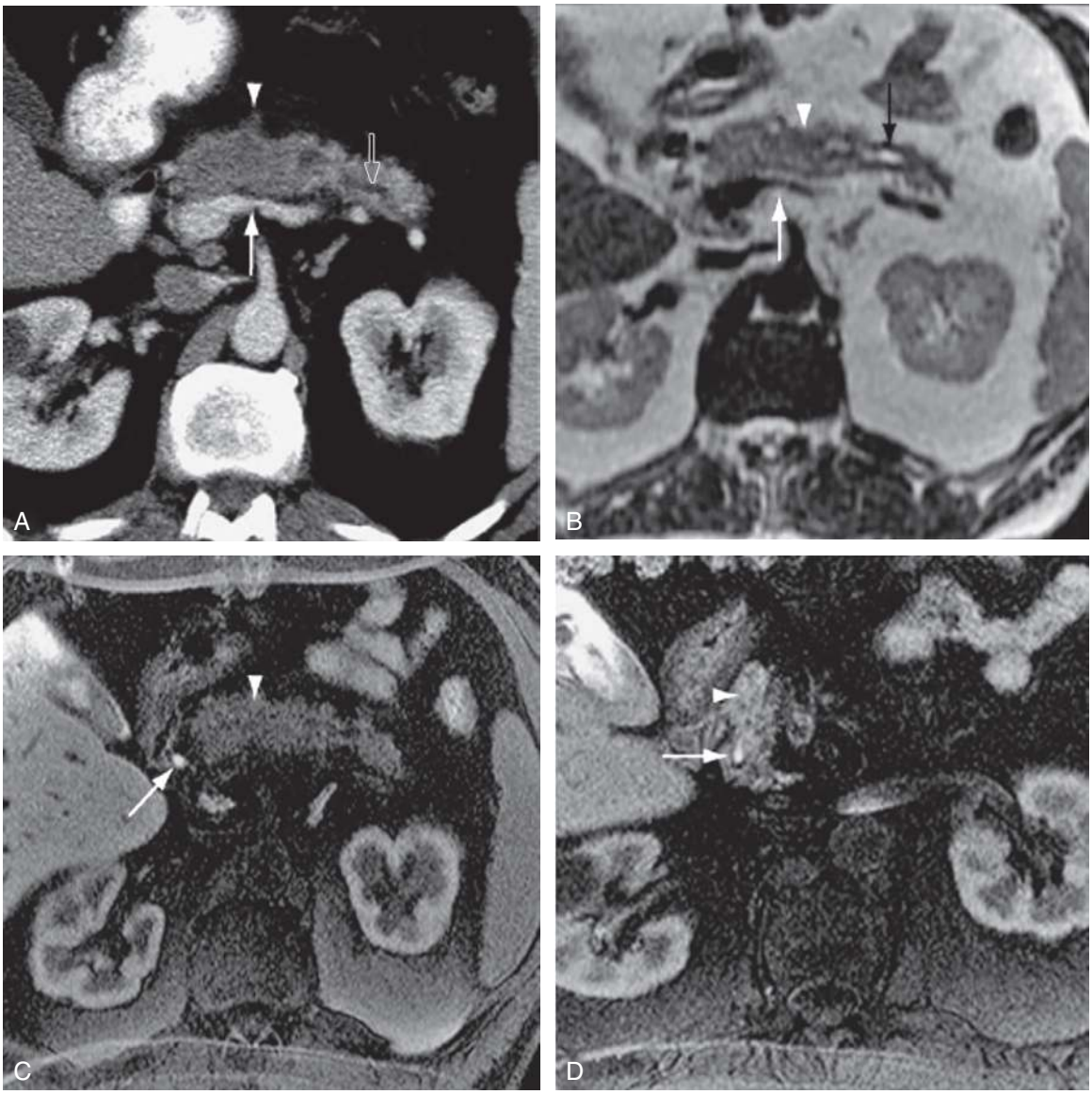

FIG 81.4 Pancreatic cancer. **A,** Axial contrast-enhanced computed tomography scan shows a large pancreatic body mass (*arrowhead*) with distal ductal dilation (*open arrow*). The splenic vein (*arrow*) is not involved. **B,** Axial T1-weighted magnetic resonance imaging (MRI) shows similar findings: pancreatic body mass (*arrowhead*), distal ductal dilation (*black arrow*), and a clear fat plane from the splenic vein (*white arrow*). **C,D,** Postmanganese T1-weighted fat-saturated MRI. The pancreatic mass does not enhance (*arrowhead*). The common bile duct is enhanced and is seen as a high-signal structure (*arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging.* 3rd ed. Philadelphia: Saunders; 2006:2672.)

pancreatitis. Arterial blood gas analysis can identify respiratory failure and metabolic acidosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes perforated peptic ulcer, acute cholecystitis, bowel obstruction, renal calculi, myocardial infarction, mesenteric infarction, diabetic ketoacidosis, and pneumonia. Rarely, the collagen vascular diseases, including systemic lupus erythematous and polyarteritis nodosa, may mimic chronic pancreatitis. Because the pain of acute herpes zoster may precede the rash by 3–7 days, it may erroneously be attributed to chronic pancreatitis in patients who have had previous bouts of the disease. In addition, the clinician should always consider the possibility of malignant pancreatic disease (Figs. 81.4 and 81.5).

TREATMENT

The initial management of chronic pancreatitis focuses on alleviating pain and treating malabsorption. As with acute pancreatitis, the pancreas is allowed to rest by giving the

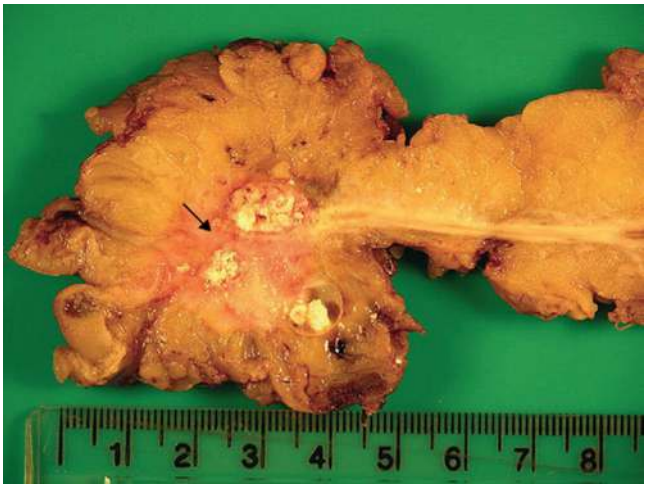

FIG 81.5 Pancreatic adenocarcinoma. Resection specimen showing pancreatic adenocarcinoma (*arrow*) arising within hereditary chronic pancreatitis. Ductal calcification and cyst formation are also seen. (From French JJ, Charnley RM. Chronic pancreatitis. *Surgery (Oxford)* 2013;31(6):304–309.)

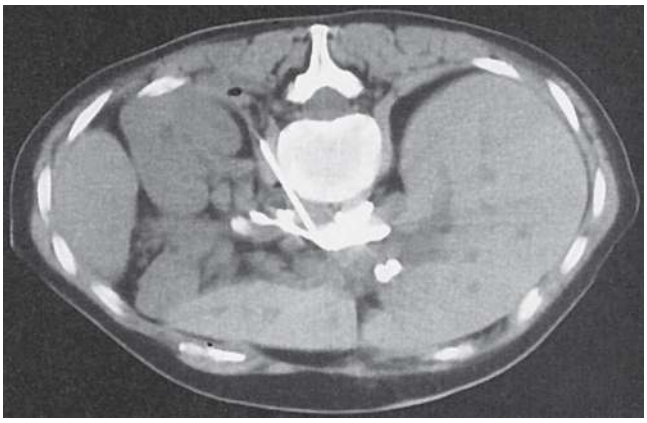

FIG 81.6 Computed tomography–guided celiac plexus block. (From Waldman SD. *Atlas of interventional pain management*. 2nd ed. Philadelphia: Saunders; 2004:288.)

patient nothing by mouth to decrease serum gastrin secretion and, if ileus is present, instituting nasogastric suction. Short-acting opioid analgesics such as hydrocodone are a reasonable next step if conservative measures do not control the patient's pain. If ileus is present, a parenteral opioid such as meperidine is a good alternative. Because the opioid analgesics have the potential to suppress the cough reflex and respiration, the patient must be closely monitored and instructed in pulmonary toilet techniques. The use of opioid analgesics must be monitored carefully, because the potential for misuse and dependence is high.

If symptoms persist, CT-guided celiac plexus block with local anesthetic and steroid is indicated and may decrease the mortality and morbidity associated with this disease (Fig. 81.6). If the relief from this technique is short-lived, neurolytic CT-guided celiac plexus block with alcohol or phenol is a reasonable next step. Alternatively, continuous thoracic epidural block with local anesthetic, opioid, or both may provide adequate pain control and allow the patient to avoid the respiratory depression associated with systemic opioid analgesics.

Hypovolemia should be treated aggressively with crystalloid and colloid infusions. For prolonged cases of chronic pancreatitis, parenteral nutrition is indicated to avoid malnutrition. Secondary insulin-dependent diabetes mellitus may require insulin supplementation. Exopancreatic enzyme supplementation may also be required. Surgical drainage and removal of necrotic tissue may be required if severe necrotizing pancreatitis fails to respond to these treatment modalities.

COMPLICATIONS AND PITFALLS

The major pitfall is failure to recognize the severity of the patient's condition and to identify and aggressively treat the extrapancreatic manifestations of chronic pancreatitis. Hypovolemia, hypocalcemia, and renal and respiratory failure occur with sufficient frequency that the clinician must actively seek these potentially fatal complications and treat them aggressively. If opioids are used, the clinician must constantly watch for overuse and dependence, especially if the underlying cause of the chronic pancreatitis is alcohol abuse.

CLINICAL PEARLS

Chronic pancreatitis is a common cause of abdominal pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking serious extrapancreatic complications. The judicious use of opioid analgesics is usually adequate to control the pain of acute exacerbations. If necessary, celiac plexus block and thoracic epidural block are straightforward techniques that can produce dramatic pain relief.

SUGGESTED READINGS

Esposito I, Hruban Ralph H, Caroline Verbeke, et al. Risk of recurrent pancreatitis and progression to chronic pancreatitis after a first episode of acute pancreatitis. *Clin Gastroenterol Hepatol*. 2016;14(5):738–746.

French JJ, Charnley RM. Chronic pancreatitis. *Surgery (Oxford)*. 2013;31(6):304–309.

Haeberle L, Saluja Ashok, Castillo Carlos Fernandez-del, et al. Guidelines on the histopathology of chronic pancreatitis. Recommendations from the working group for the international consensus guidelines for chronic pancreatitis in collaboration with the International Association of Pancreatology, the American Pancreatic Association, the Japan Pancreas Society, and the European Pancreatic Club. *Pancreatology*. 2020;20(4):586–593. https://doi.org/10.1016/j.pan.2020.04.009.

Irwan Iqbal Rivai M, Mitra Juni, et al. A rare case of tropical chronic pancreatitis with giant pseudocyst: case report. *Ann Med Surg*. 2021;71:102947. https://doi.org/10.1016/j.amsu.2021.102947.

Johnston M, Ravindran R. Chronic pancreatitis. *Surgery (Oxford)*. 2022;40(4):266–273. https://doi.org/10.1016/j.mpsur.2022.01.007.

Majumder S, Chari ST. Chronic pancreatitis. *Lancet*. 2016;387(10031):1957–1966.

Samokhvalov AV, Rehm J, Roerecke M. Alcohol consumption as a risk factor for acute and chronic pancreatitis: a systematic review and a series of meta-analyses. *Ebio Med*. 2015;2(12):1996–2002.

Tuck NL, Teo K, Kuhlmann L, et al. Pain patterns in chronic pancreatitis and chronic primary pain. *Pancreatology*. 2022;22(5):572–582.

Waldman SD. Celiac plexus block: single needle transaortic approach. In: *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2016:401–407.

Waldman SD. Celiac plexus block. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:476–477.

Waldman SD. Chronic pancreatitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:279–280.

Waldman SD. The celiac plexus. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:117–118.

82

Abdominal Aortic Aneurysm

ICD-10 CODE I71.40

THE CLINICAL SYNDROME

Abdominal aortic aneurysms have been called the silent widow-maker because they are almost always symptomatic until they expand or rupture. Abdominal aortic aneurysms are most often the result of proteolytic degeneration of the media layer of the aorta with an associated inflammatory response causing biomechanical stress to the aortic wall which ultimately leads to the gradual dilatation of the vessel lumen (Fig. 82.1). A disease of the elderly, abdominal aortic aneurysm occurs more frequently in white males, with smoking a major risk factor for the development of this potentially life-threatening disorder (Fig. 82.2). Atherosclerosis is a common finding associated with the

FIG 82.1 Intraoperative photographs. **A,** The patient was placed in the right lateral decubitus position. **B,** A large amount of intramural thrombus was evacuated. **C,** Final placement of aortic graft before closure of the aneurysmal sac around the graft. (From Ng JI, Nguyen T, Tarpara A, et al. Giant abdominal aortic aneurysms. *J Vasc Surg Cases Innov Tech*. 2021;7(4):659–664.)

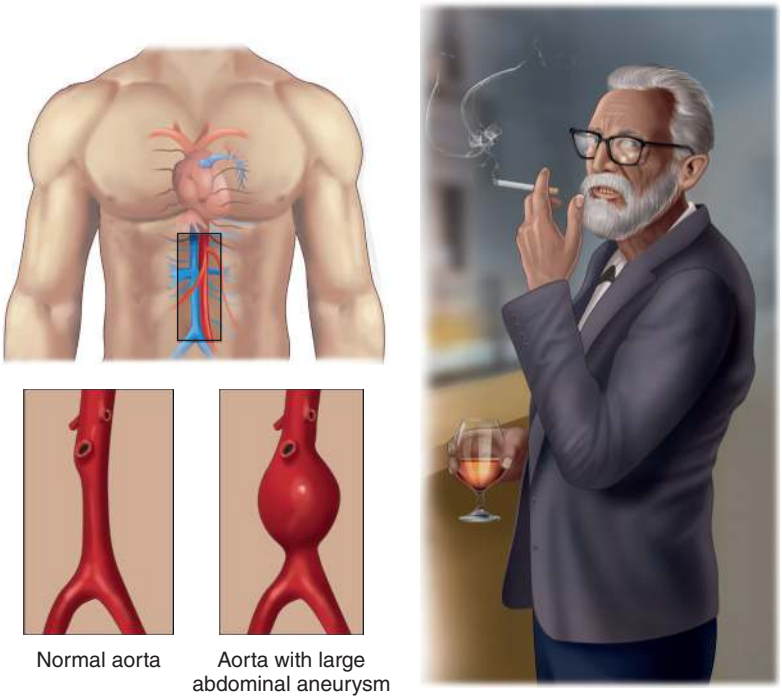

FIG 82.2 A disease of the elderly, abdominal aortic aneurysm occurs more frequently in white males, with smoking a major risk factor for the development of this potentially life-threatening disorder.

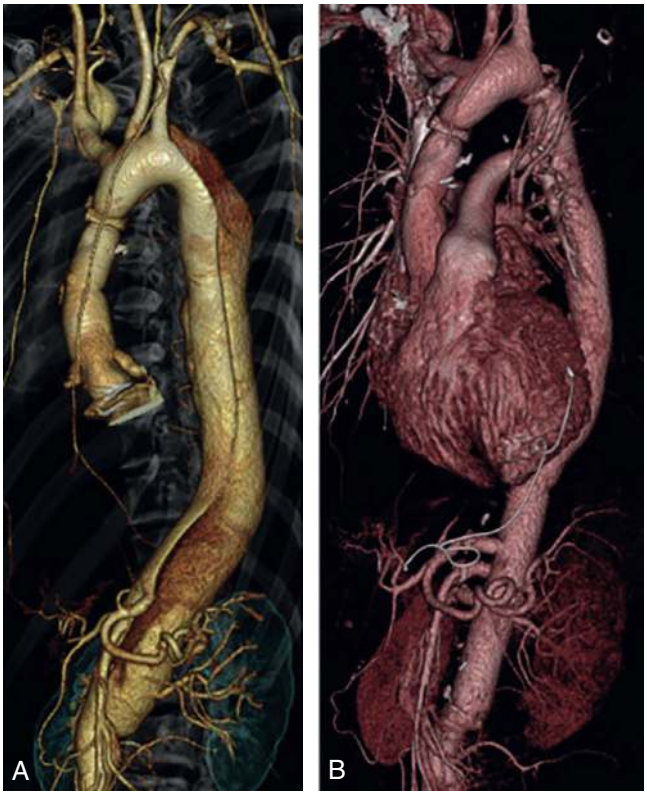

FIG 82.3 Aortic aneurysm in patient with Marfan syndrome. Three-dimensional computed tomography. **A,** Before thoracoabdominal aorta replacement showing Crawford extent II chronic aortic dissection. **B,** After graft replacement. (From Ando M, Yamauchi H, Hoshino Y, Ono M. Thoracoabdominal aorta replacement for a patient with Marfan syndrome with poor left ventricular function. *JTCVS Tech*. 2021;7:45–48.)

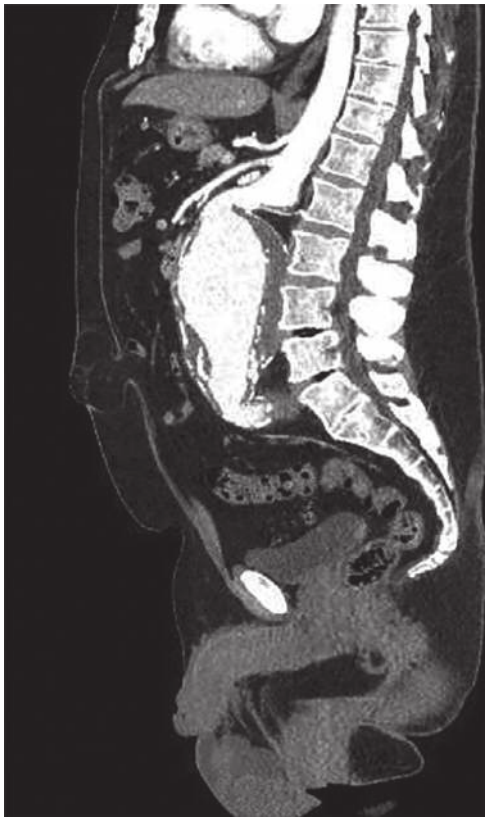

FIG 82.4 Sagittal view of CT angiogram showing 9.0 cm AAA with evidence of "draping aorta" over spinal column to suggest evidence of posterior rupture. *AAA*, Abdominal aortic aneurysm; *CT*, computed tomography. (From Cherniwchan H, Huang B, Ginting N. Chronic contained posterior rupture of large abdominal aortic aneurysm missed on pre-operative CT imaging. *Ann Vasc Surg Brief Rep Innov*. 2022;2(2):100083.)

development of abdominal aortic aneurysm as is the presence of chronic obstructive pulmonary disease, coronary artery disease, and hypertension. There is also an increased incidence of abdominal aortic aneurysm in patients suffering from Marfan, Ehlers–Danlos syndrome, and collagen vascular disease (Fig. 82.3). Most abdominal aortic aneurysms are asymptomatic and are identified as an incidental finding on physical examination or on diagnostic imaging testing obtained for other reasons.

SIGNS AND SYMPTOMS

In most patients, abdominal aortic aneurysms are asymptomatic until they enlarge or impinge on surrounding structures (Fig. 82.4). At his point, the aneurysm will be palpable on abdominal examination. Nonspecific back, abdominal, flank, and groin pain may be attributed to musculoskeletal causes. Occasionally, compression of displacement of abdominal viscera may cause the patient to experience early satiety, nausea, vomiting, urinary symptoms, or edema secondary to venous compression. Abdominal bruit may be present with a thrill identifiable in an occasional patient. Blue toe syndrome may occur if thromboembolic phenomenon occurs (Fig. 82.5 and

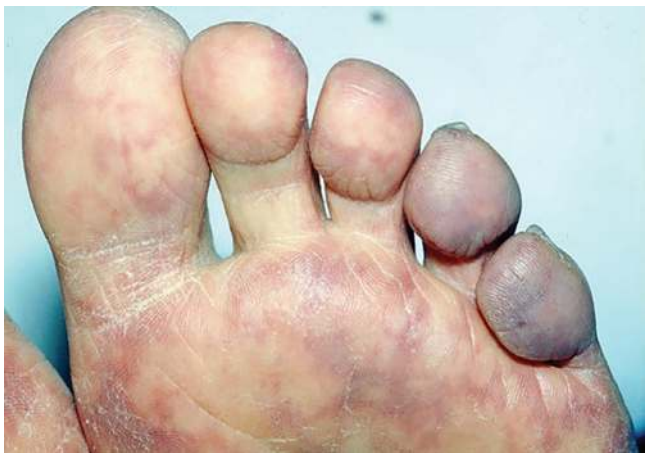

FIG 82.5 Blue toes and livedo reticularis from atheroemboli. (From Hirschmann JV, Raugi GJ. Blue (or purple) toe syndrome. *J Am Acad Dermatol*. 2009;60(1):1–20.)

Box 82.1). Progressive symptoms associated with abdominal aortic aneurysms represent a medical emergency as they may signal the possibility of rapid expansion and imminent rupture. With retroperitoneal bleeding from the aneurysm, Grey Turner sign may be present indicating retroperitoneal

BOX 82.1 Causes of Blue Toe Syndrome

Embolism
Atherosclerosis
Talc emboli
Aortic aneurysm
Atrial fibrillation

Thrombosis
Warfarin-induced skin necrosis
Thrombotic thrombocytopenic purpura
Disseminated intravascular coagulation
Toxic shock syndrome
Antiphospholipid syndrome
Vasoconstrictive disorders
Ergotism
Acryocynosis
Bechet syndrome
Syphillis
Frostbite
Perniosis (Trench foot)
Paraneoplastic acral vascular syndrome
Vasculitis secondary to collagen vascular disease

Impaired Venous Outflow
Phlegmasia cerulea dolens
Venous thrombosis
Venous gangrene

Abnormal Circulating Blood
Cryofibrinogenemia
Cryoglobulinemia
Cold agglutinins
Essential thrombocythemia

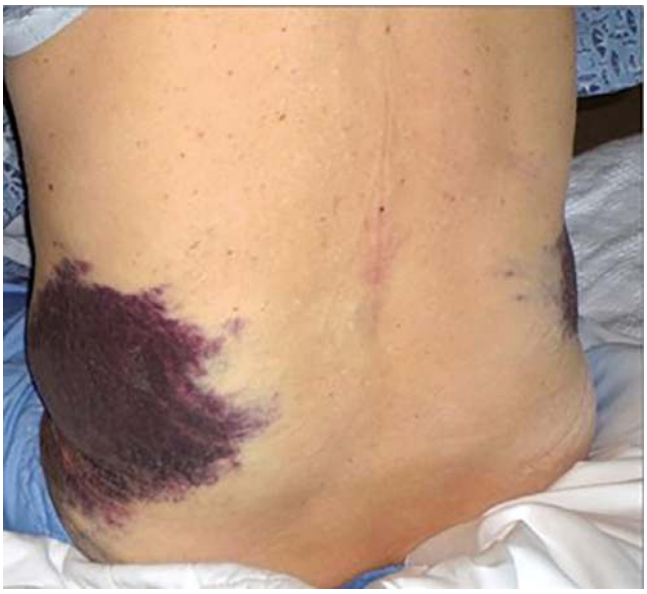

FIG 82.6 Grey Turner sign. (From Masha L, Bernard S. Grey Turner's sign suggesting retroperitoneal haemorrhage. *Lancet*. 2014;383(9932):1920.)

hemorrhage (Fig. 82.6). The pain associated with the rupture of abdominal aortic aneurysms is severe and may be associated with syncope.

TESTING

Laboratory testing is utilized to identify preexisting comorbidities that may impact the operative risk of elective or emergent repair of an abdominal aortic aneurysm. Pulmonary function testing will also help with risk stratification given the high incidence of smoking associated with abdominal aortic aneurysm. Ultrasonography is the gold standard for the diagnosis of abdominal aortic aneurysm, with a sensitivity of 100% and a specificity of 96% (Fig. 82.7). Ultrasonography can also aid in the identification of other intra-abdominal pathology including fistula, branch artery involvement, suprarenal extension of the aneurysm, and vascular compression as well as identifying the presence of free blood in the peritoneal cavity. Computerized tomography also has a sensitivity of almost 100% in the diagnosis of abdominal aortic aneurysm with the advantage of a larger field of view making the assessment of the rostral-caudal extent of the aneurysm as well as evaluation of involvement of other visceral vessels much easier (Fig. 82.8). Three-dimensional computed tomography (CT) and CT angiography may offer added insights into the best approach to surgical repair of more complex abdominal aortic aneurysms (Fig. 82.9).

DIFFERENTIAL DIAGNOSIS

The variable and nonspecific clinical presentation of the abdominal aortic aneurysm can lead to diagnostic misadventures. Musculoskeletal, abdominal, urinary, and cardiac disorders are often the working diagnosis, with the diagnosis of abdominal aortic aneurysm entertained when a pulsatile mass is identified on physical examination or it is identified on medical imaging for other indications. Common mimics of abdominal aortic aneurysm are listed in Box 82.2.

TREATMENT

Prophylactic repair of abdominal aortic aneurysms using open surgical, minimally invasive laparotomy, and endovascular stenting is the treatment of choice given that long-term medical management ultimately has a mortality of 100% due to the probability of rupture (Fig. 82.10). Rupture risk is greatly increased as the diameter of the abdominal aortic aneurysm increases. For abdominal aortic aneurysms with a diameter below 4.9 cm, annual surveillance with watchful waiting is a reasonable first step. For abdominal aortic aneurysms with a diameter greater than 6 cm, surgical repair is

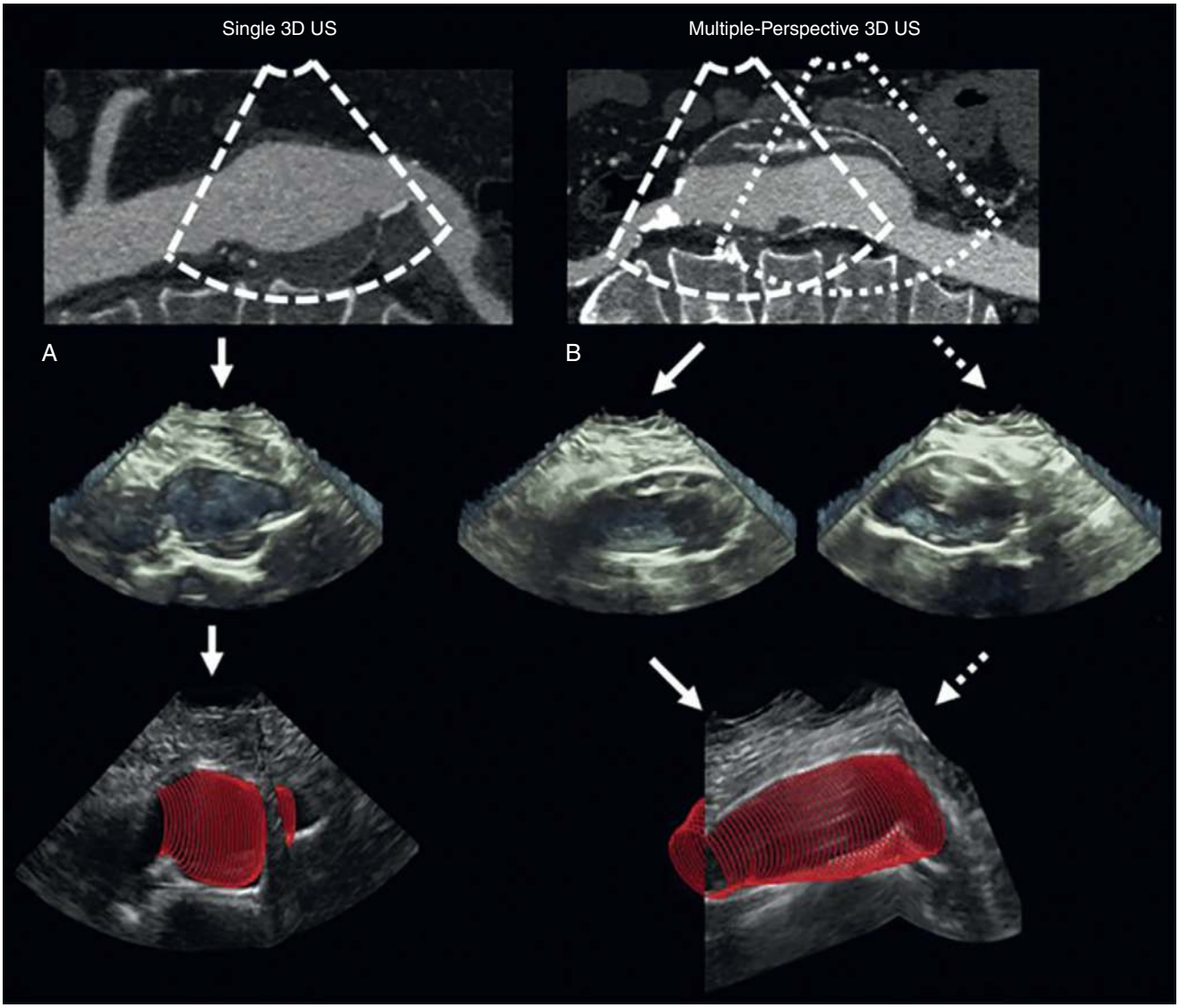

FIG 82.7 Ultrasound of abdominal aortic aneurysm. Example of a relatively small AAA **(A)** and a larger AAA **(B)**. Computed tomography data are shown (*top row*) with the field of view of the 3D US scan in the *x–z* plane (*dashed white lines*), as well as the corresponding 3D US volume data (*middle row*). Finally, automatic segmentation of the US data for the single volume and the merged 3D volumes are shown (*bottom row*). 3D, Three dimensional; *AAA*, abdominal aortic aneurysm; US, ultrasound. (From van Disseldorp EMJ, van Dronkelaar JJ, Pluim JPW, et al. Ultrasound based wall stress analysis of abdominal aortic aneurysms using multiperspective imaging. *Eur J Vasc Endovasc Surg*. 2020;59(1):81–91.)

indicated. Patients elder than 80 years of age, especially those with significant comorbidities, will most likely benefit from endovascular stenting of the aneurysm. Endovascular stents are placed within the lumen of the abdominal aortic aneurysm and serve to contain aortic flow and decrease the aneurysmal endovascular pressure (Fig. 82.11). Unfortunately, continued leakage around the endovascular stent can occur and these endoleaks can allow the increased pressurization of the abdominal aortic aneurysm (endotension) to continue unabated placing the patient at continued risk of rupture (Fig. 82.12).

COMPLICATIONS AND PITFALLS

Prehospital rupture of an abdominal aortic aneurysm is associated with a mortality of greater than 50%. Comorbidities including congestive heart failure and chronic obstructive pulmonary disease cause a significant negative impact on prognosis. Because the prophylactic surgical or endovascular repair of abdominal aortic aneurysm is associated with a significantly lower mortality when compared with the repair of aneurysms that have already ruptured, clinicians should focus on early treatment of this disorder

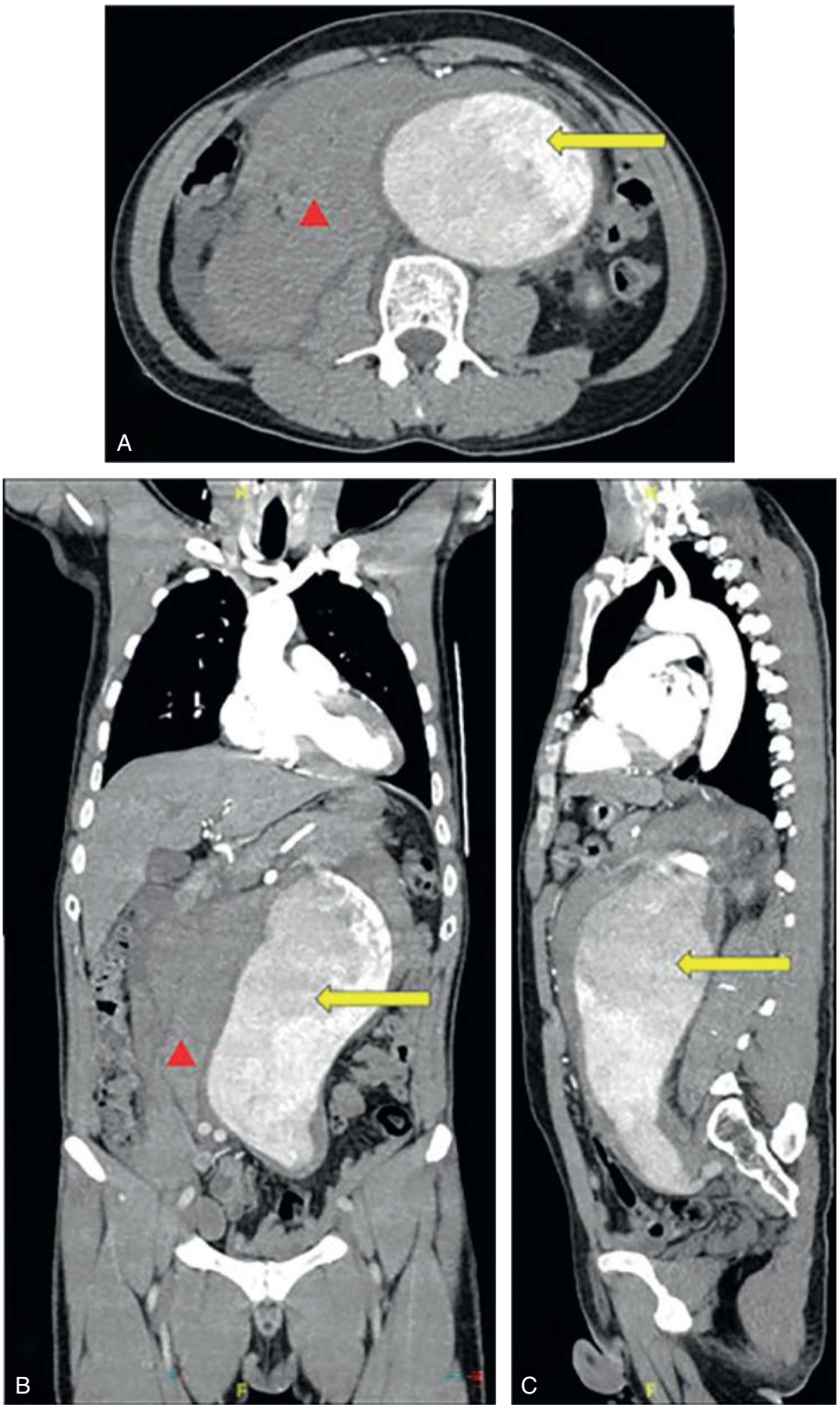

FIG 82.8 CT aortogram (**A,** axial section; **B,** coronal section; **C,** parasagital section) demonstrating the large ruptured abdominal aortic aneurysm (10.8 × 10.8 × 24.1 cm^3) (*yellow arrow*) and the retroperitoneal hematoma (*red arrowhead*). *CT*, Computed tomography. (From Lohani KR, Yan Xin GW, Cui J, Sannasi VV. Ruptured abdominal aortic aneurysm in a young male patient, a rare case report. *Int J Surg Case Rep*. 2022;90:106713.)

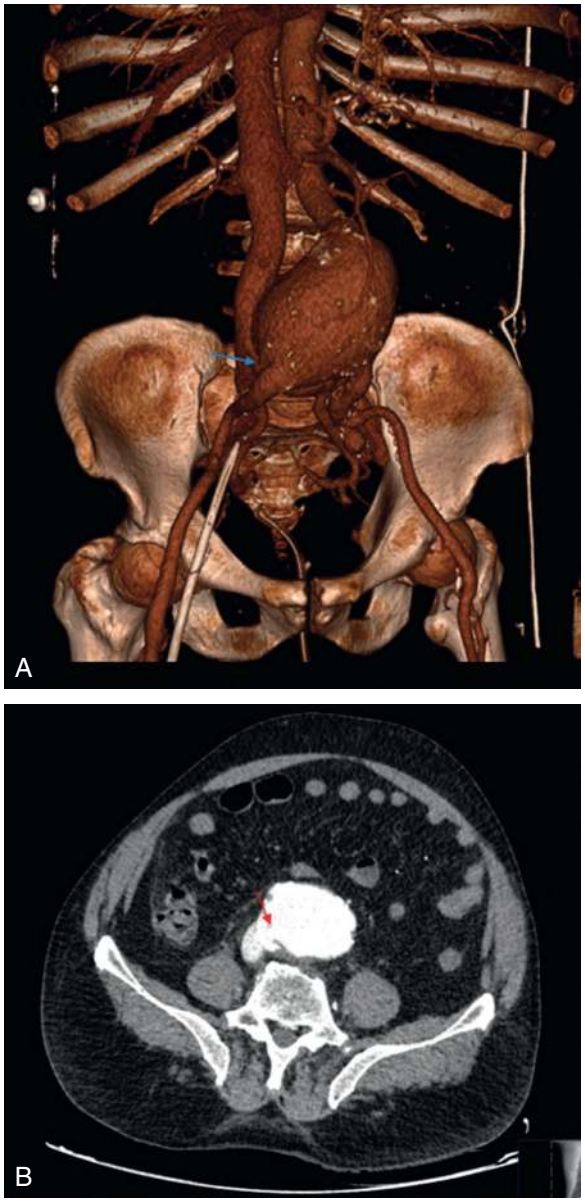

FIG 82.9 A, Volume rending 3D CT image shows a large abdominal aortic aneurysm distal to the renal arteries and the approximate location of the aortocaval fistula (*blue arrow*). B, Contrast enhanced axial CT image shows indentation and fistula line in the vena cava (*red arrow*), disappearance of the fatty planes between vena cava and aorta, and rapid simultaneous contrast passage into the vena cava from the aorta. *CT*, Computed tomography. (From Esmat HA, Naseri MW. Endovascular management of aortocaval fistula complicating abdominal aortic aneurysm presenting as an acute renal failure. *Ann Med Surg*. 2021;62:477–480.)

BOX 82.2 Differential Diagnosis of Abdominal Aortic Aneurysm

- Acute gastritis
- Cholelithiasis
- Diverticulitis pancreatitis
- Musculoskeletal complaints
- Renal calculi
- Peptic ulcer disease
- Appendicitis
- Bowel obstruction
- Myocardial infarction

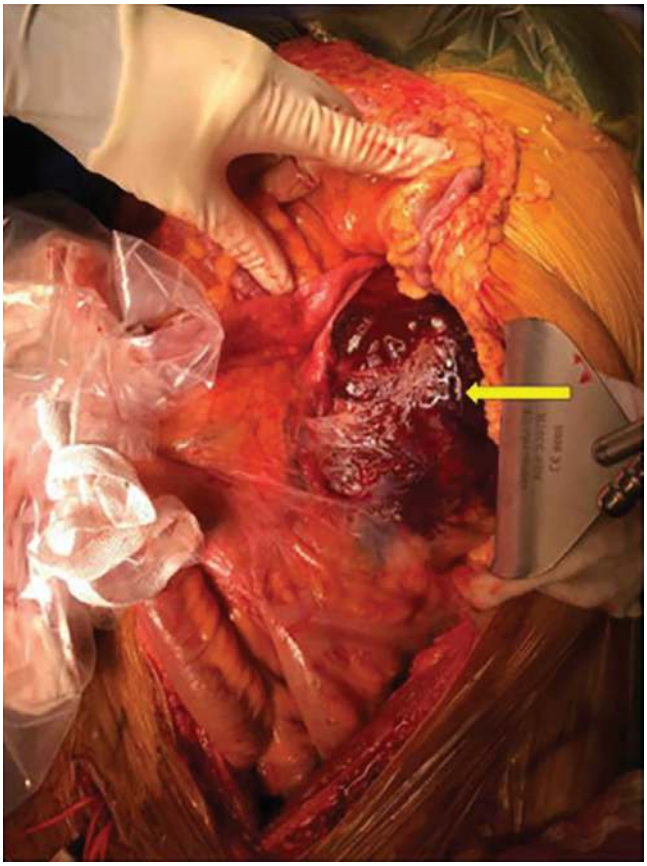

FIG 82.10 Intraoperative picture demonstrating the aneurysmal dilatation of abdominal aortic aorta (*yellow arrow*). Bowel has been eviscerated into bowel bag to the patient's right and the retractor is on the left. Surgeon's hand is holding the transverse colon cranially. (From Lohani KR, Yan Xin GW, Cui J, Sannasi VV. Ruptured abdominal aortic aneurysm in a young male patient, a rare case report. *Int J Surg Case Rep*. 2022;90:106713.)

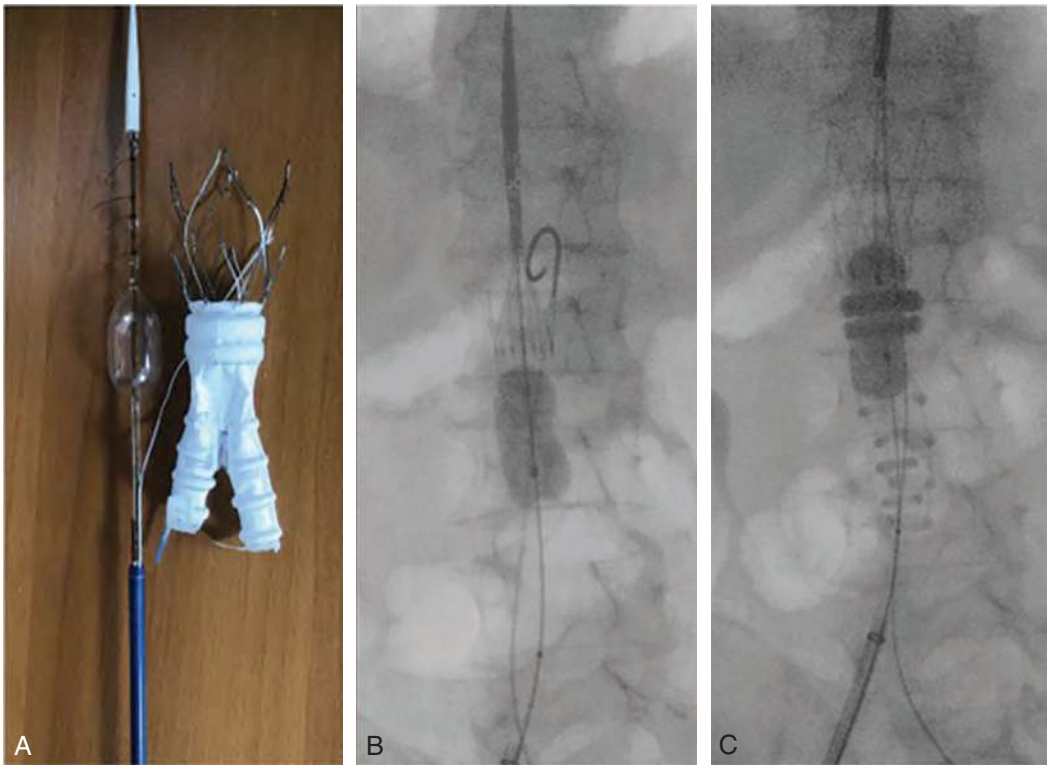

FIG 82.11 **A,** Ovation Alto endograft model with the incorporated compliant balloon inflated. **B,** Intraoperative fluoroscopy with Ovation Alto semideployed with the incorporated balloon partially inflated. **C,** Intraoperative fluoroscopy with Ovation Alto endograft main body deployed, with O rings polymer filled. The incorporated balloon is advanced 5–7 mm and fully inflated. (From de Donato G, Pasqui E, Panzano C, et al. Early experience with the new Ovation Alto stent graft in endovascular abdominal aortic aneurysm repair. *EJVES Vasc Forum*. 2022;54:7–12.)

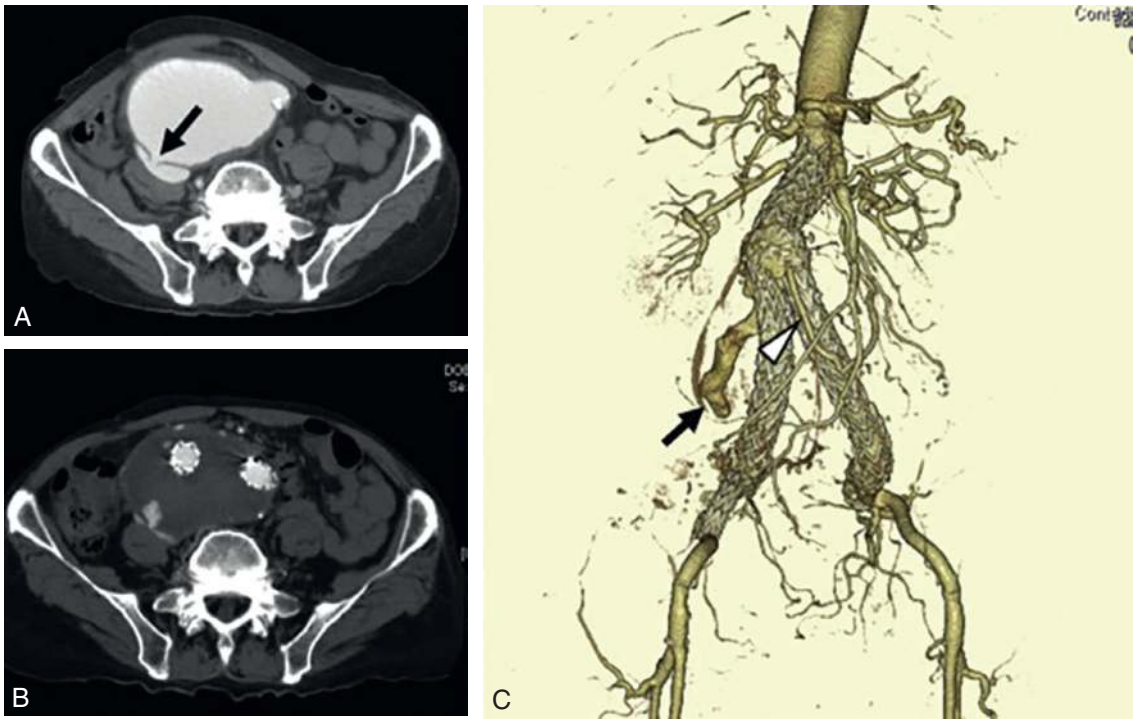

FIG 82.12 Computed tomography (CT) angiogram at the first operation. **A,** Preoperative CT scan showing abdominal aortic aneurysm (AAA) and aortocaval fistula (ACF; *arrow*). **B,** Postoperative CT scan showing a type II endoleak and persistent communication between the AAA and inferior vena cava (IVC); however, the aneurysm sac had shrunk. **C,** Three-dimensional CT angiogram showing persistent communication (*arrow*) via a meandering mesenteric artery (*open arrow*). (From Fujimiya T, Seto Y, Ishida K, Takase S, Satokawa H, Yokoyama H. Impending rupture of abdominal aortic aneurysm due to spontaneous obstruction of aortocaval fistula after endovascular abdominal aortic aneurysm repair. *J Vasc Surg Cases Innov Tech*. 2021;7(2):219–222.)

CLINICAL PEARLS

Most abdominal aortic aneurysms are asymptomatic and are identified as an incidental finding on physical examination or on diagnostic imaging testing obtained for other reasons. This in part explains the fact that over 80% of patients with ruptured abdominal aortic aneurysm do not have a previous diagnosis of this life-threatening disease. A high index of suspicion for the diagnosis of abdominal aortic aneurysm is critical to allow diagnosis and treatment prior to rupture.

SUGGESTED READINGS

Hoo AJS, Fitzgibbon JJ, Hussain MA, et al. Contemporary indications for open abdominal aortic aneurysm repair in the endovascular era. *J Vasc Surg*. 2022;76(4):923–931. e1. https://doi.org/10.1016/j.jvs.2022.03.866.

Hu YD, Jones JM, Sensenig CJ, et al. The impact of work status and age in choosing type of abdominal aortic aneurysm repair. *J Surg Res*. 2022;275:149–154. https://doi.org/10.1016/j.jss.2021.12.036.

Le Q, Kanneganti M, Tracci MC. Management of ruptured abdominal aortic aneurysm infected with *Listeria monocytogenes* in a patient with poor dentition. *Ann Vasc Surg Brief Rep Innov*. 2022;2(1):100041. https://doi.org/10.1016/j.avsurg.2022.100041.

Olson SL, Panthofer AM, Blackwelder W, et al. Role of volume in small abdominal aortic aneurysm surveillance. *J Vasc Surg*. 2022;75(4):1260–1267. e3. https://doi.org/10.1016/j.jvs.2021.09.046.

Rahim AA, Ibrahim R, Yao Lu, et al. Incidence of secondary rupture after abdominal aortic aneurysms (endovascular vs. open surgical repair). *Ann Med Surg*. 2021;70:102831. https://doi.org/10.1016/j.amsu.2021.102831.

Sen I, Franco-Mesa C, Erben Y, DeMartino RR. Abdominal aortic and visceral artery aneurysms. *Cardiol Clin*. 2021;39(4):517–525. https://doi.org/10.1016/j.ccl.2021.06.004.

83

Irritable Bowel Syndrome

ICD-10-CM DIAGNOSIS CODE K58.0

THE CLINICAL SYNDROME

Irritable bowel syndrome (IBS) is a common cause of abdominal pain, affecting one in five adults in developed countries. Although it can also occur in the pediatric and adolescent population, the diagnosis rarely occurs before the age of 50. Women are affected more frequently than men. IBS is characterized by recurrent abdominal pain, discomfort, bloating, gas, and an associated change in bowel habits that can take the form of either diarrhea or constipation (Fig. 83.1). Often mucus is present in the stool. IBS is often classified as a functional gastrointestinal disorder because of the association with depression, gastrointestinal-specific anxiety, alexithymia, mood disorders, sleep disorders, perimenstrual disorders, and sexual dysfunction. Food may serve as a trigger to IBS, with spices, chocolate, beans, cabbage, cruciferous vegetables, and fruits frequently implicated. Alterations in the gut microbiota, or dysbiosis, following antibiotic usage or gastrointestinal viral, bacterial, and parasitic infections may also contribute to the pathogenesis of IBS. There appears to be a genetic predisposition to IBS with a family history being identified in approximately 30% of patients diagnosed with IBS. It has been postulated that stress-induced alterations in the brain–gut axis affecting the central and autonomic nervous system, the enteric nervous system, and the neuroendocrine and neuroimmune systems play an important role in the evolution of this disease (Fig. 83.2).

FIG 83.1 Irritable bowel syndrome is characterized by recurrent abdominal pain, discomfort, bloating, gas, and an associated change in bowel habits that can take the form of either diarrhea or constipation.

SIGNS AND SYMPTOMS

Patients suffering from IBS will appear healthy yet may appear tense or anxious. Abdominal examination is bland, with findings of peritoneal irritation and no abnormal mass or organomegaly will be present.

TESTING

The diagnosis of IBS is usually made by taking a careful history. The Rome Scoring Criteria for IBS will help the clinician improve the specificity and sensitivity of diagnosis and guide the physical examination and the use of additional testing (Table 83.1). Basic hematology, thyroid testing, and serum chemistries are indicated in all patients suspected of having IBS. Special attention to serum calcium to rule out hyperparathyroidism and thyroid function testing is mandatory. Stool analysis to rule out all occult blood, malabsorption and viral, bacterial, and parasitic infections should also be performed. Fecal calprotectin analysis may help identify intestinal mucosal inflammation. Hydrogen glucose breath analysis and lactose intolerance testing to rule out bacterial overgrowth should be considered in those patients who developed abdominal symptoms following antibiotic therapy. Sigmoidoscopy, colonoscopy, and lower gastrointestinal barium studies may also be indicated to rule out diseases including inflammatory bowel diseases that may mimic IBS.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes inflammatory bowel disease, celiac disease, microscopic colitis, lactose intolerance, fructose intolerance, colon cancer, gastrointestinal infections, and the symptoms of hyperparathyroidism, hypothyroidism,

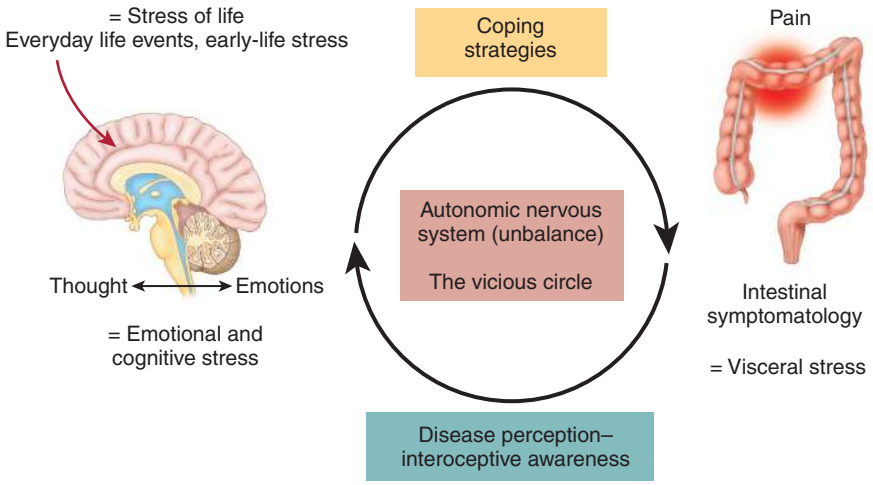

FIG 83.2 Behavioral, affective, and cognitive processes can affect brain–gut axis functioning as a vicious circle that will amplify sensitivity, motility, and anxiety. (Redrawn from Pellissier S, Bonaz B. The place of stress and emotions in the irritable bowel syndrome. In: Litwack G, ed. *Vitamins and hormones*. Vol 103. San Diego: Academic Press; 2017:327–354.)

TABLE 83.1 Diagnosis of Irritable Bowel Syndrome According to Rome III and IV Criteria for Adults and Children

Rome III Criteria	Rome IV Criteria
Adults	
Recurrent abdominal pain or discomfort with onset at least 6 months prior to diagnosis, associated with two or more of the following, at least 3 days per month in the last 3 months • improvement with defecation • onset associated with a change in frequency of stool • onset associated with a change in form (appearance) of stool	Recurrent abdominal pain with onset at least 6 months prior to diagnosis, associated with two or more of the following, at least 1 day per week in the last 3 months • related to defecation • associated with a change in frequency of stool • associated with a change in form (appearance) of stool
Children	
Abdominal discomfort or pain at least one per week, for at least 2 months before diagnosis, associated with 2 or more of the following at least 25% of time • improvement with defecation • onset associated with a change in frequency of stool • onset associated with a change in form (appearance) of stool No evidence of an inflammatory, anatomic, metabolic, or neoplastic process that explains the subject's symptoms. Additional symptoms confirming the diagnosis: • change in defecation rhythm >3/day or <3/week • change in stool consistency—excessively hard or loose • change in stool passage—tenesmus, a feeling of incomplete evacuation • stool containing mucus • bloating	Abdominal pain at least 4 days per months, for at least 2 months before diagnosis, associated with one or more of the following • related to defecation • associated with a change in frequency of stool • associated with a change in form (appearance) of stool In children with constipation, the pain does not resolve with resolution of the constipation (children in whom the pain resolves have functional constipation, not irritable bowel syndrome). After appropriate evaluation, the symptoms cannot be fully explained by another medical condition.

and neuroendocrine tumors (Fig. 83.3). The Mayo Clinic has suggested a simplified algorithm to evaluate functional gastrointestinal disorders (Fig. 83.4).

TREATMENT

The starting point in the treatment of IBS is aimed at eliminating several groups of foods that may be contributing to the patient's gastrointestinal symptoms: (1) gluten-containing foods; (2) high-gas foods; and (3) fermentable oligo-, di-, and monosaccharides and polyols. The addition of fiber supplements, such as psyllium and methylcellulose, may decrease constipation and osmotic laxatives may be added in resistant cases. If diarrhea is the predominant bowel symptom, prophylactic loperamide, and bile acid binders such as cholestyramine and colesevelam may be considered. If gut spasms are present, the judicious use of anticholinergics and antispasmodics, such as hyoscyamine and dicyclomine, may help. If pain,

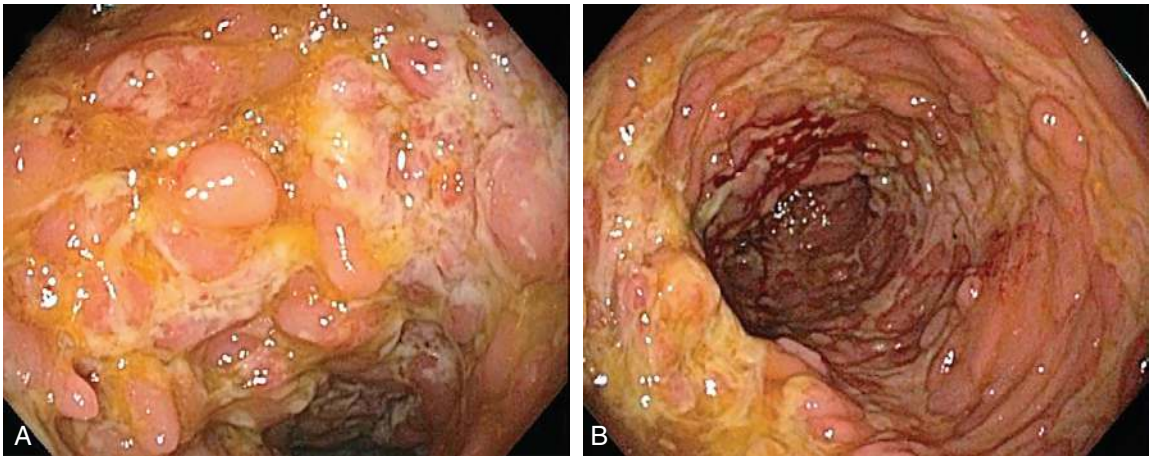

FIG 83.3 A 38-year-old man with Crohn's disease, showing pseudopolyposis **(A)** and colonic ulcers **(B)**. (From Walsh A, Mabee J, Trivedi K. Inflammatory bowel disease. *Prim Care*. 2011;38(3):415–432.)

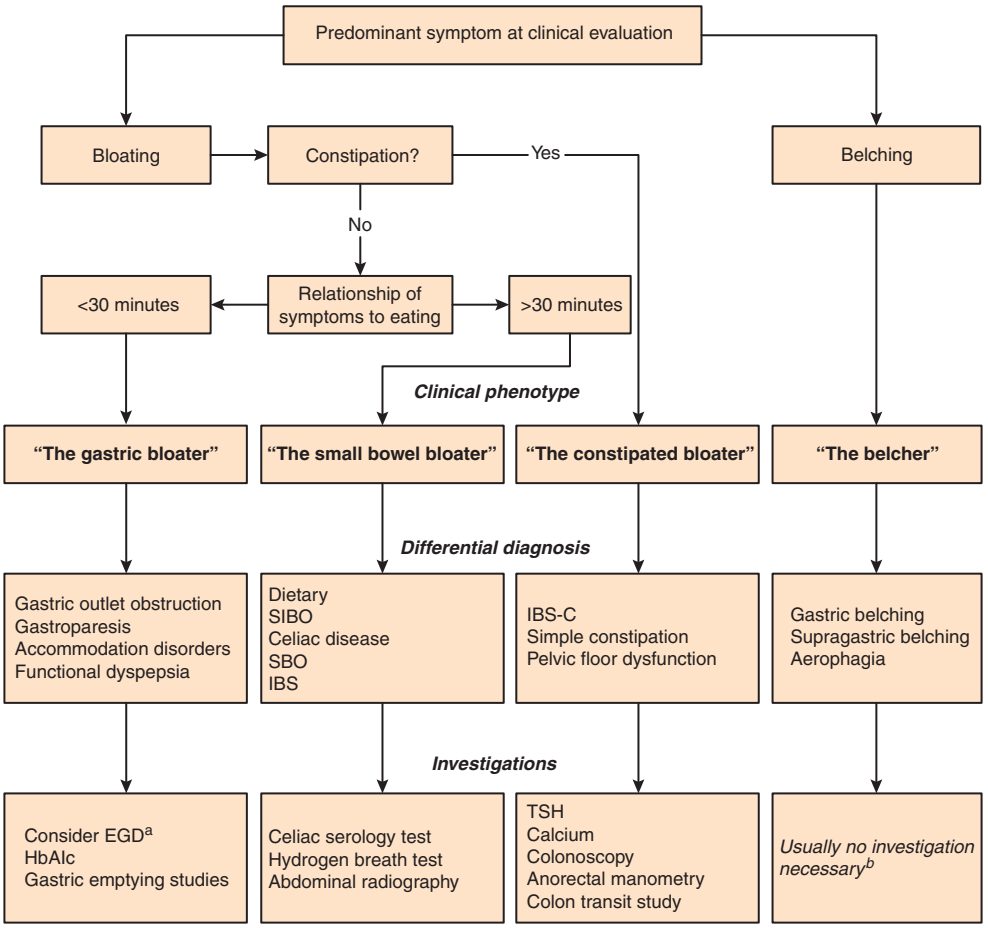

FIG 83.4 A simplified approach to the evaluation of the patient with gas-related symptoms. [a]Indicated in the presence of alarm features: Age >50 years, upper gastrointestinal (GI) malignancy in a first-degree relative, weight loss, GI bleeding, or iron deficiency anemia, dysphagia, odynophagia, persistent vomiting, abnormal imaging suggesting organic disease (American Society for Gastrointestinal Endoscopy). [b]Consider EGD ± gastric biopsies and/or gastric emptying test to rule out an organic etiology, in certain patients. *EGD*, Esophagogastroduodenoscopy; *HbA1c*, glycosylated hemoglobin; *IBS*, irritable bowel syndrome; *IBS-C*, irritable bowel syndrome–constipation; *SBO*, small bowel obstruction; *SIBO*, small intestine bacterial overgrowth; *TSH*, thyroid-stimulating hormone. (From Cotter TG, Gurney M, Loftus CG. Gas and bloating—controlling emissions: a case-based review for the primary care provider. *Mayo Clin Proc*. 2016;91(8):1105–1113.)

discomfort, and sleep disturbance are present, the use of a tricyclic antidepressant or selective serotonin reuptake inhibitor is strongly recommended. Specific treatments for IBS include alosetron, which is used in IBS with diarrhea in women. This drug is not approved for use in men. Lubiprostone, which increases the fluid secretion in the small intestine, may be used in women suffering from IBS with severe constipation. The effectiveness of this drug has not been demonstrated in men. Other newer pharmacologic treatments for IBS include eluxadoline which can decrease IBS-related diarrhea. Linaclotide and increased fluid secretion in the small intestine and may be useful in the management of IBS-related constipation. The antibiotic rifaximin may also help decrease the amount of intestinal bacterial overgrowth seen in some patients suffering from IBS. Treatment of underlying depression, anxiety, sleep disturbance, and mood disorders is crucial if long-term symptom improvement is to be achieved. Alternative treatments, including peppermint and diet supplementation with pre- or probiotics, may provide symptomatic relief in some patients suffering from IBS.

COMPLICATIONS AND PITFALLS

The major pitfall is failure to recognize the underlying cause of the patient's gastrointestinal symptoms. Red flags, including unexplained weight loss, bleeding from the gastrointestinal tract, dysphagia, odynophagia, unexplained fever, chronic vomiting, and nocturnal symptoms, should make the diagnosis of IBS one of exclusion.

CLINICAL PEARLS

IBS is a common cause of abdominal pain. Correct diagnosis is necessary to treat this painful condition properly and to avoid overlooking other causes of abdominal pain and gastrointestinal distress. The behavior components of IBS must be identified and treated if a satisfactory outcome is to be expected.

SUGGESTED READINGS

Domingo JJS. Irritable bowel syndrome. *Med Clín (Engl Ed)*. 2022;158(2):76–81. https://doi.org/10.1016/j.medcle.2021.04.015.

Goldstein RS, Cash BD. Making a confident diagnosis of irritable bowel syndrome. *Gastroenterol Clin N Am*. 2021;50(3):547–563. https://doi.org/10.1016/j.gtc.2021.03.004.

Liu JJ, Brenner DM. Focus on pharmacotherapy for irritable bowel syndrome with constipation. *Gastroenterol Clin N Am*. 2021;50(3):639–653. https://doi.org/10.1016/j.gtc.2021.04.004.

McGowan A, Harer KN. Irritable bowel syndrome and eating disorders: a burgeoning concern in gastrointestinal clinics. *Gastroenterol Clin N Am*. 2021;50(3):595–610. https://doi.org/10.1016/j.gtc.2021.03.007.

Ringel Y. The gut microbiome in irritable bowel syndrome and other functional bowel disorders. *Gastroenterol Clin N Am*. 2017;46(1):91–101.

Sayuk GS. Medical therapies for diarrhea-predominant irritable bowel syndrome. *Gastroenterol Clin N Am*. 2021;50(3):611–637. https://doi.org/10.1016/j.gtc.2021.04.003.

Sharma A, Herekar A, Yan Y, et al. Dyssynergic defecation and other evacuation disorders. *Gastroenterol Clin N Am*. 2022;51(1):55–69. https://doi.org/10.1016/j.gtc.2021.10.004.

Videlock EJ, Chang L. Latest insights on the pathogenesis of irritable bowel syndrome. *Gastroenterol Clin N Am*. 2021;50(3):505–522. https://doi.org/10.1016/j.gtc.2021.04.002.

84

Anterior Cutaneous Nerve Entrapment

ICD-10 CODE G58.9

CLINICAL SYNDROME

Entrapment of the anterior cutaneous branch of the intercostal nerve is an uncommon and a frequently overlooked cause of anterior abdominal wall pain. The most common site of entrapment is the lateral border of the rectus abdominus muscle. Entrapment of the anterior cutaneous nerve by the lateral border of the rectus abdominus muscle produces a constellation of symptoms consisting of severe knife-like pain emanating from the anterior abdominal wall and associated with the physical finding of point tenderness over the affected anterior cutaneous nerve. The pain radiates medially to the linea alba but in almost all cases does not cross the midline. This nerve entrapment syndrome occurs most commonly in young women. The patient can often localize the source of pain accurately by pointing to the spot at which the anterior cutaneous branch of the affected intercostal nerve pierces the fascia of the abdominal wall at the lateral border of the rectus abdominus muscle (Fig. 84.1). At this point, the anterior cutaneous branch of the intercostal nerve turns sharply in an anterior direction to provide innervation to the anterior wall. The nerve passes through a firm fibrous ring as it pierces the fascia, and at this point the nerve becomes subject to entrapment (Fig. 84.2). The nerve is accompanied through the fascia by an epigastric artery and vein. There is the potential for small amounts of abdominal fat to herniate through this fascial ring and become incarcerated, which results in further entrapment of the nerve. The pain of anterior cutaneous nerve entrapment is moderate to severe in intensity.

FIG 84.1 The patient can often localize the source of pain from anterior cutaneous nerve entrapment accurately by pointing to the spot at which the anterior cutaneous branch of the affected intercostal nerve pierces the fascia of the abdominal wall at the lateral border of the rectus abdominus muscle.

SIGNS AND SYMPTOMS

As mentioned earlier, the patient often can point to the exact spot where the anterior cutaneous nerve is entrapped. Palpation of this point often elicits sudden, sharp, lancinating pain in the distribution of the affected anterior cutaneous nerve. Voluntary contraction of the abdominal muscles puts additional pressure on the nerve and may elicit the pain. The patient attempts to splint the affected nerve by keeping the thoracolumbar spine slightly flexed to avoid increasing tension on the abdominal musculature. Having the patient do a sit-up often reproduces the pain, as does a Valsalva maneuver. Patients suffering from anterior cutaneous nerve entrapment will also exhibit a positive Carnett's test when the patient is asked to tense his or her abdominal musculature which is indicative of abdominal wall pain rather than pain with an intraabdominal nidus (Fig. 84.3A and B).

TESTING

Plain radiographs are indicated in all patients who present with pain thought to be emanating from the lower costal cartilage and ribs to rule out occult bony pathology, including rib fracture and tumor. Radiographic evaluation of the

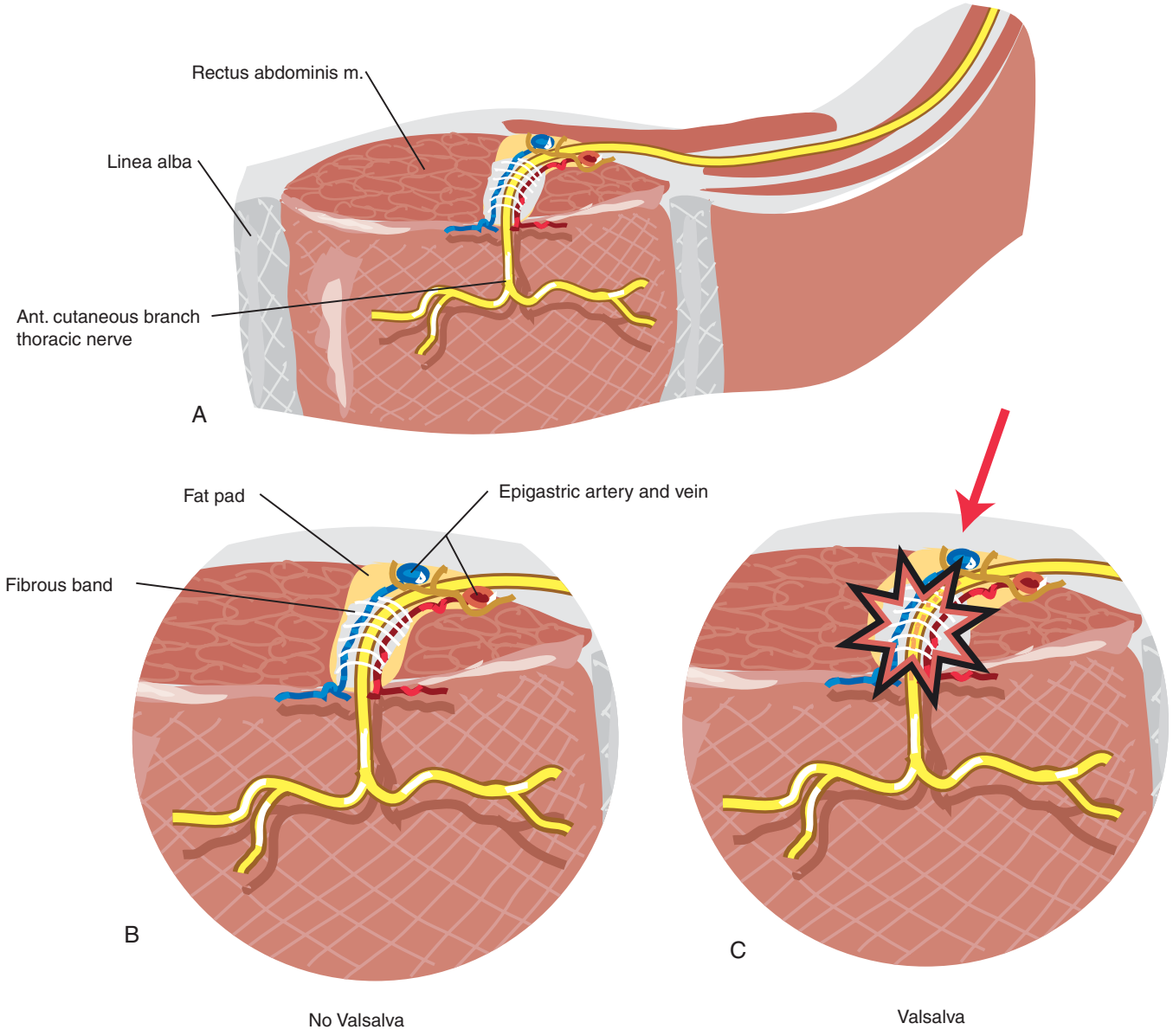

FIG 84.2 A–C, Contraction of the abdominal muscles or an increase in intraabdominal pressure puts additional traction on the anterior cutaneous branch of the intercostal nerve and may elicit sudden, sharp, lancinating pain in the distribution of the affected anterior cutaneous nerve. (From Waldman SD. *Pain management*. Philadelphia: WB Saunders; 2007.)

gallbladder is indicated if cholelithiasis is suspected. Based on the patient's clinical presentation, additional tests, including complete blood cell count, rectal examination with stool guaiac, erythrocyte sedimentation rate, and antinuclear antibody testing, may be indicated. Ultrasonography and computed tomography (CT) scan of the abdomen are indicated if intraabdominal pathology or occult mass is suspected. Injection of the anterior cutaneous nerve with or without ultrasound guidance at the point at which it pierces the fascia serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of anterior cutaneous nerve entrapment should consider ventral hernia, peptic ulcer disease, cholecystitis, intermittent bowel obstruction, renal calculi, angina, mesenteric vascular insufficiency, diabetic polyneuropathy, and pneumonia (Table 84.1). Rarely, collagen vascular diseases, including systemic lupus erythematosus and polyarteritis nodosa, may cause intermittent abdominal pain; porphyria also may cause intermittent abdominal pain.

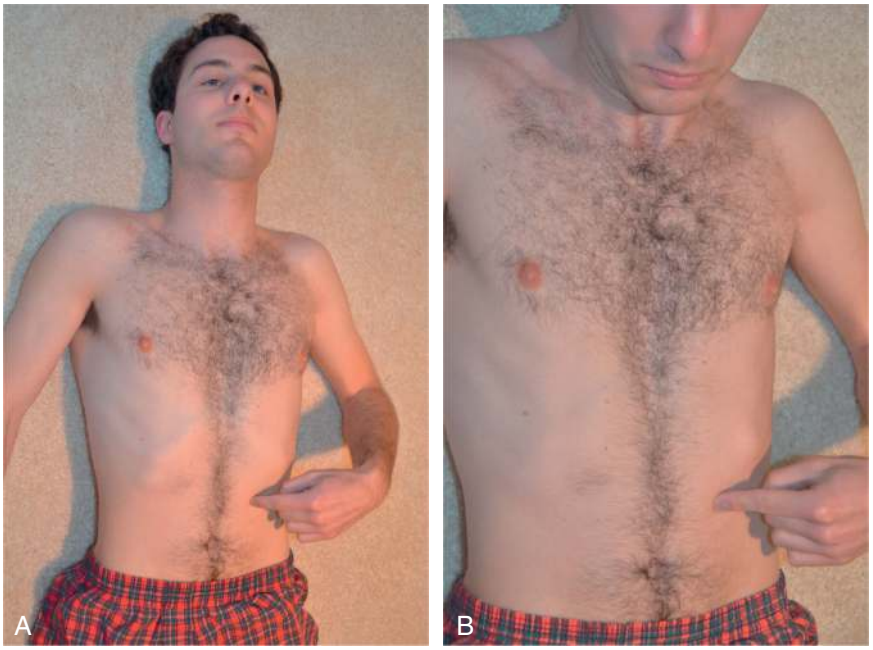

FIG 84.3 **A,** Carnett's sign. The patient is asked to completely relax his or her abdominal muscles and point with one finger to the most painful area. **B,** The patient is then asked to maximally tense his or her abdominal muscles. Carnett's test is positive if the localized pain increases at the previously identified painful area.

TABLE 84.1 The Differential Diagnosis of Anterior Cutaneous Nerve Entrapment Syndrome

Differential Diagnosis	Investigations and Characteristics
Anterior cutaneous nerve entrapment syndrome	Carnett's test, injection of local anesthetics
Thoracic lateral cutaneous nerve entrapment	History of previous surgery, clinical examination
Ilioinguinal or iliohypogastric nerve entrapment	History of previous groin surgery, clinical examination, injection of local anesthetics
Endometriosis	History of cyclic abdominal pain, laparoscopy
Myofascial pain syndrome	Clinical examination, myofascial strain
Slipping rib syndrome	Hypermobile, luxating 8th to 10th ribs, clinical examination
Diabetic radiculopathy	Paraspinal EMG, patient with diabetes mellitus
Abdominal wall tear	History of acute pain related to lifting or stretching, athletes
Abdominal wall or rectus sheath hematoma	Abdominal ultrasound or CT scan, after laparoscopy, after coughing in anticoagulated patient
Herpes zoster	History and clinical examination, dermatomal
Abdominal wall tumor (lipoma, desmoid, metastasis)	History and clinical examination, abdominal CT scan
Spinal nerve irritation	Referred pain by thoracic spine pathology
Hernia	Abdominal ultrasound, clinical examination
Traction symphysitis or pubalgia	Positive findings on MRI or scintigraphy, athletes

CT, Computed tomographic; *EMG*, electromyography; *MRI*, magnetic resonance imaging.

Because the pain of acute herpes zoster may precede the rash by 24–72 hours, the pain may be attributed erroneously to anterior cutaneous nerve entrapment.

TREATMENT

Initial treatment of the pain and functional disability associated with anterior cutaneous entrapment syndrome should include a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or the cyclooxygenase-2 (COX-2) inhibitors and physical therapy. Local application of heat and cold also may be beneficial. The repetitive movements that incite the syndrome should be avoided. For patients who do not respond to these treatment modalities, injection of the anterior cutaneous nerve at the point at which the nerve pierces the fascia with a local anesthetic and steroid may be a reasonable next step. Ultrasound guidance may improve the accuracy of needle placement and reduce complications (Figs. 84.4 and 84.5). If the symptoms of anterior cutaneous entrapment syndrome persist, surgical exploration and decompression of the anterior cutaneous nerve are indicated.

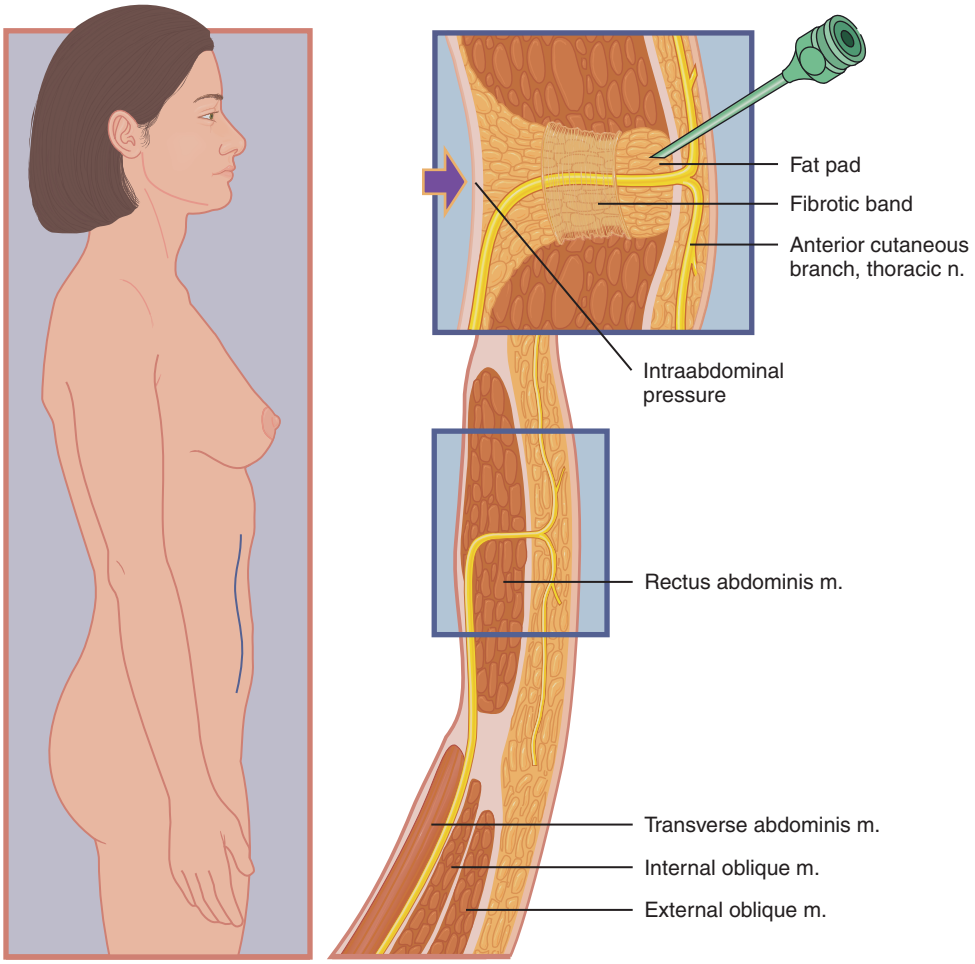

FIG 84.4 Injection technique for anterior cutaneous nerve entrapment syndrome. (From Waldman SD. *Atlas of pain management injection techniques*. 4th ed. Philadelphia: WB Saunders; 2017.)

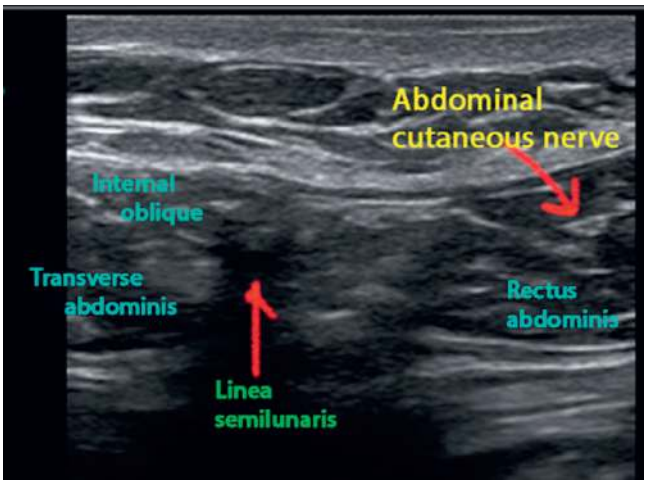

FIG 84.5 Ultrasound image demonstrating the relationship of the anterior cutaneous nerve to the rectus abdominus muscle and the peritoneum.

CLINICAL PEARLS

Patients suffering from pain emanating from anterior cutaneous nerve entrapment by the rectus abdominus muscle often attribute their pain symptoms to a gallbladder attack or ulcer disease. Reassurance is required, although it should be remembered that this musculoskeletal pain syndrome and intraabdominal pathology can coexist. The use of physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes this injection technique for anterior cutaneous nerve entrapment syndrome. Vigorous exercises should be avoided because they would exacerbate the patient's symptoms. Simple analgesics and NSAIDs may be used concurrently with the aforementioned injection technique. Radiographic evaluation for intraabdominal pathology is indicated in patients with anterior abdominal pain of unclear etiology.

SIDE EFFECTS AND COMPLICATIONS

The major complications associated with anterior cutaneous entrapment syndrome fall into two categories: (1) iatrogenically induced complications secondary to incorrect diagnosis and (2) failure of the clinician to recognize that a hernia coexists with the nerve entrapment until bowel ischemia occurs.

SUGGESTED READINGS

Cofano G, LaCourt S, Nguyen S. Conservative management of anterior cutaneous nerve entrapment syndrome: a case report. *J Bodyw Mov Ther*. 2021;26:141–146. https://doi.org/10.1016/j.jbmt.2020.12.012.

García-Carmona JA, Sánchez-Lucas J. Role of the Carnett sign in the diagnosis of anterior cutaneous nerve entrapment syndrome

misdiagnosed as somatic symptom disorder: a case report. *Neurología (English Edition)*. 2021;36(2):179–180. https://doi.org/10.1016/j.nrleng.2020.02.009.

Hall MW, Sowden DS, Gravestock N, et al. Abdominal wall tenderness test. *Lancet*. 1991;337(8757):1606–1607.

Kanakarajan S, High K, Nagaraja R. Chronic abdominal wall pain and ultrasound-guided abdominal cutaneous nerve infiltration: a case series. *Pain Med*. 2011;12(3):382–386.

Kuan L-C, Li Y-T, Chen F-M, et al. Efficacy of treating abdominal wall pain by local injection. *Taiwan J Obstet Gynecol*. 2006;45:239–243.

Mol FMU, Jansen CH, van Dijk W, et al. Factors predicting outcome after anterior neurectomy in patients with chronic abdominal pain due to anterior cutaneous nerve entrapment syndrome (ACNES). *Surgery*. 2019;165(2):417–422. https://doi.org/10.1016/j.surg.2018.08.011.

Mooney DP. The technique of cutaneous neurectomy for anterior cutaneous nerve entrapment syndrome. *J Pediatr Surg*. 2020;55(6):1142–1144. https://doi.org/10.1016/j.jpedsurg.2020.02.013.

Srinivasan R, Greenbaum DS. Chronic abdominal wall pain: a frequently overlooked problem. Practical approach to diagnosis and management. *Am J Gastroenterol*. 2002;97:824–830.

Diverticulitis

ICD-10 CODE K57.32

THE CLINICAL SYNDROME

Diverticulitis is a common cause of acute abdominal pain in western and industrialized countries. It is found more commonly in women, the disease occurs more commonly after the fourth decade. Diverticulitis occurs when small herniations of the colonic mucosa and submucosa, known as diverticula, become inflamed or tear (Fig. 85.1). It is estimated that approximately 75% of patients will have diverticula by the age of 80, as there is age-related weakening of the abdominal wall in areas of insertion of the vasa recta. Decreased bowel motility of senescence may also play a role in increasing intracolonic pressure, as may change in the microbiome of the gastrointestinal tract.

Patients with diverticulitis will develop abdominal pain that is usually located in the left lower quadrant, although there is an increased incidence of right-sided diverticular disease in Asians (Fig. 85.2). Constipation is present approximately 50% of the time, with diarrhea occurring in 25% of patients suffering from acute diverticulitis. Abdominal tenderness is invariably present as are fever and chills. The pain of diverticulitis is proportional to the extent of inflammation, with the pain ranging from mild, intermittent pain to severe, unremitting pain with frank signs of peritonitis including rebound tenderness. Lower gastrointestinal bleeding, which may be significant, may also be present.

Factors that increase the risk of developing diverticulitis include advancing age, low-fiber high-fat diet, obesity, smoking, and the use of corticosteroids and nonsteroidal antiinflammatory agents. Diets high in vitamin D and the use of statins and calcium channel blocker may exert a protective effect. Mild cases of diverticulitis are managed conservatively, but approximately 25% of patients with acute diverticulitis will develop complications that may include abscess formation, stenosis, bowel obstruction, peritonitis, and sepsis.

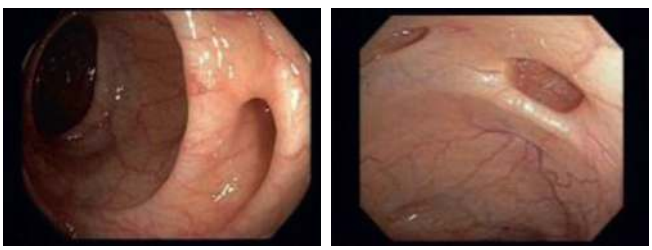

FIG 85.1 Diverticulosis on colonoscopy. (From Feuerstein JD, Falchuk KR. Diverticulosis and diverticulitis. *Mayo Clin Proc.* 2016;91(8):1094–1104.)

SIGNS AND SYMPTOMS

Left-sided abdominal pain is present in most patients with acute diverticulitis, although patients of Asian descent have an increased incidence of right-sided diverticulitis, which may mimic acute appendicitis. The pain of acute diverticulitis is associated with anorexia and a change in bowel habits and gastrointestinal symptoms of constipation, diarrhea, bloating, flatulence, and nausea and vomiting. A small percentage of patients will complain of urinary urgency and frequency

FIG 85.2 The patient with acute diverticulitis will suffer from left-sided abdominal pain associated with a change in bowel habits. Fever and chills are often present.

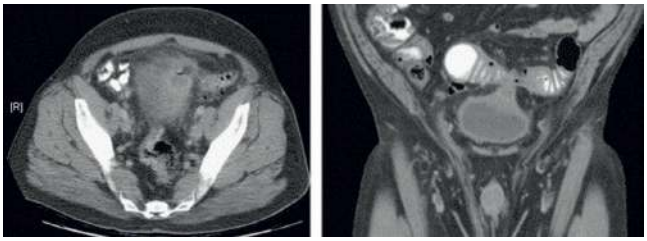

FIG 85.3 Computed tomography scan (anterior, posterior, and coronal views) showing colovesical fistula. (From Chapman JR, Wolff BG. The management of complicated diverticulitis. *Adv Surg*. 2006;40:285–297.)

secondary to irritation of the adjacent urinary tract. Often the patient will flex the hip on the affected side owing to irritation of the psoas muscle. Mild diverticulitis may produce minimal constitutional symptoms, but if the disease progresses, fever and chills may be pronounced.

On physical examination, the extent of abdominal findings will be in proportion to the extent of the diverticulitis. Small microperforations of left-sided diverticula will produce diffuse left lower quadrant pain with minimal peritoneal findings. With more severe diverticulitis, the pain will become more localized to the left lower quadrant and pelvis with rebound tenderness a prominent physical finding. If a peridiverticular abscess or phlegmon forms, a tender, palpable mass may be identified. The abdomen may be distended and tympanic to percussion, with bowel sounds diminished or absent. If a fistula into the genitourinary tract forms, fecaluria or pneumaturia may be present with colovaginal fistulas occurring in females (Fig. 85.3).

TESTING

Computed tomography of the abdomen and pelvis has replaced barium enema as the preferred imaging modality to diagnose diverticulitis because not only can it diagnose the disease with a high degree of specificity and sensitivity, but can also identify complications as well as pericolonic abscess and other pathologic processes that may mimic the diverticulitis. The Hinchey classification can help define the severity of complicated diverticulitis and guide treatment (Table 85.1 and Fig. 85.4). Colonoscopy can also diagnose diverticulitis and post-diverticulitis complications such as stenosis, but cannot identify potential serious complications as intraabdominal or retroperitoneal abscess or fecal peritonitis (Fig. 85.5). Based on the patient's clinical presentation, additional testing, including a complete blood count to identify leukocytosis with a left shift, urinalysis, and serum chemistries are indicated. Blood cultures should be obtained if fever is present. A pregnancy test must be obtained on all females of child-bearing age to rule out ectopic pregnancy. If abscess formation is suspected, imaging of adjacent structures (e.g., hip, bladder) should be obtained sooner than later.

TABLE 85.1 The Hinchey Classification Of Diverticulitis

Stage I	**Pericolic abscess or phlegmon**
Stage II	**Pelvic, intraabdominal, or retroperitoneal abscess**
Stage III	**Generalized purulent peritonitis**
Stage IV	**Generalized fecal peritonitis**

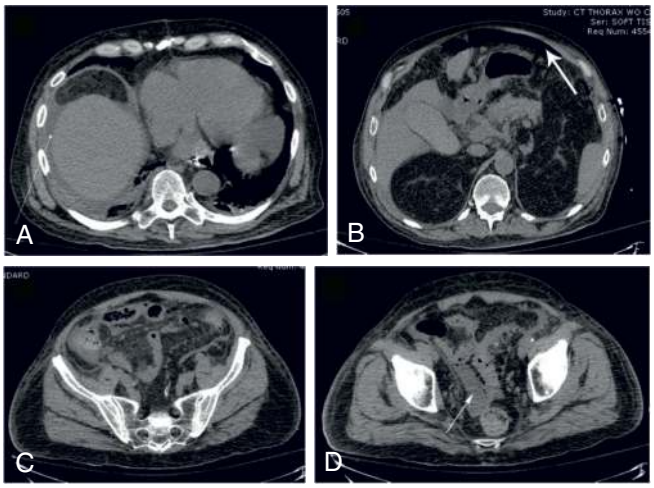

FIG 85.4 **A,** Modified Hinchey stage III diverticulitis. *Arrow* points to free fluid. **B,** Modified Hinchey stage III diverticulitis. *Arrow* points to free air. **C,** Modified Hinchey stage III diverticulitis. Demonstrates intraabdominal free fluid. **D,** Modified Hinchey stage III diverticulitis. *Arrow* points to pelvic fluid. (From Hall J, Hammerich K, Roberts P. New paradigms in the management of diverticular disease. *Curr Probl Surg*. 2010; 47(9):680–735.)

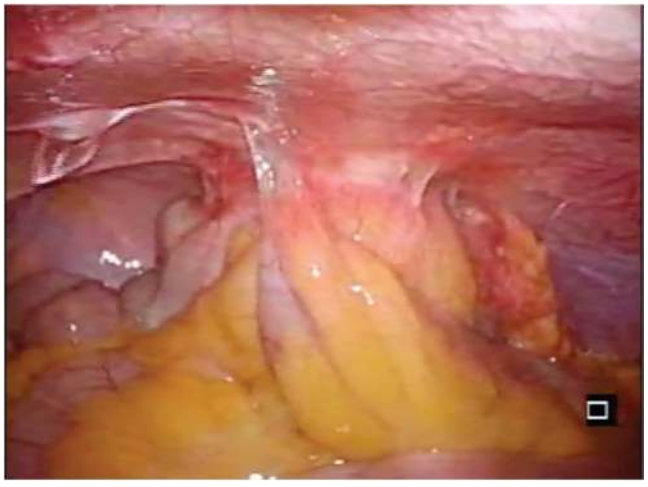

FIG 85.5 Laparoscopic findings demonstrating post-diverticulitis stenosis. Note that the stenotic area of the ascending colon had adhered to the abdominal wall. (From Yoshida S, Hiyama K, Kirino I, et al. Ascending colon stenosis caused by repeated diverticulitis that clinically mimicked advanced colon cancer: a case report. *Int J Surg Case Rep*. 2022;95:107184.)

BOX 85.1 Diseases That May Mimic Acute Diverticulitis

Appendicitis
Inflammatory bowel disease
Irritable bowel syndrome
Colorectal malignancies
Acute gastroenteritis
Ectopic pregnancy
Ischemic colitis
Abdominal angina
Tubo-ovarian abscess
Pelvic inflammatory disease
Ureteral calculi
Volvulus
Ovarian torsion
Endometriosis

DIFFERENTIAL DIAGNOSIS

Most causes of acute abdominal pain can mimic the diverticulitis and are listed in Box 85.1. Most commonly, acute gastroenteritis, inflammatory bowel disease, irritable bowel syndrome, ectopic pregnancy, ischemic colitis, and mesenteric artery ischemia are misdiagnosed as diverticulitis. Acute appendicitis can also mimic right-sided diverticulitis.

TREATMENT

The treatment of diverticulitis is based on the severity of the disease and must be individualized to the specific patient. In uncomplicated diverticulitis presenting with mild symptoms, patients are treated with a clear liquid diet for 7–10 days and oral broad-spectrum antibiotics, such as ciprofloxacin and metronidazole, that cover anaerobic microorganisms. Opioids should be avoided, as they decrease bowel motility.

For patients with more severe symptomatology, including fever and chills, the immediate use of broad-spectrum intravenous antibiotics and the drainage of any abscess are mandatory (Fig. 85.6). If the abscess cannot be drained percutaneously, or if significant perforation, fistula, or bowel obstruction is present, emergent surgical treatment consisting of primary bowel resection is indicated. Occasionally, a diverting colostomy will be required to allow resolution of severe diverticulitis.

COMPLICATIONS AND PITFALLS

Many other causes of an acute abdomen can mimic the presentation of diverticulitis. The failure to correctly identify the source of the patient's abdominal symptoms can lead to significant morbidity and mortality. It should be remembered that right-sided diverticulitis is common in patients of Asian descent and may present identically to acute appendicitis. Early identification and drainage of abscess is essential to avoid more serious complications when treating diverticulitis.

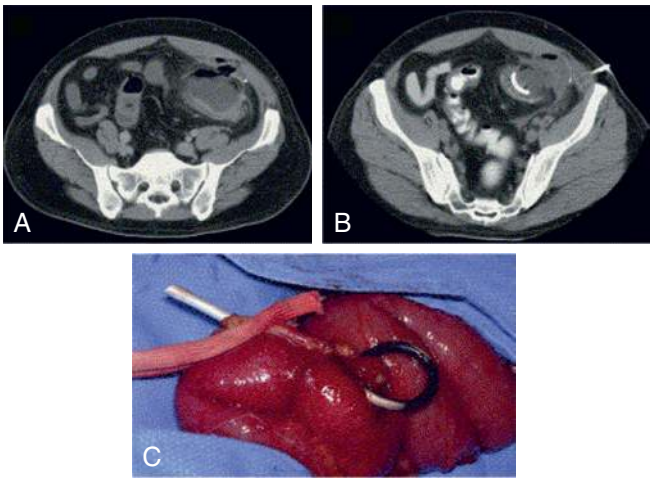

FIG 85.6 Diverticular abscess. **A,** Diagnostic computed tomography. **B,** Percutaneous drain placed. **C,** Intraoperative resection of bowel with percutaneous drain in place. (From Chapman JR, Wolff BG. The management of complicated diverticulitis. *Adv Surg*. 2006;40:285–297.)

CLINICAL PEARLS

Diverticulitis is a common cause of acute abdominal pain. Its clinical presentation can range from a mild self-limited disease to a life-threatening illness. Because of the number of other diseases that mimic diverticulitis, diagnosis and treatment may be delayed, leading to increased morbidity and, rarely, mortality. Early implementation of broad-spectrum antibiotics and identification and drainage of pericolonic abscess are mandatory to decrease more serve complications.

SUGGESTED READINGS

Abet E, Bawjeski S, Planche L, et al. Evaluation of a treatment pathway for the management of uncomplicated diverticulitis. *J Visc Surg*. 2022. https://doi.org/10.1016/j.jviscsurg.2021.10.005.

Ferrara F, Bollo J, Vanni LV, et al. Diagnosis and management of right colonic diverticular disease: a review. *Cir Esp*. 2016; 94(10):553–559.

Feuerstein JD, Falchuk KR. Diverticulosis and diverticulitis. *Mayo Clin Proc*. 2016;91(8):1094–1104.

Kazemi SN, Raoufi M, Samsami M, et al. A case-report on diverticulitis misdiagnosed as tubo-ovarian abscess. *Ann Med Surg*. 2021;72:103049. https://doi.org/10.1016/j.amsu.2021.103049.

Khan A, Hawkins AT. Challenging surgical dogma: controversies in diverticulitis. *Surg Clin N Am*. 2021;101(6):967–980. https://doi.org/10.1016/j.suc.2021.05.024.

Morgan A, Yglesias B, DeVito P. A case report: colonoscopy-induced perforated diverticulitis. *Int J Surg Open*. 2022;42:100466. https://doi.org/10.1016/j.ijso.2022.100466.

Roig JV, Salvador A, Frasson M, et al. Surgical treatment of acute diverticulitis. A retrospective multicentre study. *Cir Esp*. 2016; 94(10):569–577.

Sahli H, Azhar N, Lydrup N-L, et al. Risk factors for recurrence and complications in acute uncomplicated diverticulitis: a retrospective

cohort study. *Int J Surg Open*. 2022;43:100471. https://doi.org/10.1016/j.ijso.2022.100471.

Tan JPL, Barazanchi AWH, Singh PP, et al. Predictors of acute diverticulitis severity: a systematic review. *Int J Surg*. 2016;26:43–52.

Yoshida S, Hiyama K, Kirino I, et al. Ascending colon stenosis caused by repeated diverticulitis that clinically mimicked advanced colon cancer: a case report. *Int J Sur Case Rep*. 2022;95:107184. https://doi.org/10.1016/j.ijscr.2022.107184.

Spigelian Hernia

ICD-10 CODE B02.22

THE CLINICAL SYNDROME

Spigelian hernia is a rare hernia of the anterior abdominal wall, with inguinal, umbilical, and femoral hernias occurring much more commonly (Fig. 86.1). Also known as lateral ventral hernias, Spigelian hernia occurs anywhere the Spigelian line, the area just lateral to the rectus abdominis muscle when the transverse abdominis muscle transitions to aponeurosis. It is believed that the crescent moon-shaped and variable width of the Spigelian aponeurosis along its crainocaudal path predisposes the formation of hernia at this anatomic location. Because the more narrow cranial portion of the Spigelian aponeurosis is reinforced by fibers of the internal oblique and transverse abdominis muscles, Spigelian hernias tend to occur more commonly along the more caudad portion of the aponeurosis below the umbilicus (Fig. 86.2). The hernia sac of the Spigelian hernia almost always comprised peritoneum and preperitoneal fat with small bowel or omentum occasionally present (Fig. 86.3). Rarely, portions of organs or other intraabdominal contents and embryologic remnants may be present and contribute to the patient's symptoms (Fig. 86.4). Spigelian hernia formation has also been associated with conditions which lead to stretching of

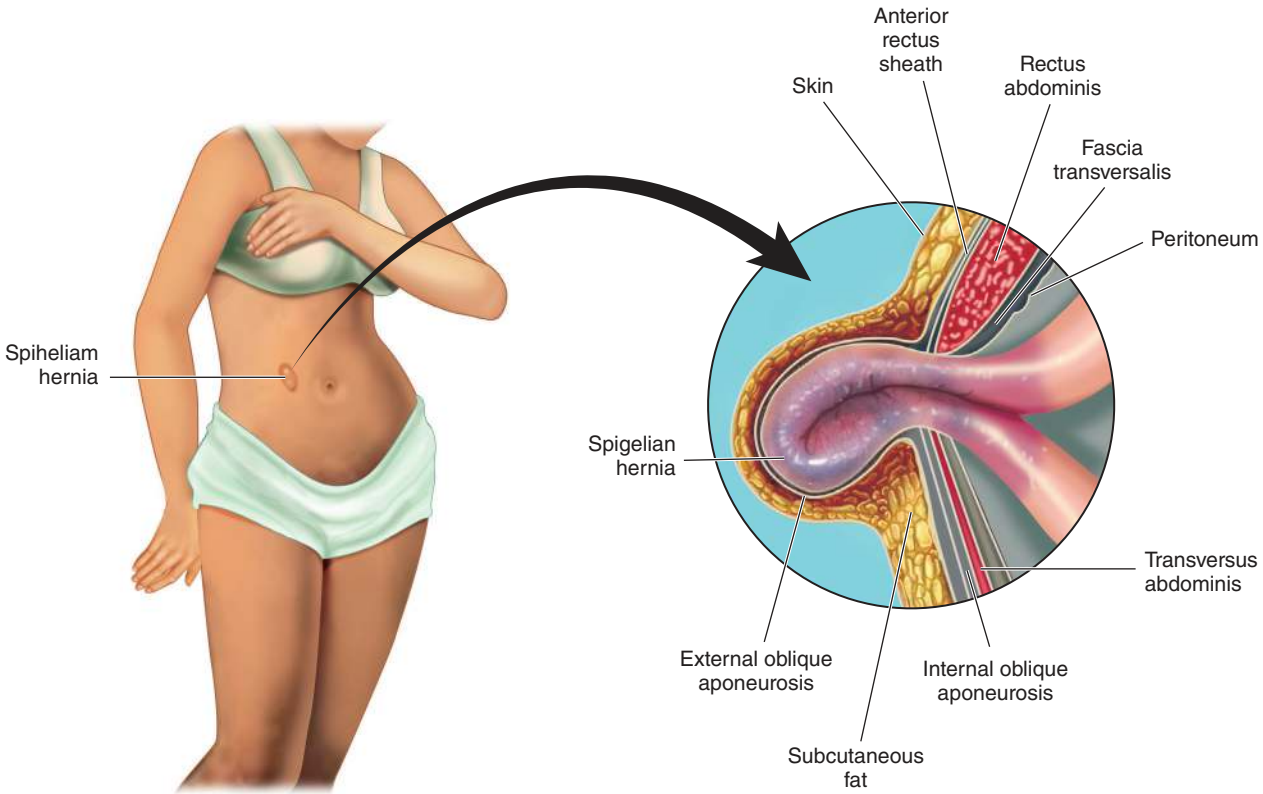

FIG 86.1 Spigelian hernia is a rare hernia of the anterior abdominal wall, with inguinal, umbilical, epigastric, and femoral hernias occurring much more commonly.

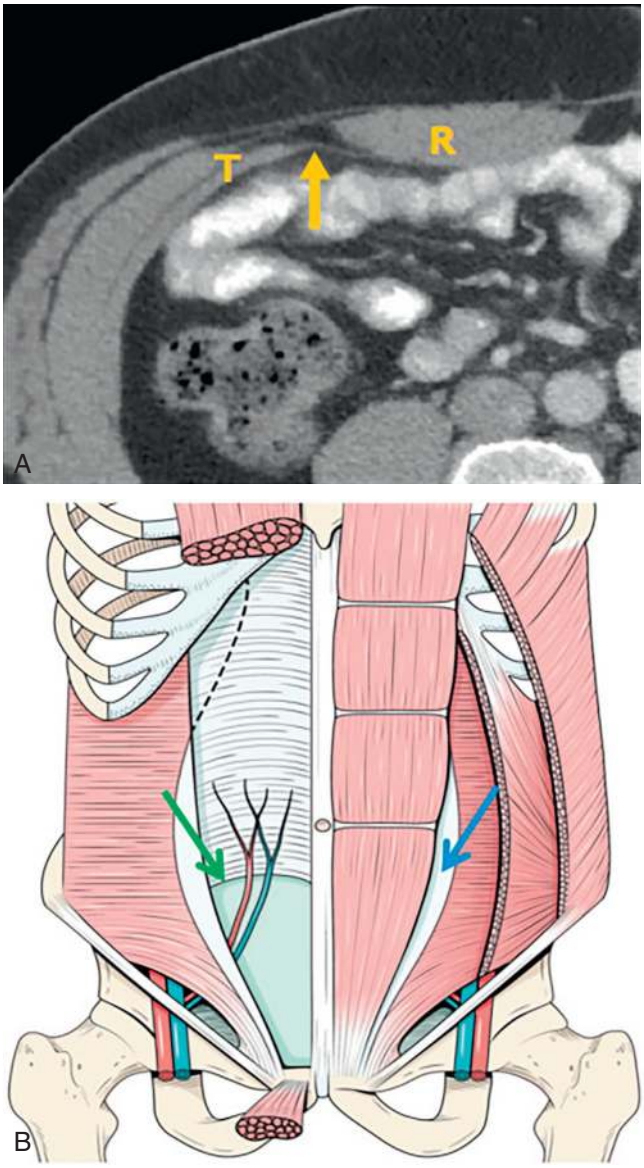

FIG 86.2 **A,** Axial computed tomography scan image of the anterior abdominal wall. *Yellow arrow*, Spigelian aponeurosis; *R*, rectus abdominis muscle; *T*, transversus abdominis muscle. **B,** Coronal artist drawing of the Spigelian Aponeurosis shown by the *blue arrow* and semicircular line (arcuate line or line of Douglas) by the *green arrow*. (From Azar SF, Jamadar DA, Wasnik AP, et al. MDCT imaging in Spigelian hernia, clinical, and surgical implications. *Clin Imaging*. 2021;74:131–138.)

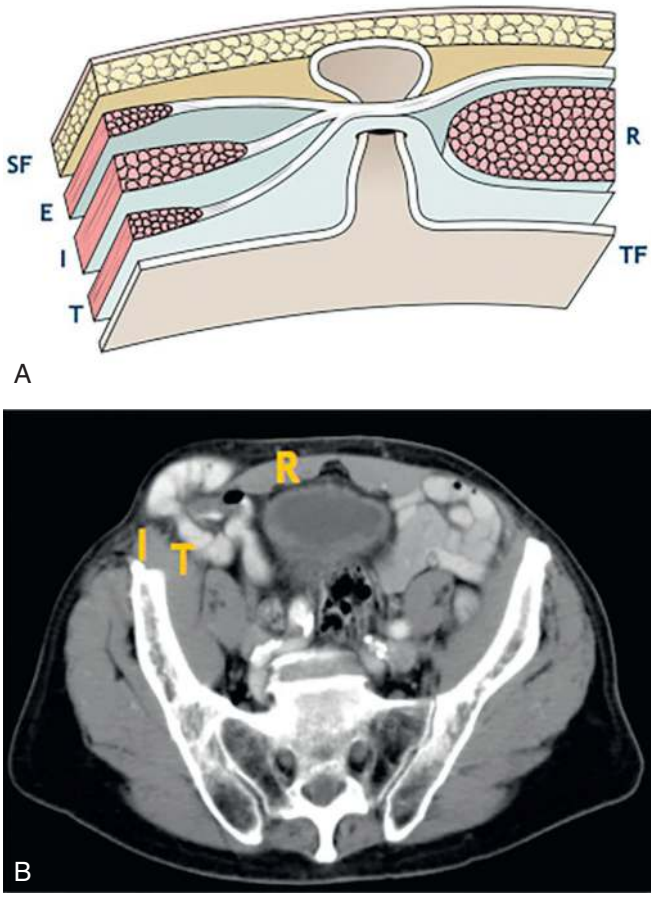

FIG 86.3 **A,** Artist rendition of **(B)**. Axial computed tomography scan image of 63 year-old-male with right-sided subcutaneous Spigelian hernia, below the level of the semicircular line (arcuate line or line of Douglas) containing uncomplicated small bowel loops. *R*, Rectus abdominis muscle; *T*, transversus abdominis muscle; *I*, internal oblique muscle; *E*, external oblique muscle; *SF*, subcutaneous fat; *TF*, transversalis fascia. (From Azar SF, Jamadar DA, Wasnik AP, et al. MDCT imaging in Spigelian hernia, clinical, and surgical implications. *Clin Imaging*. 2021;74:131–138.)

the abdominal wall including obesity, chronic cough, sit-ups, pregnancy, and ascites. Previous surgical or traumatic damage to the nerves of the anterolateral abdominal wall may cause weakness of the Spigelian aponeurosis and predispose the patient to the development of Spigelian hernia.

SIGNS AND SYMPTOMS

The majority of patients suffering from Spigelian hernia are asymptomatic and the diagnosis is only made when the patient or clinician identify a visible or palpable mass along the aponeurosis or in the rare instance when the contents of the hernia sac incarcerate and subsequently strangulate. Pain associated with Spigelian hernia is variable and has been described as dull, with neuritic, shock-like pain occasionally present if the hernia sac entraps a nerve of the abdominal wall (Fig. 86.5). If a visible mass is present, it should be more pronounced with coughing, straining or Valsalva, disappearing when the patient assumes the supine position unless the hernia sac is incarcerated. Identification of the hernia defect by palpation is difficult due to the small size of the abdominal wall defect. Some patients suffering from Spigelian hernia will experience point tenderness to palpation of the hernia defect and may notice allodynia just medial to the defect due to repeated mechanical irritation of adjacent nerves. The phenomenon is known as the Valleix sign.

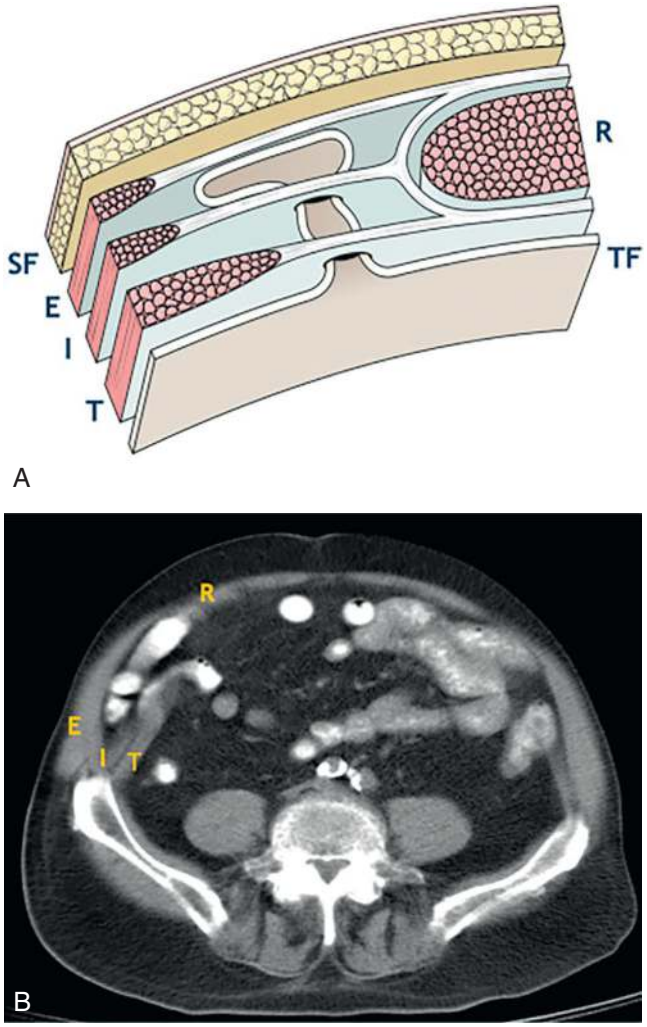

FIG 86.4 Axial computed tomography scan image of a 60-year-old male with a right-sided interparietal-type Spigelian hernia. *R*, Rectus abdominis muscle; *T*, transversus abdominis muscle; *I*, internal oblique muscle; *E*, external oblique muscle; *SF*, subcutaneous fat; *TF*, transversalis fascia. (From Azar SF, Jamadar DA, Wasnik AP, et al. MDCT imaging in Spigelian hernia, clinical, and surgical implications. *Clin Imaging*. 2021;74:131–138.)

TESTING

Ultrasonography is considered the diagnostic modality of choice in patients suspected of suffering from Spigelian hernia. It has the advantages of being noninvasive, radiation-free, and having the capability to easily identify hernia defects, the presence and content of associated hernia sacs as well as identifying other occult pathology of the abdominal wall and abdomen (Fig. 86.6). Computerized tomography is a reasonable additional diagnostic modality to confirm the diagnosis of Spigelian hernia and identify other occult pathology of the abdominal wall and abdomen (Figs. 86.7). Computerized tomography will also help identify other types of abdominal hernias (Fig. 86.8). Rarely, surgical exploration is required to diagnose Spigelian hernia.

DIFFERENTIAL DIAGNOSIS

Given the nonspecific clinical presentation of Spigelian hernia, the diagnosis becomes one of exclusion absent obvious clinical and/or confirmatory medical imaging findings. Appendicitis, ruptured ovarian cysts, ectopic pregnancy, biliary colic, pregnancy, and pancreatitis are frequently the initial working diagnosis in patients subsequently found to be suffering from Spigelian hernia. Unusual tumors of the abdominal wall such as desmoid tumors must be considered as should spontaneous hematoma of the abdominal wall in those patients taking anticoagulants.

TREATMENT

Given that risk of incarceration and strangulation remains an everpresent possibility, surgical repair should be considered in almost all patients suffering from Spigelian hernia. Open as well as laparoscopic procedures have been described, with most surgeons recommending the use of mesh to repair the defect and decrease the incidence of recurrence (Figs. 86.9 and 86.10). Robotic-assisted techniques have also been described.

COMPLICATIONS AND PITFALLS

Spigelian hernias tend to remain asymptomatic until the hernia becomes large enough to create a visible mass or the hernia incarcerates or becomes strangulated. Because Spigelian hernia mimics the presentation of many diseases that cause abdominal and pelvic pain, the clinician must maintain a high index of suspicion to avoid diagnostic misadventures. A failure to misdiagnose the strangulated Spigelian hernia can lead to significant mortality and morbidity.

CLINICAL PEARLS

Given the nonspecific clinical presentation of Spigelian hernia, the diagnosis becomes one of exclusion absent obvious clinical and/or confirmatory medical imaging findings. Appendicitis, ruptured ovarian cysts, ectopic pregnancy, biliary colic, pregnancy, and pancreatitis are frequently the initial working diagnosis in patients subsequently found to be suffering from Spigelian hernia. Unusual tumors of the abdominal wall such as desmoid tumors must be considered as should spontaneous hematoma of the abdominal wall in those patients taking anticoagulants.

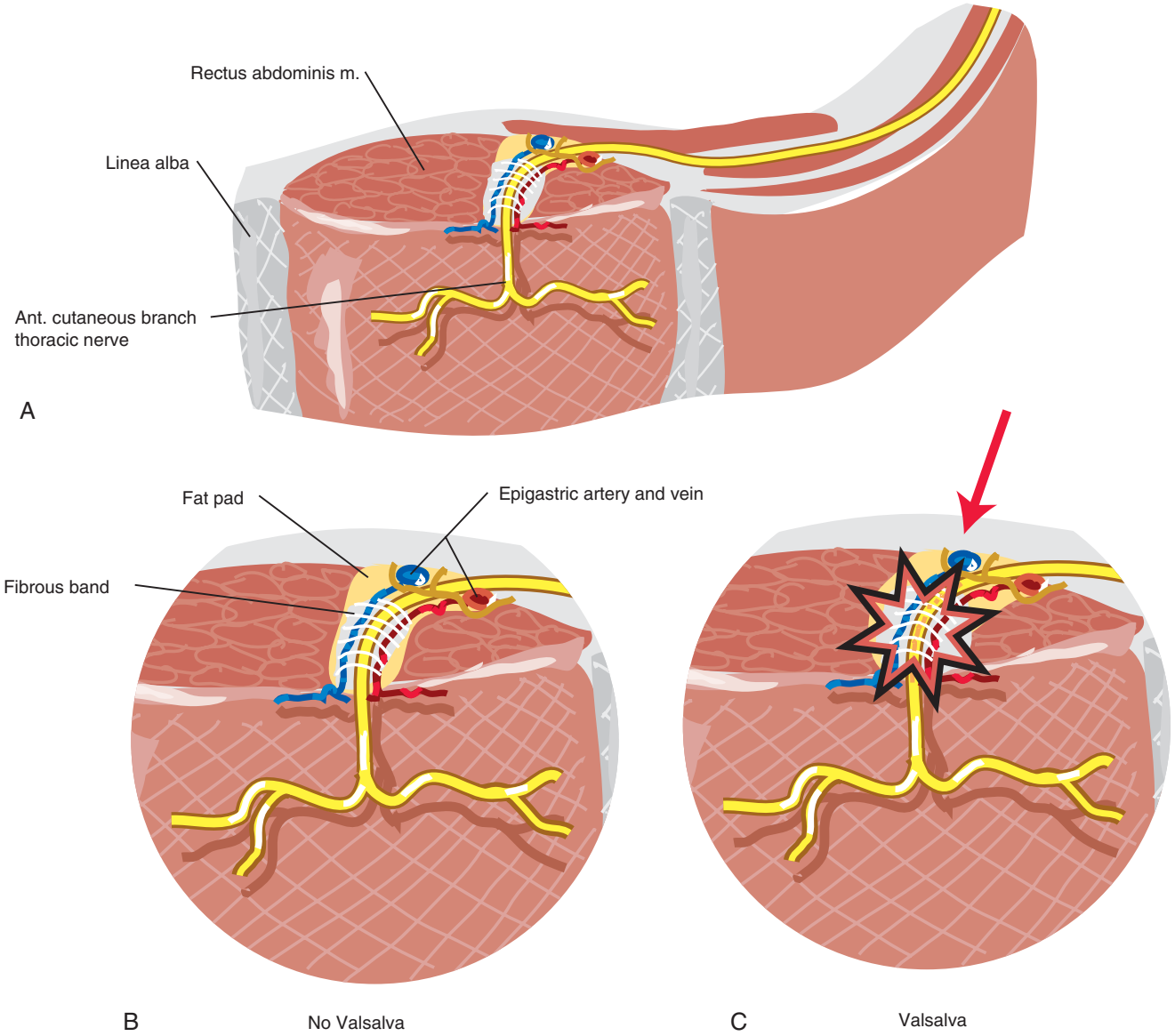

FIG 86.5 (A, B, C) Contraction of the abdominal muscles or an increase in intraabdominal pressure results in additional traction being placed on the anterior cutaneous branch of the intercostal nerve.

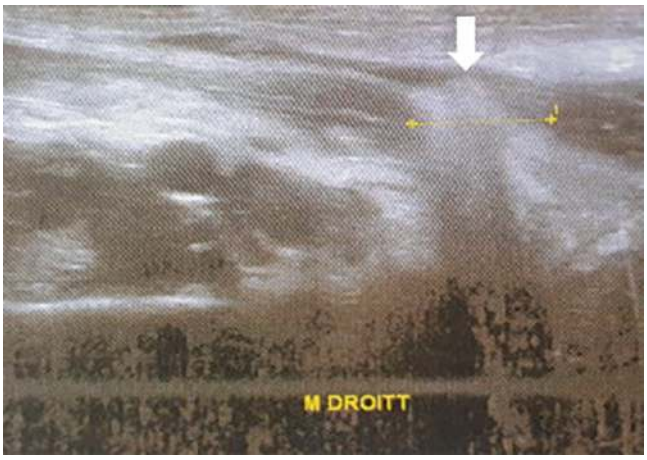

FIG 86.6 Diastasis between the rectus abdominis muscle and the ipsilateral oblique and transverse muscles with evidence of Spigelian hernia (*arrow*). Use Figure 1, https://ars.els-cdn.com/content/image/1-s2.0-S2213576621003201-gr3.jpg (From Thamri F, Houidi S, Zouaoui A, et al. Traumatic Spigelian hernia in a child. *J Pediatr Surg Case Rep*. 2021;75:102099.)

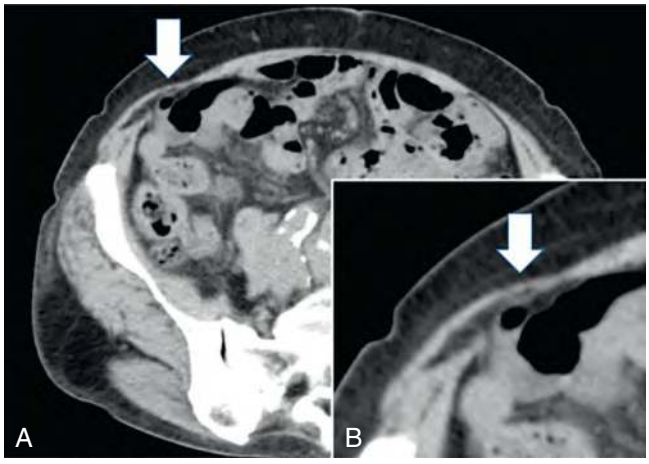

FIG 86.7 Computed tomography of Spigelian hernia. **A,** A defect in the abdominal wall at the lateral edge of the right rectus abdominis muscle and prolapse of fatty tissue (*arrow*). **B,** Enlarged view of Spigelian hernia (*arrow*). (From Takayama J, Okada S, Nakatani K, et al. The advantage of laparoscopic surgery in the treatment of Spigelian hernia: a report of two cases. *Int J Surg Case Rep*. 2021;82:105903. ISSN 2210-2612, https://doi.org/10.1016/j.ijscr.2021.105903.)

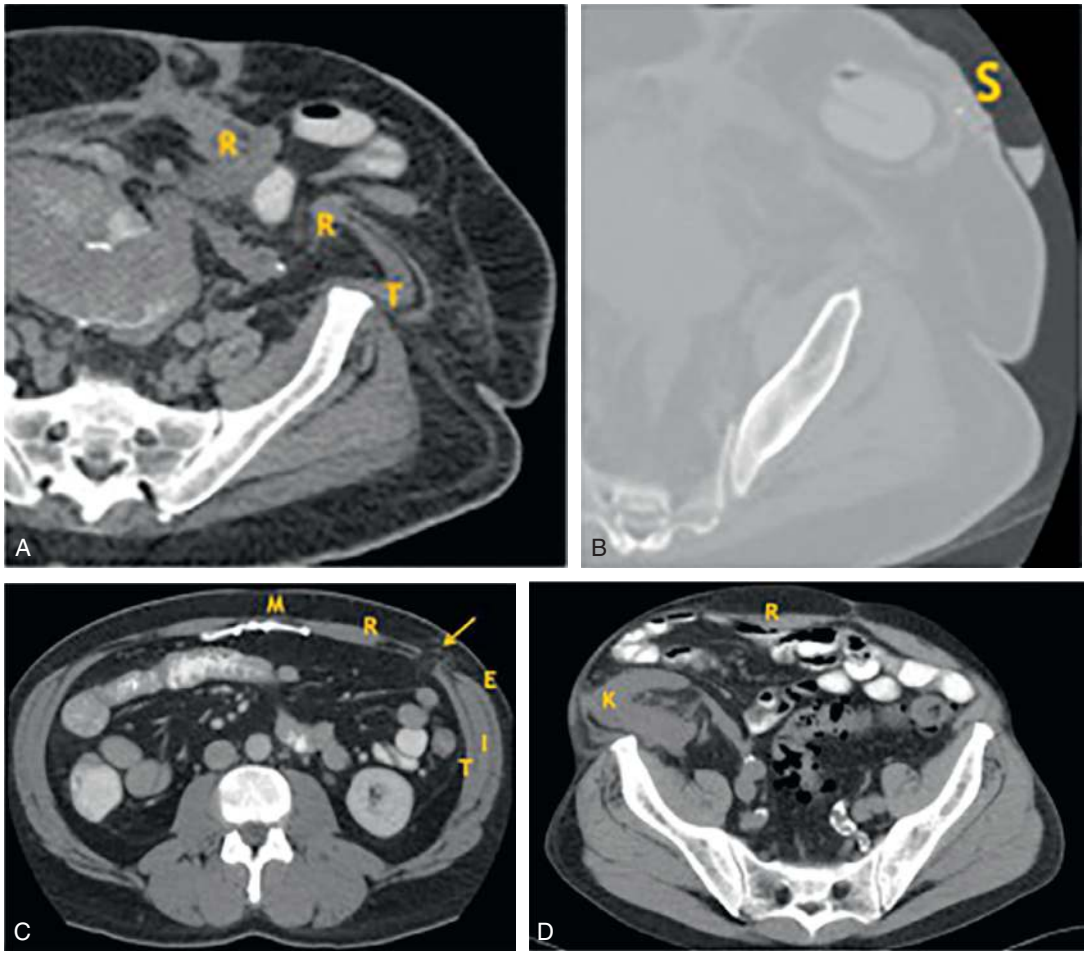

FIG 86.8 Axial CT scan images of incisional hernias. **A,** Parastomal hernia, stoma through rectus muscle, with intact Spigelian aponeurosis. **B,** Stoma sac with an air-fluid level, best seen on lung window. **C,** Laparoscopic trocar insertion through Spigelian aponeurosis for umbilical herniorrhaphy with midline mesh. **D,** Incisional hernia at kidney transplantation incision along Spigelian aponeurosis. *R*, Rectus abdominis muscle; *T*, transversus abdominis muscle; *I*, internal oblique muscle; *E*, external oblique muscle; *M*, mesh material; *S*, stoma bag; *K*, kidney transplant; *yellow arrow*, laparoscopic port hernia defect. (From Azar SF, Jamadar DA, Wasnik AP, et al. MDCT imaging in Spigelian hernia, clinical, and surgical implications. *Clin Imaging*. 2021;74:131–138.)

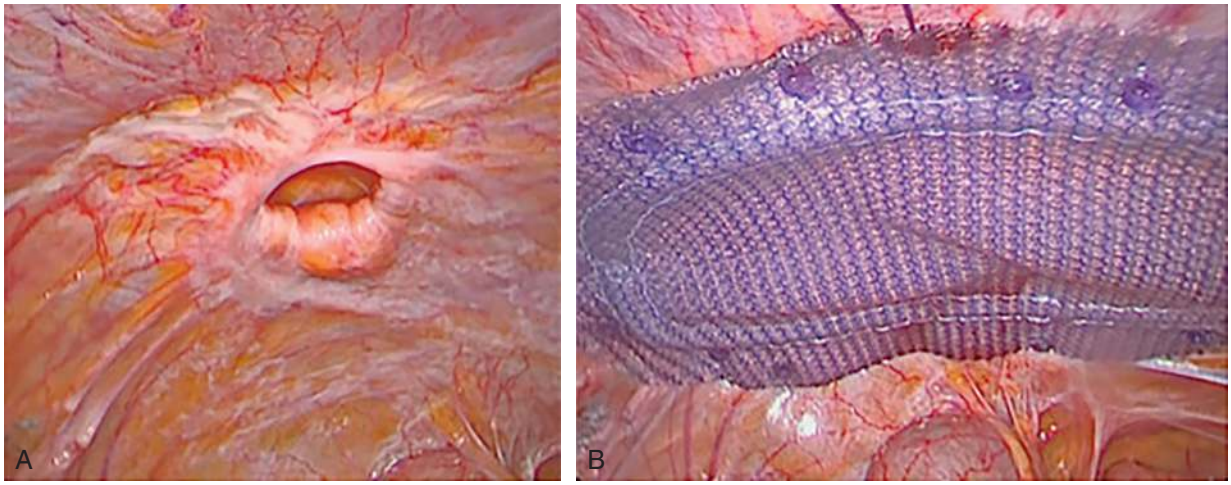

FIG 86.9 Spigelian hernia. Laparoscopic repair. **A,** Through a laparoscope, a 12-mm round defect is seen at the lateral edge of the right rectus abdominis muscle. **B,** The defect is fully covered with the BARD VENTRIO ST. (From Takayama J, Okada S, Nakatani K, et al. The advantage of laparoscopic surgery in the treatment of Spigelian hernia: a report of two cases. *Int J Surg Case Rep.* 2021;82:105903. ISSN 2210-2612, https://doi.org/10.1016/j.ijscr.2021.105903.)

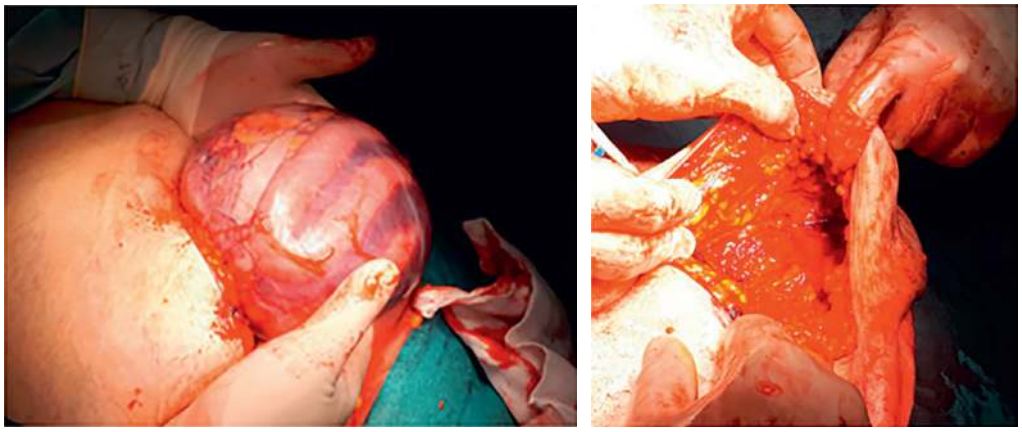

FIG 86.10 Intraoperative view of the hernia sac including several viable small loops and fluid (*upper part)* and abdominal wall showing the defect (Spigelian hernia). (From Di Furia M. Giant Spigelian hernia presenting as small bowel obstruction: case report and review of literature. *Int J Surg Case Rep*. 2019;63:118–121.)

SUGGESTED READINGS

Cervantes BYH, Lambert DM, Lopez MR, et al. Giant intraparietal inguinal hernia misdiagnosed as Spigelian hernia in an old woman. *Pan Afr Med J*. 2020;36:117. https://doi.org/10.11604/pamj.2020.36.117.21652.

Di Furia M. Giant Spigelian hernia presenting as small bowel obstruction: case report and review of literature. *Int J Surg Case Rep*. 2019;63:118–121. https://doi.org/10.1016/j.ijscr.2019.09.026.

Griffin AS, Schaefer NR, Jeyarajan E, Cross T. Concurrent Spigelian hernia and falciform ligament hernia in a 67-year-old female. *Int J Surg Case Rep*. 2015;13:27–29. https://doi.org/10.1016/j.ijscr.2015.05.035. ISSN 2210–2612.

Khadka P, Dhakal SKS. Case report of ovary and fallopian tube as content of a Spigelian hernia – a rare entity. *Int J Surg Case Rep*. 2017;31:206–208. https://doi.org/10.1016/j.ijscr.2017.01.015.

Rankin A, Kostusiak M, Sokker A. Spigelian hernia: case series and review of the literature. *Visc Med*. 2019;35:133–136. https://doi.org/10.1159/000494280.

Skandalakis PN, Zoras O, Skandalakis JE, Mirilas P. Spigelian hernia: surgical anatomy, embryology, and technique of repair. *Am Surg*. 2006;72(1):42–48.

Takayama J, Okada S, Nakatani K, et al. The advantage of laparoscopic surgery in the treatment of Spigelian hernia: a report of two cases. *Int J Surg Case Rep*. 2021;82:105903 https://doi.org/10.1016/j.ijscr.2021.105903. ISSN 2210–2612.

Ilioinguinal Neuralgia

ICD-10 CODE G57.90

THE CLINICAL SYNDROME

Ilioinguinal neuralgia is one of the most common causes of lower abdominal and pelvic pain encountered in clinical practice. Ilioinguinal neuralgia is caused by compression of the ilioinguinal nerve, and the most common causes of compression are traumatic injury to the nerve, including direct blunt trauma and damage during inguinal herniorrhaphy and pelvic surgery. Rarely, ilioinguinal neuralgia occurs spontaneously.

The ilioinguinal nerve is a branch of the L1 nerve root, with a contribution from T12 in some patients (Table 87.1). The nerve follows a curvilinear course that takes it from its origin at the L1 (or occasionally T12) somatic nerves to inside the concavity of the ileum. The ilioinguinal nerve continues anteriorly to perforate the transverse abdominal muscle at the level of the anterior superior iliac spine. The nerve may interconnect with the iliohypogastric nerve as it continues to pass along its course medially and inferiorly, where it accompanies the spermatic cord through the inguinal ring and into the inguinal canal. The distribution of the sensory innervation of the ilioinguinal nerves varies from patient to patient, and overlap with the iliohypogastric nerve may be considerable.

TABLE 87.1 Distribution of the Ilioinguinal, Iliohypogastric, and Genitofemoral Nerves

Nerve	Nerve Roots	Cutaneous Innervation	Motor Innervation
Iliohypogastric	T12–L1	Lower abdomen Superior mons pubis Lateral gluteal	Internal oblique
Ilioinguinal	T12–L1	Superomedial thigh Base of penis Anterior scrotum Labium majora Mons pubis	Internal oblique
Genitofemoral	L1–L2	Superior anterior thigh Scrotum Labia	Cremaster

In general, the ilioinguinal nerve provides sensory innervation to the skin of the upper inner thigh and the root of the penis and upper scrotum in men or the mons pubis and lateral labia in women.

SIGNS AND SYMPTOMS

Ilioinguinal neuralgia manifests as paresthesias, burning pain, and occasionally numbness over the lower abdomen that radiates into the scrotum or labia and occasionally into the upper inner thigh; pain does not radiate below the knee. The pain of ilioinguinal neuralgia worsens with extension of the lumbar spine, which puts traction on the nerve; thus patients often assume a bent-forward, novice skier's position (Figs. 87.1 and 87.2). If the condition remains untreated, progressive motor deficit, consisting of bulging of the anterior abdominal wall muscles, may occur. This bulging may be confused with inguinal hernia.

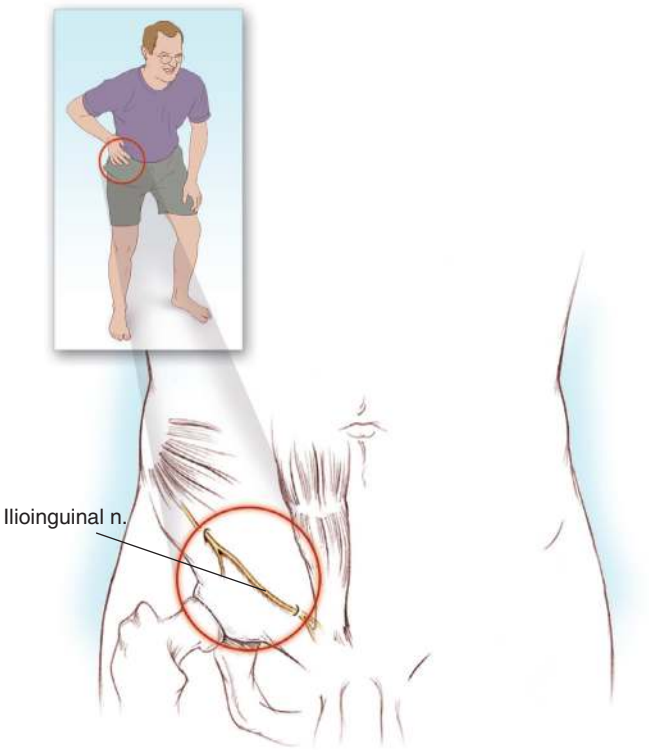

FIG 87.1 Patients suffering from ilioinguinal neuralgia often bend forward in the novice skier's position to relieve the pain.

FIG 87.2 Patients suffering from ilioinguinal neuralgia will often assume the novice skier position to relieve pressure or tension on the affected ilioinguinal nerve.

Physical findings include sensory deficit in the inner thigh, scrotum, or labia in the distribution of the ilioinguinal nerve. Weakness of the anterior abdominal wall musculature may be present. Tinel's sign may be elicited by tapping over the ilioinguinal nerve at the point where it pierces the transverse abdominal muscle.

TESTING

Electromyography (EMG) can distinguish ilioinguinal nerve entrapment from lumbar plexopathy, lumbar radiculopathy, and diabetic polyneuropathy. Plain radiographs of the hip and pelvis are indicated in all patients who present with ilioinguinal neuralgia, to rule out occult bony disease. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Ultrasound imaging of the ilioinguinal nerve may provide important information as to the cause of nerve entrapment (Fig. 87.3). Magnetic resonance imaging (MRI) of the lumbar spine and lumbar plexus is indicated if tumor or hematoma is suspected (Fig. 87.4). The injection technique described later serves as both a diagnostic and a therapeutic maneuver (Fig. 87.5).

DIFFERENTIAL DIAGNOSIS

Lesions of the lumbar plexus caused by trauma, hematoma, tumor, diabetic neuropathy, or inflammation can mimic the pain, numbness, and weakness of ilioinguinal neuralgia and must be excluded (Fig. 87.6).

Further, significant variability exists in the anatomy of the ilioinguinal nerve, which can result in significant variation in the clinical presentation.

TREATMENT

Initial treatment of ilioinguinal neuralgia consists of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors. Avoidance of repetitive activities thought to exacerbate the pain (e.g., squatting or sitting for prolonged periods) may also ameliorate the patient's symptoms. Pharmacologic treatment is usually disappointing, however, in which case ilioinguinal nerve block with local anesthetic and steroid is required.

Ilioinguinal nerve block is performed with the patient in the supine position; a pillow can be placed under the patient's knees if lying with the legs extended increases the pain because of traction on the nerve. The anterior superior iliac spine is identified by palpation, and a point 2 inches medial and 2 inches inferior to it is identified and prepared with antiseptic solution. A 1½-inch, 25-gauge needle is advanced at an oblique angle toward the pubic symphysis (see Fig. 87.5). A total of 5 to 7-mL of 1% preservative-free lidocaine in solution with 40-mg methylprednisolone is injected in a fanlike manner as the needle pierces the fascia of the external oblique muscle. Care must be taken not to insert the needle too deeply, which risks entering the peritoneal cavity and perforating the abdominal viscera. Because of the overlapping innervation of the ilioinguinal and iliohypogastric nerves, it is usually not necessary to block branches of each nerve. After injection of the solution, pressure is applied to the injection site to decrease the incidence of ecchymosis and hematoma formation, which can be quite dramatic, especially in anticoagulated patients. If anatomic landmarks are unclear, the use of fluoroscopic or ultrasound guidance should be considered to increase the accuracy of needle placement and decrease needle-related complications (Fig. 87.7).

For patients who do not rapidly respond to ilioinguinal nerve block, consideration should be given to epidural steroid injection of the T12–L1 segments.

COMPLICATIONS AND PITFALLS

Because of the anatomy of the ilioinguinal nerve, damage to or entrapment of the nerve anywhere along its course can produce a similar clinical syndrome. Therefore a careful search for pathologic processes at the T12–L1 spinal segments and along the path of the nerve in the pelvis is mandatory in all patients who present with ilioinguinal neuralgia without a history of inguinal surgery or trauma to the region.

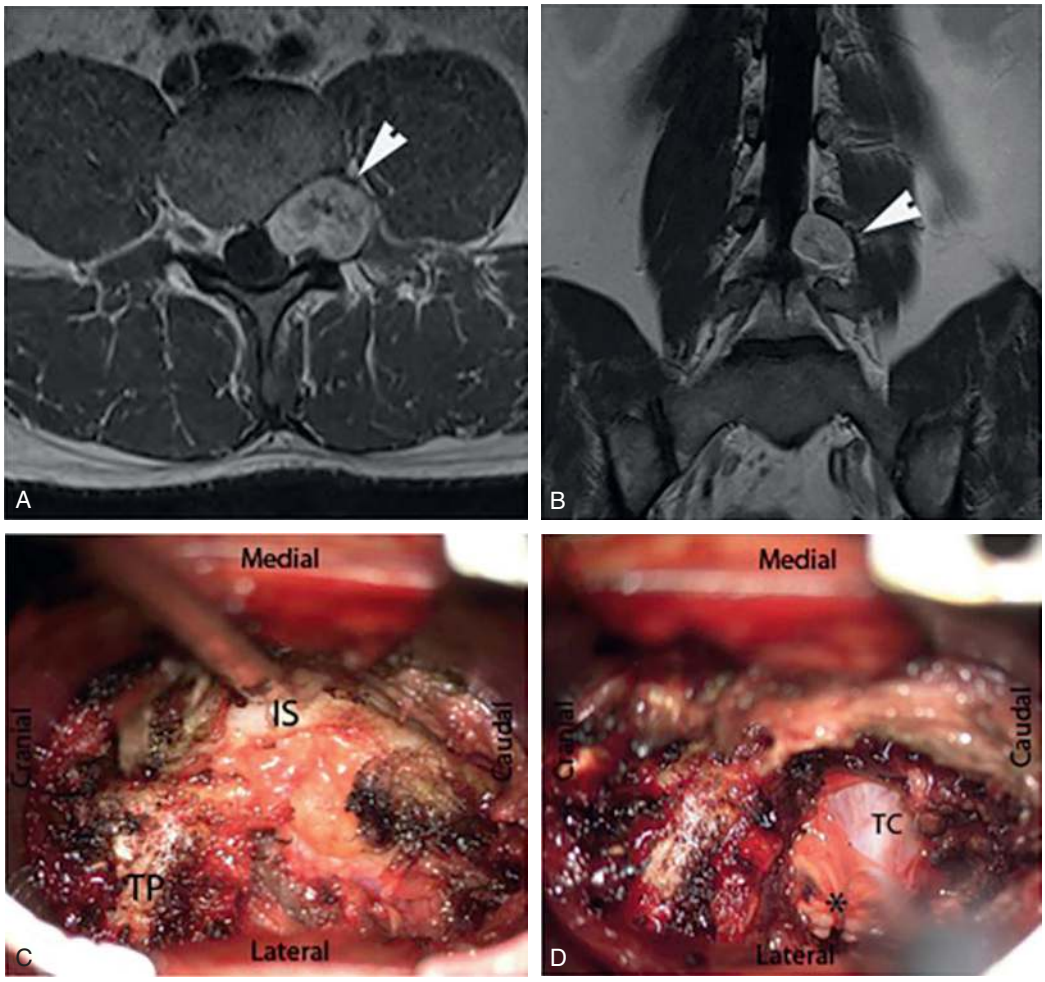

FIG 87.3 Preoperative T1-weighted MRI on axial **(A)** and coronal **(B)** plane, revealing a large dumbbell tumor in the left L4 foramen (patient #8). The motor nerve root could be identified surrounding the superior and anterior border of the tumor (*arrows*). Intraoperative photographs demonstrating the relevant anatomy **(C, D)**. After placement of the working tube, the relevant anatomy is exposed **(C)**: transverse process of L4 (TP) and lateral border of the isthmus (IS). After partial resection and retraction of the tumor capsule (TC), the motor root could be identified using the intraoperative neurostimulation (*) **(D)**. *MRI*, Magnetic resonance imaging. (From Zairi F, Troux C, Sunna T, et al. Minimally invasive resection of large dumbbell tumors of the lumbar spine: advantages and pitfalls. *Clin Neurol Neurosurg.* 2018;168:91–96. https://doi.org/10.1016/j.clineuro.2018.03.005.)

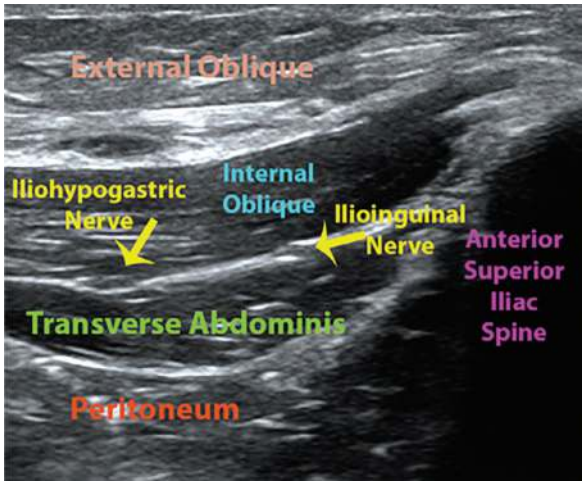

FIG 87.4 Ultrasound imaging of the ilioinguinal and iliohypogastric nerves. Note the relationship of the ilioinguinal nerve to the anterior superior iliac spine.

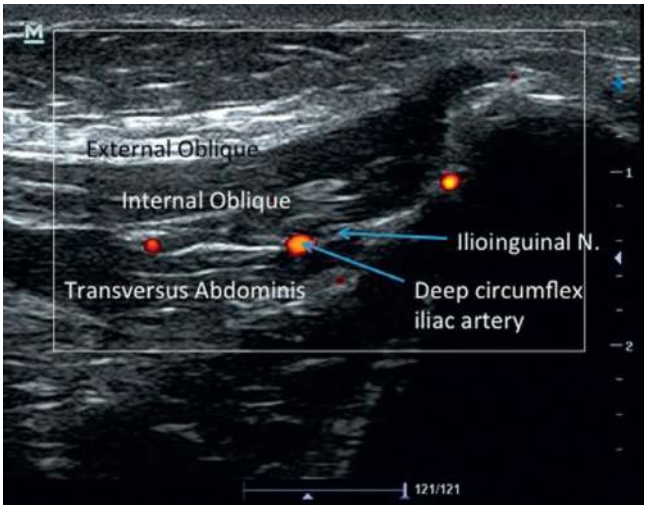

FIG 87.5 Ultrasound-guided ilioinguinal nerve block anatomy. Color Doppler image demonstrating the deep circumflex iliac artery which lies in the fascial plane between the internal oblique and transversus abdominis muscles adjacent to the ilioinguinal nerve.

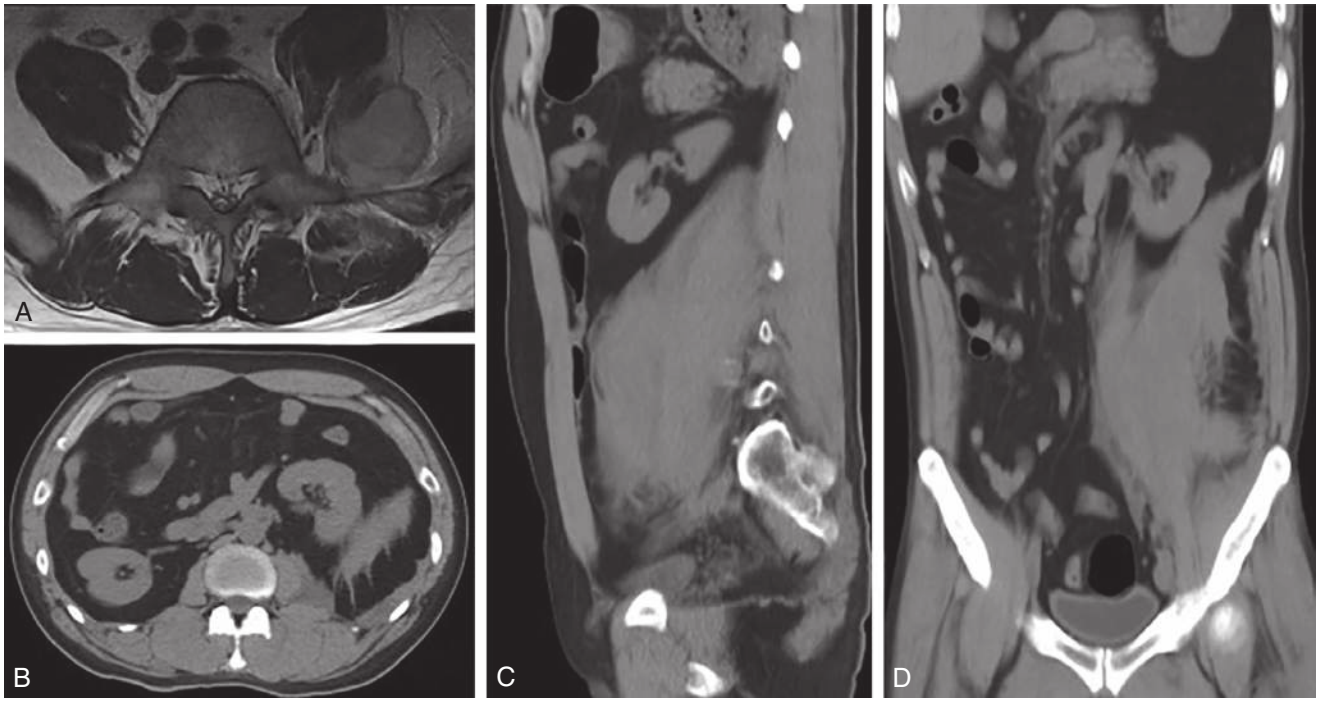

FIG 87.6 Axial MRI showed an anterior shift of the left psoas muscle caused by the retroperitoneal hematoma **(A, C)**. At 7 hr after surgery, the change of the hematoma to a diffuse-type was confirmed on coronal abdominal CT images **(B, D)**. The volume was measured to be 810 mL (20 cm × 9 cm × 9 cm × 0.5cm), and the hematoma was pushing the left kidney in a superior and ventral direction. (From Bae D-H, Eun S-S, Lee S-H, Lee S-M. Two cases of retroperitoneal hematoma after transforaminal percutaneous endoscopic lumbar discectomy. *Interdiscip Neurosurg.* 2020;20:100649. https://doi.org/10.1016/j.inat.2019.100649.)

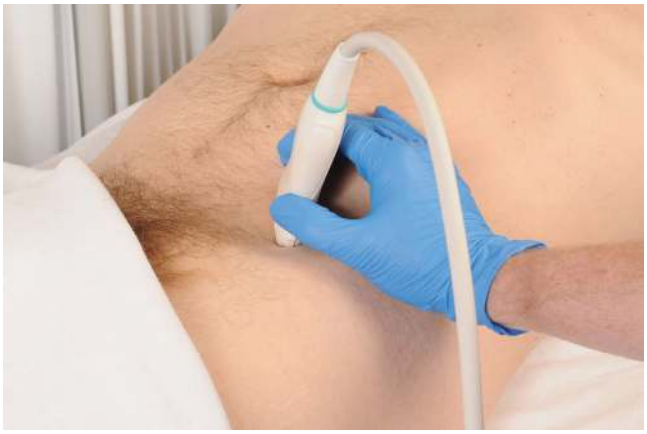

FIG 87.7 Proper ultrasound transducer placement for ultrasound-guided ilioinguinal nerve block.

The major complications of ilioinguinal nerve block are ecchymosis and hematoma formation. If the needle is too deep and enters the peritoneal cavity, perforation of the colon may result in the formation of an intraabdominal abscess and fistula. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Ilioinguinal neuralgia is a common cause of lower abdominal and pelvic pain, and ilioinguinal nerve block is a simple technique that can produce dramatic pain relief. If a patient presents with pain suggestive of ilioinguinal neuralgia and does not respond to ilioinguinal nerve block, a lesion more proximal in the lumbar plexus or an L1 radiculopathy should be considered. Such patients often respond to epidural steroid blocks. EMG and MRI of the lumbar plexus are indicated in this patient population to rule out other causes of ilioinguinal pain, including malignant disease invading the lumbar plexus or epidural or vertebral metastatic disease at T12–L1.

SUGGESTED READINGS

Bhatia N, Sen IM, Mandal B, Batra A. Postoperative analgesic efficacy of ultrasound-guided ilioinguinal-iliohypogastric nerve block compared with medial transverse abdominis plane block in inguinal hernia repair: a prospective, randomised trial. *Anaesth Crit Care Pain Med.* 2018:238–241.

Waldman SD. Ilioinguinal nerve entrapment. In: *Waldman's Comprehensive Atlas of Diagnostic Ultrasound of*

Painful Conditions. Philadelphia: Wolters Kluwer; 2016: 587–594.

Waldman SD. Iliohypogastric nerve entrapment. In: *comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:587–595.

Waldman SD. Ilioinguinal nerve block. In: *Pain review*. 2nd ed. Philadelphia: WB Saunders; 2017:474–476.

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Genitofemoral Neuralgia

ICD-10 CODE G57.90

THE CLINICAL SYNDROME

Genitofemoral neuralgia is one of the most common causes of lower abdominal and pelvic pain encountered in clinical practice. It may be caused by compression of or damage to the genitofemoral nerve anywhere along its path. The most common causes of genitofemoral neuralgia involve traumatic injury to the nerve, including direct blunt trauma and damage during inguinal herniorrhaphy and pelvic surgery. Rarely, genitofemoral neuralgia occurs spontaneously.

The genitofemoral nerve arises from fibers of the L1 and L2 nerve roots and passes through the substance of the psoas muscle, where it divides into a genital and a femoral branch. The femoral branch passes beneath the inguinal ligament, along with the femoral artery, and provides sensory innervation to a small area of skin on the inner thigh. The genital branch passes through the inguinal canal to provide innervation to the round ligament of the uterus and labia majora in women. In men, the genital branch passes with the spermatic cord to innervate the cremasteric muscles and provides sensory innervation to the bottom of the scrotum.

SIGNS AND SYMPTOMS

Genitofemoral neuralgia manifests as paresthesias, burning pain, and occasionally numbness over the lower abdomen that radiates to the inner thigh in both men and women and into the labia majora in women and the bottom of the scrotum and cremasteric muscles in men (Fig. 88.1); the pain does not radiate below the knee. The pain of genitofemoral

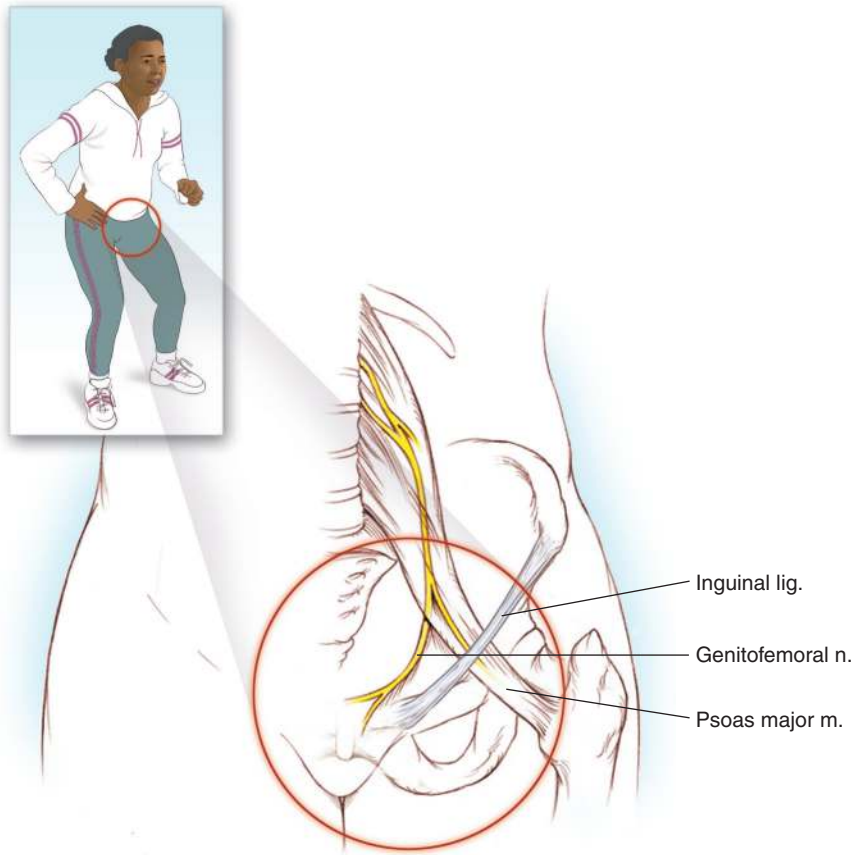


FIG 88.1 The pain of genitofemoral neuralgia radiates into the inner thigh of men and women and into the labia majora in women and the inferior scrotum in men.

neuralgia worsens with extension of the lumbar spine, which puts traction on the nerve. Therefore patients with genitofemoral neuralgia often assume a bent-forward, novice skier's position (see Fig. 88.1).

Physical findings include sensory deficit in the inner thigh, base of the scrotum, or labia majora in the distribution of the genitofemoral nerve. Weakness of the anterior abdominal wall musculature may be present. Tinel sign may be elicited by tapping over the genitofemoral nerve at the point where it passes beneath the inguinal ligament.

TESTING

Electromyography (EMG) can distinguish genitofemoral nerve entrapment from lumbar plexopathy, lumbar radiculopathy, and diabetic polyneuropathy. Plain radiographs of the hip and pelvis are indicated in all patients who present with genitofemoral neuralgia, to rule out occult bony disease. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) of the lumbar plexus is indicated if tumor or hematoma is suspected. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Lesions of the lumbar plexus caused by trauma, hematoma, tumor, diabetic neuropathy, or inflammation can mimic the pain, numbness, and weakness of genitofemoral neuralgia and must be excluded. Further, significant variability exists in the anatomy of the genitofemoral nerve, which can result in significant variation in the clinical presentation.

TREATMENT

Initial treatment of genitofemoral neuralgia consists of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors. Avoidance of repetitive activities thought to exacerbate the pain (e.g., squatting or sitting for prolonged periods) may also ameliorate the patient's symptoms. Pharmacologic treatment is usually disappointing, however, in which case genitofemoral nerve block with local anesthetic and steroid is required.

Genitofemoral nerve block is performed with the patient in the supine position; a pillow can be placed under the patient's knees if lying with the legs extended increases the pain because of traction on the nerve. The genital branch of the genitofemoral nerve is blocked as follows: The pubic tubercle is identified by palpation, and a point just lateral to it is identified and prepared with antiseptic solution. A 1½-inch, 25-gauge needle is advanced at an oblique angle toward the pubic symphysis (Fig. 88.2). A total of 3 to 5 mL of 1% preservative-free lidocaine in solution with 80-mg methylprednisolone is injected in a fanlike manner as the needle pierces the inguinal ligament. Care must be taken not to insert the needle deeply enough to enter the peritoneal cavity and perforate the abdominal viscera. Ultrasound needle guidance will help increase the accuracy of needle placement as well as decrease the incidence of needle-related complications (Fig. 88.3).

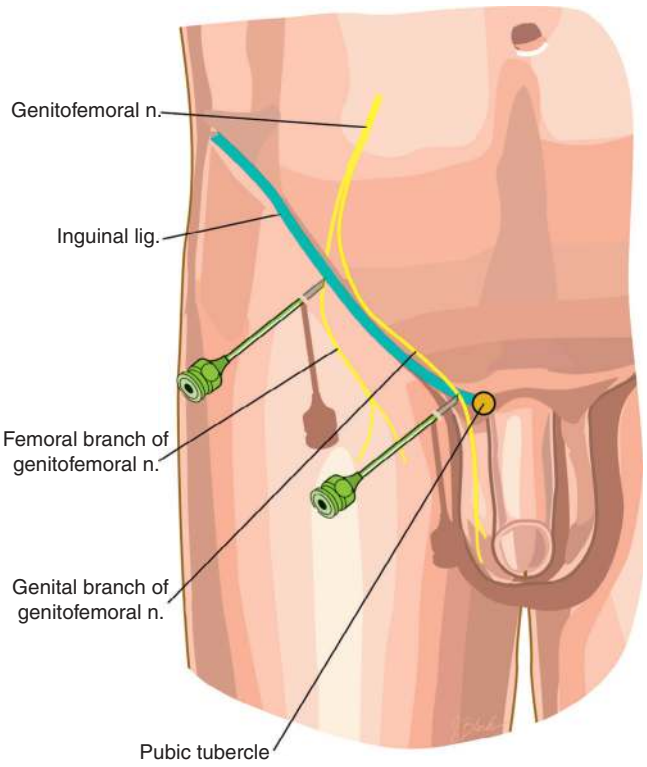


FIG 88.2 Correct needle placement for genitofemoral nerve block. (From Waldman SD. *Atlas of interventional pain management*. Philadelphia: Saunders; 1998:374.)

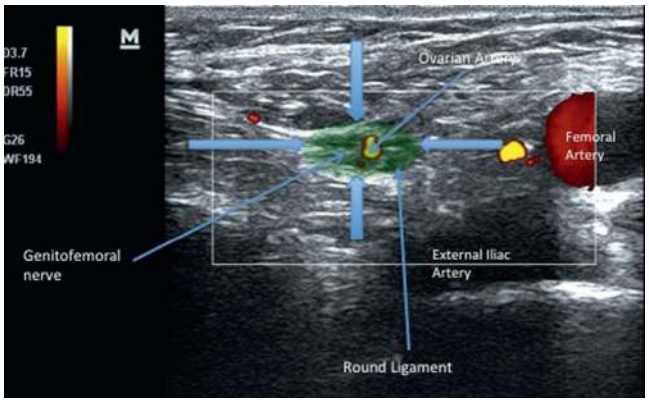


FIG 88.3 Relationship of the genitofemoral nerve to the femoral artery. Color Doppler image of the femoral artery as it begins to descend beneath the inguinal ligament into the abdominal cavity as it becomes the external iliac artery.

The femoral branch of the genitofemoral nerve is blocked by identifying the middle third of the inguinal ligament. After preparation of the skin with antiseptic solution, 3 to 5 mL of 1% lidocaine is infiltrated subcutaneously just below the ligament (see Fig. 88.2). Care must be taken not to enter the femoral artery or vein or to block the femoral nerve

inadvertently. The needle must be kept in a subcutaneous position to avoid entering the peritoneal cavity and perforating the abdominal viscera. If the patient has an inflammatory component to the pain, the local anesthetic is combined with 80-mg methylprednisolone and injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, by substituting 40-mg methylprednisolone for the initial 80-mg dose. Because of overlapping innervation of the ilioinguinal and iliohypogastric nerves, it is usually not necessary to block branches of each nerve during genitofemoral nerve block. After injection of the solution, pressure is applied to the injection site to decrease the incidence of ecchymosis and hematoma formation, which can be quite dramatic, especially in anticoagulated patients. Ultrasound needle guidance will help increase the accuracy of needle placement as well as decrease the incidence of needle-related complication when blocking the femoral branch of the genitofemoral nerve.

For patients who do not rapidly respond to genitofemoral nerve block, consideration should be given to epidural steroid injection of the L1–L2 segments.

COMPLICATIONS AND PITFALLS

Because of the anatomy of the genitofemoral nerve, damage to or entrapment of the nerve anywhere along its course can produce a similar clinical syndrome. Therefore a careful search for pathologic processes at the L1–L2 spinal segments and along the path of the nerve in the pelvis is mandatory in all patients who present with genitofemoral neuralgia without a history of inguinal surgery or trauma to the region.

The major complications of genitofemoral nerve block are ecchymosis and hematoma formation. If the needle is too deep and enters the peritoneal cavity, perforation of the colon may result in the formation of an intraabdominal abscess and fistula. Early detection of infection is crucial to avoid potentially life-threatening sequelae.

CLINICAL PEARLS

Genitofemoral neuralgia is a common cause of lower abdominal and pelvic pain; genitofemoral nerve block is a simple technique that can produce dramatic pain relief. If a patient presents with pain suggestive of genitofemoral neuralgia and does not respond to genitofemoral nerve block, lesions more proximal in the lumbar plexus or an L1 radiculopathy should be considered. Such patients often respond to epidural steroid blocks. EMG and MRI of the lumbar plexus are indicated in this patient population to rule out other causes of genitofemoral pain, including malignant disease invading the lumbar plexus or epidural or vertebral metastatic disease at T12–L1.

SUGGESTED READINGS

Ahmadian A, Abel N, Dakwar E. Injuries to the nerves of the abdominopelvic region. In: Tubbs RS, Rizk E, Shoja MM, et al., eds. *Nerves and nerve injuries*. San Diego: Academic Press; 2015:545–555.

Belanger GV, VerLee GT. Diagnosis and surgical management of male pelvic, inguinal, and testicular pain. *Surg Clin North Am*. 2016;96(3):593–613.

Kretschmer T, Heinen C. Iatrogenic injuries of the nerves. In: Tubbs RS, Rizk E, Shoja MM, et al., eds. *Nerves and nerve injuries*. San Diego: Academic Press; 2015:557–585.

Nguyen DK, Amid PK, Chen DC. Groin pain after inguinal hernia repair. *Adv Surg*. 2016;50(1):203–220.

Waldman SD. Genitofemoral nerve block. In: *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2017:470–473.

Waldman SD. Genitofemoral neuralgia. In: *Pain review*. 2nd ed. Philadelphia; 2017:285–286.

89

Lumbar Radiculopathy

ICD-10 CODE M54.16

THE CLINICAL SYNDROME

Lumbar radiculopathy is a constellation of symptoms including neurogenic back and lower extremity pain emanating from the lumbar nerve roots. In addition to pain, patients may experience numbness, weakness, and loss of reflexes. The causes of lumbar radiculopathy include herniated disk, foraminal stenosis, tumor, osteophyte formation, and, rarely, infection. Many patients and their physicians refer to lumbar radiculopathy as *sciatica*.

SIGNS AND SYMPTOMS

Patients suffering from lumbar radiculopathy complain of pain, numbness, tingling, and paresthesias in the distribution of the affected nerve root or roots (Table 89.1). Patients may also note weakness and lack of coordination in the affected extremity. Muscle spasms and back pain, as well as pain referred into the buttocks, are common. Decreased sensation, weakness, and reflex changes are demonstrated on physical examination. Patients with lumbar radiculopathy commonly experience a reflex shifting of the trunk to one side, called *list* (Fig. 89.1). Lasègue's straight leg raising sign is almost always positive in patients with lumbar radiculopathy (Fig. 89.2). Occasionally, patients suffering from lumbar radiculopathy experience compression of the lumbar spinal nerve roots and cauda equina that results in lumbar myelopathy or cauda equina syndrome; if so, they experience varying degrees of lower extremity weakness and bowel and bladder symptoms. This condition represents a neurosurgical emergency and should be treated as such.

TESTING

Magnetic resonance imaging (MRI) provides the best information about the lumbar spine and its contents and should be performed in all patients suspected of suffering from lumbar radiculopathy (Fig. 89.3). MRI is highly accurate and can identify abnormalities that may put the patient at risk for the development of lumbar myelopathy. In patients who cannot undergo MRI (e.g., those with pacemakers), computed tomography (CT) or myelography is a reasonable alternative. Diskography may also help clarify the diagnosis in difficult cases (Fig. 89.4). Radionuclide bone scanning and plain radiography are indicated if fracture or a bony abnormality, such as metastatic disease, is being considered.

Although MRI, CT, and myelography can supply useful neuroanatomic information, electromyography (EMG) and nerve conduction velocity testing provide neurophysiologic information about the actual status of each individual nerve root and the lumbar plexus. EMG can also distinguish plexopathy from radiculopathy and can identify a coexistent entrapment neuropathy, such as tarsal tunnel syndrome, which may confuse the diagnosis.

If the diagnosis of lumbar radiculopathy is in question, laboratory testing consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, human leukocyte antigen (HLA)-B27 screening, and automated blood chemistry should be performed to rule out other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Lumbar radiculopathy is a clinical diagnosis supported by a combination of clinical history, physical examination, radiography, and MRI. Pain syndromes that may mimic lumbar radiculopathy include low back strain; lumbar bursitis; lumbar fibromyositis; inflammatory arthritis; and disorders of the lumbar spinal cord, roots, plexus, and nerves (Table 89.2 and Box 89.1).

TREATMENT

Lumbar radiculopathy is best treated with a multimodality approach. Physical therapy, including heat modalities and deep sedative massage, combined with nonsteroidal antiinflammatory drugs and skeletal muscle relaxants is a good starting point. If necessary, caudal or lumbar epidural nerve blocks can be added; nerve blocks with local anesthetic and steroid are extremely effective in the treatment of lumbar radiculopathy (Fig. 89.5). Underlying sleep disturbance and depression are best treated with a tricyclic antidepressant such as nortriptyline, which can be started at a single bedtime dose of 25-mg.

TABLE 89.1 Clinical Features of Lumbar Radiculopathy

Lumbar Root	Pain	Sensory Changes	Weakness	Reflex Changes
L4	Back, shin, thigh, and leg	Shin numbness	Ankle dorsiflexors	Knee jerk
L5	Back, posterior thigh, and leg	Numbness of top of foot and first web space	Extensor hallucis longus	None
S1	Back, posterior calf, and leg	Numbness of lateral foot	Gastrocnemius and soleus	Ankle jerk

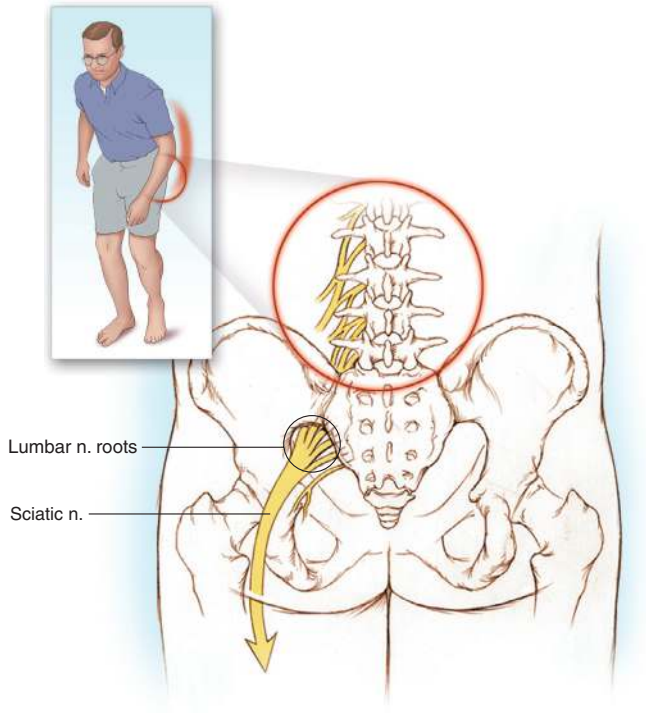


FIG 89.1 Patients suffering from lumbar radiculopathy often assume an unnatural posture in an attempt to take pressure off the affected nerve root and relieve pain.

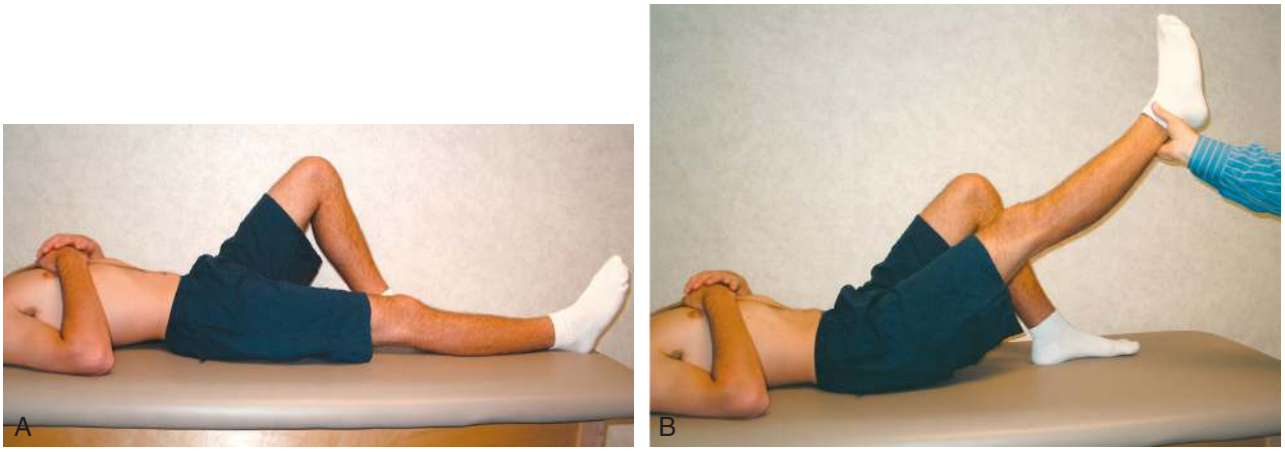


FIG 89.2 Lasègue's straight leg raising test. **A,** With the patient in the supine position, the unaffected leg is flexed 45 degrees at the knee, and the affected leg is placed flat against the table. **B,** With the ankle of the affected leg placed at 90 degrees of flexion, the affected leg is slowly raised toward the ceiling while the knee is kept fully extended. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:243–244.)

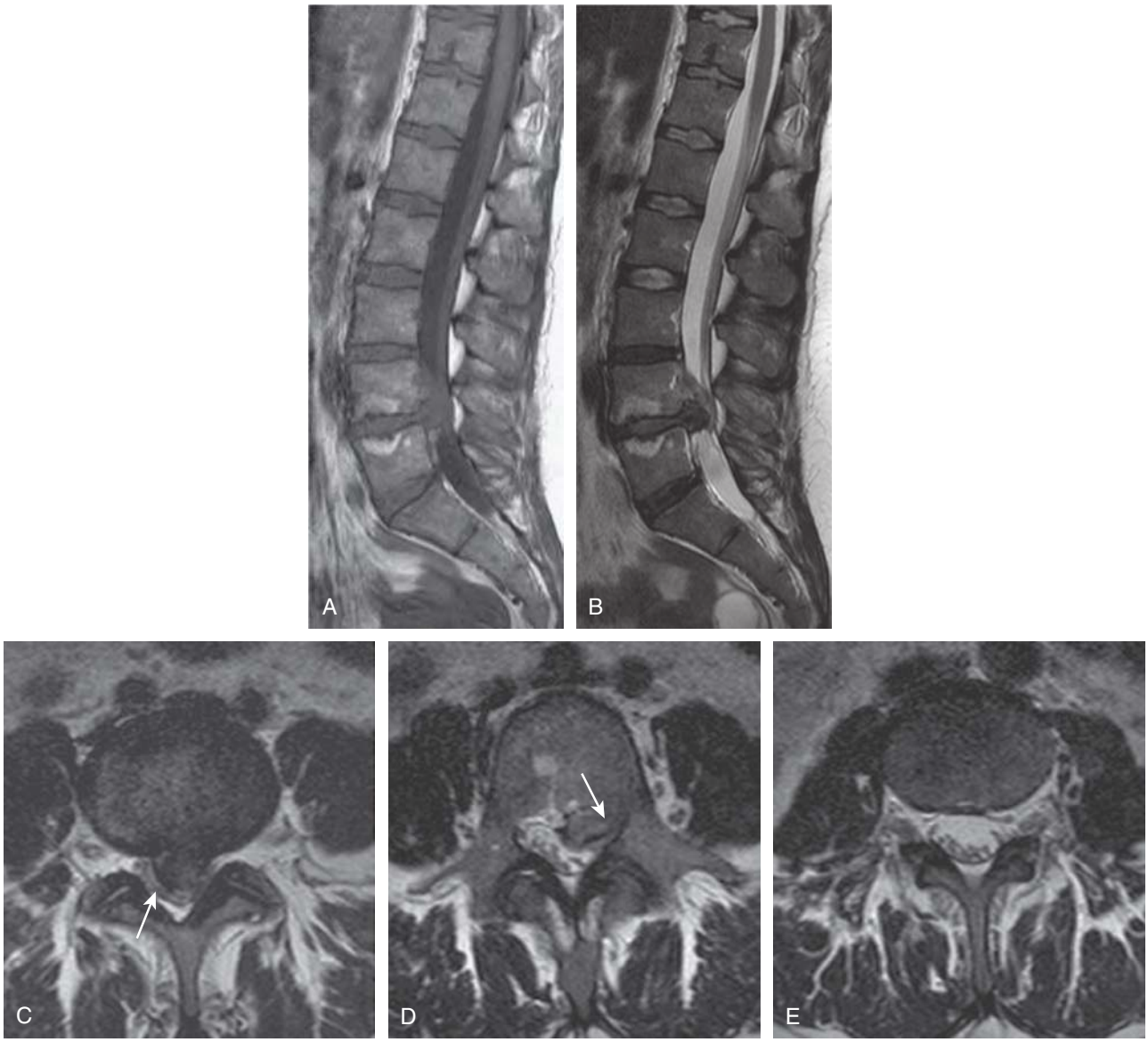


FIG 89.3 Sagittal T1W **(A)** and T2W **(B)** magnetic resonance (MR) images of a young woman with cauda equina syndrome and radicular left leg pain. There is a large disk extrusion at the L4–L5 level, which has intermediate SI on both the T1W and T2W MR images. **C,** An axial T2W MR image shows compression of the thecal sac, which is displaced to the right (*white arrow*). **D,** More proximally, the disk protrusion obliterates the traversing nerve root within the lateral recess (*white arrow*). Compared with the normal appearance of the thecal sac at the L3–L4 level **(E)**. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011:50–51.)

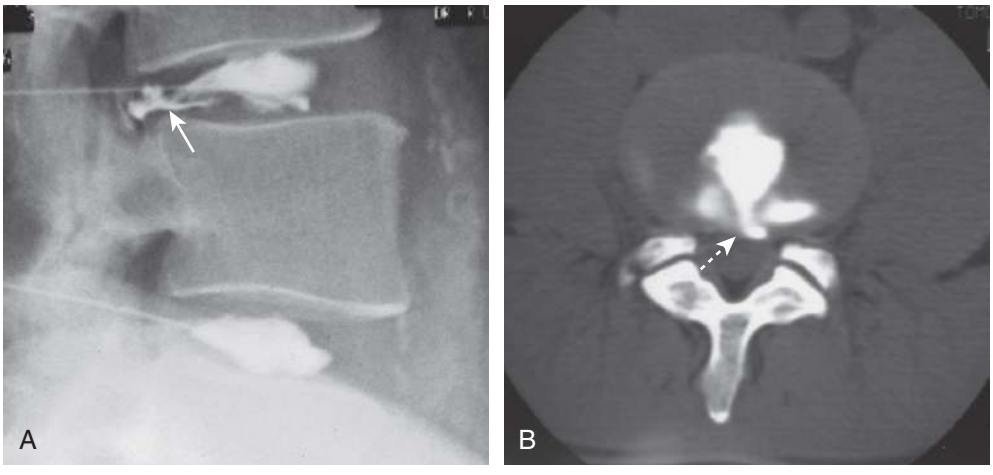


FIG 89.4 A, Diskogram demonstrating an annular fissure extending from the nucleus pulposus to the posterior disk margin (*white arrow*). **B,** The same appearance is seen on a computed tomography (CT) diskogram, with high-density contrast medium extending into a posterior annular fissure (*broken arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011:F49–F42.)

TABLE 89.2 Pain Syndromes that may Mimic Lumbar Radiculopathy

Localized Pathology	Primary Hip Pathology	Systemic Disease	Sympathetically Mediated Pain	Pain Referred from Other Body Areas
Disk Space Pathology	Bursitis	Rheumatoid arthritis	Complex regional pain syndrome Type I	Pancreatitis
Primary bone tumor	Tendinitis	Collagen vascular disease	Complex regional pain syndrome Type II	Malignancy of the retroperitoneal space
Facet joint disease	Aseptic necrosis	Reiter syndrome	Postthrombophlebitis pain (milk leg)	Lumbar Plexopathy
Localized or generalized degenerative arthritis	Osteoarthritis	Gout		Fibromyalgia
Osteophyte formation	Joint instability	Ankylosing spondylitis		Intraabdominal tumors
Vertebral compression fracture	Muscle strain	Other crystal arthropathies		Myofascial pain syndromes
Disk space infection	Muscle sprain	Charcot neuropathic arthritisMultiple sclerosis		Entrapment neuropathies
Herniated lumbar disk	Periarticular infection	Ischemic pain secondary to vascular insufficiency		
Degenerative disk disease				
Primary spinal cord and/or cauda equina pathology				
Osteomyelitis				
Epidural abscess				
Epidural hematoma				

From Waldman SD. *Physical Diagnosis of Pain: An Atlas of Signs and Symptoms*. 3rd ed. St. Louis: Elsevier; 2016, Table 137.1.

BOX 89.1 Causes of Lumbar Radiculopathy

- Herniated disk
- Foraminal stenosis
- Tumor
- Osteophyte formation
- Infection (rare)

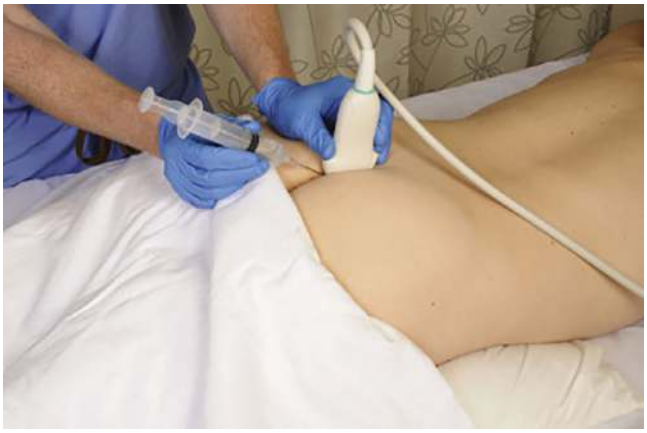


FIG 89.5 Ultrasound-guided caudal epidural block. After the sacral hiatus, sacrococcygeal ligament, and caudal canal are identified in the longitudinal ultrasound view, a 22- or 25-gauge, 2-inch needle is inserted through the skin approximately 1 cm below the inferior border of the transducer using an in-plane approach. The needle is advanced at a 45-degrees angle to the skin through the sacrococcygeal ligament into the caudal canal under real-time ultrasound guidance until the entire needle tip rests just beyond the sacrococcygeal ligament within the caudal canal. (From Waldman SD. Caudal epidural block: prone position. Ch 115. In: *Atlas of Interventional Pain Management*. 5th ed. Philadelphia: Elsevier; 2023:663.)

COMPLICATIONS AND PITFALLS

Failure to diagnose lumbar radiculopathy accurately may put the patient at risk for the development of lumbar myelopathy, which, if untreated, may progress to paraparesis or paraplegia.

CLINICAL PEARLS

Tarsal tunnel syndrome must be differentiated from lumbar radiculopathy involving the lumbar nerve roots, which may also mimic tibial nerve compression. Further, lumbar radiculopathy and tibial nerve entrapment may coexist in the double-crush syndrome.

SUGGESTED READINGS

Barr K. Electrodiagnosis of lumbar radiculopathy. *Phys Med Rehabil Clin North Am*. 2013;24(1):79–91.

Waldman SD. Functional anatomy of the lumbar spine. In: *Physical Diagnosis of Pain: An Atlas of Signs and Symptoms*. 4th ed. Philadelphia: Elsevier; 2020.

Waldman SD. Lumbar radiculopathy. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:256–257.

Waldman SD. Lumbar epidural nerve block: interlaminar approach. In: *Atlas of Interventional Pain Management*. 5th ed. Philadelphia: Elsevier; 2023:597–611.

Waldman SD. The Lesegue straight leg raiding test for lumbar radiculopathy. In: *Physical Diagnosis of Pain: An Atlas of Signs and Symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:235–236.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the lumbar spine. In: *Imaging of Pain*. Philadelphia: Elsevier; 2011:109–110.

Latissimus Dorsi Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The latissimus dorsi muscle is a broad, sheetlike muscle whose primary function is to extend, adduct, and medially rotate the arm; its secondary function is to aid in deep inspiration and expiration. The latissimus dorsi muscle originates on the spine of T7; the spinous processes and supraspinous ligaments of all lower thoracic, lumbar, and sacral vertebrae; the lumbar fascia; the posterior third iliac crest; the last four ribs; and the inferior angle of the scapula. The muscle inserts on the bicipital groove of the humerus and is innervated by the thoracodorsal nerve.

The latissimus dorsi muscle is susceptible to myofascial pain syndrome, which most often results from repetitive microtrauma to the muscle during such activities as vigorous use of exercise equipment or tasks that require reaching in a forward and upward motion (Fig. 90.1). Blunt trauma to the muscle may also incite latissimus dorsi myofascial pain syndrome.

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment.

The trigger point is pathognomonic of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of

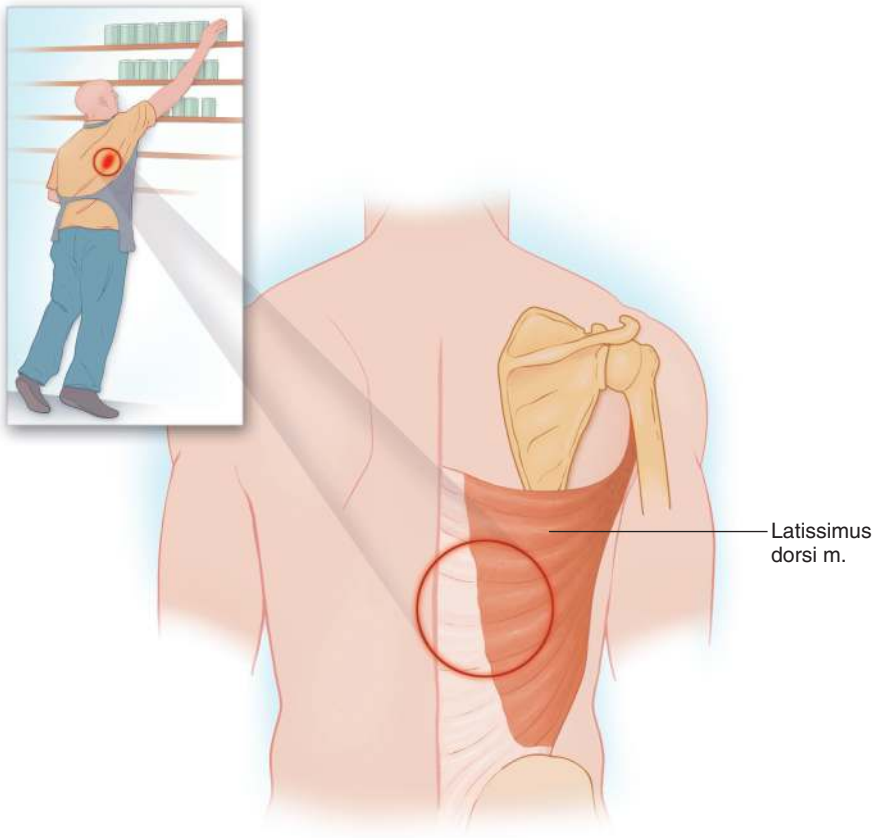


FIG 90.1 Latissimus dorsi syndrome is usually caused by repetitive microtrauma to the muscle during such activities as vigorous use of exercise equipment or tasks that require reaching forward and upward.

the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, often occurs and is also characteristic of myofascial pain syndrome.

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to be the result of microtrauma to the affected muscle. This trauma may result from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The latissimus dorsi muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point, the pathologic lesion of latissimus dorsi syndrome, is characterized by a local point of exquisite tenderness at the inferior angle of the scapula; this pain is referred to the axilla and the back of the ipsilateral upper extremity into the dorsal aspect of the ring and little fingers (Fig. 90.2). Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain and referred pain. In addition, the jump sign is often present.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with latissimus dorsi syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing has revealed

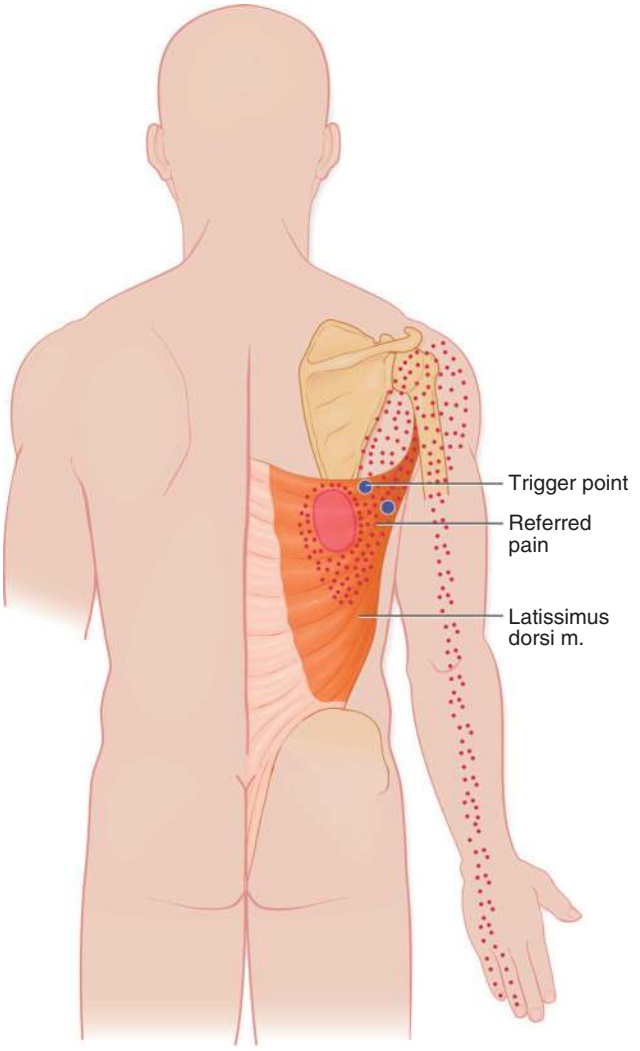


FIG 90.2 The trigger point of latissimus dorsi syndrome is at the inferior angle of the scapula, with pain referred to the axilla and the back of the ipsilateral upper extremity into the dorsal aspect of the ring and little fingers. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:299.)

an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic latissimus dorsi syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of latissimus dorsi syndrome is based on clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from latissimus dorsi syndrome. The clinician must rule out other coexisting disease processes that may mimic latissimus dorsi syndrome, including primary inflammatory muscle disease,

multiple sclerosis, and collagen vascular disease. The use of electrodiagnostic and radiographic testing can identify coexisting pathologic processes such as subscapular bursitis, cervical radiculopathy, herniated nucleus pulposus, and rotator cuff tear. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms of latissimus dorsi syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger point and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

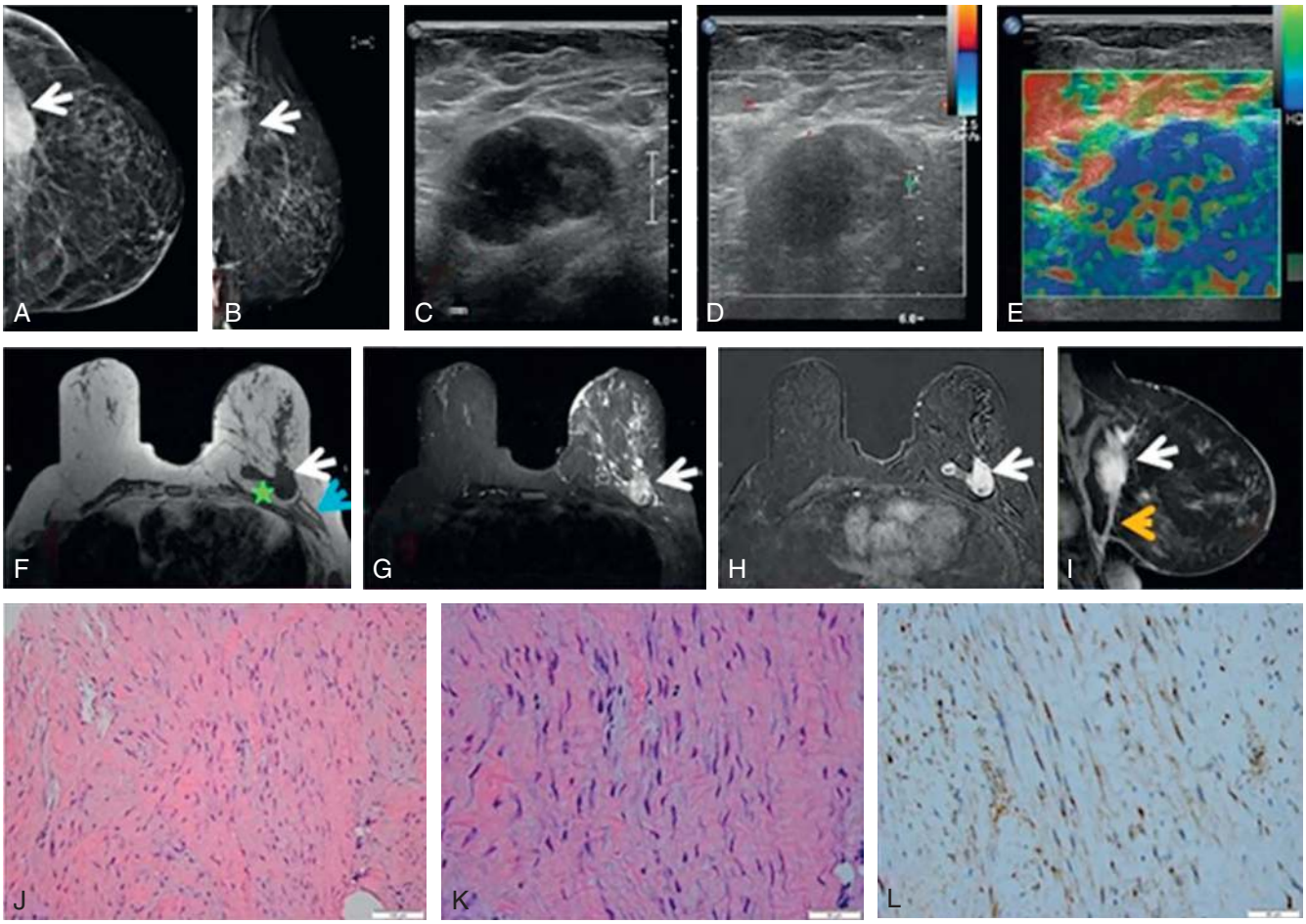


FIG 90.3 Desmoid tumor in the left breast with latissimus dorsi flap found in 2016. Craniocaudal **(A)** and mediolateral oblique **(B)** views of mammography showed noncalcified mass lesion (*white arrow*) with irregular borders in the upper outer quadrant of the left breast; ultrasound images demonstrated a hypoechoic oval mass **(C)** with circumscribed margins, measuring 3.5 cm without internal vascularity **(D),** intermediate stiffness on elastography **(E)**; breast MRI images showed a lobulated mass lesion which was isointense on T1-weighted image **(F)** (*green star*—preserved fat plane between lesion and pectoralis muscle; *blue arrow*—latissimus dorsi flap), hyper-intense on T2-weighted image **(G)**, with intense contrast enhancement **(H)**, and tail-like extension of the lesion (*yellow arrow*) probably to the flap on sagittal multiplanar image **(I)**; H&E stained images at 20× **(J)** and 40× magnification **(K)**; Immunohistochemical staining with anti-β-catenin antibody stained image at 40× magnification **(L)** demonstrated histologic and immunophenotypic features of fibromatosis of the breast. (From Canan A, Wang X. Recurrent desmoid tumor arising from latissimus dorsi flap: a case report. *Clin Imag.* 2019;53:191–194, ISSN 0899-7071. https://doi.org/10.1016/j.clinimag.2018.10.025. https://www.sciencedirect.com/science/article/pii/S089970711830295X.)

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid pneumothorax during injection of trigger points in proximity to the underlying pleural space.

CLINICAL PEARLS

Although latissimus dorsi syndrome is a common disorder, it is often misdiagnosed. Therefore, in patients suspected of suffering from latissimus dorsi syndrome, a careful evaluation to identify underlying disease processes is mandatory (Fig. 90.3). Latissimus dorsi syndrome often coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Arnold LM. The pathophysiology, diagnosis and treatment of fibromyalgia. *Psychiatr Clin North Am*. 2010;33(2):375–408.

Canan A, Wang X. Recurrent desmoid tumor arising from latissimus dorsi flap: a case report. *Clin Imaging*. 2019;53:191–194. https://doi.org/10.1016/j.clinimag.2018.10.025.

Cors-Serra S, Blanco-Hernández T, Cano-Teuler M, et al. Jerks of the latissimus dorsi muscle and intercostal neuralgia after posterolateral thoracotomy. *Clin Neurophysiol Pract*. 2021;6:203–205. https://doi.org/10.1016/j.cnp.2021.06.003.

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208:87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Ge H-Y, Nie H, Madeleine P, et al. Contribution of the local and referred pain from active myofascial trigger points in fibromyalgia syndrome. *Pain*. 2009;147(1–3):233–240.

Waldman SD. Injection technique for latissimus dorsi syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:347–349.

Spinal Stenosis

ICD-10 CODE M48.06

THE CLINICAL SYNDROME

Spinal stenosis is the result of congenital or acquired narrowing of the spinal canal. It occurs most commonly at the L5 vertebral level, with women affected more commonly than men (Fig. 91.1). Clinically, spinal stenosis usually manifests in a characteristic manner as pain and weakness in the legs when walking. This neurogenic pain is called pseudoclaudication or neurogenic claudication (Fig. 91.2). These symptoms are usually accompanied by lower extremity pain emanating from the lumbar nerve roots. In addition, patients with spinal stenosis may experience numbness, weakness, and loss of reflexes. The causes of spinal stenosis include bulging or herniated disk, facet arthropathy, and thickening and buckling of the interlaminar ligaments (Box 91.1). All these inciting factors tend to worsen with age.

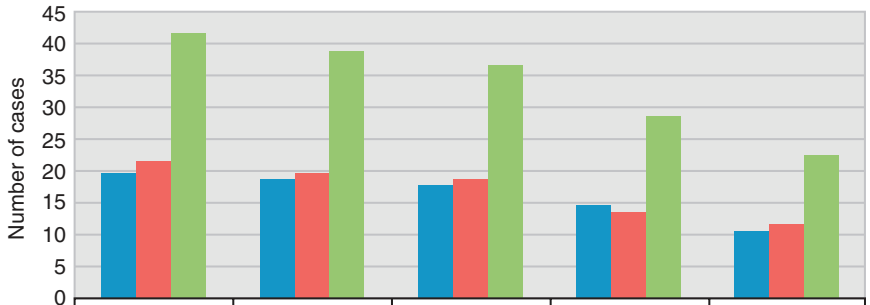
SIGNS AND SYMPTOMS

Patients suffering from spinal stenosis complain of calf and leg pain and fatigue when walking, standing, or lying supine. These symptoms disappear if they flex the lumbar spine or assume the sitting position. Frequently, patients suffering from spinal stenosis exhibit a simian posture, with a forward-flexed trunk and slightly bent knees when walking, to decrease the symptoms of pseudoclaudication (Fig. 91.3). Extension of the spine may cause an increase in symptoms. Patients also complain of pain, numbness, tingling, and paresthesias in the distribution of the affected nerve root or roots. Weakness and lack of coordination in the affected extremity may be noted. Patients often have a positive stoop test for spinal stenosis (Fig. 91.4). Muscle spasms and back pain, as well as pain referred to the trapezius and interscapular region, are common. Decreased sensation, weakness, and reflex changes are demonstrated on physical examination.

Occasionally, patients suffering from spinal stenosis experience compression of the lumbar spinal nerve roots and cauda equina, with resulting lumbar myelopathy or cauda equina syndrome. These patients experience varying degrees of lower extremity weakness and bowel and bladder symptoms. This condition represents a neurosurgical emergency and should be treated as such, although the onset of symptoms is often insidious.

TESTING

Magnetic resonance imaging (MRI) provides the best information about the lumbar spine and its contents and should be performed in all patients suspected of having spinal stenosis. MRI is highly accurate and can identify



	L5	L4	L3	L2	L1
MALE	20	19	18	15	11
FEMALE	22	20	19	14	12
TOTAL	42	39	37	29	23

FIG 91.1 Incidence of lumbar spinal stenos at each vertebral level by gender. (From Singh V, Sethi R, Chauhan BKS, et al. Lumbar spinal stenosis and morphometry of lumbar vertebral canal. *J Anat Soc India.* 2016;65(1):33–37.)

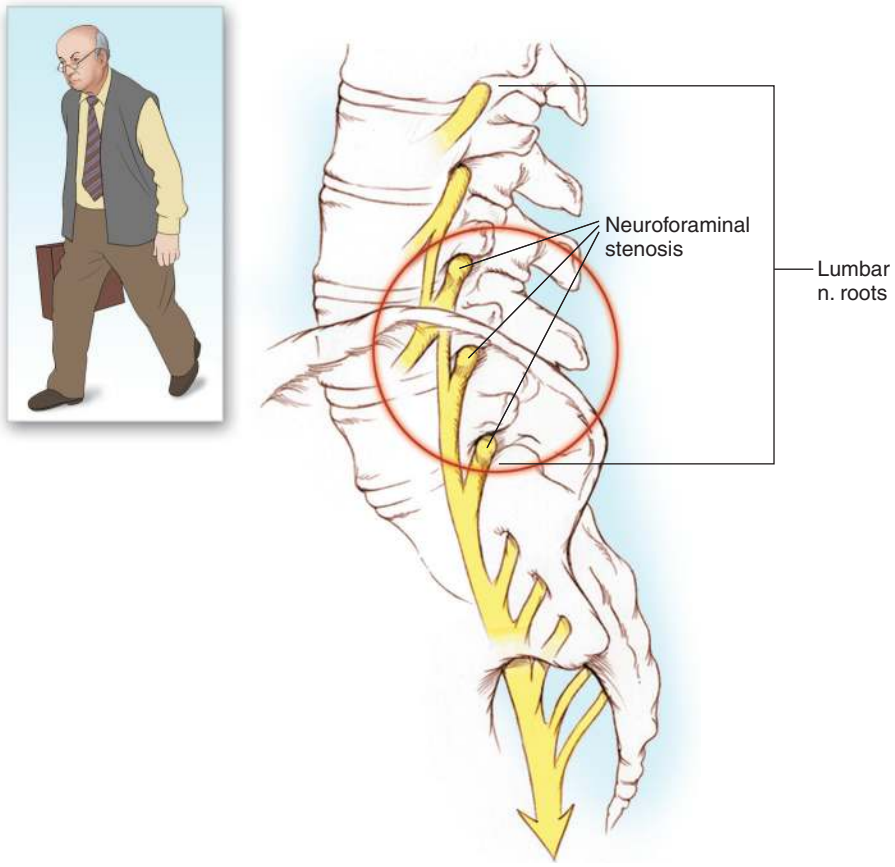


FIG 91.2 Pseudoclaudication is the sine qua non of spinal stenosis.

BOX 91.1 Causes of Lumbar Spinal Stenosis

- Congenital
  - Idiopathic
  - Achondroplastic
- Acquired
- Degenerative
  - Spondylosis
  - Spondylolisthesis
  - Disk degeneration
  - Disk herniation
  - Hypertrophy of facet joints
  - Hypertrophy of ligamentum flavum
  - Scoliosis
- Traumatic
- Iatrogenic
  - Postlaminectomy
  - Postsurgical fusion
  - Postsurgical perineural fibrosis
  - Postmyelogram arachnoiditis
- Infectious
  - Discitis
  - Paraspinal tubercular abscess
  - Osteomyelitis
- Rheumatic/Miscellaneous
  - Miscellaneous space-occupying lesions
  - Ankylosing spondylitis
  - Rheumatoid arthritis
  - Fluoride deposition
- Metabolic
  - Paget disease
  - Acromegaly
  - Diffuse idiopathic skeletal hyperostosis
  - Hyperparathyroidism
  - Diffuse idiopathic hyperostosis
  - Epidural lipomatosis
  - X-linked hypophosphatemic osteomalacia

FIG 91.3 Patients suffering from spinal stenosis often assume a simian posture, with a forward-flexed trunk and slightly bent knees when walking, to decrease the symptoms of pseudoclaudication. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:260.)

A B

FIG 91.4 Stoop test for spinal stenosis. A, Extension of the lumbar spine exacerbates the pain of spinal stenosis. B, Flexion of the lumbar spine relieves the pain of spinal stenosis. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:261.)

abnormalities that may put the patient at risk for lumbar myelopathy (Fig. 91.5). In patients who cannot undergo MRI (e.g., those with pacemakers), computed tomography (CT), or myelography is a reasonable alternative (Figs. 91.6 and 91.7). Radionuclide bone scanning and plain radiography are indicated if a coexistent fracture or bony abnormality, such as metastatic disease, is being considered.

Although MRI, CT, and myelography can supply useful neuroanatomic information, electromyography (EMG) and nerve conduction velocity testing provide neurophysiologic information about the actual status of each individual nerve root and the lumbar plexus. EMG can also distinguish plexopathy from radiculopathy and can identify a coexistent entrapment neuropathy that may confuse the diagnosis.

If the diagnosis is in question, laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, human leukocyte antigen-B27 screening, and automated blood chemistry should be performed to rule out other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Spinal stenosis is a clinical diagnosis supported by a combination of clinical history, physical examination, radiography, and MRI. Pain syndromes that may mimic spinal stenosis include low back strain; lumbar bursitis; lumbar fibromyositis; inflammatory arthritis; and disorders of the lumbar spinal cord, roots, plexus, and nerves, including diabetic femoral neuropathy.

TREATMENT

Spinal stenosis is best treated with a multimodality approach. Physical therapy, including heat modalities and deep sedative massage, combined with nonsteroidal antiinflammatory drugs and skeletal muscle relaxants is a reasonable starting point. If necessary, caudal or lumbar epidural nerve blocks can be added (Fig. 91.8). Caudal epidural blocks with local anesthetic and steroid are extremely effective in the treatment of spinal stenosis. Underlying sleep disturbance and depression are best treated with a tricyclic antidepressant, such as nortriptyline, which can be started at a single bedtime dose of 25 mg. Recent clinical experience suggests that injection of type A botulinum toxin into the gastrocnemius muscles may also provide symptomatic relief of nocturnal leg cramps associated with lumbar spinal stenosis.

COMPLICATIONS AND PITFALLS

Failure to diagnose spinal stenosis accurately may put the patient at risk for the development of lumbar myelopathy or cauda equina syndrome, which, if untreated, may progress to paraparesis or paraplegia.

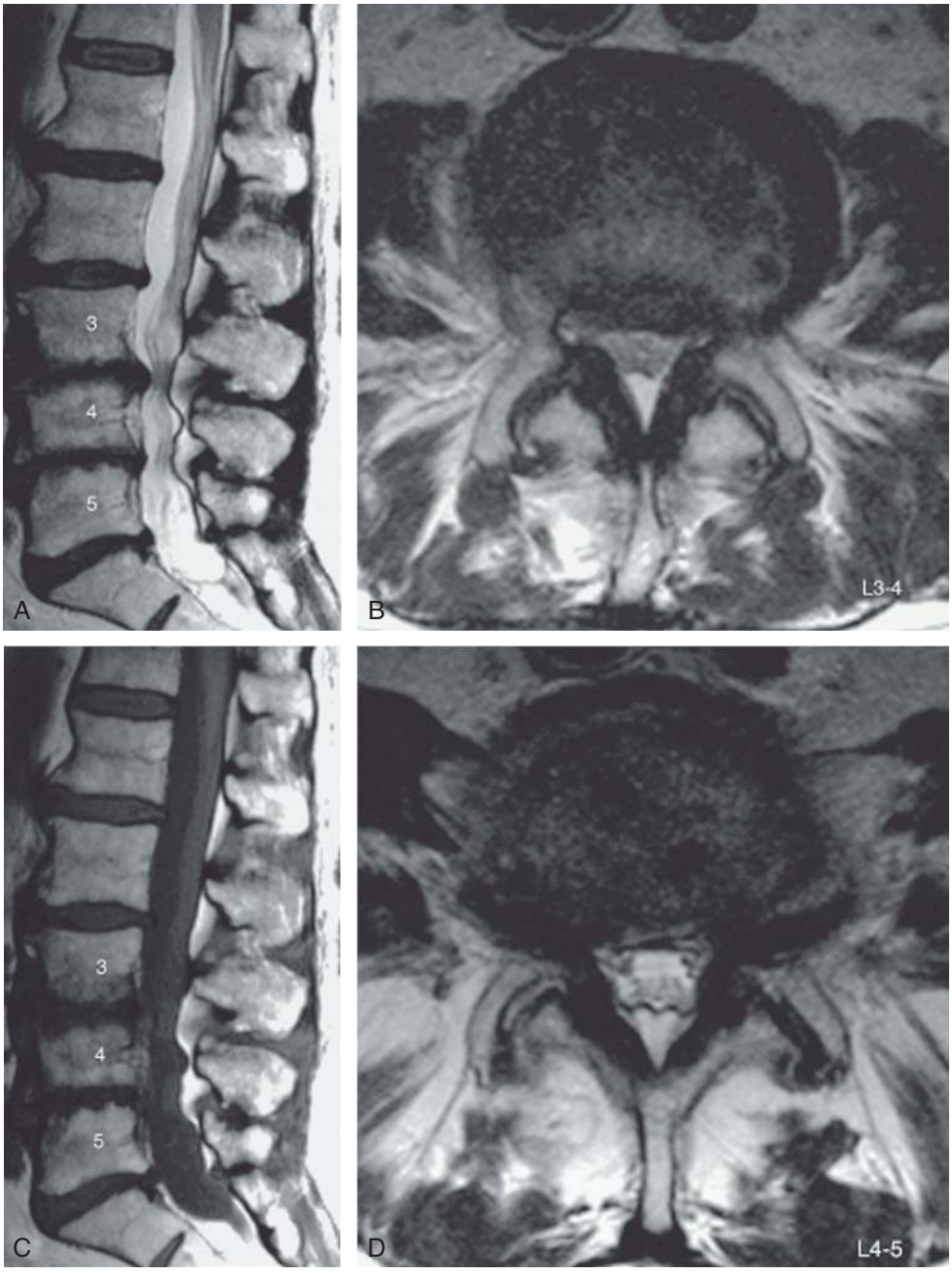


FIG 91.5 Acquired degenerative spinal stenosis. **A, B,** Sagittal T2- and T1-weighted magnetic resonance imaging (MRI) demonstrates severe disk degeneration at L3–L4 and L4–L5, with disk desiccation, disk space narrowing, irregularity of the adjacent vertebral end plates, and posterior bulging disks. The nerve roots of the cauda equina have an undulating or wavy appearance because of the marked constriction at L3–L4. **C, D,** On axial T2-weighted MRI, the combination of posterior bulging disks, facet hypertrophy, and thickened ligamenta flava causes severe spinal canal stenosis at L3–L4 and moderate stenosis at L4–L5. Also note severe compromise of the lateral recesses at L3–L4 bilaterally. (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2228.)

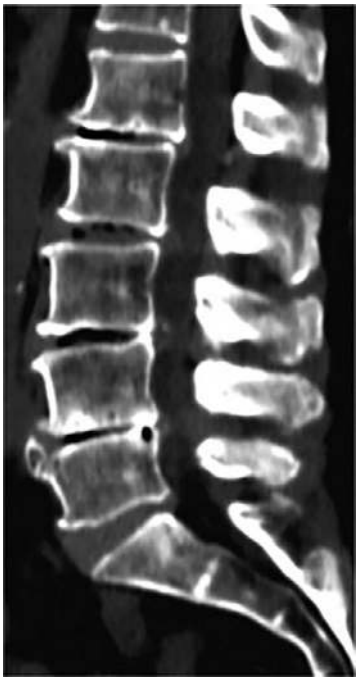


FIG 91.6 Sagittal computed tomography of a patient with lumbar spinal stenosis that shows disk degeneration with evident loss of disk height, multilevel disk protrusion, along with osteophyte formation, rendering the spinal canal stenotic. (From Biller J, Ferro J. *Handbook of clinical neurology*. Vol 119. Amsterdam: Elsevier; 2014, Fig. 35.1.)

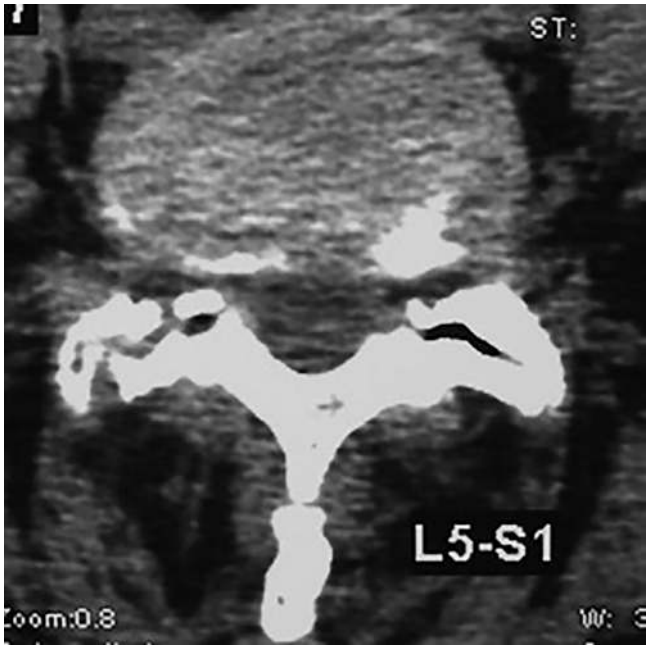


FIG 91.7 The transaxial computed tomography study at the L5S1 level shows marked spondyloarthrotic changes involving the L5S1 facet joints accompanied by anteroposterior diameter and lateral recess stenosis and a focal far lateral right-sided soft herniated disk. (From Quiñones-Hinojosa A. Schmidek and sweet operative neurosurgical techniques. 6th ed. Philadelphia: Saunders; 2016, Fig. 163.16.)

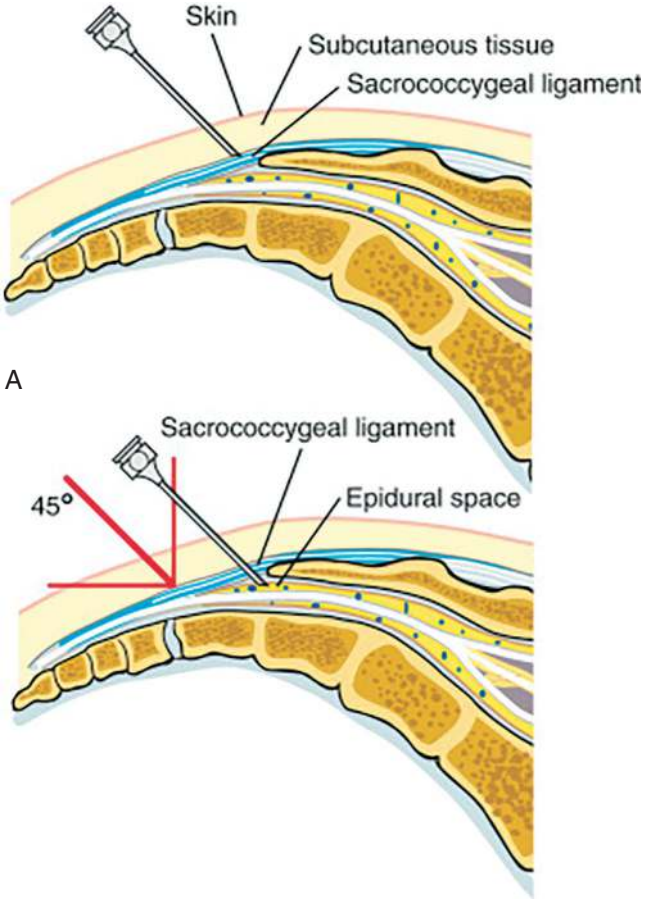


FIG 91.8 Caudal epidural block. **A,** After the sacral hiatus is located, a 25-gauge, 1½-inch needle is inserted through the anesthetized area at a 45-degree angle into the sacrococcygeal ligament. **B,** The needle is then advanced through the ligament into the sacral canal. As the sacrococcygeal ligament is penetrated, a pop will be felt.

CLINICAL PEARLS

Spinal stenosis is a common cause of back and lower extremity pain, and the finding of pseudoclaudication should point the clinician toward this diagnosis. This syndrome tends to worsen with age. The onset of lumbar myelopathy or cauda equina syndrome may be insidious, so careful questioning and physical examination are required to avoid missing these complications.

SUGGESTED READINGS

Cowley P. Neuroimaging of spinal canal stenosis. *Magn Reson Imaging Clin N Am*. 2016;24(3):523–539.

Genevay S, Atlas SJ. Lumbar spinal stenosis. *Best Pract Res Clin Rheumatol*. 2010;24(2):253–265.

Singh V, Sethi R, Chauhan BKS, et al. Lumbar spinal stenosis and morphometry of lumbar vertebral canal. *J Anat Soc India*. 2016;65(1):33–37.

Waldman SD. Spinal stenosis. In: *Pain review*. Philadelphia: Saunders; 2009:202–203.
Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the lumbar spine. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:109–110.
Waldman SD. Lumbar radiculopathy. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:223–224.
Waldman SD. Caudal epidural block: prone. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2023:663–675.
Waldman SD. Lumbar epidural nerve block. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2023:587–611.

Arachnoiditis

ICD-10 CODE G03.9

THE CLINICAL SYNDROME

Arachnoiditis consists of thickening, scarring, and inflammation of the arachnoid membrane. These abnormalities may be self-limited or may lead to compression of the nerve roots and spinal cord. In addition to pain, patients with arachnoiditis may experience numbness, weakness, loss of reflexes, and bowel and bladder symptoms. The exact cause of arachnoiditis is unknown, but it may be related to herniated disk, infection, tumor, myelography, spinal surgery, or intrathecal administration of drugs. Anecdotal reports of arachnoiditis after epidural and subarachnoid administration of methylprednisolone have surfaced as have reports of arachnoiditis after repeat high-volume epidural blood patches to treat postdural puncture headache.

SIGNS AND SYMPTOMS

Patients suffering from arachnoiditis complain of pain, numbness, tingling, and paresthesias in the distribution of the affected nerve root or roots (Table 92.1). Weakness and lack of coordination in the affected extremity may be noted; muscle spasms, back pain, and pain referred to the buttocks are common. Decreased sensation, weakness, and reflex changes are demonstrated on physical examination. Occasionally, patients with arachnoiditis experience compression of the lumbar spinal cord, nerve roots, and cauda equina, with resulting lumbar myelopathy or cauda equina syndrome (Fig. 92.1). These patients experience varying degrees of lower extremity weakness and bowel and bladder symptoms.

TESTING

Magnetic resonance imaging (MRI) provides the best information about the lumbar spine and its contents and should be performed in all patients suspected of suffering from arachnoiditis (Fig. 92.2). MRI is highly accurate and can identify abnormalities that may put the patient at risk for the development of lumbar myelopathy or cauda equina syndrome. In patients who cannot undergo MRI (e.g., those with pacemakers), computed tomography (CT) or myelography is a reasonable alternative (Figs. 92.3 and 92.4). Radionuclide bone scanning and plain radiography are indicated if a fracture or bony abnormality, such as metastatic disease, is being considered.

Although MRI, CT, and myelography can supply useful neuroanatomic information (Fig. 92.5), electromyography (EMG) and nerve conduction velocity testing provide neurophysiologic information about the actual status of each individual nerve root and the lumbar plexus. EMG and somatosensory-evoked potentials can also distinguish plexopathy from arachnoiditis and can identify a coexistent entrapment neuropathy that may confuse the diagnosis.

If the diagnosis is in question, laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, antinuclear antibody testing, human leukocyte antigen-B27 screening, and automated blood chemistry should be performed to rule out other causes of the patient's pain.

DIFFERENTIAL DIAGNOSIS

Arachnoiditis is a clinical diagnosis supported by a combination of clinical history, physical examination, radiography, and MRI. Conditions that may mimic arachnoiditis include tumor; infection; and disorders of the lumbar spinal cord, roots, plexus, and nerves.

TREATMENT

Little consensus exists on the best treatment of arachnoiditis; most efforts are aimed at decompressing the nerve roots and spinal cord and treating the inflammatory component of

TABLE 92.1 Clinical Features of Arachnoiditis

Lumbar Root	Pain	Sensory Changes	Weakness	Reflex Changes
L4	Back, shin, thigh, and leg	Shin numbness	Ankle dorsiflexors	Knee jerk
L5	Back, posterior thigh, and leg	Numbness of top of foot and first web space	Extensor hallucis longus	None
S1	Back, posterior calf, and leg	Numbness of lateral foot	Gastrocnemius and soleus	Ankle jerk

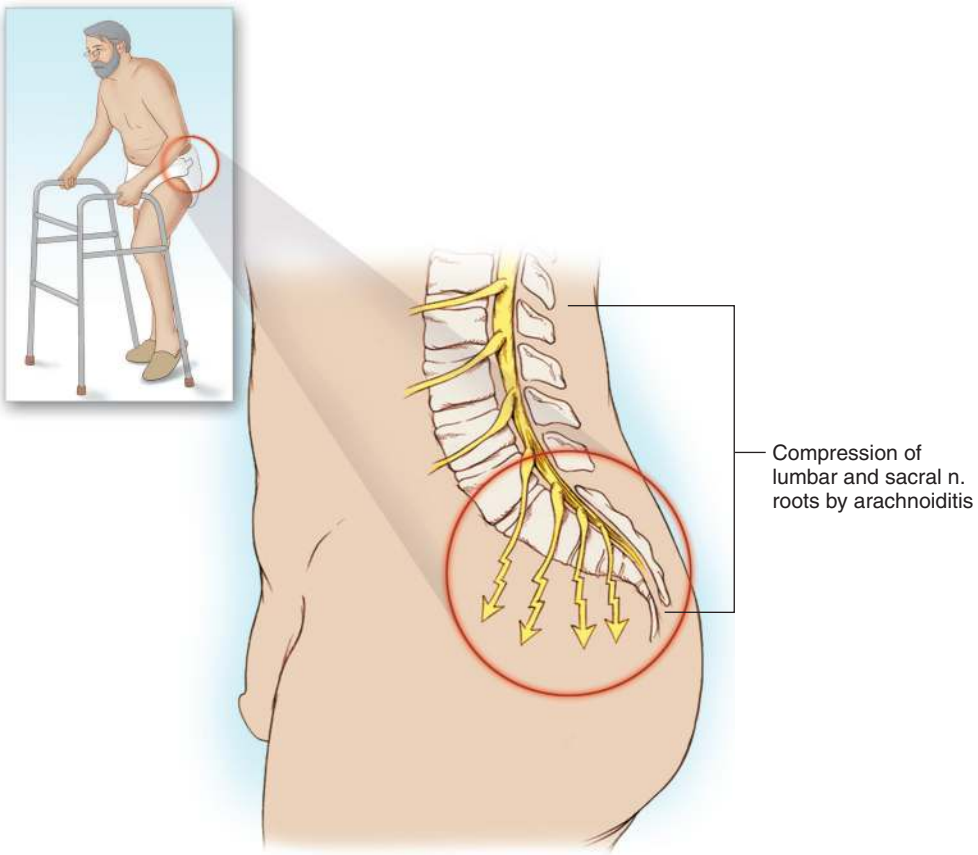


FIG 92.1 Arachnoiditis may result in lumbar myelopathy or cauda equina syndrome.

A B C

FIG 92.2 Postoperative chronic arachnoiditis. **A,** Sagittal short repetition time/echo time (TR/TE) (600/15) conventional spin-echo image magnetic resonance imaging (MRI) demonstrates degeneration of the L4–L5 intervertebral disk. **B,** Axial short TR/TE (600/15) conventional spin-echo MRI at the level of L3 demonstrates spondylosis with no associated abnormality. **C,** Axial short TR/TE (600/15) conventional spin-echo MRI after gadolinium administration shows enhancement of matted nerve roots forming a thickened cord (*arrow*), indicating the presence of adhesive arachnoiditis.

FIG 92.2 cont'd D, Oblique view from a water-soluble contrast myelogram shows the partially empty sac and the cordlike matting of the nerve roots (*arrow*). E, Axial computed tomography scan obtained through L3 shows matting of the nerve roots into a cordlike structure (*arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al. eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2274.)

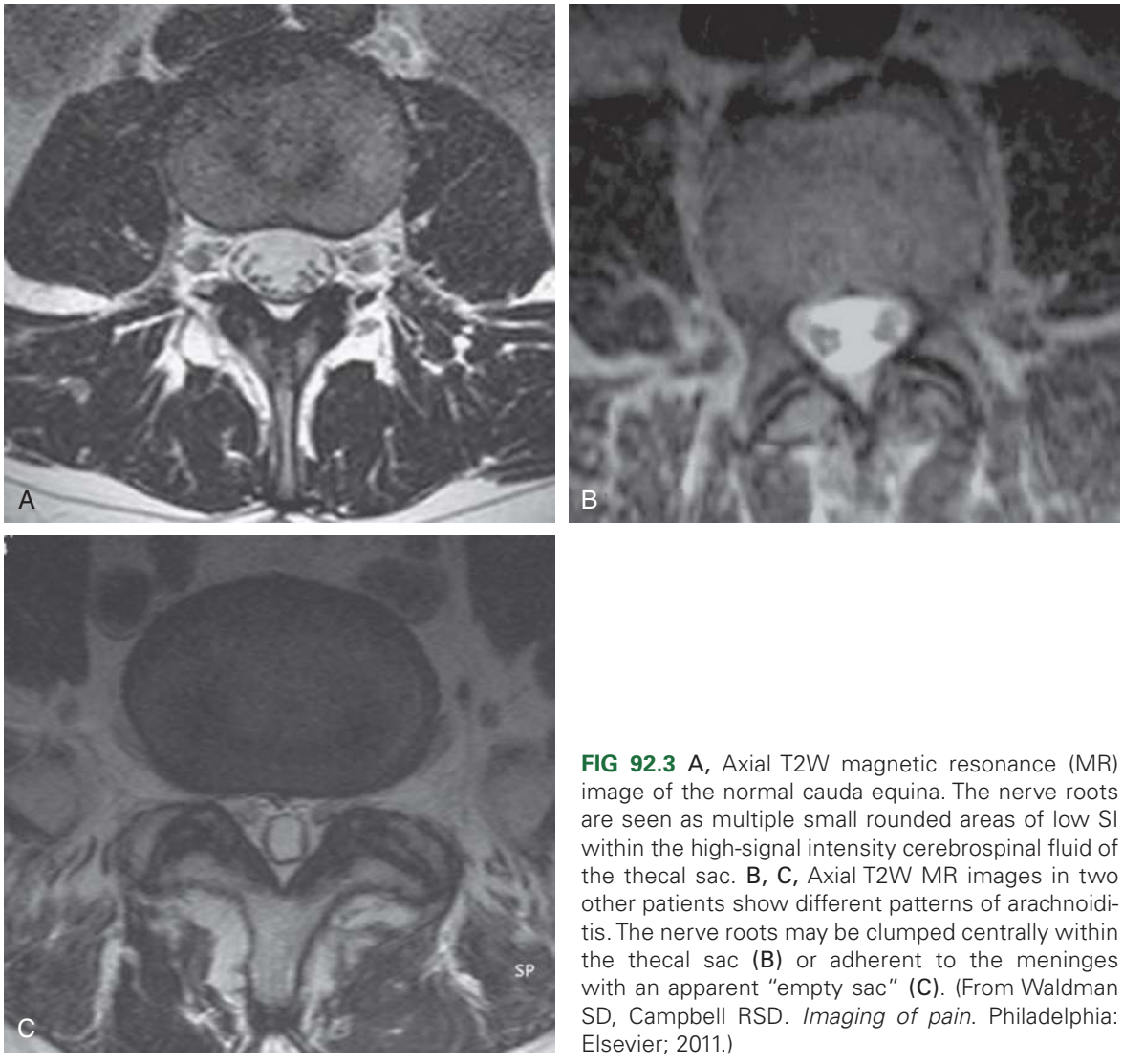


FIG 92.3 A, Axial T2W magnetic resonance (MR) image of the normal cauda equina. The nerve roots are seen as multiple small rounded areas of low SI within the high-signal intensity cerebrospinal fluid of the thecal sac. B, C, Axial T2W MR images in two other patients show different patterns of arachnoiditis. The nerve roots may be clumped centrally within the thecal sac (B) or adherent to the meninges with an apparent "empty sac" (C). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

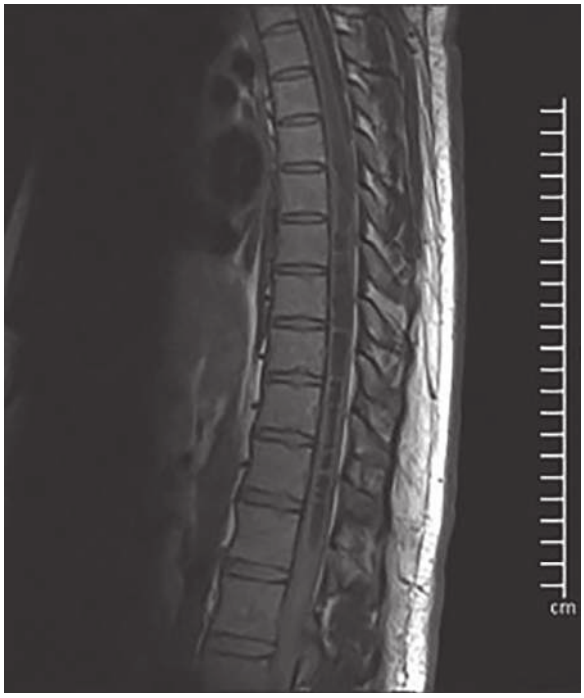


FIG 92.4 Sagittal T1W MRI shows an expansile intramedullary lesion of low signal intensity seen extending between T3 and L1 level with thinning of spinal cord parenchyma. The lesion shows high signal intensity with some heterogeneous signal intensity (due to CSF pulsation artifact). (From Safi S, Thabat A, Arshad M, Hanoun M. Arachnoiditis—a challenge in diagnosis and success in outcome—case report, *Interdiscip Neurosurg*. 2021;25:101219.)

the disease. Epidural neurolysis or the caudal administration of steroids may decompress the nerve roots if the pathologic process is localized. More generalized arachnoiditis often requires surgical decompressive laminectomy. The results of these treatment modalities are disappointing at best.

Underlying sleep disturbance and depression are treated with a tricyclic antidepressant such as nortriptyline, which can be started at a single bedtime dose of 25-mg. Neuropathic pain associated with arachnoiditis may respond to gabapentin. Spinal cord stimulation may also provide symptomatic relief. Opioid analgesics should be used with caution, if at all.

COMPLICATIONS AND PITFALLS

Failure to diagnose arachnoiditis accurately may put the patient at risk for the development of lumbar myelopathy or cauda equina syndrome, which, if untreated, may progress to paraparesis or paraplegia.

CLINICAL PEARLS

Arachnoiditis is a potentially devastating disease that may erroneously be attributed to the clinician's efforts to diagnose and treat low back and lower extremity pain. For this reason, MRI and EMG should be obtained early in patients who have no clear cause of their symptoms.

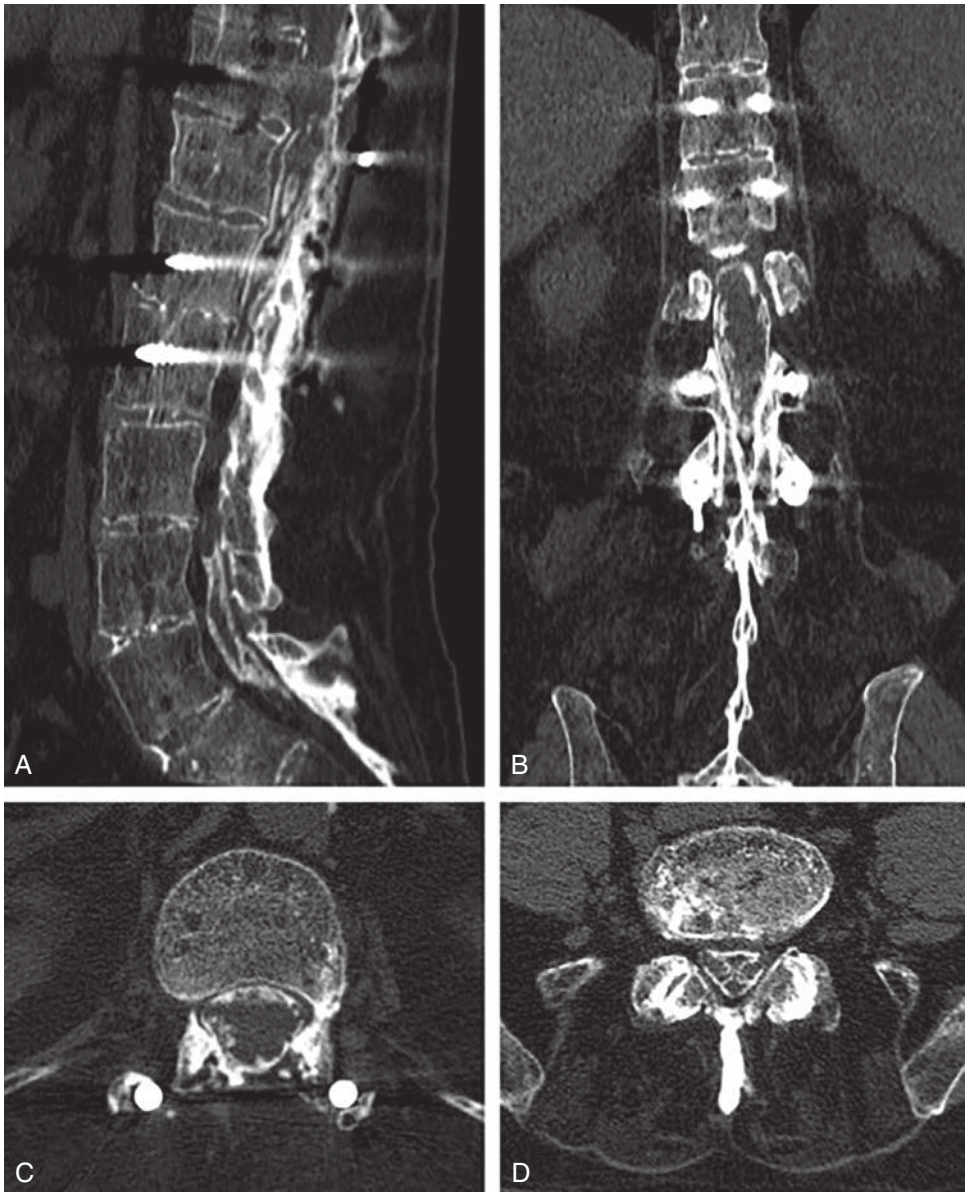


FIG 92.5 Preoperative sagittal, coronal, and axial computed tomography images showing thecal sac and arachnoid ossification (AO) **(A–D)** and two different ossification patterns in the same patient. In type II AO, concentric ossification surrounds the thecal sac **(C)**. In type III AO, there is a "honeycomb" appearance in which nerve roots may be encased in arachnoid ossification **(D)**. (From Scalia G, Certo F, Maione M, Barbagallo GMV. Spinal arachnoiditis ossificans: report of quadruple-triggered case. *World Neurosurg*. 2019;123:1–6.)

SUGGESTED READINGS

Benyaich Z, Laghmari M, Benali SA. Arachnoiditis ossificans of the lumbar spine: a rare cause of progressive cauda equina syndrome. *World Neurosurg*. 2021;148:116–117. https://doi.org/10.1016/j.wneu.2021.01.057.

Carlswärd C, Darvish B, Tunelli J, et al. Chronic adhesive arachnoiditis after repeat epidural blood patch. *Int J Obstet Anesth*. 2015;24(3):280–283.

Crum BA, Strommen JA. Nerve root assessment with SEP and MEP. *Handb Clin Neurophysiol*. 2008;8:455–463.

Escobales N, Grudzinski K, Jain I, Abreu-Sosa S. Post-procedural adhesive arachnoiditis with subsequent incomplete paraplegia. *Arch Phys Med Rehabil*. 2021;102(10):e91–e92. https://doi.org/10.1016/j.apmr.2021.07.745.

Parenti V, Huda F, Richardson PK, et al. Lumbar arachnoiditis: does imaging associate with clinical features? *Clin Neurol Neurosurg*. 2020;192:105717. https://doi.org/10.1016/j.clineuro.2020.105717.

Safi S, Thabat A, Arshad M, Hanoun M. Arachnoiditis—a challenge in diagnosis and success in outcome—case report. *Interdiscip Neurosurg*. 2021;25:101219. https://doi.org/10.1016/j.inat.2021.101219.

Waldman SD. Arachnoiditis. In: *Pain Review*. 2nd ed. Philadelphia: Saunders; 2017:303–304.

Waldman SD, Campbell RSD. Arachnoiditis. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:169–170.

93

Discitis

ICD-10 CODE M46.47

THE CLINICAL SYNDROME

Discitis is an often misdiagnosed cause of spine pain that, if undiagnosed, can result in paralysis or life-threatening complications. Although discitis can occur anywhere in the spine, the lumbar spine is affected most often, followed by the thoracic, then the cervical spine. Discitis occurs more commonly in males and can occur spontaneously by hematogenous seeding, most frequently as a result of urinary tract infections that spread to the spinal epidural space through Batson's plexus. More commonly, discitis occurs after instrumentation of the spine, including surgery, diskography, and epidural nerve blocks. There is a bimodal age distribution in patients suffering from discitis with a peak before the age of 20 and a second peak between the ages of 50 and 70.

Not surprisingly, the infectious agents most frequently responsible for discitis are the same agents that cause urinary tract infections. The literature has suggested that the administration of steroids into the epidural space causes immunosuppression, with a resultant increase in the incidence of discitis. Although this suggestion is theoretically plausible, the statistical evidence, given the thousands of epidural steroid injections performed around the United States on a daily basis, calls this concept into question. Discitis has a 2:1 male predominance in adult patients. The average age of occurrence in children is approximately 7 years, and in adults it is the fifth decade of life. Untreated, the mortality associated with discitis approaches 10%.

The patient with discitis initially presents with ill-defined pain and spasm of the paraspinous musculature in the segment of the spine affected (e.g., cervical, thoracic, or lumbar) (Fig. 93.1). This pain becomes more intense and localized as the infection involves more of the disks and adjacent vertebral bodies and compresses neural structures. Low-grade fever and vague constitutional symptoms, including malaise and anorexia, progress to frank sepsis with high-grade fever, rigors, and chills. At this point, the patient begins to experience sensory and motor deficits, as well as bowel and bladder symptoms as a result of neural compromise. As the infection continues to expand into the epidural space, compromise of the vascular supply to the affected spinal cord and nerve occurs, with resultant ischemia and, if untreated, spinal cord infarction and permanent neurologic deficits. Even with antibiotic therapy, there is an approximate 3% mortality associated with discitis.

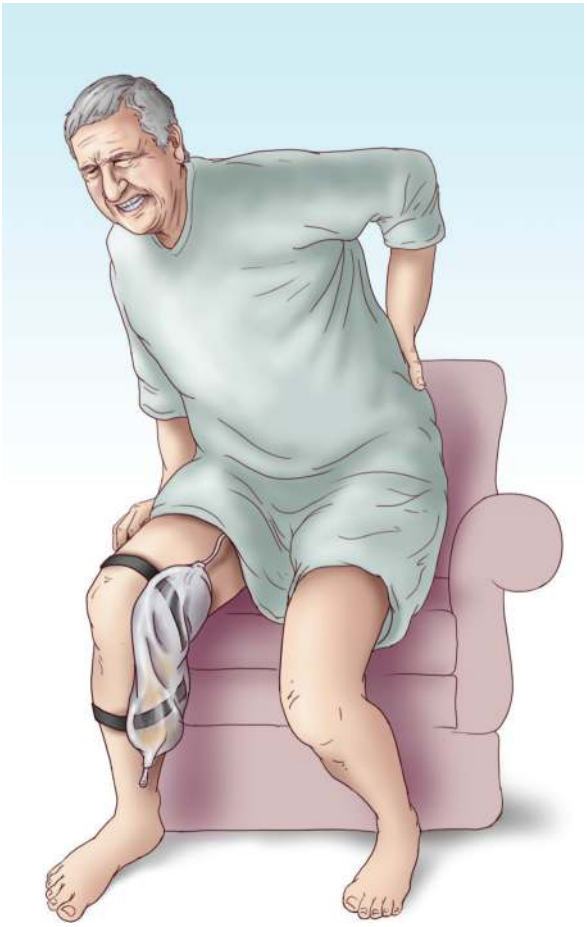


FIG 93.1 If discitis is not promptly diagnosed, compression of the involved neural structures may continue, and the patient's neurologic status may deteriorate rapidly. If diagnosis is not made and treatment initiated, irreversible motor and sensory deficit will result.

SIGNS AND SYMPTOMS

The patient with discitis initially presents with ill-defined pain in the general area of the infection. At this point, the patient may have mild pain on the range of motion of the affected segments. The neurologic examination is within normal limits. A low-grade fever or night sweats may be noted. Theoretically, if the patient has received steroids, these constitutional symptoms may be attenuated or their onset may be delayed. As the abscess increases in size, the patient appears

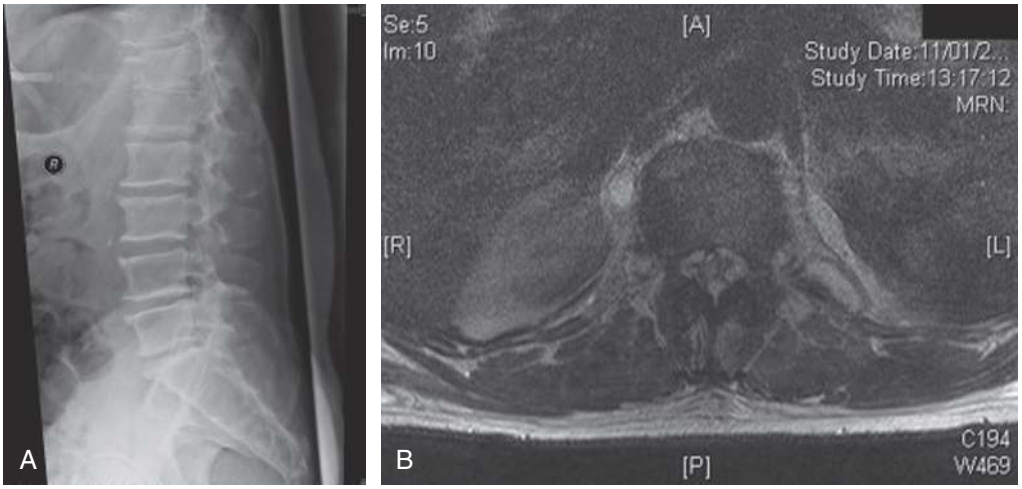


FIG 93.2 A, Plain film shows loss of disk space and end-plate destruction at T12–L1. This appearance is highly suggestive of infection. B, Axial magnetic resonance imaging at the same level indicates a paravertebral collection. (From Cottle L, Riordan T. Infectious spondylodiscitis. *J Infect*. 2008;56(6):402–412.)

acutely ill, with fever, rigors, and chills. The clinician may be able to identify neurologic findings suggestive of spinal nerve root or spinal cord compression. Subtle findings that point to the development of myelopathy (e.g., Babinski's sign, clonus, and decreased perineal sensation) may be overlooked if not carefully sought. As compression of the involved neural structures continues, the patient's neurologic status may deteriorate rapidly. If the correct diagnosis is not made, irreversible motor and sensory deficit will result.

TESTING

Plain radiographs often reveal the evidence of disk space narrowing and end-plate changes that are suggestive of discitis; however, these changes may not be present early in the course of the disease (Fig. 93.2). Early diagnosis is best made with the use of radionucleotide scanning with gallium-67 and technetium-99m (Fig. 93.3). Both magnetic resonance imaging (MRI) and computed tomography (CT) are highly accurate in the diagnosis of discitis and are probably more accurate than myelography in the diagnosis of intrinsic disease of the spinal cord and spinal tumor, among other disorders (Fig. 93.4). Needle or open surgical biopsy for culture should strongly be considered in all patients thought to be suffering from discitis, but antibiotic treatment should not be delayed if these procedures are not readily available (Fig. 93.5).

All patients suspected of suffering from discitis should undergo laboratory testing consisting of complete blood cell count, sedimentation rate, C-reactive protein, and automated blood chemistries. Blood and urine cultures should be immediately obtained in all patients thought to have discitis, to allow immediate implementation of antibiotic therapy while the workup is in progress. Gram stains and cultures of the abscess material should also be obtained, but antibiotic treatment should not be delayed while waiting for this information. Echocardiography to rule out subacute bacterial endocarditis should also be considered, especially in intravenous drug abusers.

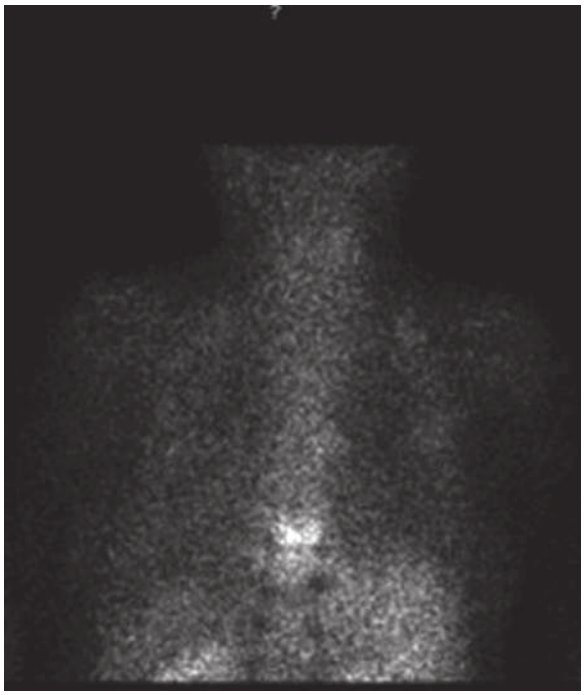


FIG 93.3 Planar gallium-67 citrate scan with uptake at T11 consistent with discitis and osteomyelitis. (From Stieber JR, Schweitzer ME, Errico TJ. The imaging of spinal infections. *Semin Spine Surg*. 2007;19(2):106–112.)

TREATMENT

The rapid initiation of treatment of discitis is mandatory if the patient is to avoid the sequelae of permanent neurologic deficit or death. The treatment of discitis is aimed at

FIG 93.4 Magnetic resonance imaging scan, sagittal sections: T1-weighted sequence **(A),** T1-weighted fat saturation sequence after gadolinium injection **(B),** and T2-weighted fat saturation sequence **(C)**. The findings are typical of L1–L2 discitis with epidural involvement. (From Millot F, Bonnaire B, Clavel G, et al. Hematogenous *Staphylococcus aureus* discitis in adults can start outside the vertebral body. *Joint Bone Spine*. 2010;77(1):76–77.)

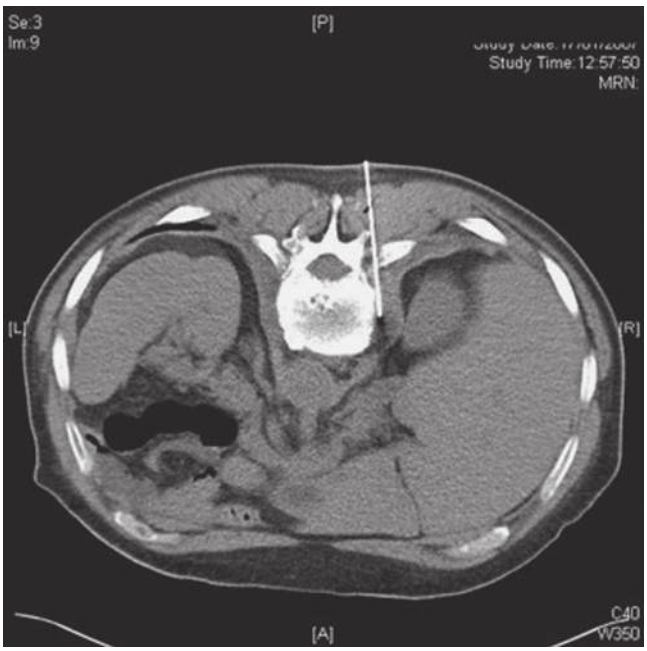


FIG 93.5 Paravertebral fluid collection being aspirated under computed tomography guidance in a patient with discitis. (From Cottle L, Riordan T. Infectious spondylodiscitis. *J Infect*. 2008;56(6):402–412.)

two goals: (1) treatment of the infection with antibiotics and (2) drainage of any abscess formation to relieve compression on neural structures (Fig. 93.6). Because most cases of discitis are caused by *Staphylococcus aureus*, antibiotics such as vancomycin that treat staphylococcal infection should be started immediately after blood and urine culture samples are taken. Antibiotic therapy can be tailored to the culture and sensitivity reports as they become available (Fig. 93.7). As mentioned, antibiotic therapy should not be delayed while waiting for definitive diagnosis if discitis is being considered as part of the differential diagnosis. Many atypical infectious agents (e.g., *Mycobacterium tuberculosis*, fungi) can cause discitis, especially in immunocompromised patients, and such pathogens should be considered in the patient who does not respond to treatment with traditional antibiotic regimens. Bed rest and use of orthotic devices to stabilize affected spinal segments should help improve the long-term outcome of patients suffering from discitis.

Antibiotics alone are rarely successful in the treatment of discitis unless the diagnosis is made very early in the course of the disease. Therefore drainage of any epidural abscess is required to affect full recovery. Drainage is usually accomplished by decompression laminectomy and evacuation of the abscess. More recently, interventional radiologists have been

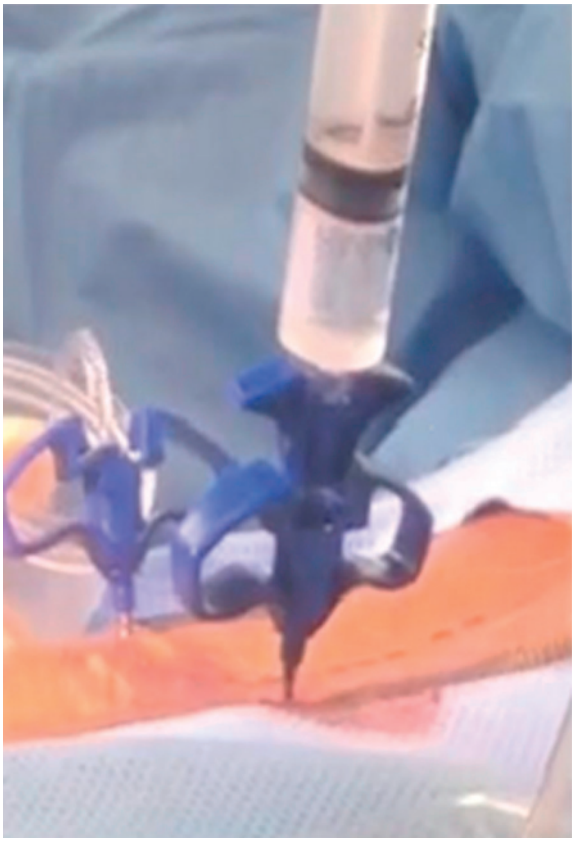


FIG 93.6 Intraoperative picture of bilateral transpedicular Jamshidi needle placement into the vertebral body abscess. Continuous irrigation is demonstrated with antibiotic solution delivered through the right Jamshidi needle in this image with clear outflow visualized streaming from the left Jamshidi needle. (From Urakov TM, Casabella AM, Levene HB. Percutaneous drainage of chronic destructive lumbar osteomyelitis abscess via the use of bilateral transpedicular trocar access. *World Neurosurg.* 2016;92;583.e1–583.e5.)

successful in draining epidural abscess percutaneously by using drainage catheters placed under CT or MRI guidance. Serial CT or MRI scans are useful in following the resolution of discitis and should be repeated immediately at the first sign of a negative change in the patient's neurologic status. If compression of the spinal cord and of associated neural structures is suspected, the clinician should follow the emergency treatment algorithm set forth in Box 93.1. Recent clinical reports suggest that intradiscal vancomycin may help prevent discitis.

DIFFERENTIAL DIAGNOSIS

The diagnosis of discitis should be strongly considered in any patient with spine pain and fever, especially if the patient has

BOX 93.1 Algorithm for Spinal Cord Compression Resulting From Discitis

- Immediately obtain blood and urine cultures.
- Immediately start high-dose antibiotics that cover *Staphylococcus aureus*.
- Immediately obtain the most readily available spinal imaging technique that can confirm the presence of spinal cord compression, such as abscess, tumor, and others:
  - Computed tomography
  - Magnetic resonance imaging
  - Myelography
- Simultaneously obtain emergency consultation from a spinal surgeon.
- Continuously and carefully monitor the patient's neurologic status.
- If any of the foregoing is unavailable, arrange emergency transfer of the patient to a tertiary care center by the most rapidly available transportation.
- Repeat imaging and obtain a repeat surgical consultation if any deterioration is noted in the patient's neurologic status.

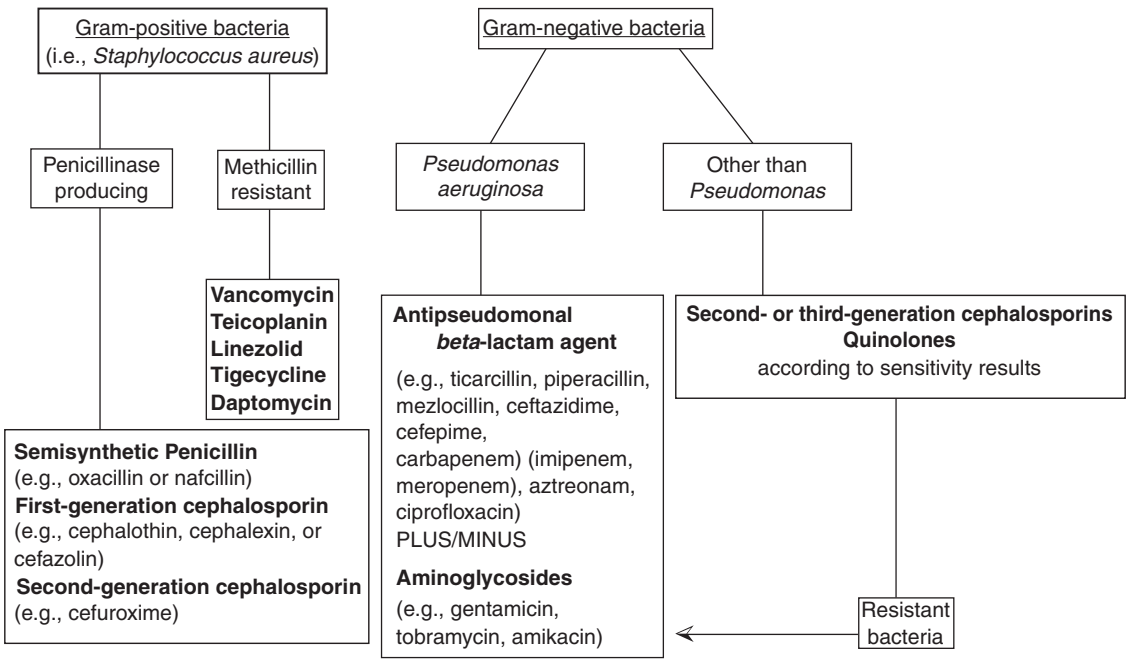


FIG 93.7 Antimicrobial treatment in pyogenic spontaneous spondylodiscitis. (From Skaf GS, Domloj NT, Fehlings MG, et al. Pyogenic spondylodiscitis: an overview. *J Infect Public Health.* 2010;3(1):5–16.)

undergone spinal instrumentation or epidural nerve blocks for either surgical anesthesia or pain control. Other pathologic processes that must be considered in the differential diagnosis include intrinsic disease of the spinal cord (e.g., demyelinating disease and syringomyelia), as well as other processes that can result in compression of the spinal cord and exiting nerve roots (e.g., metastatic tumor, Paget's disease, and neurofibromatosis). As a general rule, unless the patient has concomitant infection, none of these diseases will routinely be associated with fever, just with back pain.

COMPLICATIONS AND PITFALLS

Failure to diagnose and treat discitis rapidly and accurately can result only in disaster for the clinician and the patient alike. The insidious onset of neurologic deficit associated with discitis can lull the clinician into a sense of false security. If abscesses or other causes of spinal cord compression are suspected, a heightened index of suspicion for a subtle change in neurologic status must be considered.

CLINICAL PEARLS

Delay in diagnosis and treatment puts the patient and clinician at tremendous risk for a poor outcome. The clinician should assume that all patients who present with fever and back pain have discitis until proven otherwise and should treat these patients accordingly. Overreliance on a single negative or equivocal imaging test result is a mistake. Serial CT or MRI testing is indicated should any deterioration occur in the patient's neurologic status.

SUGGESTED READINGS

Cottle L, Riordan T. Infectious spondylodiscitis. *J Infect*. 2008;56(6):402–412.

Hoppe S, Oswald KAC, Renz N, et al. Neisseria meningitidis-induced discitis at L5–S1 mimicking lumbar disc herniation. *Lancet Infect Dis*. 2021;21(12):1758. https://doi.org/10.1016/S1473-3099(21)00352-2.

Luzzati R, Giacomazzi D, Danzi MC, et al. Diagnosis, management and outcome of clinically suspected spinal infection. *J Infect*. 2009;58(4):259–265.

Matthews E, Stokes O. Discitis and spinal infection. *Surgery (Oxford)*. 2018;36(7):352–356. https://doi.org/10.1016/j.mpsur.2018.03.017.

Mylona E, Samarkos M, Kakalou E, et al. Pyogenic vertebral osteomyelitis: a systematic review of clinical characteristics. *Semin Arthritis Rheum*. 2009;39(1):10–17.

Prodi E, Grassi R, Iacobellis F, et al. Imaging in spondylodiskitis. *Magn Reson Imaging Clin N Am*. 2016;24(3):581–600.

Samy DA, Gandham S, DeMatas M. The diagnosis and management of discitis and spinal infection. *Surgery (Oxford)*. 2021;39(8):540–546. https://doi.org/10.1016/j.mpsur.2021.07.001.

Sewell J, Sathish R, Seneviratne Epa D, et al. Rothia aeria vertebral discitis/osteomyelitis in an immunocompetent adult: case report and literature review. *IDCases*. 2022;27:e01459. https://doi.org/10.1016/j.idcr.2022.e01459.

Sharma SK, Jones JO, Zeballos PP, et al. The prevention of discitis during discography. *Spine J*. 2009;9(11):936–943.

Shroyer S, Boys G, April MD, et al. Imaging characteristics and CT sensitivity for pyogenic spinal infections. *Am J Emerg Med*. 2022. https://doi.org/10.1016/j.ajem.2022.05.050.

Skaf GS, Domloj NT, Fehlings MG, et al. Pyogenic spondylodiscitis: an overview. *J Infect Public Health*. 2010;3(1):5–16.

Tali ET, Oner AY, Koc AM. Pyogenic spinal infections. *Neuroimaging Clin N Am*. 2015;25(2):193–208.

Tirrell S, Handa S. Spinal infections: vertebral osteomyelitis, epidural abscess, diskitis. *Hosp Med Clin*. 2013;2(4):e509–e524.

Urakov TM, Casabella AM, Levene HB. Percutaneous drainage of chronic destructive lumbar osteomyelitis abscess via the use of bilateral transpedicular trocar access. *World Neurosurg*. 2016;92:583.e1–583.e5.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the lumbar spine. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:109–110.

Waldman SD, Campbell RSD. Bacterial diskitis and osteomyelitis of the lumbar spine. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:145–146.

Sacroiliac Joint Pain

ICD-10 CODE M53.3

THE CLINICAL SYNDROME

Pain from the sacroiliac joint commonly occurs when lifting in an awkward position that puts strain on the joint, its supporting ligaments, and soft tissues. The sacroiliac joint is also susceptible to the development of arthritis from various conditions that can damage the joint cartilage. Osteoarthritis is the most common form of arthritis that results in sacroiliac joint pain; rheumatoid arthritis and posttraumatic arthritis are also common causes of sacroiliac joint pain. Less common causes include the collagen vascular diseases such as ankylosing spondylitis, infection, and Lyme disease. Collagen vascular disease generally manifests as polyarthropathy rather than as monarthropathy limited to the sacroiliac joint, although sacroiliac pain secondary to ankylosing spondylitis responds exceedingly well to the intra-articular injection technique described later. Occasionally, patients present with iatrogenically induced sacroiliac joint dysfunction resulting from overaggressive bone graft harvesting for spinal fusion.

SIGNS AND SYMPTOMS

Most patients presenting with sacroiliac joint pain secondary to strain or arthritis complain of pain localized around the sacroiliac joint and upper leg that radiates into the posterior buttocks and backs of the legs (Fig. 94.1); the pain does not radiate below the knees. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. On physical examination, the affected sacroiliac joint is tender to palpation.

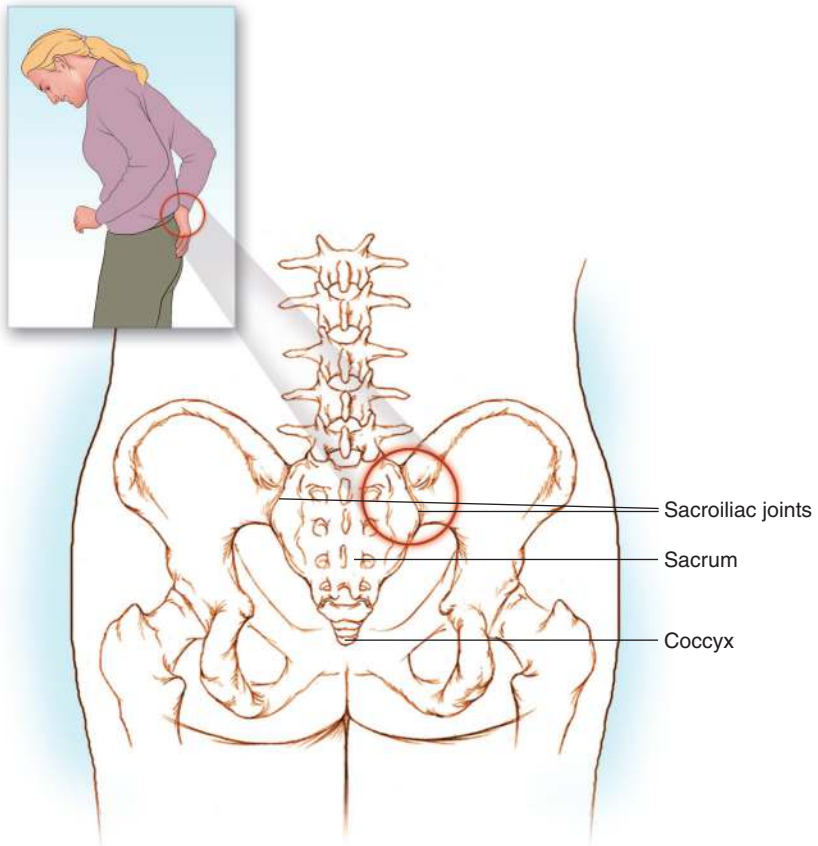


FIG 94.1 Sacroiliac joint pain radiates into the buttock and upper leg.

The patient often favors the affected leg and lists toward the unaffected side. Spasm of the lumbar paraspinal musculature is often present, as is limited range of motion of the lumbar spine in the erect position; the range of motion improves in the sitting position owing to relaxation of the hamstring muscles.

Patients with pain emanating from the sacroiliac joint exhibit a positive pelvic rock test result. This test is performed by placing the examiner's hands on the iliac crests and the thumbs on the anterior superior iliac spines and then forcibly compressing the patient's pelvis toward the midline. A positive test result is indicated by the production of pain around the sacroiliac joint. Other physical examination tests for sacroiliac joint dysfunction include the Yeoman, Gaenslen, Stork, Piedailu, and Van Durson tests (Fig. 94.2).

TESTING

Plain radiography is indicated in all patients who present with sacroiliac joint pain (Fig. 94.3). Because the sacrum is susceptible to stress fractures and to the development of infection and both primary and secondary tumors, magnetic resonance imaging of the distal lumbar spine and sacrum is indicated if the cause of the patient's pain is in question (Fig. 94.4). Computeed tomographic scanning and ultrasound imaging may also provide valuable clinical information (Fig. 94.5). Radionuclide bone scanning should also be considered in such patients to rule out tumor and insufficiency fractures that may be missed on conventional radiographs. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, human leukocyte antigen (HLA)-B27 screening, antinuclear antibody testing, and automated blood chemistry.

FIG 94.2 The Van Durson standing flexion test: as the patient bends forward, stress is placed on the sacroiliac joints. The patient will elevate the sacroiliac joint on the affected side in an effort to relieve the stress and resulting pain emanating from the painful joint. This will cause the examiner's thumb to rise. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier.)

DIFFERENTIAL DIAGNOSIS

Pain emanating from the sacroiliac joint can be confused with low back strain; lumbar bursitis; lumbar fibromyositis; piriformis syndrome; ankylosing spondylitis; inflammatory arthritis; and disorders of the lumbar spinal cord, roots, plexus, and nerves.

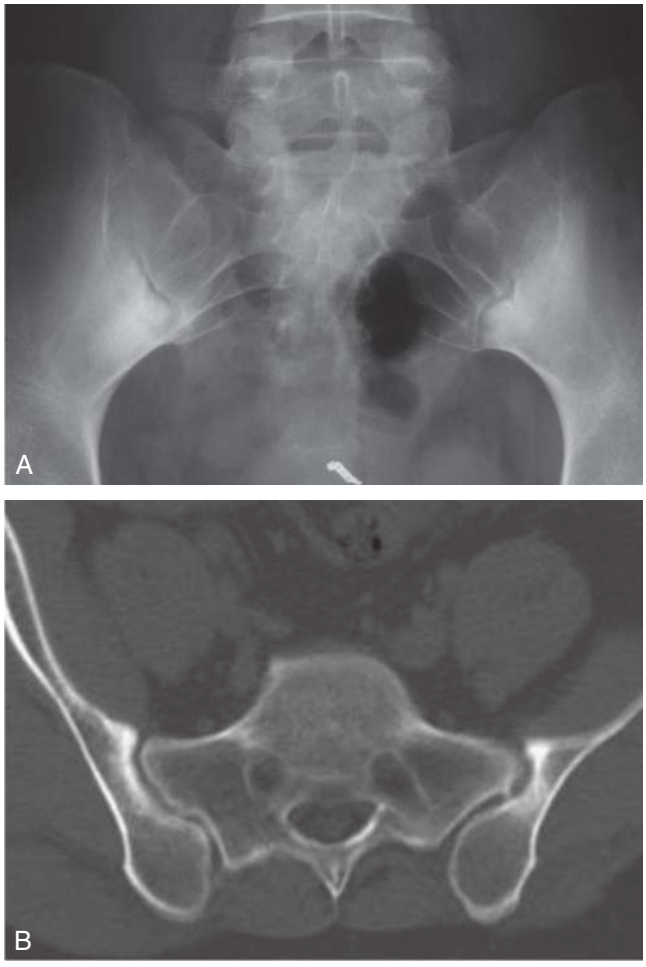


FIG 94.3 **A,** Anteroposterior (AP) radiograph of a young postpartum woman with sacroiliac joint (SIJ) pain. Sclerosis confined to the iliac aspect of both SIJs is caused by osteitis condensans ilii. **B,** An axial computed tomography (CT) scan in a different patient with stress-induced changes owing to athletic activity shows the same features of sclerosis of the iliac aspect of the SIJ. Note that, in both cases, the joint space is preserved and there is no loss of clarity of the subchondral bone plate and no erosive change. (From Waldman SD, Campbell RSD. Sacroiliac joint pain. In: *Imaging of pain*. Philadelphia: Elsevier; 2011.)

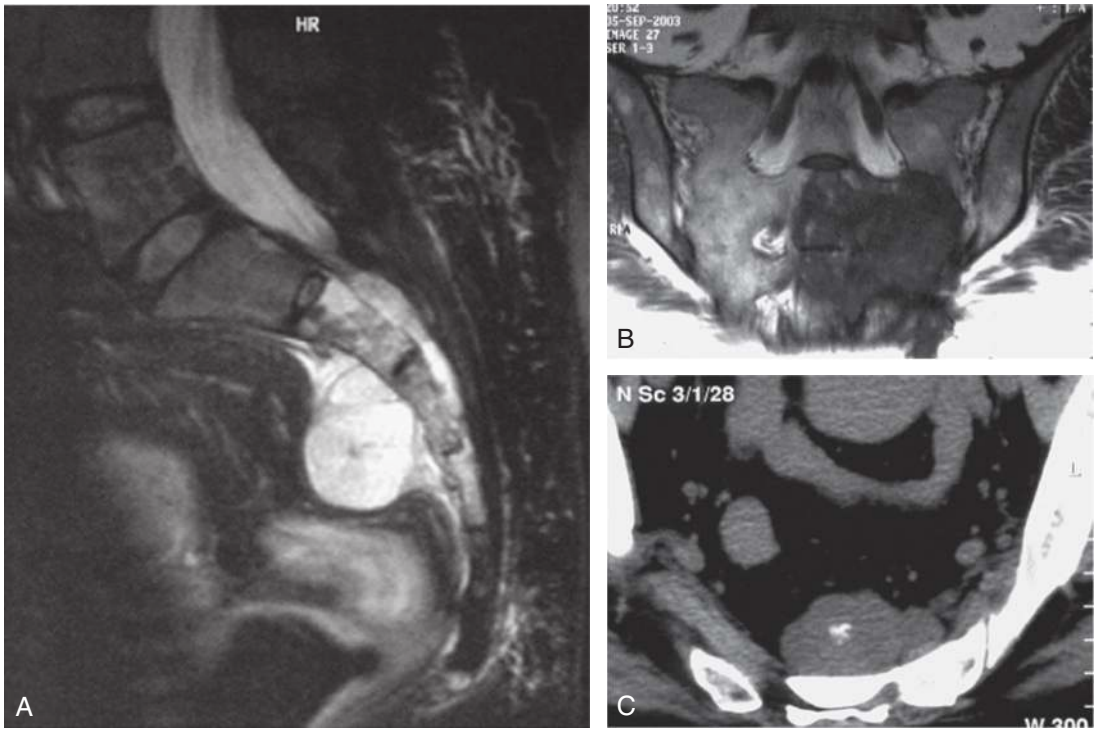


FIG 94.4 Sacral chordoma. Sagittal, fast spin-echo, T2-weighted **(A)** and axial T1-weighted **(B)** magnetic resonance images show a large soft tissue mass arising from the sacrum, with bony destruction. The bulk of the tumor is presacral. Axial computed tomography scan **(C)** demonstrates bony involvement of the left half of the sacrum by a large, midline, presacral mass with calcification. (From Edelman RR, Hesselink JR, Zlatkin MB, et al. eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2333.)

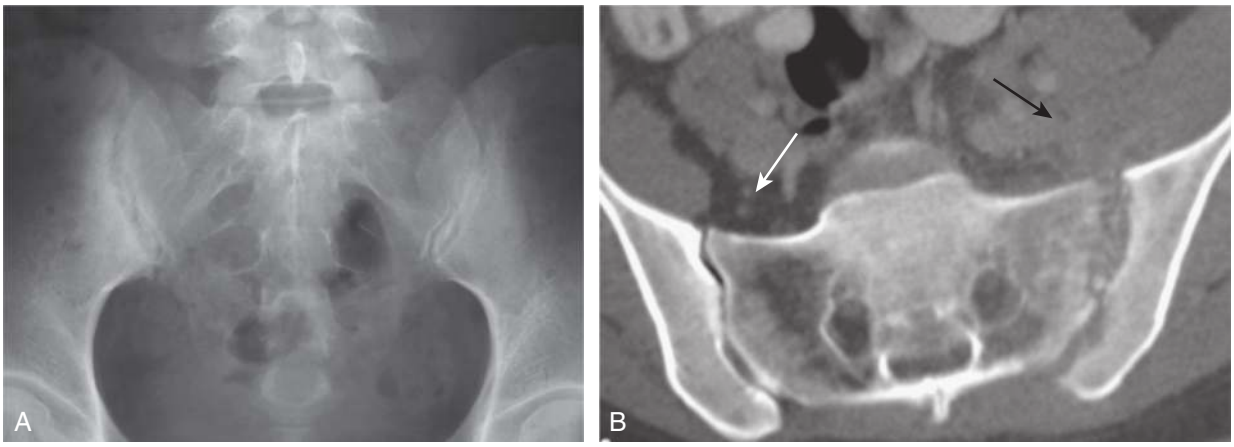


FIG 94.5 **A,** Anteroposterior (AP) radiograph of a patient with right-sided unilateral infective sacroiliitis. There is widening of the joint with loss of the crisp margin of the subchondral bone plate and ill-defined sclerosis. **B,** The axial computed tomography (CT) scan of a different patient with sacroiliac joint (SIJ) infection shows the same features in the left SIJ. Compare the anterior soft tissue inflammatory mass (*black arrow*) with the normal low-attenuation retroperitoneal fat on the opposite side (*white arrow*). (From Waldman SD, Campbell RSD. Sacroiliac joint pain. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:Fig. 78.3.)

TREATMENT

Initial treatment of the pain and functional disability of sacroiliac joint pain includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

Injection of the sacroiliac joint is carried out by placing the patient in the supine position and preparing the skin overlying the affected sacroiliac joint space with antiseptic solution. A sterile syringe containing 4-mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 25-gauge needle by using strict aseptic technique. The posterior superior spine of the ilium is identified. At this point, the needle is carefully advanced through the skin and subcutaneous tissues at a 45-degree angle toward the affected sacroiliac joint (Fig. 94.6). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly and slightly more laterally. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of fluoroscopy, computed tomography, and ultrasound guidance may be required in patients in whom the anatomic landmarks are difficult to identify (Figs. 94.7–94.9).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for sacroiliac pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. For instance, if the needle is inserted too laterally, it may traumatize the sciatic nerve. The major complication of intra-articular injection of the sacroiliac joint is infection, although it should be exceedingly rare if strict aseptic technique is followed, as well as universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after intra-articular injection, and patients should be warned of this possibility.

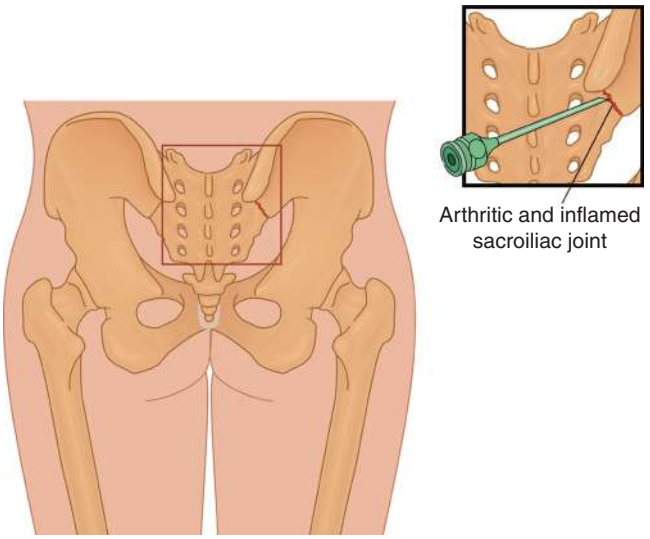


FIG 94.6 Correct needle placement for injection of the sacroiliac joint. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

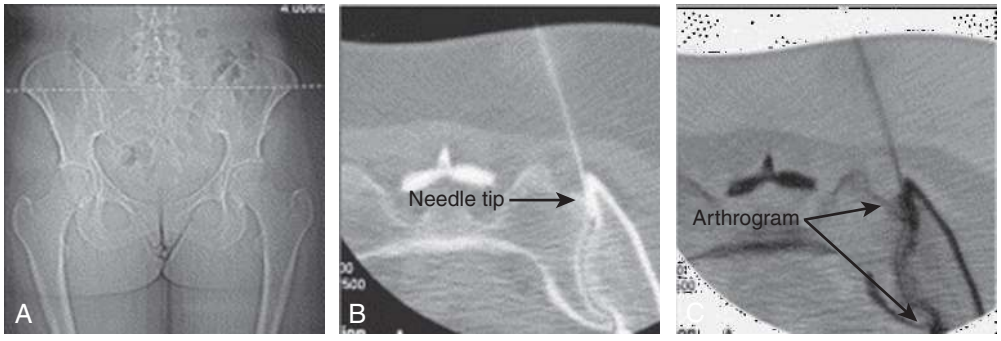


FIG 94.7 Computed tomography (CT) images from sacroiliac joint (SIJ) injection from an illustrative case. **A,** Scout tomogram for planned injection. **B,** Needle (22-gauge) directly inserted into the SIJ. **C,** CT image after injection of 1-mL Omnipaque 300. (From Block BM, Hobelmann G, Murphy KJ, et al. An imaging review of sacroiliac joint injection under computed tomography guidance. *Reg Anesth Pain Med*. 2005;30(3):295–298.)

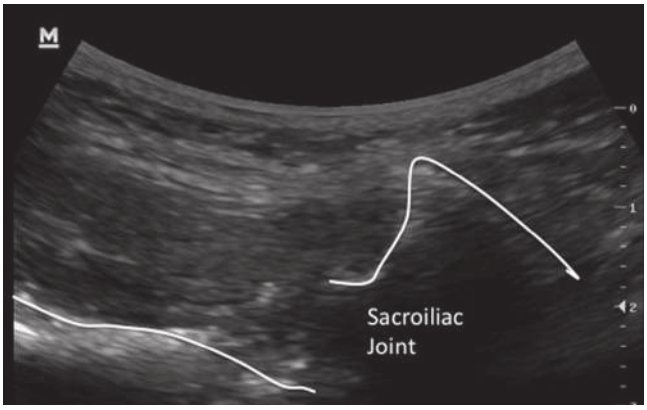


FIG 94.8 Ultrasound-guided injections may improve the accuracy of needle placement. Ultrasound image of the sacroiliac joint.

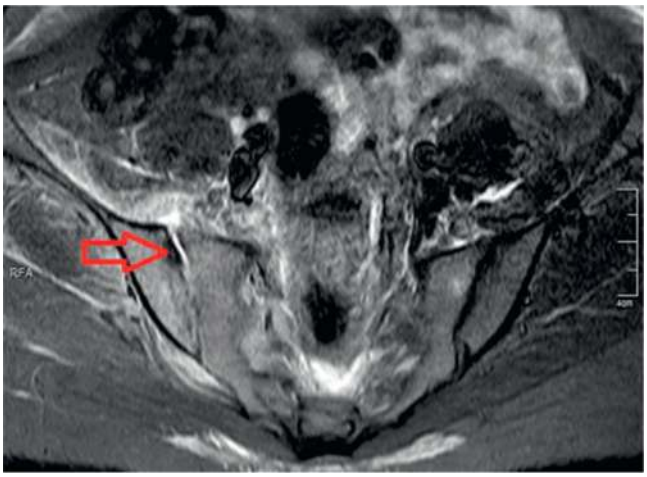


FIG 94.9 Simple nuclear magnetic resonance with inversion-recovery sequence (STIR) of sacroiliac joints. The *arrow* shows the increase of joint fluid as well as an erosion of the iliac bone. (From Carvajal-Flechas F, Sarmiento-Monroy JC, Rojas-Villarraga A, et al. Septic sacroiliitis in the late postpartum due to *Escherichia coli. Rev Colomb Reumatol (English Edition)*. 2016;23(2):131–136.)

CLINICAL PEARLS

Disorders of the sacroiliac joint can be distinguished from those of the lumbar spine by having the patient bend forward while seated. Patients with sacroiliac pain can bend forward with relative ease because of the relaxation of the hamstring muscles in this position. In contrast, patients with lumbar spine pain experience an exacerbation of symptoms when they bend forward while seated.

The injection technique described is extremely effective in the treatment of sacroiliac joint pain. Coexistent bursitis and tendinitis may contribute to sacroiliac pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Carvajal-Flechas F, Sarmiento-Monroy JC, Rojas-Villarraga A, et al. Septic sacroiliitis in the late postpartum due to *Escherichia coli*. *Rev Colomb Reumatol (English Edition)*. 2016;23(2):131–136.

Petrides S. Non-inflammatory sacroiliac joint disorders. *Indian J Rheumatol*. 2014;9(suppl 2):S54–S63.

Prakash J. Sacroiliac tuberculosis—a neglected differential in refractory low back pain—our series of 35 patients. *J Clin Orthop Trauma*. 2014;5(3):146–153.

Waldman SD. Intra-articular injection of the sacroiliac joint. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:446–450.

Waldman SD. Lumbar radiculopathy. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:223–224.

Waldman SD. Sacroiliitis and other abnormalities of the sacrococcygeal joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:710–716.

Waldman SD. The yeoman test for sacroiliac pain. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:254–255.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the sacroiliac joint and pelvis. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:192–196.

Waldman SD, Campbell RSD. Sacroiliac joint pain. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:197–200.

95

Osteitis Pubis

ICD-10 CODE M85.30

THE CLINICAL SYNDROME

Osteitis pubis causes localized tenderness over the symphysis pubis, pain radiating into the inner thigh, and a waddling gait. Radiographic changes consisting of erosion, sclerosis, and widening of the symphysis pubis are pathognomonic for osteitis pubis (Fig. 95.1). This is a disease of the second through fourth decades, and girls and women are affected more frequently than are boys and men. Osteitis pubis occurs most commonly after bladder, inguinal, or prostate surgery and is thought to result from the hematogenous spread of infection to the relatively avascular symphysis pubis. Osteitis pubis can also occur without an obvious inciting factor or infection.

SIGNS AND SYMPTOMS

On physical examination, patients exhibit point tenderness over the symphysis pubis, and the pain may radiate into the inner thigh with palpation of the symphysis pubis. Patients may also have tenderness over the anterior pelvis. The pain of osteitis pubis is aggravated by running, kicking, pivoting on one leg, and lying on the side. Patients often adopt a waddling gait to avoid movement of the symphysis pubis (Fig. 95.2). This dysfunctional gait may result in lower extremity bursitis and tendinitis, which can confuse the clinical picture and add to the patient's pain and disability.

TESTING

Plain radiography is indicated in all patients who present with pain thought to be emanating from the symphysis pubis to rule out occult bony disorders and tumor. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, C-reactive protein, serum protein electrophoresis, and antinuclear antibody testing. Magnetic resonance imaging of the pelvis is indicated if an occult mass

FIG 95.1 This 26-year-old woman developed pain and tenderness in the symphysis pubis during the third trimester of pregnancy. **A,B** Radiographs obtained 2 years apart reveal partial resolution of the abnormalities of osteitis pubis. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:2133.)

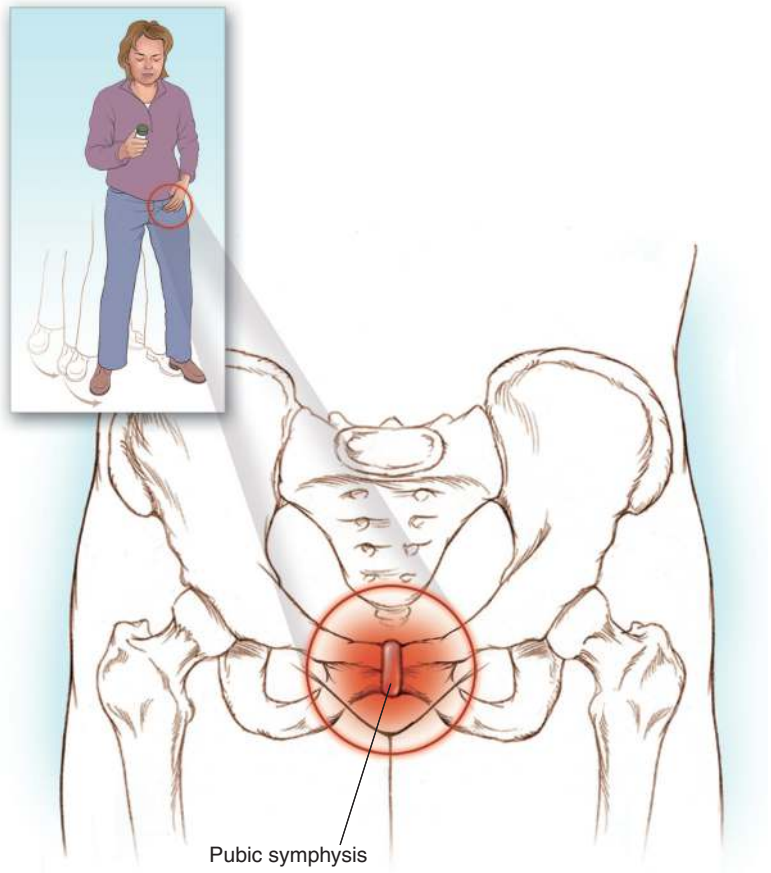


FIG 95.2 Patients with osteitis pubis often develop a waddling gait.

or tumor is suspected (Figs. 95.3–95.5). Radionuclide bone scanning may be useful to exclude stress fractures not visible on plain radiographs. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

A pain syndrome that is clinically similar to osteitis pubis may occur in patients with rheumatoid arthritis or ankylosing spondylitis; however, the characteristic radiographic changes of osteitis pubis are lacking. Adductor muscle strain and avulsion fractures may mimic the presentation of osteitis pubis. Multiple myeloma and metastatic tumor may also mimic the pain and radiographic changes of osteitis pubis. Insufficiency fractures of the pubic rami should be considered if generalized osteoporosis is present.

TREATMENT

Initial treatment of the pain and functional disability associated with osteitis pubis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

Injection for osteitis pubis is carried out by placing the patient in the supine position. The midpoints of the pubic bones and the symphysis pubis are identified by palpation, and the overlying skin is prepared with antiseptic solution. A syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 3½-inch, 25-gauge needle. The needle is advanced very slowly through the previously identified point at a right angle to the skin, directly toward the center of the symphysis pubis. Once the needle impinges on the fibroelastic cartilage of the joint, it is withdrawn slightly out of the joint. After careful aspiration for blood, and if no paresthesia is present, the contents of the syringe are gently injected. Resistance to injection should be minimal. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. This technique can also be used for prolotherapy of the pubic symphysis and for obtaining cultures of the joint (Fig. 95.6).

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics, NSAIDs, and antimyotonic agents such as tizanidine may be used concurrently with this injection technique.

B

FIG 95.3 In this 61-year-old woman with osteitis pubis, local pain, and tenderness about the symphysis pubis were the major clinical abnormalities. The radiograph **(A)** reveals considerable bone sclerosis on both sides of the symphysis, with narrowing of the joint space. A marked increase in the accumulation of a bone-seeking radiopharmaceutical agent is observed **(B)**. In this 34-year-old woman, a routine radiograph **(C)** shows unilateral osteitis pubis. A coronal T1-weighted spin-echo magnetic resonance image **(D)** shows low signal intensity in the involved bone. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:2132.)

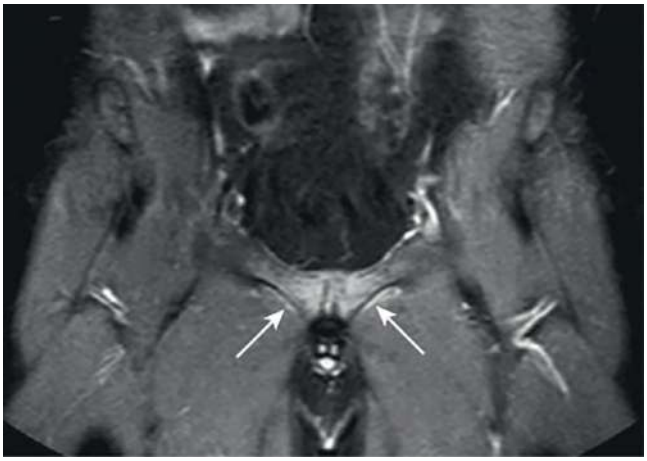


FIG 95.4 Coronal stir MR image demonstrates high signal intensity marrow edema in both pubic bones as well as periosteal edema consistent with osteitis pubis. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011:208. Fig. 82–2.)

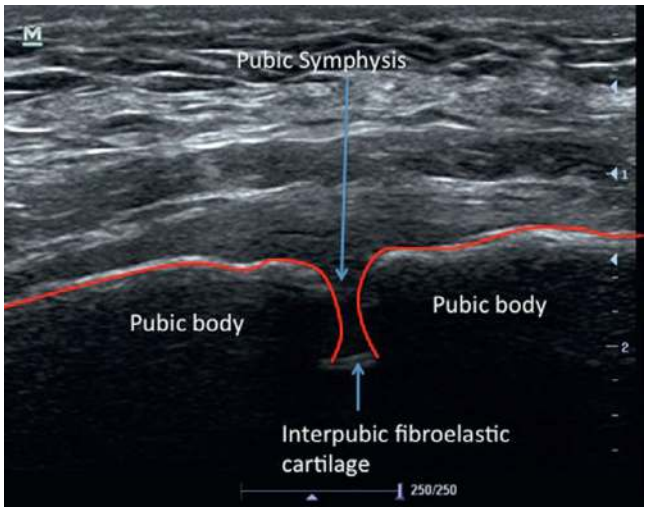


FIG 95.5 Transverse ultrasound view of the pubic symphysis and pubic bodies.

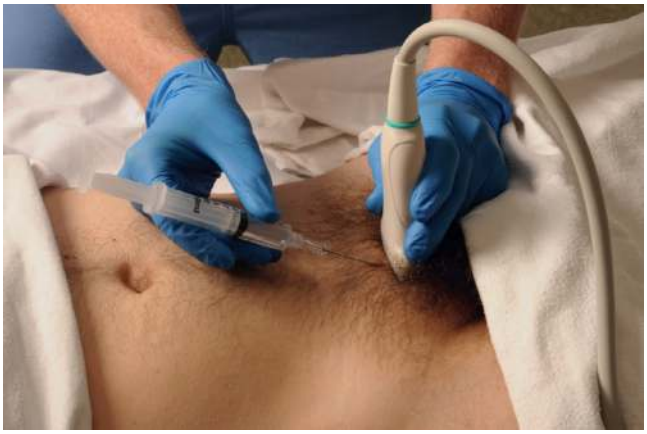


FIG 95.6 Proper needle placement for an ultrasound-guided out-of-plane injection for osteitis pubis.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The proximity to the pelvic contents makes it imperative that injection for osteitis pubis be performed only by those familiar with the regional anatomy and experienced in such techniques. Reactivation of latent infection, although rare, can occur; therefore strict attention to sterile technique is mandatory, along with universal precautions to minimize any risk to the operator. Most complications of the injection technique are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Many patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Osteitis pubis should be suspected in patients presenting with pain over the symphysis pubis in the absence of trauma. The injection technique described is an extremely effective treatment.

SUGGESTED READINGS

Budak MJ, Oliver TB. There's a hole in my symphysis—a review of disorders causing widening, erosion, and destruction of the symphysis pubis. *Clin Radiol*. 2013;68(2):173–180.

Elshout PJ, Verleyen P, Putzeys G. Osteitis pubis after TURP: a rare complication difficult to recognize. *Urol Case Rep*. 2016;4:55–58.

Glasser JG. Case report: osteitis/osteomyelitis pubis simulating acute appendicitis. *Int J Surg Case Rep*. 2018;53:269–272.

Waldman SD. Osteitis pubis and other abnormalities of the pubic symphysis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Kluwer Wolters; 2016:640–646.

Waldman SD. Injection technique for Osteitis pubis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:458–461.

Waldman SD. Osteitis pubis. In: *Atlas of common pain syndromes*. 4th ed. Philadelphia: Elsevier; 2017:311–314.

Waldman SD. Osteitis pubis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:296–297.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the sacroiliac joint and pelvis. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:192–196.

Waldman SD, Campbell RSD. Osteitis pubis. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:207–208.

96

Gluteus Maximus Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The gluteus maximus muscle's primary function is hip extension. It originates at the posterior aspect of the dorsal ilium, the posterior superior iliac crest, the posterior inferior aspect of the sacrum and coccyx, and the sacrotuberous ligament. The muscle inserts on the fascia lata at the iliotibial band and the gluteal tuberosity on the femur. The muscle is innervated by the inferior gluteal nerve.

The gluteus maximus muscle is susceptible to trauma and to wear and tear from overuse and misuse and to the development of myofascial pain syndrome, which may also be associated with gluteal bursitis. Such pain is usually the result of repetitive microtrauma to the muscle during such activities as running on soft surfaces, overuse of exercise equipment, or other repetitive activities that require hip extension (Fig. 96.1). Blunt trauma to the muscle may also incite gluteus maximus myofascial pain syndrome.

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective

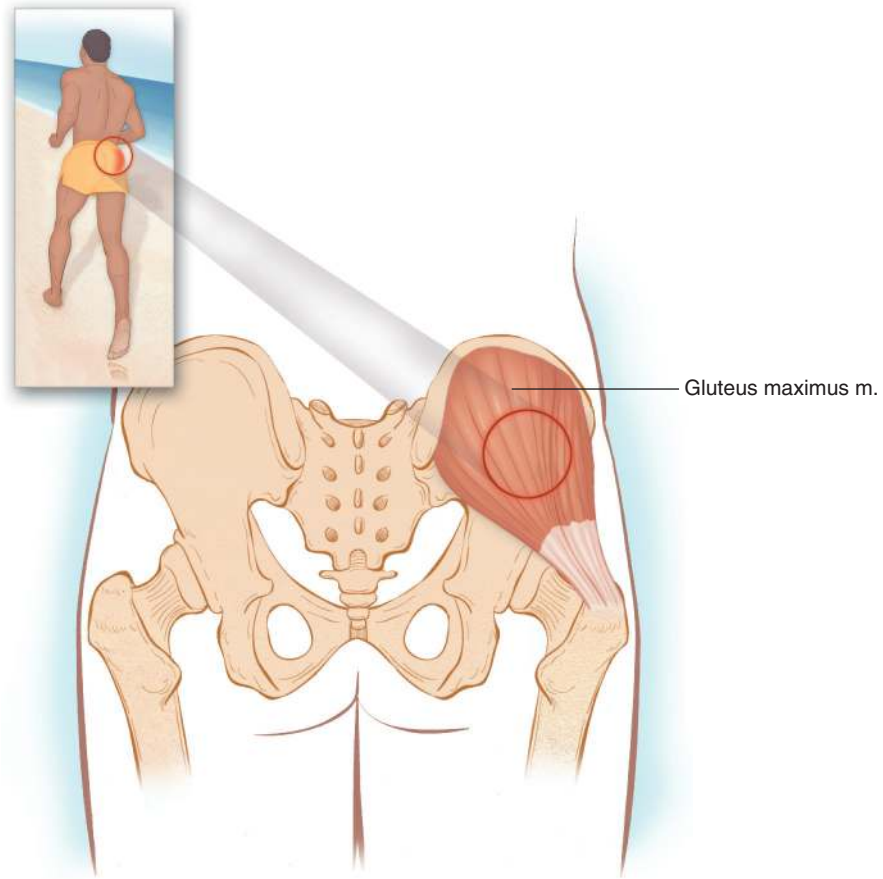


FIG 96.1 Gluteus maximus syndrome usually results from repetitive microtrauma to the muscle during such activities as running on soft surfaces, overuse of exercise equipment, or other repetitive activities that require hip extension.

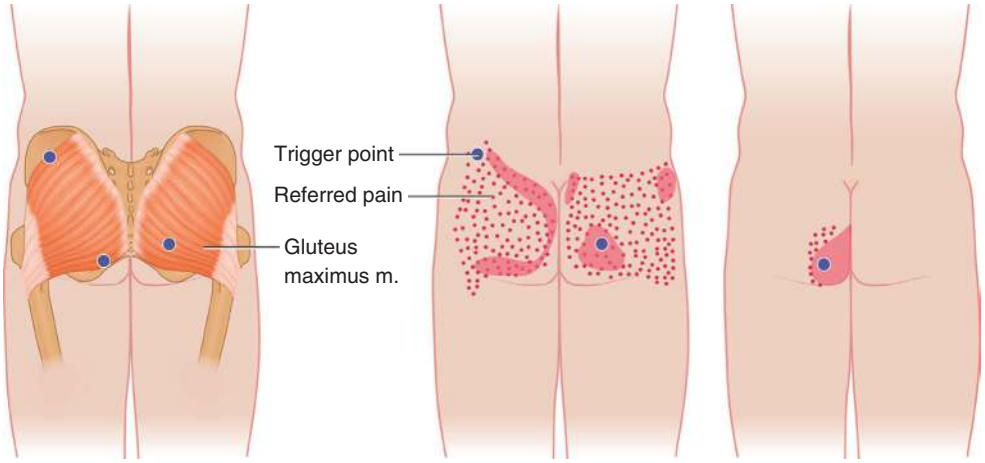


FIG 96.2 Patients with myofascial pain syndrome involving the gluteus maximus have primary pain in the medial and lower aspects of the muscle that is referred across the buttocks and into the coccygeal area. (From Waldman SD. Gluteus maximus syndrome. In: *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2007:379.)

treatment. Patients with myofascial pain syndrome involving the gluteus maximus have primary pain in the medial and lower aspects of the muscle that is referred across the buttocks and into the coccygeal area (Fig. 96.2).

The trigger point is the pathognomonic lesion of myofascial pain syndrome and is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, often occurs and is characteristic of myofascial pain syndrome. Patients with gluteus maximus syndrome have a trigger point over the upper, medial, and lower aspects of the muscle (see Fig. 96.1).

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to be caused by microtrauma to the affected muscle. This trauma may result from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The gluteus maximus muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point is the pathognomonic lesion of gluteus maximus syndrome, and it is characterized by a local point of exquisite tenderness in the gluteus maximus muscle. Mechanical stimulation of the trigger point by palpation or stretching produces both intense local pain in the medial and lower aspects of the muscle and referred pain across the buttocks and into the coccygeal area (see Fig. 96.2). In addition, the jump sign is often present.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger points has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with gluteus maximus syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic gluteus maximus syndrome (see "Differential Diagnosis").

DIFFERENTIAL DIAGNOSIS

The diagnosis of gluteus maximus syndrome is based on clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from gluteus maximus syndrome. The clinician must rule out other coexisting disease processes that may mimic gluteus maximus syndrome, including primary inflammatory muscle disease, primary hip disorders, gluteal bursitis, occult tumors, and gluteal nerve entrapment (Figs. 96.3 and 96.4). The use of electrodiagnostic and radiographic testing can identify coexisting disorders such as rectal or pelvic tumors or lumbosacral nerve lesions. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms associated with gluteus maximus syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the

A

FIG 96.3 Possible entrapment of the superior gluteal nerve. **A,** Transverse, T1-weighted, spin-echo magnetic resonance imaging (MRI) shows denervation hypertrophy of the tensor fasciae latae muscle (*arrow*). **B,** Similar hypertrophy and high signal intensity are seen in the muscle (*arrow*) on transverse, fat-suppressed, T1-weighted, spin-echo MRI obtained after intravenous gadolinium administration. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3551.)

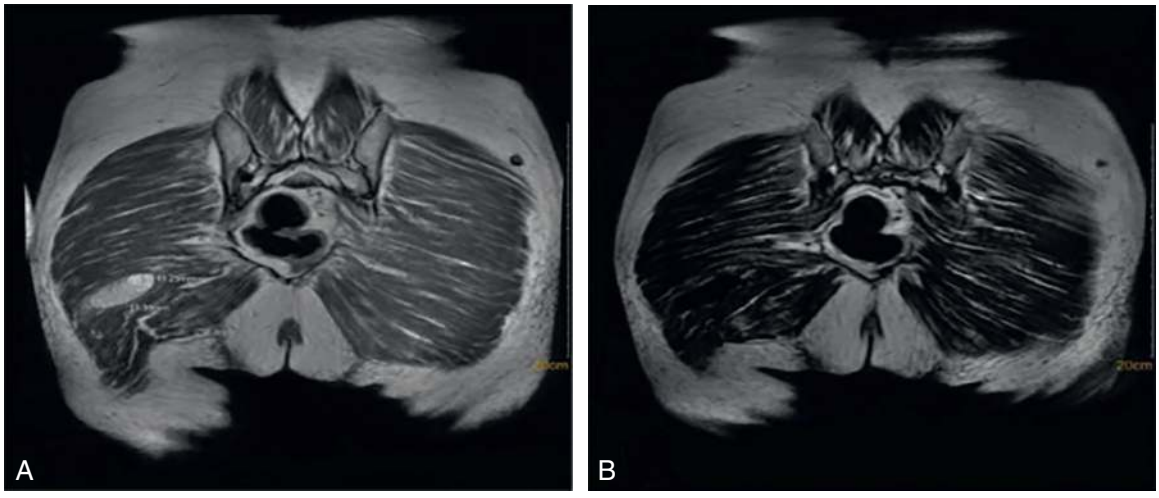


FIG 96.4 Magnetic resonance imaging revealing a 1.9 cm × 4.9 cm mass in the right gluteus maximus (**A**, **B**). (From Liu W, Xu X, Zhang Z, et al. Gluteus maximus metastasis from sacrococcygeal chordoma: a case report. *Radiol Case Rep*. 2022;17(1):46–49.)

starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin-norepinephrine reuptake inhibitor, has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections and antidepressants to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid trauma to the sciatic nerve.

CLINICAL PEARLS

Although gluteus maximus syndrome is a common disorder, it is often misdiagnosed. Therefore, in patients suspected of suffering from gluteus maximus syndrome, a careful evaluation to identify underlying disease processes is mandatory. Gluteus maximus syndrome often coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Andrade A, de Azevedo Klumb Steffens R, Torres Vilarino G, et al. Does volume of physical exercise have an effect on depression in patients with fibromyalgia? *J Affect Disord*. 2017;208:214–217.

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in fibromyalgia syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208:87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Goldenberg DL, Clauw DJ, Palmer RE, et al. Opioid use in fibromyalgia: a cautionary tale. *Mayo Clin Proc*. 2016;91(5):640–648.

Liu W, Xu X, Zhang Z, et al. Gluteus maximus metastasis from sacrococcygeal chordoma: a case report. *Radiol Case Rep*. 2022;17(1):46–49. https://doi.org/10.1016/j.radcr.2021.09.062.

Salah R, Bouya A, Bennis A, et al. Gluteus maximus abscess revealing a tuberculous arthritis: a case report and review of the literature. *Indian J Tuberc*. 2022;69(4):710–714. https://doi.org/10.1016/j.ijtb.2021.10.001.

Sluka KA, Clauw DJ. Neurobiology of fibromyalgia and chronic widespread pain. *Neuroscience*. 2016;338:114–129.

Waldman SD. Injection technique for gluteus maximis syndrome. In: *Atlas of pain management injection techniqes*. 4th ed. Philadelphia: Elsevier; 2017:435–437.

Wolfe F, Clauw DJ, Fitzcharles M-A, et al. 2016 revisions to the 2010/2011 fibromyalgia diagnostic criteria. *Semin Arthritis Rheum*. 2016;46(3):319–329.

97

Piriformis Syndrome

ICD-10 CODEG57.00

THE CLINICAL SYNDROME

Piriformis syndrome is an entrapment neuropathy that presents as pain, numbness, paresthesias, and weakness in the distribution of the sciatic nerve. It is caused by compression of the sciatic nerve by the piriformis muscle as it passes through the sciatic notch (Figs. 97.1 and 97.2). The piriformis muscle's primary function is to rotate the femur externally at the hip joint; this muscle is innervated by the sacral plexus. With internal rotation of the femur, the tendinous insertion and belly of the muscle can compress the sciatic nerve; if this compression persists, it can cause entrapment of the nerve (Fig. 97.3).

The symptoms of piriformis syndrome usually begin after direct trauma to the sacroiliac and gluteal regions. Occasionally, the syndrome is the result of repetitive hip and lower extremity motions or repeated pressure on the piriformis muscle and underlying sciatic nerve. Abscess, tumor, or hematoma in this region can mimic the clinical presentation of piriformis syndrome (Fig. 97.4).

SIGNS AND SYMPTOMS

Initial symptoms include severe pain in the buttocks that may radiate into the lower extremity and foot. Patients suffering from piriformis syndrome may develop an altered gait, leading to coexistent sacroiliac, back, and hip pain that confuses the clinical picture. Physical findings include tenderness over the sciatic notch. Palpation of the piriformis muscle reveals tenderness and a swollen, indurated muscle belly. A positive Tinel's sign over the sciatic nerve as it passes beneath the piriformis muscle is often present. A positive straight leg raising test result suggests sciatic nerve entrapment. The piriformis muscle provocation test and piriformis test can help identify

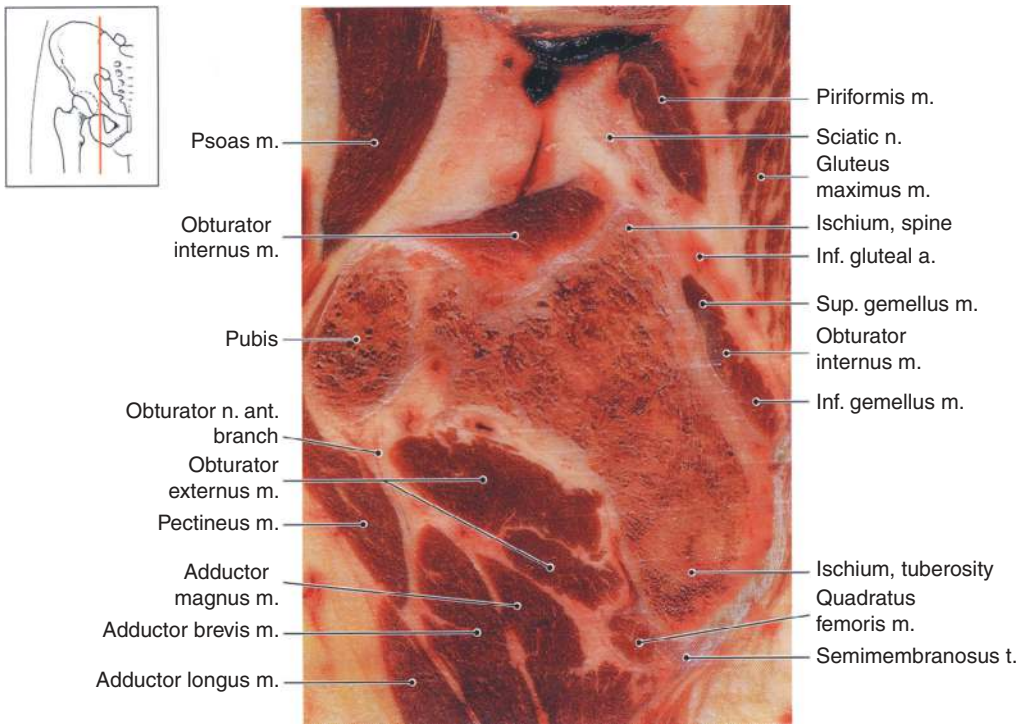


FIG 97.1 Anatomic relationship between the piriformis muscle and the sciatic nerve. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas*. 2nd ed. Philadelphia: Saunders; 2002:251.)

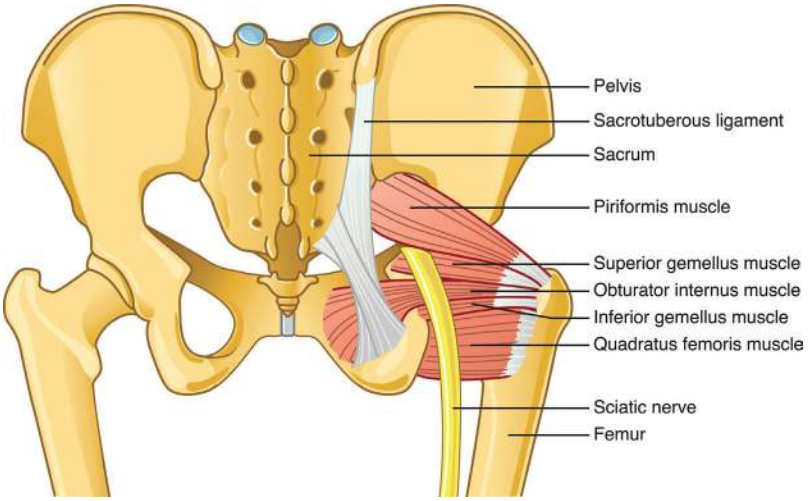


FIG 97.2 The nerve roots that make up the sciatic nerve fuse in front of the anterior surface of the lateral sacrum on the anterior surface of the piriformis muscle. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015.)

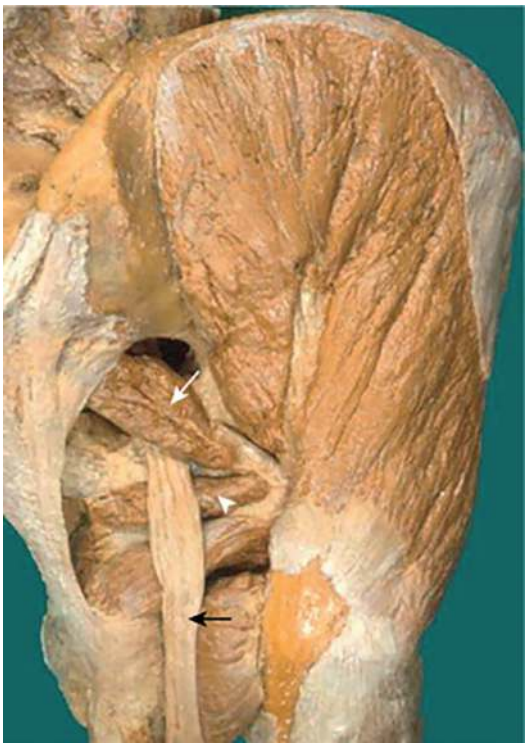


FIG 97.3 Posterior view of the right gluteal region. The sciatic nerve (*black arrow*) passes through the infrapiriform foramen, bordered superiorly by the piriformis muscle (*white arrow*) and inferiorly by the portion of the obturator internus muscle that is outside the pelvis (*arrowhead*). (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. St Louis: Elsevier; 2016.)

the piriformis muscle as the possible cause of sciatic nerve entrapment (Figs. 97.5 and 97.6).

Lifting or bending at the waist and hips increases the pain in most patients suffering from piriformis syndrome (Fig. 97.7). Weakness of the affected gluteal muscles and lower extremity and, ultimately, muscle wasting are seen in patients with advanced, untreated cases of piriformis syndrome.

TESTING

Electromyography (EMG) can distinguish lumbar radiculopathy from piriformis syndrome. Plain radiographs of the back, hip, and pelvis are indicated in all patients who present with piriformis syndrome to rule out occult bony disorders. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) of the back is indicated if herniated disk, spinal stenosis, or space-occupying lesion is suspected. MRI and ultrasound imaging of the hip and piriformis muscle may elucidate the cause of compression of the sciatic nerve (Figs. 97.8–97.11). Injection in the region of the sciatic nerve at this level serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Piriformis syndrome is often misdiagnosed as lumbar radiculopathy or primary hip disease; radiographs of the hip and EMG can make the distinction. In addition, most patients with lumbar radiculopathy have back pain associated with reflex, motor, and sensory changes, whereas patients with piriformis syndrome have only secondary back pain and no reflex changes. The motor and sensory changes of piriformis syndrome are limited to the distribution of the sciatic nerve below the sciatic notch. Lumbar radiculopathy and sciatic nerve entrapment may coexist as the double-crush syndrome.

TREATMENT

Initial treatment of the pain and functional disability associated with piriformis syndrome includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2

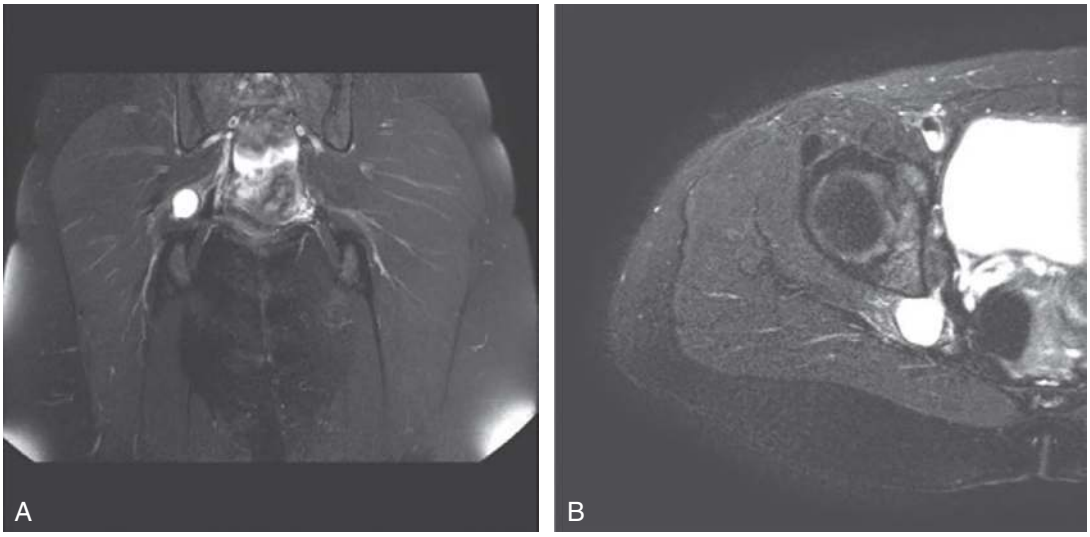


FIG 97.4 Magnetic resonance imaging (MRI) findings of a 32-year-old female patient. Coronal **(A)** and axial **(B)** MRI images demonstrating about a 2 cm sized multilobulated cystic mass located in the piriformis muscle that showed high intensity on T2-weighted and low intensity on T1-weighted image. (From Park JH, Jeong HJ, Shin HK, et al. Piriformis ganglion: an uncommon cause of sciatica. *Orthop Traumatol Surg Res*. 2016;102(2):257–260.)

FIG 97.5 The piriformis syndrome provocation test is positive in patients suffering from piriformis syndrome. To perform the piriformis syndrome provocation test, the patient is placed in the modified Sims position with the affected leg superior. The hip of the affected leg is then flexed approximately 50 degrees and, while stabilizing the pelvis, the affected leg is pushed downward.

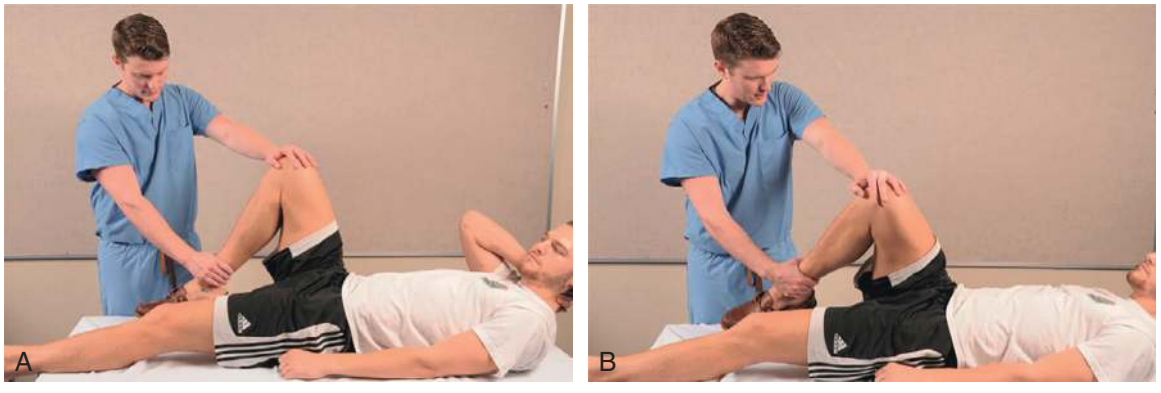


FIG 97.6 **A, B,** The piriformis test. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. St Louis: Elsevier; 2016.)

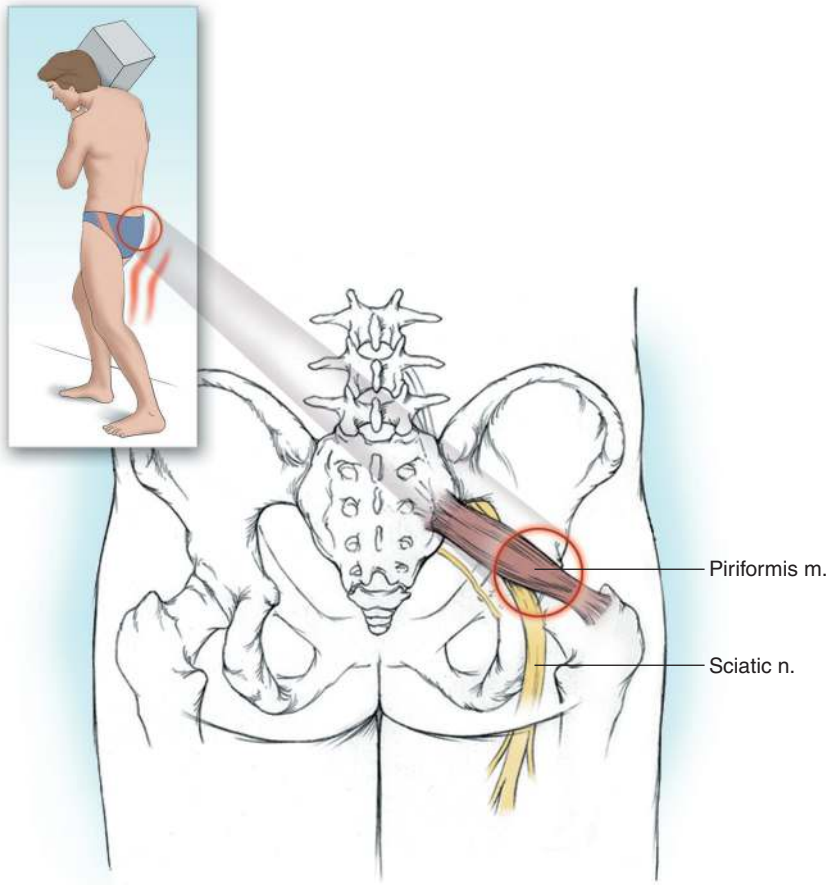


FIG 97.7 The pain of piriformis syndrome can be exacerbated by lifting.

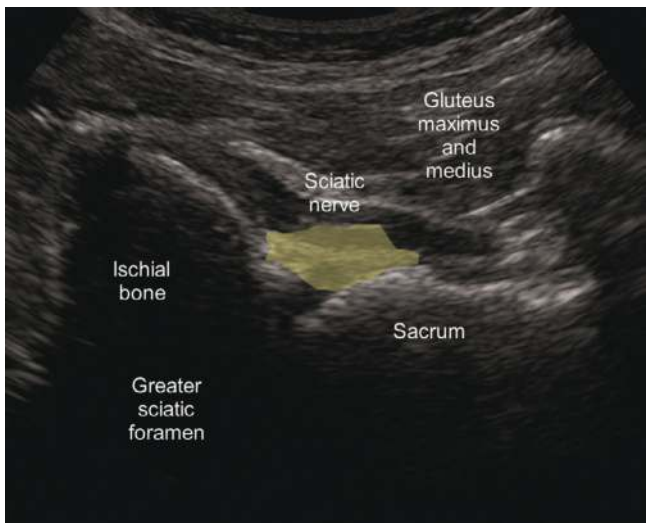


FIG 97.8 Transverse ultrasound image showing the curved hyperechoic margins of the sacrum and ischial bone. The sciatic nerve is visualized as a flattened hyperechoic structure lying between the hyperechoic curves of the sacrum and ischium. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015.)

inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. If the patient sleeps on his or her side, placing a pillow between the legs may be helpful. If the patient is suffering from significant paresthesias, gabapentin may be added. For patients who do not respond to these treatment modalities, injection of local anesthetic and methylprednisolone in the region of the sciatic nerve at the level of the piriformis muscle is a reasonable next step (Fig. 97.12). Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-induced complications. Rarely, surgical release of the entrapment is required to obtain relief.

COMPLICATIONS AND PITFALLS

The main complications of injection in the region of the sciatic nerve are ecchymosis and hematoma. Because paresthesia is elicited with the injection technique, needle-induced trauma to the sciatic nerve is a possibility. By advancing the needle slowly and withdrawing the needle slightly away from the nerve, injury to the sciatic nerve can be avoided.

FIG 97.9 **A,** Axial T1-weighted pelvic magnetic resonance neurography (MRN) showing left piriformis muscle asymmetry and atrophy. **B,** Axial postcontrast fat-saturated T1-weighted pelvic MRN showing left ischial bone marrow edema and hamstring tendinopathy. The *left arrow* in **(A)** shows an atrophic and asymmetric piriformis muscle, and the *arrow* in **(B)** shows bone marrow edema and tendinopathy. (From Kulcu DG, Naderi S. Differential diagnosis of intraspinal and extraspinal non-discogenic sciatica. *J Clin Neurosci*. 2008;15(11):1246–1252.)

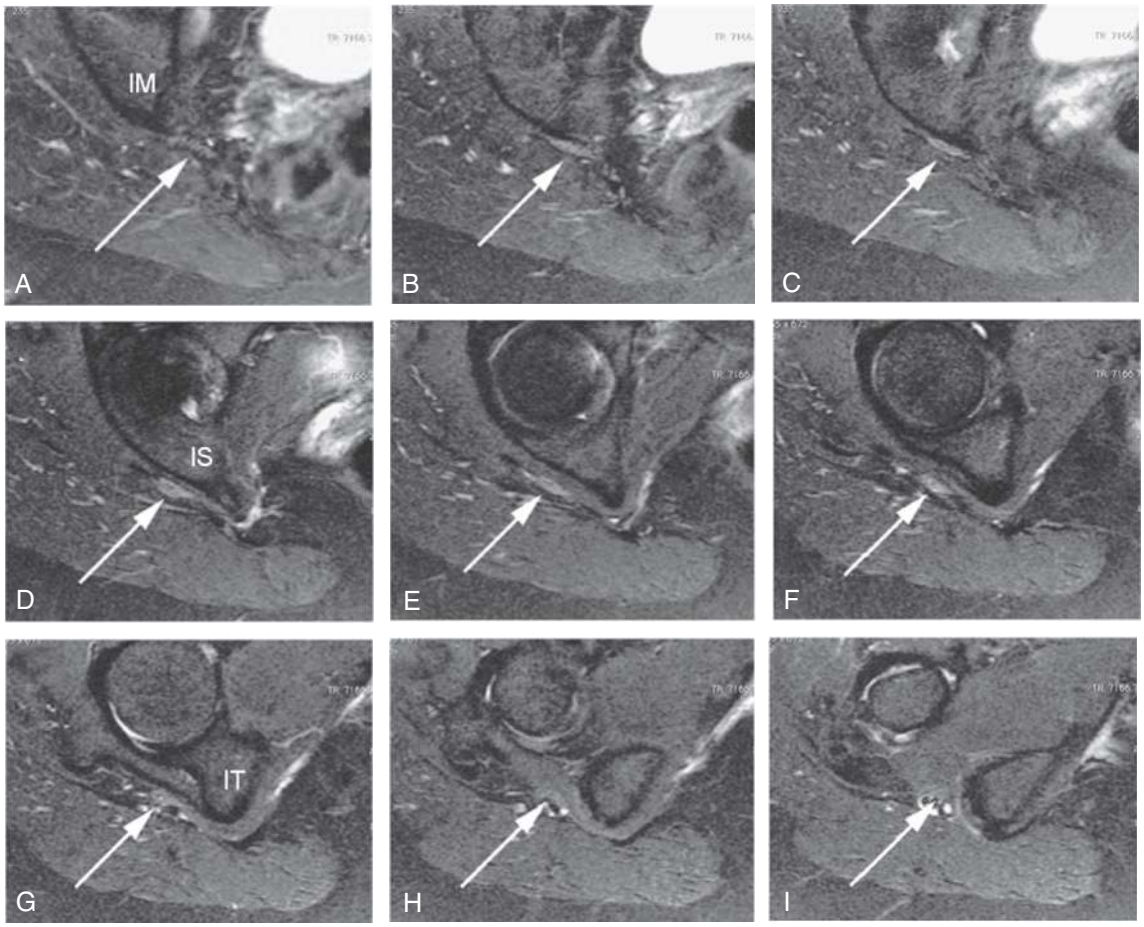


FIG 97.10 Typical findings of irritative abnormalities of the sciatic nerve in piriformis syndrome (nerve perpendicular oblique view, magnetic resonance neurographic acquisition sequence). **A,** The sciatic nerve is bowed over the medial surface of the piriformis muscle. **B,** Nerve image intensity increases as the nerve passes between the piriformis tendon and the ischial margin. **C–F,** The image intensity increase persists as the nerve descends through the ischial tunnel. **G–I,** Nerve image intensity progressively normalizes, with the nerve becoming isointense with surrounding muscle as it descends into the upper thigh. (*Arrows*) indicate the sciatic nerve. *IM*, Ischial margin; *IS*, ischial spine; *IT*, ischial tuberosity. (From Filler AG. Piriformis and related entrapment syndromes: diagnosis and management. *Neurosurg Clin N Am*. 2008;19(4):609–622; Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015.)

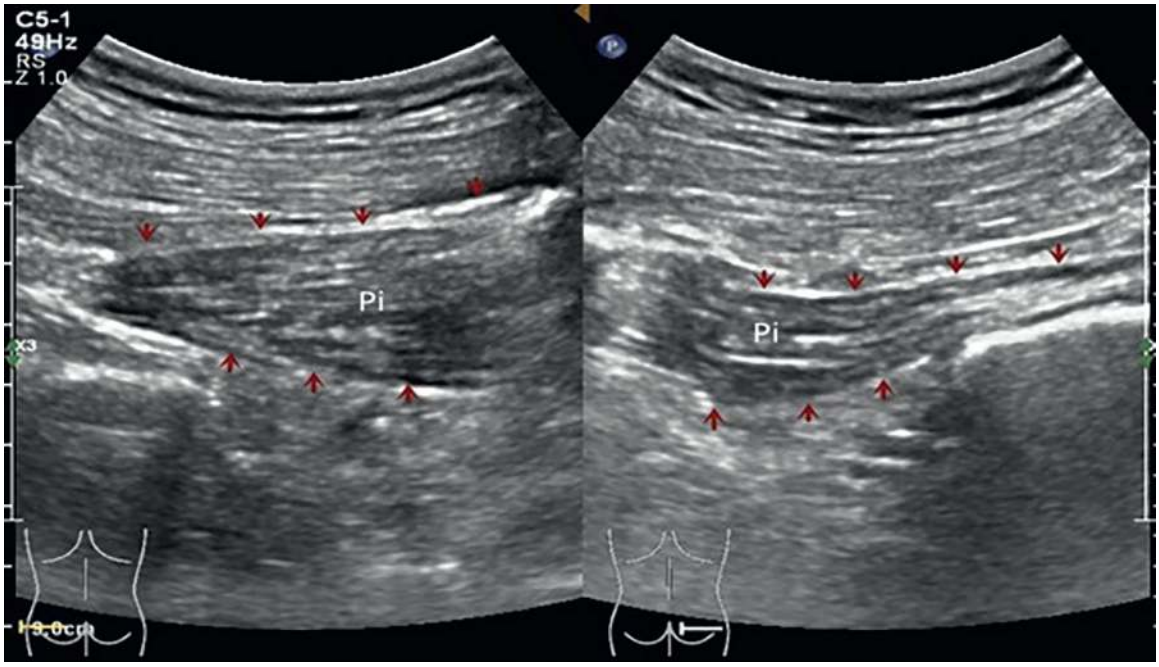


FIG 97.11 Representative ultrasound images of the piriformis (Pi) of one patient with piriformis syndrome in the long-axis view. The patient was a 36-year-old man; a driver with pain in the left buttock for 1 month's duration. Real-time duplex ultrasound scan showing the long-axis view of the piriformis on two sides. The piriformis on the abnormal side (*left*) was thicker than that on the asymptomatic side (*right*), accompanied by a decreased echo intensity and blurry muscular texture inside. *Red arrow* indicates the epimysium of the piriformis. (From Wu Y-Y, Guo X-Y, Chen K, et al. Feasibility and Reliability of an ultrasound examination to diagnose piriformis syndrome. *World Neurosurg*. 2020;134:e1085–e1092.)

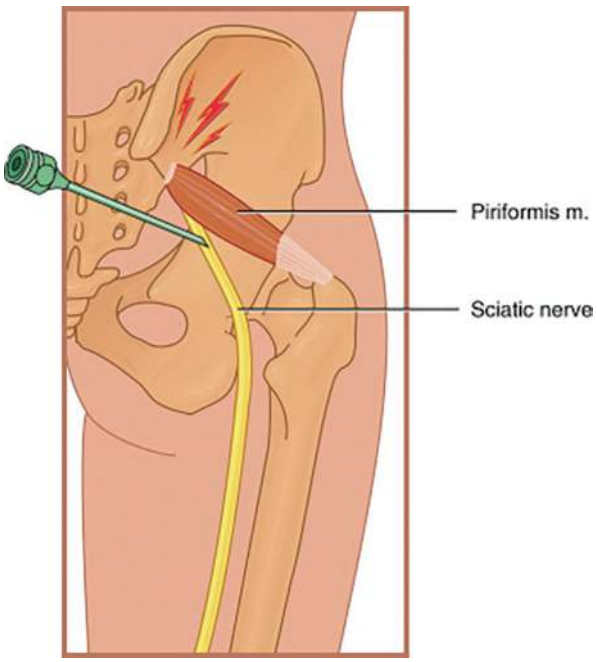


FIG 97.12 Injection technique for piriformis syndrome. (From Waldman SD. *Atlas of pain management injection techniques*. 4th ed. St Louis: Elsevier; 2017.)

CLINICAL PEARLS

Because patients suffering from piriformis syndrome may develop an altered gait, resulting in coexistent sacroiliac, back, and hip pain, careful physical examination and appropriate testing are required to sort out the diagnostic possibilities.

SUGGESTED READINGS

Fahmi A, Rahmadhan MA, Aprianto DR, et al. Complete resolution of recurrent piriformis syndrome after piriformis resection with 3 years' follow up: a case report. *Int J Surg Case Rep*. 2020;77:576–579. https://doi.org/10.1016/j.ijscr.2020.11.099.

Fritz J, Chhabra A, Wang KC, et al. Magnetic resonance neurography–guided nerve blocks for the diagnosis and treatment of chronic pelvic pain syndrome. *Neuroimaging Clin N Am*. 2014;24(1):211–234.

Michel F, Decavel P, Toussirot E, et al. The piriformis muscle syndrome: an exploration of anatomical context, pathophysiological hypotheses and diagnostic criteria. *Ann Phys Rehabil Med*. 2013;56(4):300–311.

Najdi H, Mouarbes D, Abi-akl J, et al. EMG in piriformis syndrome diagnosis: reliability of peroneal H-reflex according to results obtained after surgery, Botox injection and medical treatment.

J Clin Neurosci. 2019;59:55–61. https://doi.org/10.1016/j.jocn.2018.11.007.

Park JH, Jeong HJ, Shin HK, et al. Piriformis ganglion: an uncommon cause of sciatica. *Orthop Traumatol Surg Res*. 2016;102(2):257–260.

Vanermen F, Van Melkebeek J. Endoscopic treatment of piriformis syndrome results in a significant improvement in pain visual analog scale scores. *Arthrosc Sports Med Rehabil*. 2022;4(2):e309–e314. https://doi.org/10.1016/j.asmr.2021.10.002.

Waldman SD. Injection technique for piriformis syndrome. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:531.

Waldman SD. Piriformis syndrome. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:297–298.

Wu Y-Y, Guo X-Y, Chen K, et al. Feasibility and reliability of an ultrasound examination to diagnose piriformis syndrome. *World Neurosurg*. 2020;134:e1085–e1092. https://doi.org/10.1016/j.wneu.2019.11.098.

Ischiogluteal Bursitis

ICD-10 CODE M70.70

THE CLINICAL SYNDROME

The ischial bursa lies between the gluteus maximus muscle and the bone of the ischial tuberosity. It may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The ischial bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are often caused by direct trauma to the bursa from falls onto the buttocks or by overuse, such as prolonged riding of horses or bicycles. Running on uneven or soft surfaces such as sand may also cause ischiogluteal bursitis (Fig. 98.1). If inflammation of the ischial bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Patients suffering from ischiogluteal bursitis frequently complain of pain at the base of the buttock with resisted extension of the lower extremity. The pain is localized to the area over the ischial tuberosity; referred pain is noted in the hamstring muscle, which may develop coexistent tendinitis. Patients are often unable to sleep on the affected hip and may complain of a sharp, catching sensation when they extend and flex the hip, especially on first awakening. Physical examination may reveal point tenderness over the ischial tuberosity. Passive straight leg raising and active resisted extension of the affected lower extremity reproduce the pain (Fig. 98.2).

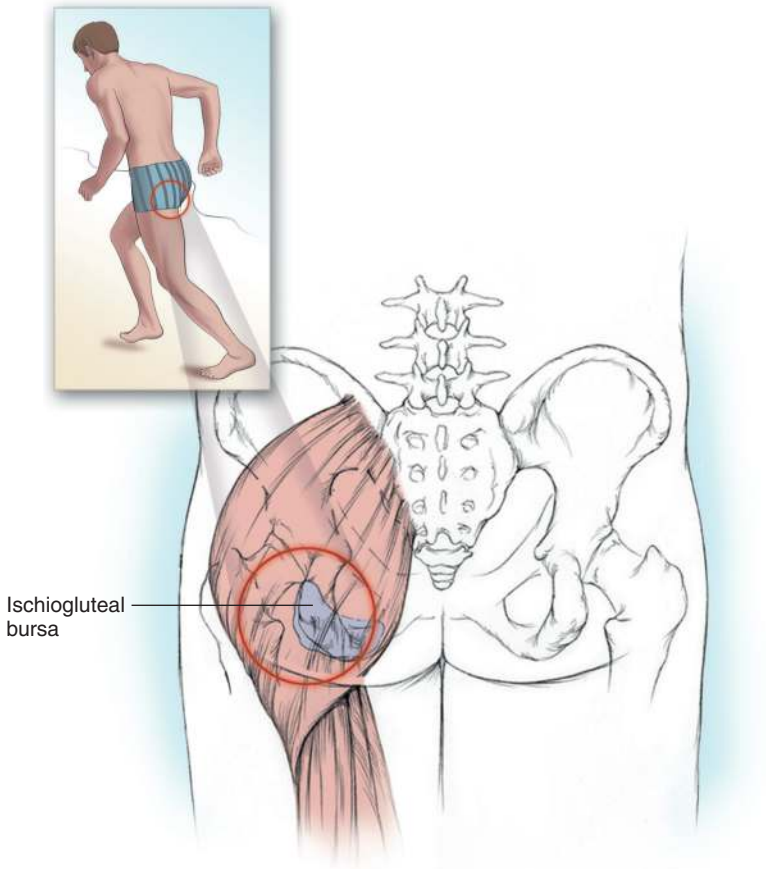


FIG 98.1 Ischiogluteal bursitis can be caused by running on soft, uneven surfaces. It manifests clinically as point tenderness over the ischial tuberosity.

Sudden release of resistance during this maneuver causes a marked increase in pain.

TESTING

Plain radiographs of the hip may reveal calcification of the bursa and associated structures, consistent with chronic inflammation (Fig. 98.3). Magnetic resonance and ultrasound imaging are indicated if disruption of the hamstring musculotendinous unit is suspected as well as to confirm the diagnosis (Figs. 98.4 and 98.5). The injection technique described later serves as both a diagnostic and a therapeutic maneuver and is also used to treat hamstring tendinitis. Laboratory tests, including a complete blood count,

FIG 98.2 Resisted hip extension test for ischiogluteal bursitis. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:309.)

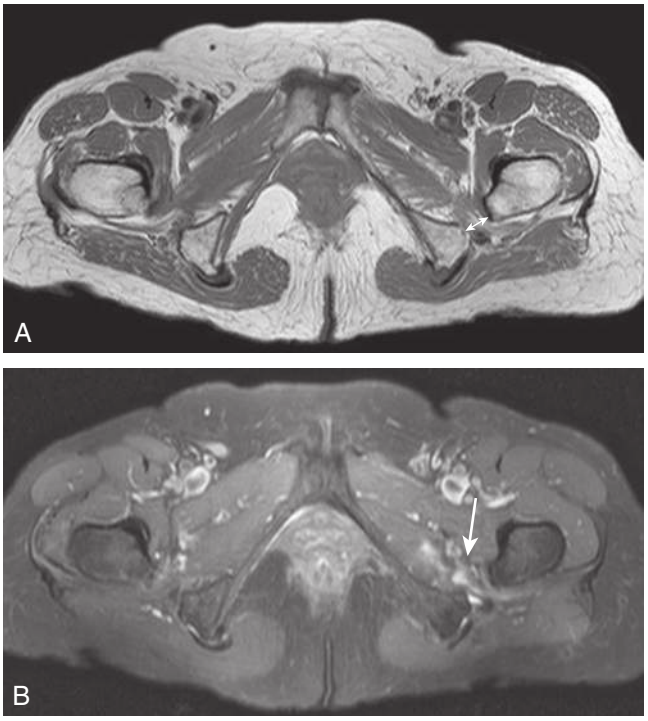


FIG 98.4 **A,** Axial T1-weighted magnetic resonance (MR) image of a middle-aged woman with poorly localized hip pain demonstrates reduced space between the lesser trochanter and the ischium on the left side (*double-headed white arrow*) owing to ischiofemoral impingement. **B,** The axial fat-suppressed T2-weighted MR image shows high-SI edema within the quadratus femoris muscle and adjacent ischiogluteal bursa (*white arrow*). (From Waldman SD, Campbell RSD. Ischiogluteal bursitis. In: *Imaging of pain*. Philadelphia: Elsevier; 2011.)

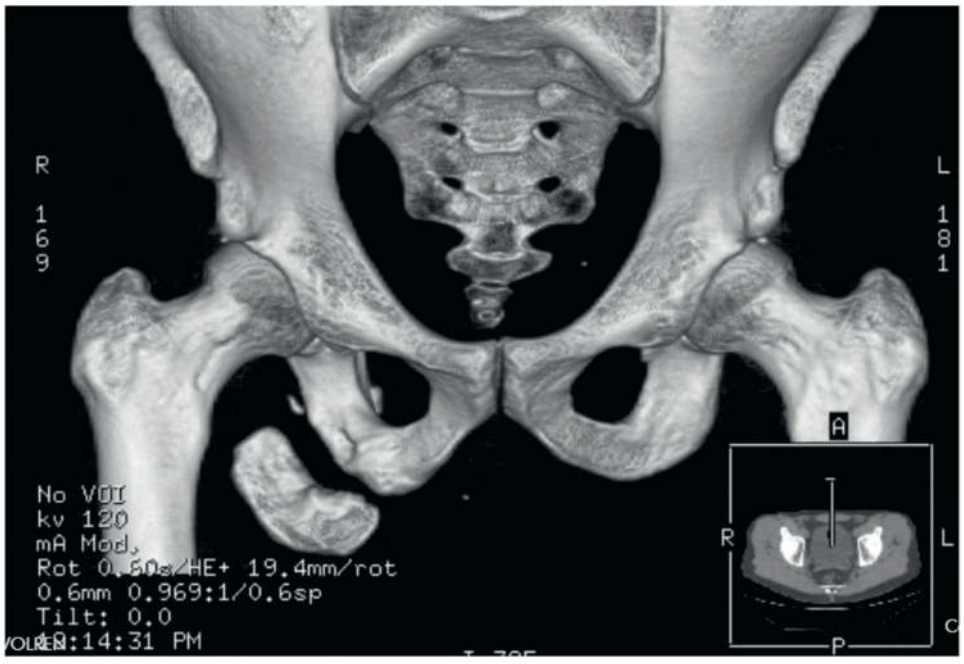


FIG 98.3 Three-dimensional reconstruction computed tomography scan showing a fracture large nonunion of the right ischium. (From Erik J. Stapleton, Julie Winn, Hervey L. Kimball, Suzanne L. Miller, Chronic Ischial Avulsion Fracture Excision With Primary Proximal Hamstring Repair: A Technique. *Arthroscopy Techniques*. 2022;11(10):e1801–e1809.)

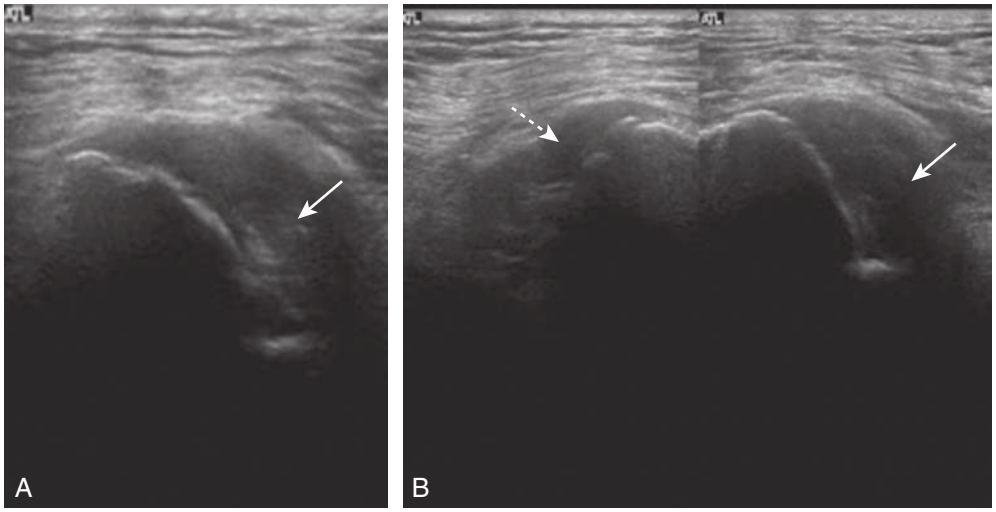


FIG 98.5 A, Transverse ultrasound (US) image of a young athlete with buttock pain and thickening of the hamstring tendon (*white arrow*) and some surrounding low-echo fluid within the ischiogluteal bursa. B, A comparative image shows the symptomatic side (*white arrow*) and the asymptomatic side (*broken white arrow*). (Image courtesy Dr. M. Reijnierse, Leiden, the Netherlands. From Waldman SD, Campbell RDS. Ischiogluteal bursitis. In: *Imaging of pain*. Philadelphia: Elsevier; 2011.)

erythrocyte sedimentation rate, and antinuclear antibody testing, are indicated if collagen vascular disease is suspected. Plain radiography and radionuclide bone scanning are indicated in the presence of trauma or if tumor is a possibility.

DIFFERENTIAL DIAGNOSIS

Although the diagnosis of ischiogluteal bursitis is usually straightforward, this painful condition is occasionally confused with sciatica, primary disease of the hip, insufficiency fractures of the pelvis, and tendinitis of the hamstrings (Box 98.1). Tumors of the hip and pelvis should also be considered in the differential diagnosis of ischiogluteal bursitis.

BOX 98.1 Differential Diagnosis of Ischiogluteal Bursitis

- Hamstring tendinopathy
- Hamstring tears
- Gluteal bursitis
- Avulsion fractures of the ischial tuberosity
- Primary and metastatic tumors involving the ischial tuberosity
- Myofascial pain
- Arthritides of the hip
- Acetabular labral tear
- Avascular necrosis of the femoral head
- Septic arthritis
- Septic bursitis
- Gluteal medius tendinopathy
- Impingement syndromes
- Piriformis tendinopathy
- Piriformis syndrome
- Iliopsoas tendinopathy
- Cluneal nerve entrapment
- Sacroiliac joint dysfunction
- Lumbar spine dysfunction

TREATMENT

Initial treatment of the pain and functional disability associated with ischiogluteal bursitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Any repetitive activity that may exacerbate the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To inject the ischiogluteal bursa, the patient is placed in the lateral position with the affected side upward and the affected leg flexed at the knee. The skin overlying the ischial tuberosity is prepared with antiseptic solution. A syringe containing 4 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle. The ischial tuberosity is identified with a sterilely gloved finger. Before needle placement, the patient should be instructed to say "There!" immediately if he or she feels paresthesia in the lower extremity, indicating that the needle has impinged the sciatic nerve. Should paresthesia occur, the needle is immediately withdrawn and repositioned more medially. The needle is then carefully advanced at that point through the skin, subcutaneous tissues, muscle, and tendon until it impinges on the bone of the ischial tuberosity. Care must be taken to keep the needle in the midline and not to advance it laterally to avoid contacting the sciatic nerve. After careful aspiration, and if no paresthesia is present, the contents of the syringe are gently injected into the bursa. Ultrasound needle guidance will improve the accuracy

FIG 98.6 Ultrasound-guided injection of the ischial bursa.

of needle placement and decrease the incidence of needle-related complications (Fig. 98.6).

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics, NSAIDs, and antimyotonic agents such as tizanidine may be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. Because of the proximity to the sciatic nerve, injection for ischiogluteal bursitis should be performed only by those familiar with the regional anatomy and experienced in the technique. Many patients complain of a transient increase in pain after injection of the affected bursa and tendons, and patients should be warned of this possibility. If patients continue to engage in the repetitive activities responsible for ischiogluteal bursitis, improvement will be limited.

CLINICAL PEARLS

To distinguish ischiogluteal bursitis from hamstring tendinitis, the clinician should remember that ischiogluteal bursitis manifests with point tenderness over the ischial bursa, whereas the tenderness of hamstring tendinitis is more diffuse over the upper muscle and tendons. The treatment is the same, however. Injection is extremely effective in relieving the pain of both ischiogluteal bursitis and hamstring tendinitis.

SUGGESTED READINGS

Lormeau C, Cormier G, Sigaux J, et al. Management of septic bursitis. *Joint Bone Spine*. 2018:341–347.

Waldman SD. Ischial bursitis. In: *Imaging of pain*. Philadelphia: Saunders Elsevier; 2011:349–350.

Waldman SD. Ischial bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:455–459.

Waldman SD. Ischial bursitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:510–511.

Waldman SD. Injection technique for ischial bursitis. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:534–538.

Wisniewski SJ, Hurdle M, Erickson JM, et al. Ultrasound-guided ischial bursa injection: technique and positioning considerations. *PMR*. 2014;6(1):56–60.

Endometriosis

ICD-10 CODE N80.9

THE CLINICAL SYNDROME

Endometriosis is a common cause of pelvic, low back, and abdominal pain caused by the implantation of normal uterine endometrial mucosa in abnormal locations. Endometriosis occurs in approximately 8% of women with approximately 30% of these women completely asymptomatic. An estrogen-dependent disease, it usually affects reproductive-aged women with an active hypothalamic–pituitary–ovarian axis. It is identified in approximately 35% of infertile women and 80% of women suffering from chronic pelvic pain. There is a 10-fold increased incidence of endometriosis in those women who have a first-degree relative suffering from the disease with a locus on chromosome 7p15.2 lined to an increased incidence of endometriosis in women of European descent. There is also concordance in monozygotic twins. A suggestion is that there is an increased incidence of endometriosis and specific phenotypic traits including red hair, nevi, freckles, and sensitivity to sun exposure. Other risk factors for endometriosis include early menarche, prolonged heavy menstrual flow, and delayed first birth.

The symptomatology associated with endometriosis is summarized in Box 99.1. The pain of endometriosis is cyclical in that it accompanies menstruation. The onset of pain usually precedes menstrual flow by 48 hours and begins to resolve after 2 days of menstruation. The ameliorating effects of pregnancy and menopause are the rule, although hormone replacement therapy may cause a recurrence of the symptoms associated with endometriosis. The pain of endometriosis is not related to the load of abnormal implanted endometrial mucosa and stroma, but to the location and depth of each endometrial implant (Fig. 99.1). There may also be bidirectional crosstalk between abnormal endometrial implants and subserving nerves as pain patterns become established (Fig. 99.2). Psychometric testing suggests that patients suffering from symptomatic endometriosis have increased anxiety and neuroticism when compared with other female control groups. These symptoms may be exacerbated if the endometriosis is associated with infertility.

BOX 99.1 Common Symptoms of Endometriosis

- Dysmenorrhea
- Pelvic pain
- Lower abdominal pain
- Lower back pain
- Groin pain
- Dyspareunia
- Dysuria
- Urinary frequency
- Dyschezia

SIGNS AND SYMPTOMS

Symptoms of endometriosis include dysmenorrhea, pelvic pain, groin pain, lower abdominal and back pain, dyspareunia, dysuria, and dyschezia with the symptom experienced related to the anatomic area of implantation. Abnormal implantation on the posterior peritoneum, uterus, and ovaries is most often associated with pelvic and lower abdominal pain, with implantation on the ureters and bladder associated with dysuria and urinary frequency. Significant implantation onto the ureters can cause obstruction of the affected ureter (Fig. 99.3). If there is significant abnormal implantation onto the colon and rectosigmoid colon, dyschezia and, rarely, partial bowel obstruction can occur (Fig. 99.4). There are clinical reports of hemoptysis from pulmonary implantation as well as cyclical umbilical bleeding from implants on the umbilicus (Fig. 99.5).

Physical examination of patients suffering from symptomatic endometriosis usually yields nonspecific findings including poorly defined abdominal and pelvic tenderness on abdominal or abdominal and pelvic examination. These findings are best identified if examinations are performed on the first day of menses. With significant abnormal pelvic implantation, tender nodules may be appreciated during palpation of the uterus, uterosacral ligaments, fallopian tubes, ovaries, and posterior cul-de-sac. On direct examination by laparoscopy or exploratory laparotomy, superficial implants will exhibit a hormonally responsive blue/black powder burn or flame-red vesicular appearance (Fig. 99.6).

TESTING

Plain radiographs of the pelvis will yield little clinically useful information and should be reserved for those patients thought to have bony abnormality (e.g., osteitis pubis).

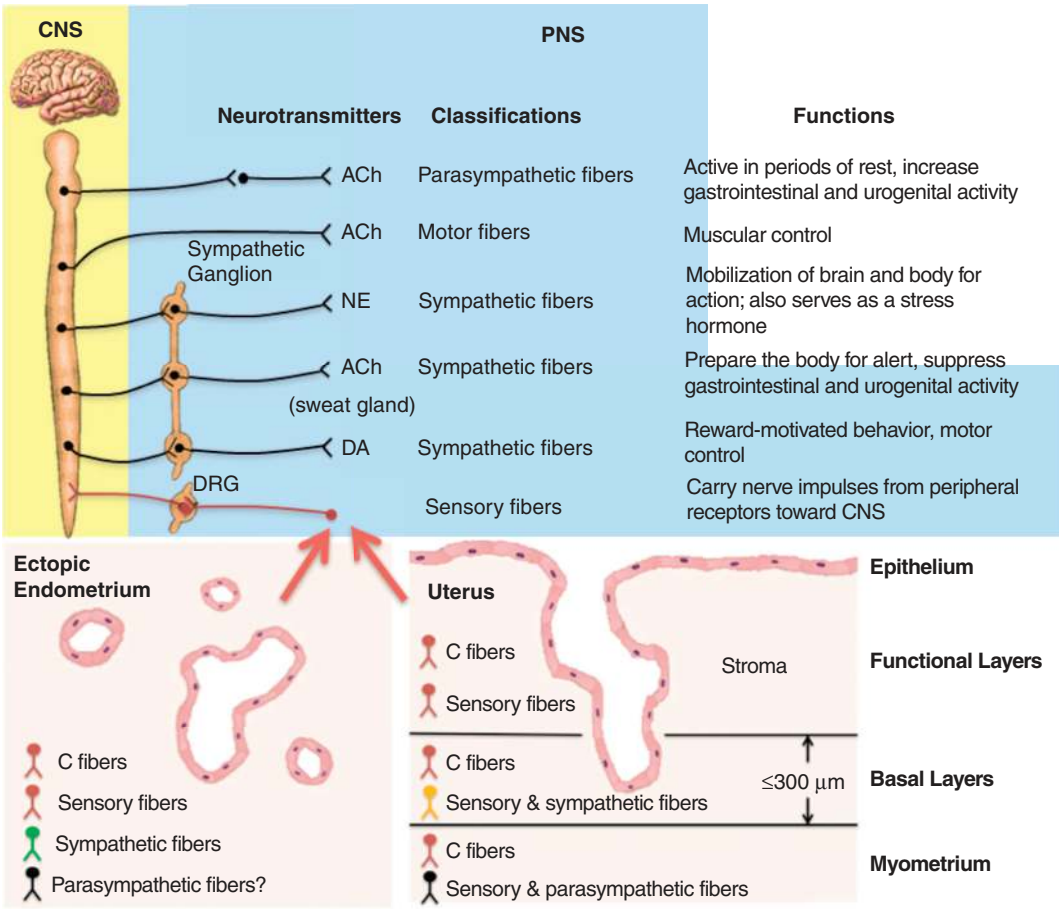


FIG 99.1 Classifications of nerve fibers and their relationship with eutopic and ectopic endometrium. ▬, Efferent fibers; ▬, afferent fibers; , increased density in women with endometriosis; , decreased density in women with endometriosis; , present in women with endometriosis only; , no differences between women with and without endometriosis. *Ach*, Acetylcholine; *CNS*, central nervous system; *DA*, dopamine; *DRG*, dorsal root ganglion; *E*, epinephrine; *NE*, norepinephrine; *PNS*, peripheral nervous system. (Modified from Yan D, Liu X, Guo S-W. Nerve fibers and endometriotic lesions: partners in crime in inflicting pains in women with endometriosis. *Eur J Obstet Gynecol Reprod Biol*. 2017;209:14–24.)

Pelvic and transvaginal ultrasound and magnetic resonance testing will identify larger abnormal endometrial implants as well as identify occult pathology that might be missed on physical examination (e.g., ovarian cancer) (Figs. 99.7 and 99.8). Ultrasound is also useful in characterizing whether an abnormal mass is solid or cystic (e.g., ovarian chocolate cysts). Computerized tomographic scanning with intravenous contrast may help identify abnormal implantations that may be obstructing ureters as well as abnormal implantation on the urinary bladder. Laparoscopy with direct visualization will provide the most accurate diagnosis, with exploratory laparotomy reserved for cases where the index of suspicion for malignancy is high. Complete blood count should be performed on all patients suspected of suffering from endometriosis to identify anemia and possible infection. Assay of the Thomsen–Friedenreich antigen (Gal beta1–3GalNAc) bearing proteins has an approximate 80% sensitivity and specificity in the ability to identify significant endometriosis.

DIFFERENTIAL DIAGNOSIS

Diseases that may mimic the clinical presentation of endometriosis include malignancies of the pelvic organs and colon, ovarian cysts, pelvic adhesions, interstitial cystitis, pelvic inflammatory disease, ovarian torsion, and ectopic pregnancy.

TREATMENT

The hormone-dependent nature of endometriosis provides the basis for initial medical therapy. Combination of oral contraceptives, progestational agents, danazol, aromatase inhibitors, and gonadotropin-releasing hormone analogues provides significant symptom relief in patients suffering from endometriosis. Combination of contraceptives act by suppressing ovarian function and may afford some protection against the increased risk of epithelial ovarian cancer seen in patients with endometriosis. Progestational agents cause

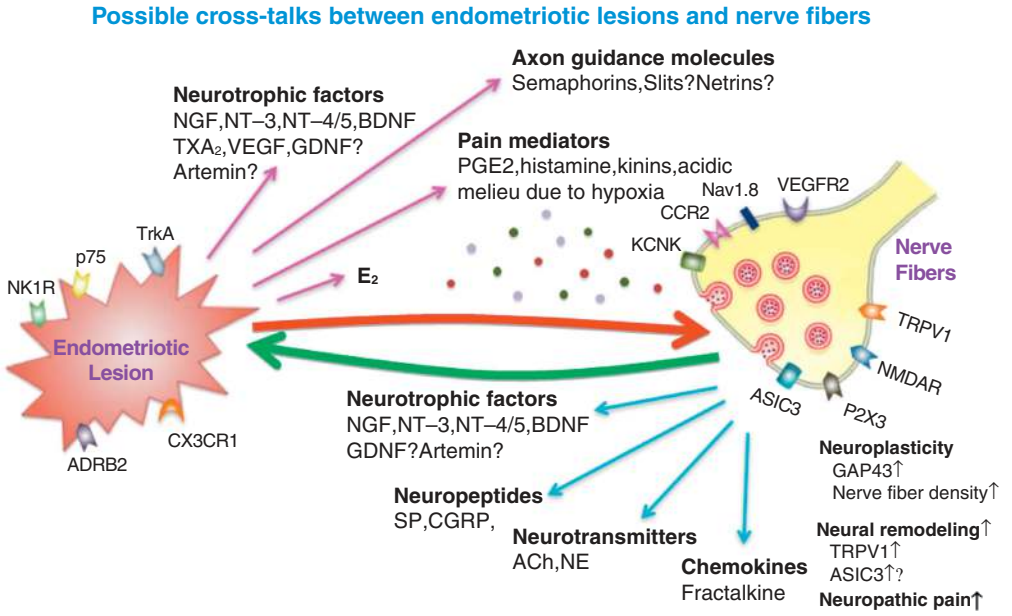


FIG 99.2 Schematic illustration of possible cross-talks between endometriotic lesions and nerve fibers. *Ach*, Acetylcholine; *ADRB2*, β² adrenergic receptor; *ASIC3*, the acid sensing ion channel 3; *BDNF*, brain-derived neurotrophic factor; *CGRP*, calcitonin gene-related peptide; *CX3CR1*, CX3C chemokine receptor 1; *GAP43*, growth-associated protein 43; *GDNF*, glial-derived neurotrophic factor; *KCNK*, potassium channel subfamily K member; *$Na_v1.8$*, sodium voltage-gated ion channels; *NE*, norepinephrine; *NGF*, nerve growth factor; *NK1R*, neurokinin receptor 1; *NMDAR*, N-methyl-D-aspartic acid receptor; *NT-3*, neurotrophin-3; *NT-4/5*, neurotrophin-4/5; *PGE_2*, prostaglandin E²; *P2XR3*, P2X purinoceptor 3; *SP*, substance P; *TRPV1*, transient receptor potential cation channel subfamily V member 1; *TXA_2*, thromboxane A²; *VEGF*, vascular endothelial growth factor. The question mark (?) indicates the role of indicated molecule in endometriosis-associated pain, if any, is yet to be investigated. (Modified from Yan D, Liu X, Guo S-W. Nerve fibers and endometriotic lesions: partners in crime in inflicting pains in women with endometriosis. *Eur J Obstet Gynecol Reprod Biol*. 2017;209:14–24.)

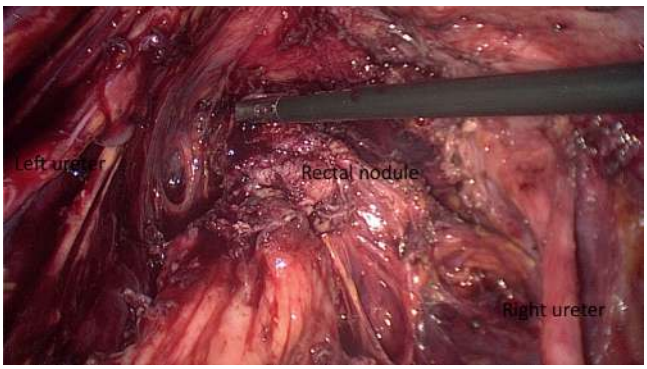


FIG 99.3 Endometriosis involving the rectosigmoid colon. Mobilization of rectovaginal septum revealing endometriotic nodule. (From Hogg S, Vyas S. Endometriosis. *Obstet Gynaecol Reprod Med*. 2015;25(5):133–141.)

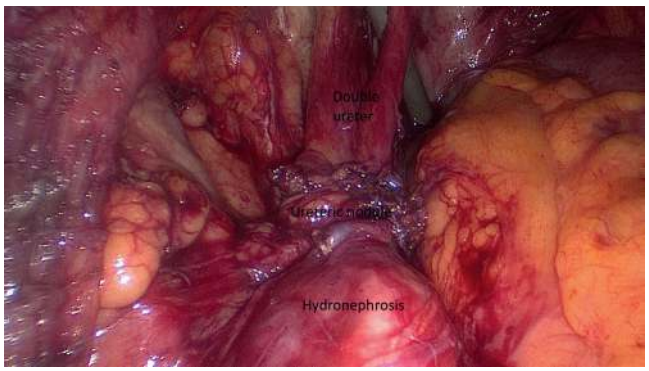


FIG 99.4 Endometriosis implants involving the ureter. Extrinsic endometriotic lesion causing hydronephrosis. (From Hogg S, Vyas S. Endometriosis. *Obstet Gynaecol Reprod Med*. 2015;25(5):133–141.)

atrophy of the endometrium and decidualization. Danazol acts by inhibiting the spike in midcycle follicle-stimulating hormone and luteinizing hormone to the detriment of the corpus luteum. Gonadotropin-releasing hormone analogues reduce the symptoms of endometriosis by producing a pituitary-induced hypogonadic state. Aromatase inhibitors inhibit the aromatase enzyme system which reduces plasma estrogen in postmenopausal women. Surgical treatments for endometriosis have two goals: (1) the surgical excision of abnormal endometrial implants to reduce load and (2) to correct obstructions and anatomic distortions of the tissues and organs that have abnormal endometrial implants.

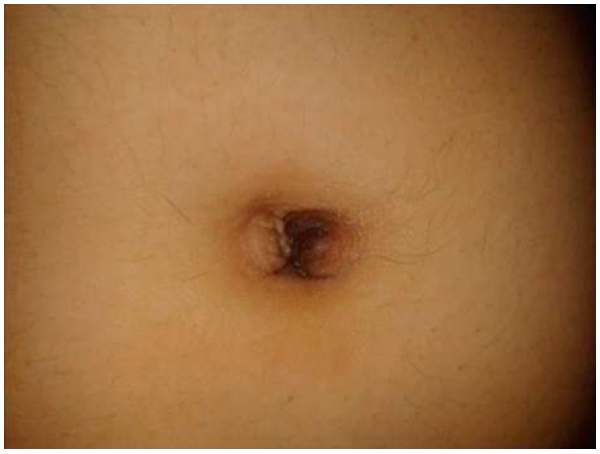


FIG 99.5 Primary umbilical endometriosis. (From Eğin S, Pektaş BA, Hot S, et al. Primary umbilical endometriosis: a painful swelling in the umbilicus concomitantly with menstruation. *Int J Surg Case Rep*. 2016;28:78–80.)

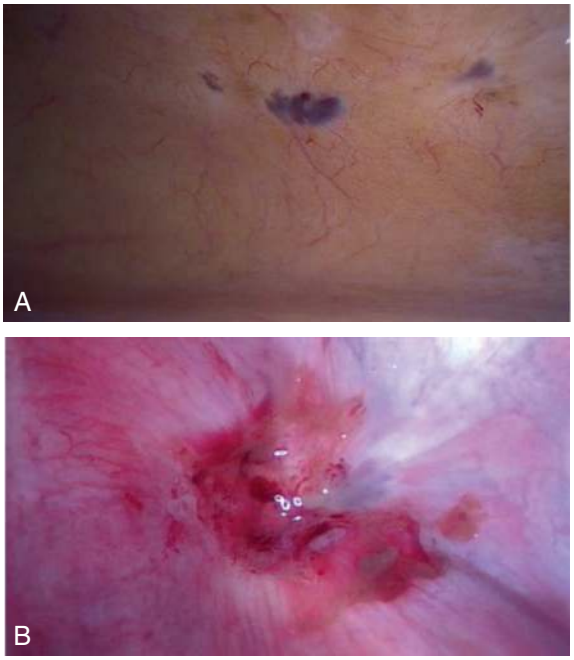


FIG 99.6 Superficial endometriosis. **A,** Powder burn appearance. **B,** Flame-red appearance with associated scarring. (From Hogg S, Vyas S. Endometriosis. *Obstet Gynaecol Reprod Med*. 2015;25(5):133–141.)

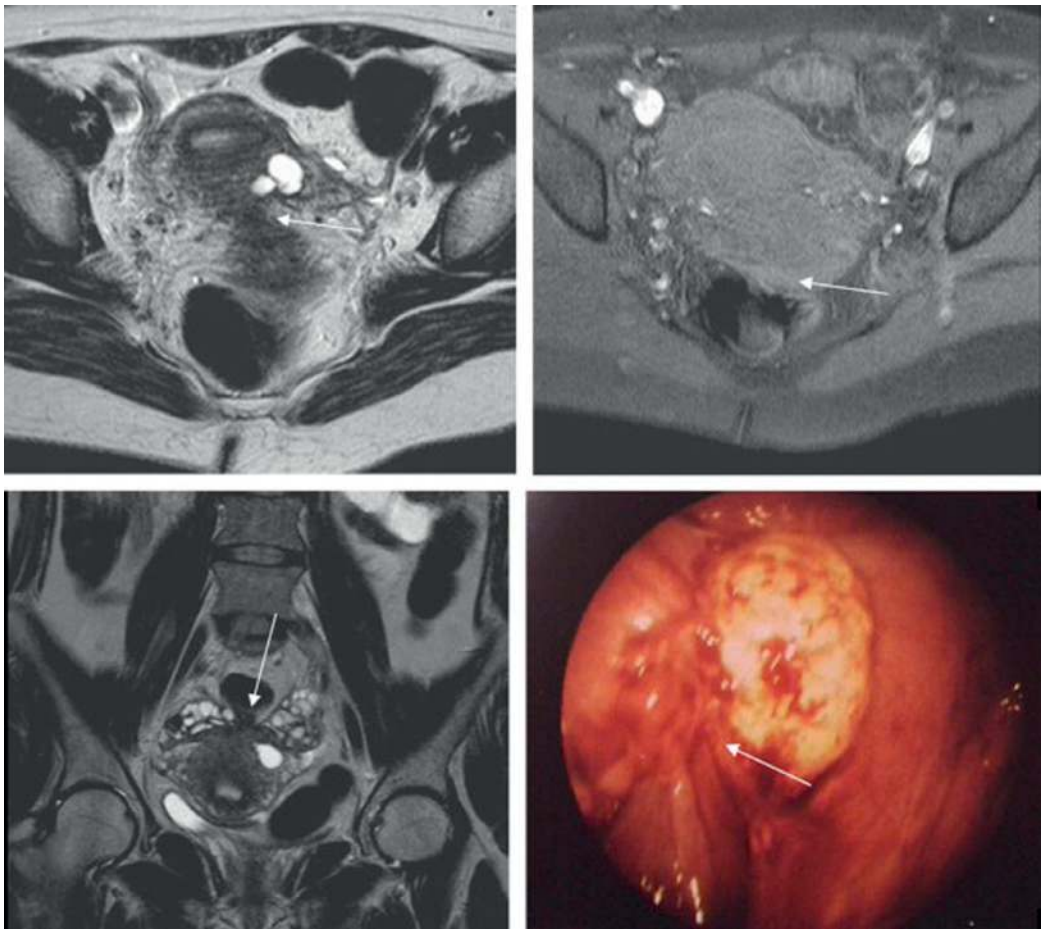


FIG 99.7 Periovarian endometriosis with fibrous adhesions to the ovarian fossae, hypointense T1- and T2-weighted images and laparoscopic correlation with whitish fibrous implants. (From Dallaudière B, Salut C, Hummel V, et al. MRI atlas of ectopic endometriosis. *Diagn Interv Imaging*. 2013;94(3):263–280.)

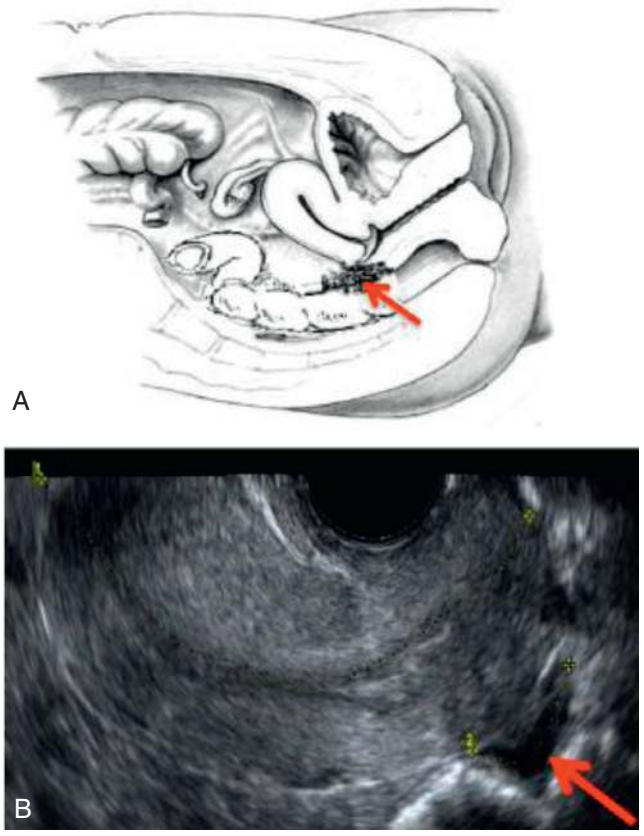


FIG 99.8 Low rectal endometriotic nodule. **A,** Anatomical schematic showing a lower or caudal rectum deep infiltrating endometriosis nodule (*red arrow*). **B,** Transvaginal ultrasound view of uterus in longitudinal section with a hypoechoic retrocervical lesion adherent to the low rectum and the uterine cervix (*red arrow*). (From Exacoustos C, Malzoni M, Di Giovanni A, et al. Ultrasound mapping system for the surgical management of deep infiltrating endometriosis. *Fertil Steril.* 2014;102(1):143–150.e2, Suppl. Fig. 2.)

The local application of heat and cold to the painful area may provide symptomatic relief of mild pain as may simple analgesics. Any repetitive activity that may exacerbate the patient's symptoms should be avoided.

COMPLICATIONS AND PITFALLS

Untreated, undertreated, or improperly treated endometriosis can result in increased morbidity for the patient. While the primary complication of endometriosis is infertility, endometriosis can result in adhesions of the pelvic structures. If these adhesions are bad enough, surgery may be required with all of the attendant surgical complications. It should be remembered that patients suffering from endometriosis have a high than expected incidence of ovarian cancer.

CLINICAL PEARLS

Endometriosis can be a challenging disease to manage. It can sometimes be mistaken for other cyclical painful conditions including pelvic inflammatory disease, ectopic pregnancy, and irritable bowel syndrome. Careful pelvic examination combined with ultrasound and magnetic resonance imaging will almost always clarify the diagnosis. If the diagnosis remains unclear after these diagnostic tests, laparoscopy may be necessary.

SUGGESTED READINGS

Boesgaard-Kjer D, Boesgaard-Kjer D, Kjer JJ. Primary umbilical endometriosis (PUE). *Eur J Obstet Gynecol Reprod Biol.* 2017;209:44–45.

Ferrero S. Endometriosis: modern management of an ancient disease. *Eur J Obstet Gynecol Reprod Biol.* 2017;209:1.

Hogg S, Vyas S. Endometriosis. *Obstet Gynaecol Reprod Med.* 2015;25(5):133–141.

Ito TE, Abi Khalil ED, Taffel M, et al. Magnetic resonance imaging correlation to intraoperative findings of deeply infiltrative endometriosis. *Fertil Steril.* 2017;107(2):e11–e12.

Morotti M, Vincent K, Becker CM. Mechanisms of pain in endometriosis. *Eur J Obstet Gynecol Reprod Biol.* 2017;209:8–13.

Parazzini F, Esposito G, Tozzi L, et al. Epidemiology of endometriosis and its comorbidities. *Eur J Obstet Gynecol Reprod Biol.* 2017;209:3–7.

Yan D, Liu X, Guo SW. Nerve fibers and endometriotic lesions: partners in crime in inflicting pains in women with endometriosis. *Eur J Obstet Gynecol Reprod Biol.* 2017;209:14–24.

100

Ectopic Pregnancy

ICD 10 CODE O00. 9

THE CLINICAL SYNDROME

Ectopic pregnancy is a common cause of pelvic and abdominal pain (Fig. 100.1). Ectopic pregnancy occurs when an ovum is fertilized and the conceptus successfully implants and begins to mature outside the uterine endometrium. Ectopic pregnancy is common, with a reported incidence of 1 in 40 pregnancies, and is the cause of 4%–8% of pregnancy-related deaths. The incidence of ectopic pregnancy increases with age, with an adjusted odds ratio for ectopic pregnancy in women over 40 at approximately 3.1. Approximately 97% of ectopic pregnancies occur within the fallopian tubes, with extratubular ectopic pregnancy, including the cervix, ovary, abdominal cavity, interstitium, and retroperitoneal space, accounting for the remaining 3% (Figs. 100.2–100.6; Box 100.1). Unlike the uterus, which is able to accommodate the growing fetus, these other structures cannot, resulting in rupture and life-threatening hemorrhage.

Factors that contribute to an increased risk of ectopic pregnancy include fallopian tubal abnormalities from infection, previous surgical trauma to the fallopian tubes including tubal ligation, the use of ovulation-inducing drugs, and the maternal use of diethylstilbestrol. A previous history of ectopic pregnancy as well as smoking, intrauterine device use, and a history of multiple sexual partners also increase the risk of ectopic pregnancy.

SIGNS AND SYMPTOMS

The classic triad of the clinical presentation of ectopic pregnancy is (1) abdominal pain; (2) amenorrhea; and (3) vaginal bleeding (Fig. 100.7). The patient may also experience other symptoms commonly associated with pregnancy, including morning sickness, breast tenderness, and breast fullness. On physical examination, the uterus may be slightly enlarged

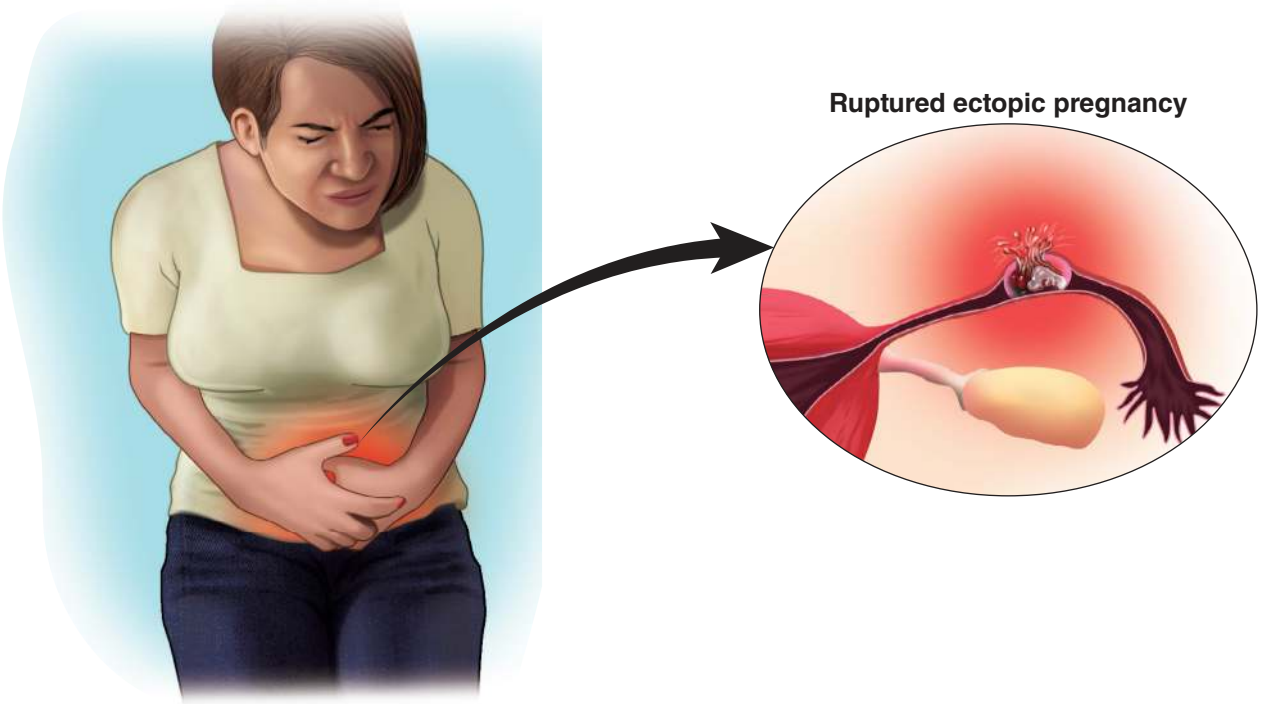


FIG 100.1 Ectopic pregnancy is a common cause of pelvic and abdominal pain. Ectopic pregnancy occurs when an ovum is fertilized and the conceptus successfully implants and begins to mature outside the uterine endometrium.

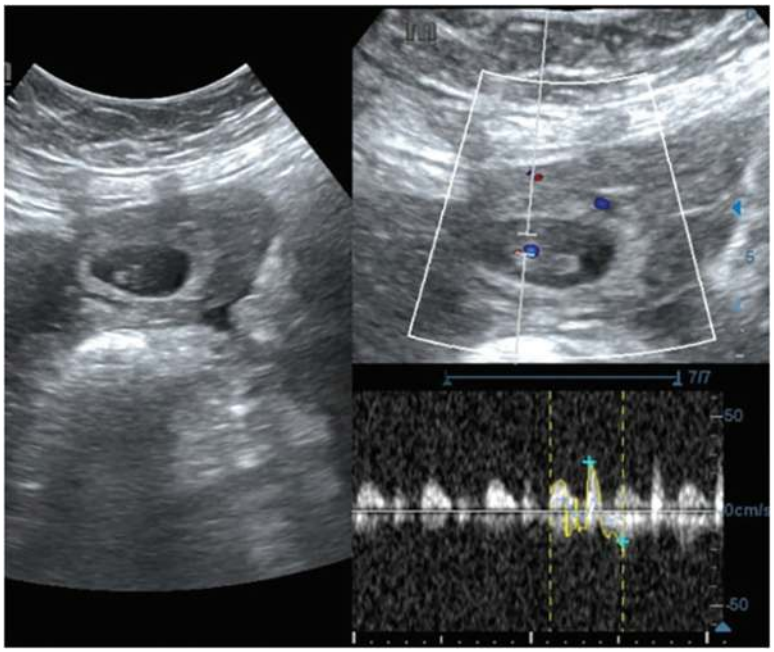


FIG 100.2 Ectopic pregnancy on ultrasonography. Right fallopian tube with pregnancy sac and 7-week embryo. (From Al Dus G, Alhamoud AU, Allah NA, Alabdalla J. Two embryos did not implant into the womb. A rare case of non-iatrogenic bilateral ectopic pregnancy (two-tailed tubal ectopic pregnancy) case report. *Ann Med Surg*. 2021;71:102840.)

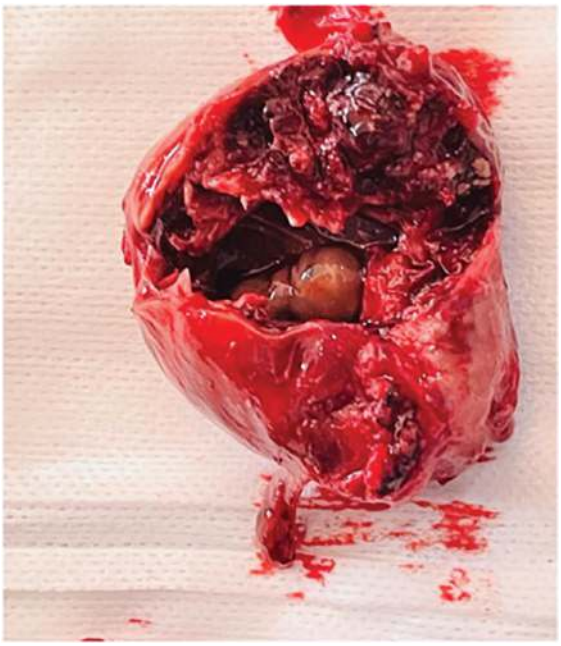


FIG 100.3 Ovarian ectopic pregnancy. Visualization of the embryo after dissection of the ovary. (From Bouab M, Touimi AB, Jalal M, Lamrissi A, Fichtali K, Bouhya S. Diagnosis and management of ectopic ovarian pregnancy: a rare case report. *Int J Surg Case Rep*. 2022;91:106742.)

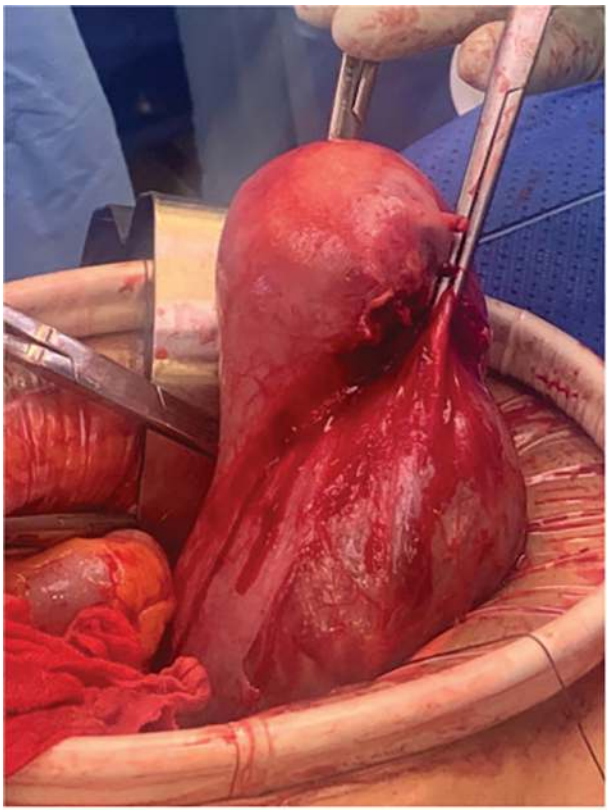


FIG 100.4 Cervical ectopic pregnancy. A normal-sized, nongravid uterus on top of a ballooned cervix indicative of a cervical ectopic pregnancy. (From Coulter-Nile S, Balachandar K, Ward H. A diagnostic dilemma of an 18-week cervical ectopic pregnancy: a case report. *Case Rep Womens Health*. 2022;33:e00385.)

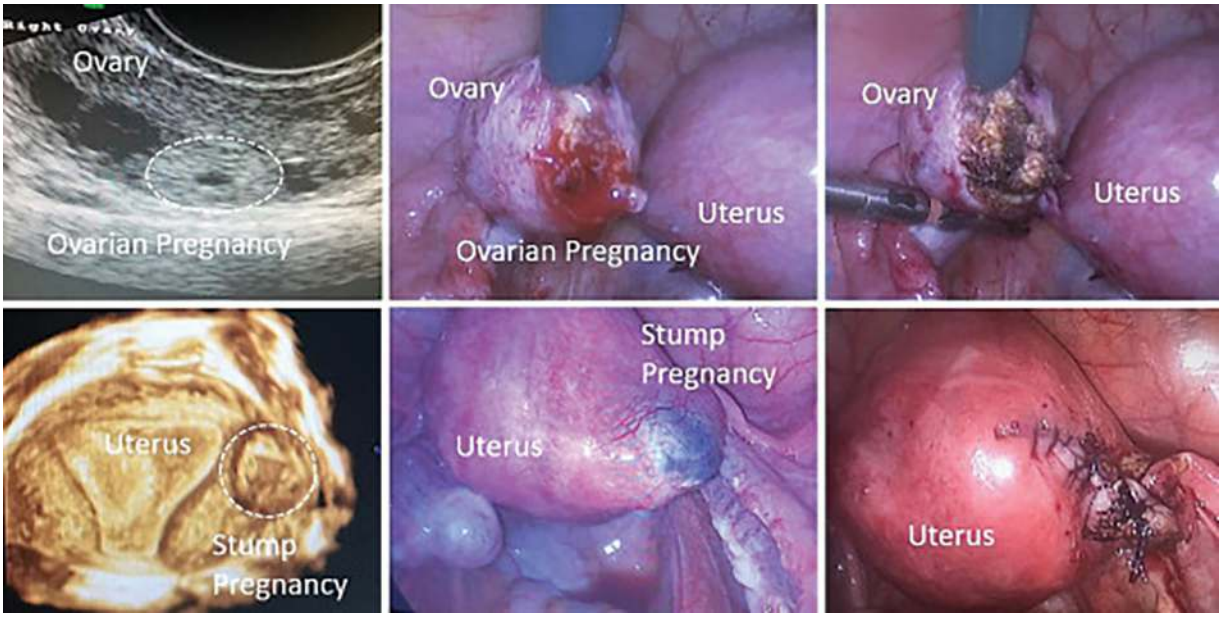


FIG 100.5 Sonographic and laparoscopic images of ovarian and tubal stump pregnancy. (From Tsviban A, Maymon R, Pekar-Zlotin M, Smorgick N, Gat I, Melcer Y. Similar but different: a comparison of rare site ectopic pregnancies. *Am J Emerg Med*. 2022;52:50–53.)

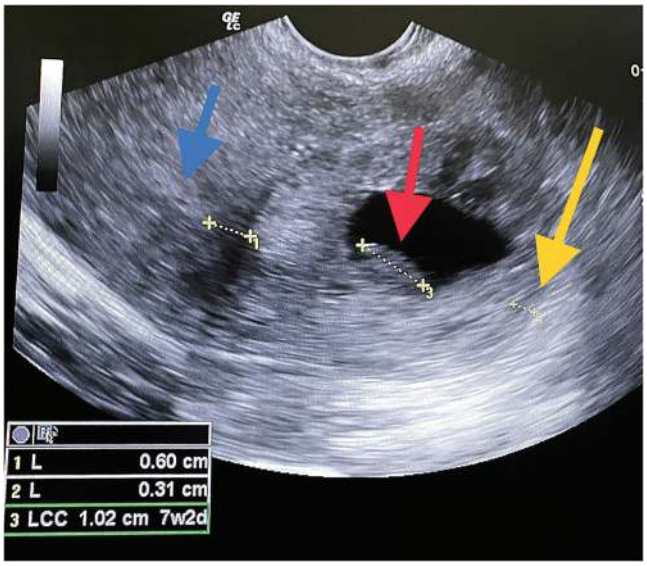


FIG 100.6 Ultrasound imaging of an interstitial pregnancy. *Blue arrow*: endometrium; *red arrow*: embryo; *yellow arrow*: surrounding myometrium. (From Slaoui A, Slaoui A, Zeraidi N, Lakhdar A, Kharbach A, Baydada A. Interstitial pregnancy is one of the most serious and uncommon ectopic pregnancies: case report. *Int J Surg Case Rep*. 2022;95:107195.)

BOX 100.1 Types of Ectopic Pregnancy

- Tubal
- Cervical
- Abdominal
- Retroperitoneal
- Ovarian
- Interstitial
- Other

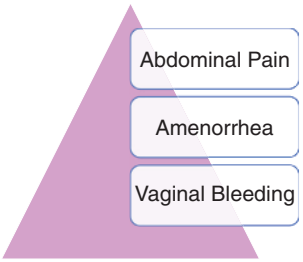


FIG 100.7 The classic clinical triad of ectopic pregnancy.

and soft with pain elicited by uterine or cervical movement. An adnexal mass may be palpated and uterine endometrial detritus may be present in the vagina due to hormonal stimulation of the endometrium from the fetus. With advanced abdominal pregnancy, fetal movement may be appreciated. If rupture and hemorrhage occur, the signs and symptoms of hypovolemic shock combined with peritoneal findings may become obvious.

TESTING

Serum beta-human chorionic gonadotropin (HCG) levels tend to rise less quickly with ectopic pregnancy when compared with a normal pregnancy. Ultrasonography is the medical imaging method of choice for the diagnosis of ectopic pregnancy and has the advantage of identifying both an intrauterine pregnancy and an ectopic pregnancy (Fig. 100.8). Specific ultrasonographic findings of tubal ectopic pregnancy include the blog and the bagel signs (Figs. 100.9 and 100.10).

There is a correlation between the level of serum HCG and the point at which an ectopic pregnancy can be reliably

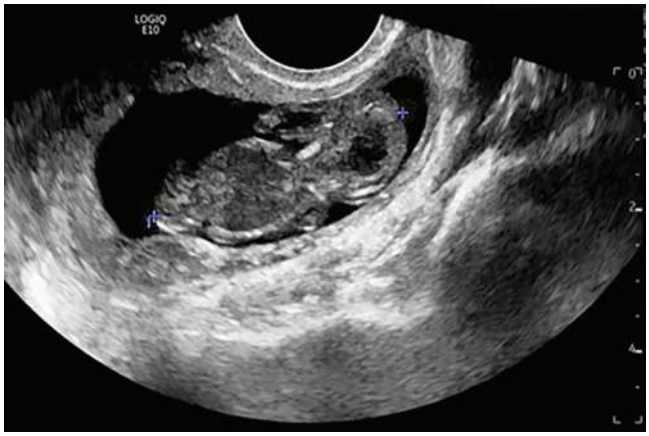


FIG 100.8 A transvaginal ultrasound demonstrating a large right adnexal mass containing an embryo with a crown-rump length of 3.9 cm corresponding to 10 weeks, 6 days gestational age. (From Haddaden M, Maharaj A, Muzzi K, Paudel K, Haas CJ. A large unruptured ectopic pregnancy. *Radiol Case Rep*. 2021;16(5):1204–1206.)

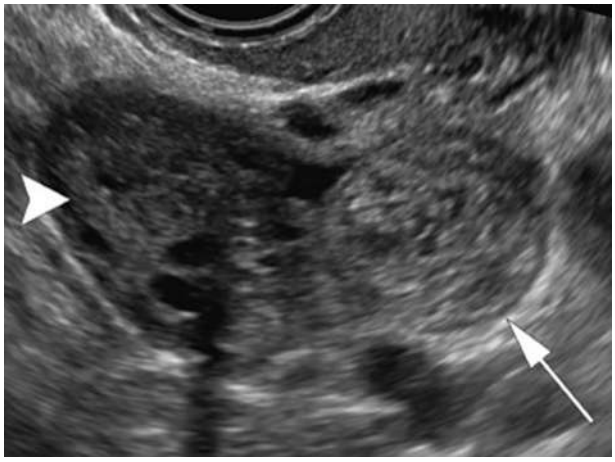


FIG 100.9 The "blob sign." A 32-year-old pregnant patient (6 weeks gestational age based on last menstrual period) presented with pelvic pain and vaginal bleeding. Beta-hCG measured 225 mIU/mL. Gray scale pelvic ultrasound of the left adnexa demonstrated a nodule or "blob" like structure (*arrow*) abutting the ovary (*arrowhead*). (From Lima D, Revels JW, Mattinson T, Wang SS. The bagel and blob signs in tubal ectopic pregnancy. *Radiol Case Rep*. 2021;16(7):1851–1853.)

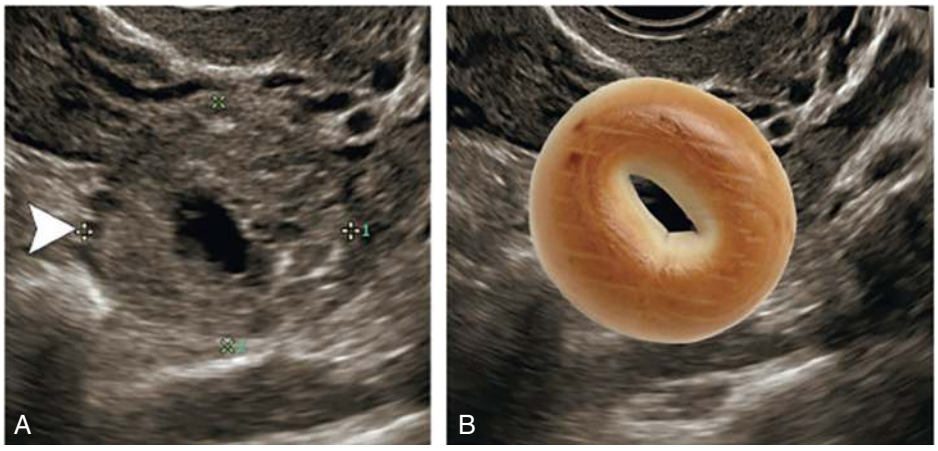


FIG 100.10 The "bagel sign." A 32-year-old pregnant patient (6 weeks gestational age based on last menstrual period) was represented with pelvic pain and vaginal bleeding. Beta-hCG measured 1657 mIU/mL compared to 225 mIU/mL 3 days prior. **A,** Gray scale pelvic ultrasound of the left adnexa demonstrates a ring-like structure with a thick, solid, hyperechoic periphery and fluid centrally: the "bagel sign" (*arrowhead*) (calipers along the periphery were placed to measure the size of the structure). **B,** Bagel animation overlaid on top of the sonographic finding. Ectopic pregnancy was confirmed clinically. (From Lima D, Revels JW, Mattinson T, Wang SS. The bagel and blob signs in tubal ectopic pregnancy. *Radiol Case Rep*. 2021;16(7):1851–1853.)

identified by transvaginal or abdominal ultrasound. This point is known as the HCG discriminatory zone and is between 6000 and 6500 mIU/mL with abdominal ultrasonography and 1500 and 1800 mIU/mL with transvaginal ultrasonography. An empty uterus on ultrasound examination with an HCG level above the HCG discriminatory zone must be assumed to be an ectopic pregnancy until proven otherwise. Laparoscopy may be required to confirm the diagnosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of ectopic pregnancy must include molar pregnancy, appendicitis, retained products of conception, complications of abortion, early pregnancy loss, tubal abscess, ovarian cysts (Fig. 100.11), placenta previa, and ovarian tumors (Box 100.2). Intrauterine pregnancy must also always be included in the differential diagnosis (Fig. 100.12).

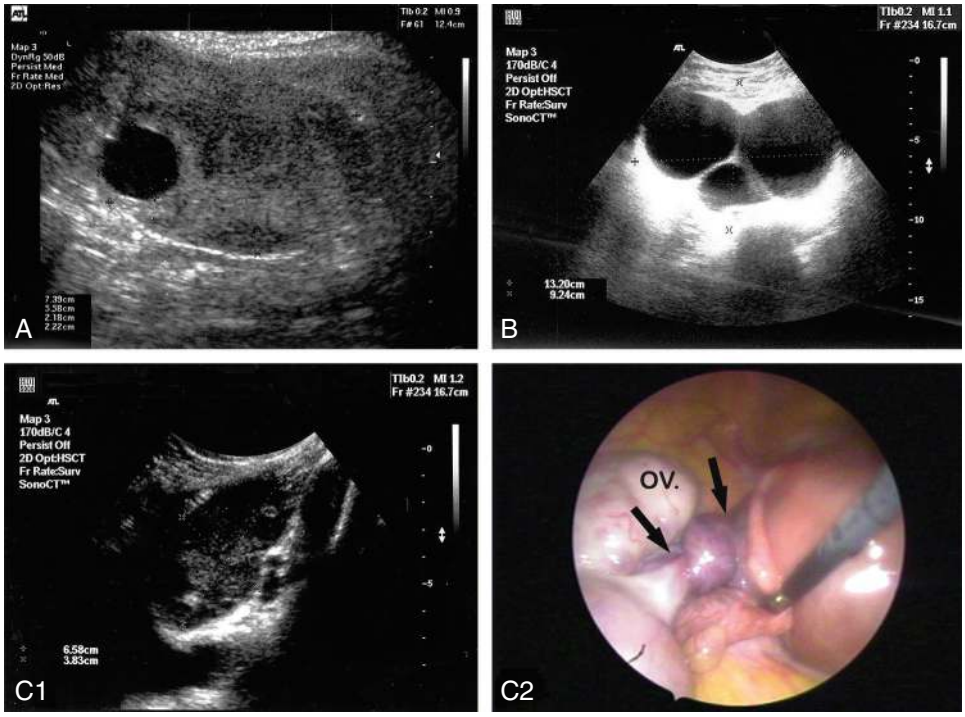


FIG 100.11 Montage plate of the three different ultrasound images of twisted ovaries in pregnancy. **A,** Transabdominal scan of an enlarged ovary with a 20-mm simple uniloclar cyst. The ovarian parenchyma appears edematous. **B,** Transabdominal scan of an enlarged 130 × 92-mm ovary with multicystic components. **C1,** Transabdominal scan of an enlarged 65-mm ovary without cystic components. Again, the ovarian parenchyma appears edematous. **C2,** Laparoscopy of the same patient as in C1. Arrowheads point to the twisted ovarian pedicle. (Reprinted by permission from the American Society for Reproductive Medicine., Noam Smorgick, Moty Pansky, Michal Feingold, Arie Herman, Reuvit Halperin, Ron Maymon. The clinical characteristics and sonographic findings of maternal ovarian torsion in pregnancy. *Fertility and Sterility*. 2009;92(6):1983–1987.)

BOX 100.2 Differential Diagnosis of Ectopic Pregnancy

- Retained products of conception
- Molar pregnancy
- Appendicitis
- Complications of abortion
- Early pregnancy loss
- Tuboovarian abscess
- Ovarian cysts
- Placenta previa
- Ovarian tumors

TREATMENT

The treatment of ectopic pregnancy is driven by whether the ectopic pregnancy is unruptured or ruptured. It should be remembered that in either circumstance, blood type, Rh type, and screening for antibodies should be done to identify those pregnant patients that need RhoGAM administration and to ensure the availability of blood products should the ectopic pregnancy suddenly rupture. In the patient with unruptured ectopic pregnancy, the administration of the

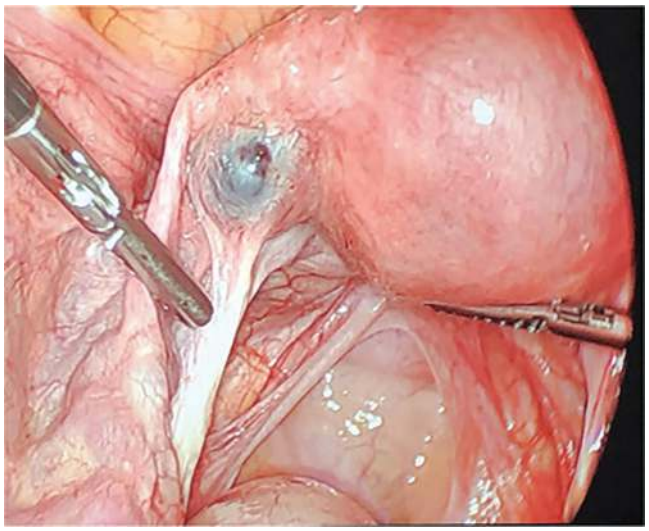


FIG 100.12 Intraoperative image of left broad ligament ectopic pregnancy between the left fallopian tube and uteroovarian ligament, unruptured and without overlying myometrium. (From Compadre AJ, Ukoha EP, Zhang W. Combined surgical and medical management of a broad ligament ectopic pregnancy: a case report. *Case Rep Womens Health*. 2021;31:e00316.)

chemotherapeutic agent methotrexate is a medical treatment for early ectopic pregnancy. Advantages of the use of methotrexate to treat early ectopic pregnancy include the avoidance of surgical interventions with their attendant risks including tubal scarring. This medical intervention for ectopic is contraindicated in patients with serum HCG levels of greater than 5000, demonstrable fetal cardiac activity, and in patients with ultrasound-documented free fluid in the cul-de-sac. Other contraindications to the use of methotrexate to treat early ectopic pregnancy include mothers who are breastfeeding, those with liver abnormalities, and those with blood dyscrasias.

Surgical treatment for ectopic pregnancy is driven by the type of ectopic pregnancy being treated. For tubal ectopic pregnancy, laparoscopic linear with milking the conceptus out of the distal tubal ampulla is performed. In instances where the patient no longer desires fertility or in the setting of severe tubal damage, total salpingectomy is indicated.

Regardless of the choice of treatment for ectopic pregnancy, weekly monitoring of quantitative HCG is required until levels return to zero to ensure that treatment is complete. This return to zero usually occurs within 3 weeks but almost always by 6 weeks posttreatment. It should be remembered that a failure to return to zero may indicate persistent or multiple ectopic pregnancies.

COMPLICATIONS AND PITFALLS

Failure to diagnose an ectopic pregnancy can result in catastrophic rupture and hemorrhage which is associated with significant morbidity and mortality. The monitoring of posttreatment serum HCG levels is critical to avoid missing the diagnosis of persistent or multiple ectopic pregnancies. Counseling and wellness checks are always indicated.

CLINICAL PEARLS

The failure to diagnose ectopic pregnancy can result in significant morbidity and mortality when the pregnancy ruptures and bleeding ensues. Although 97% of ectopic pregnancies are tubal, the clinician must maintain a high index of suspicion for ectopic pregnancy occurring in other anatomic locations such as the abdomen, ovary, cervix, and retroperitoneum.

SUGGESTED READINGS

Cardall AK, Jacobson JC, Prager S, et al. Medical management of ectopic pregnancy in a family planning clinic: a case series. *Contraception*. 2022;109:68–72. https://doi.org/10.1016/j.contraception.2021.11.010.

Coulter-Nile S, Balachandar K, Ward H. A diagnostic dilemma of an 18-week cervical ectopic pregnancy: a case report. *Case Rep Women Health*. 2022;33:e00385. https://doi.org/10.1016/j.crwh.2022.e00385.

Slaoui S, Slaoui A, Zeraidi N, et al. Interstitial pregnancy is one of the most serious and uncommon ectopic pregnancies: case report. *Int J Surg Case Rep*. 2022;95:107195. https://doi.org/10.1016/j.ijscr.2022.107195.

Tsviban A, Maymon R, Pekar-Zlotin M, et al. Similar but different: a comparison of rare site ectopic pregnancies. *Am J Emerg Med*. 2022;52:50–53. https://doi.org/10.1016/j.ajem.2021.11.025.

101

Pelvic Inflammatory Disease

ICD-10 CODE N73.9

THE CLINICAL SYNDROME

Pelvic inflammatory disease, which is often simply referred to as PID, is a painful infectious and inflammatory disease that most commonly occurs in females below 25 years of age with a history of multiple sexual partners, the failure to use contraception, and living in areas with high incidence of sexually transmitted diseases (Fig. 101.1). Additional risk factors for the development of PID include use of intrauterine devices, frequent vaginal douching, and menstruation. Starting as an asymptomatic or mildly symptomatic vaginal infection, PID ascends via the cervix to the upper genital tract, with infection and inflammatory changes of the fallopian tubes, ovaries, and uterus (Fig. 101.2). If the disease progresses, it may ultimately spread to the abdomen with a predilection to the perihepatic region. This perihepatic involvement is known as Fitz–Hugh–Curtis syndrome and is characterized by pain and classic violin adhesions (Fig. 101.3). Rarely, PID can occur in non-sexually active females.

PID is most often caused by *Chlamydia trachomatis*, but *Neisseria gonorrhoeae*, *Haemophilus influenza*, *Gardnerella vaginalis*, and bacteroides have also been implicated. Approximately 35% of cases of PID are polymicrobial in nature. Complications of PID include chronic pelvic pain, infertility, ectopic pregnancy, tubo-ovarian abscess, and perihepatitis (Fitz–Hugh–Curtis syndrome).

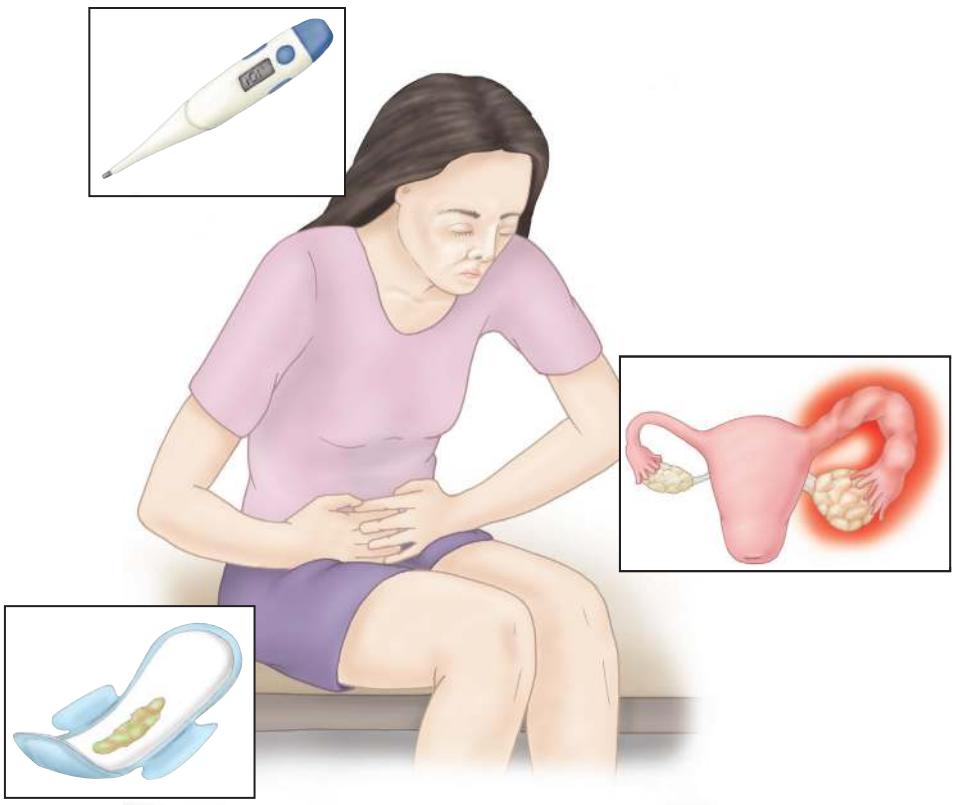


FIG 101.1 Pelvic inflammatory disease is a painful infectious and inflammatory disease that most commonly occurs in females below 25 years of age with a history of multiple sexual partners, the failure to use contraception, and living in areas with high incidence of sexually transmitted diseases.

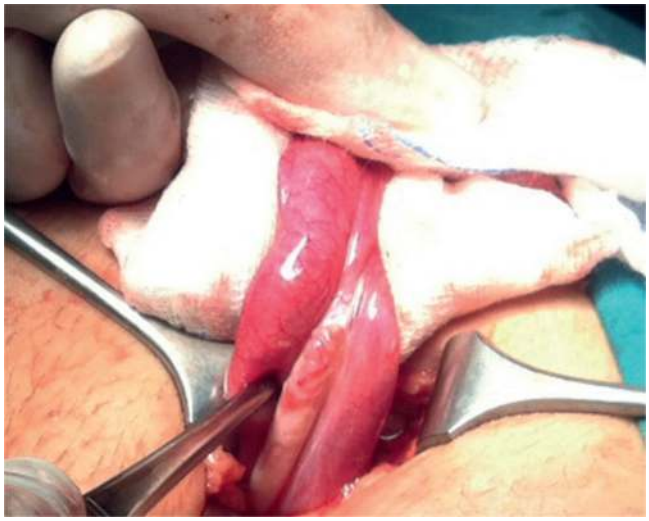


FIG 101.2 Intraoperative photograph showing the right adnexa of a patient with pelvic inflammatory disease. Notice the forceps pointing to marked hyperemia and edema of the inflamed fallopian tube and the thickening of the adjacent mesosalpinx. (From Mentessidou A, Theocharides C, Patoulias I, et al. Enterobius vermicularis-associated pelvic inflammatory disease in a child. *J Pediatr Adolesc Gynecol*. 2016;29(2):e25–e27.)

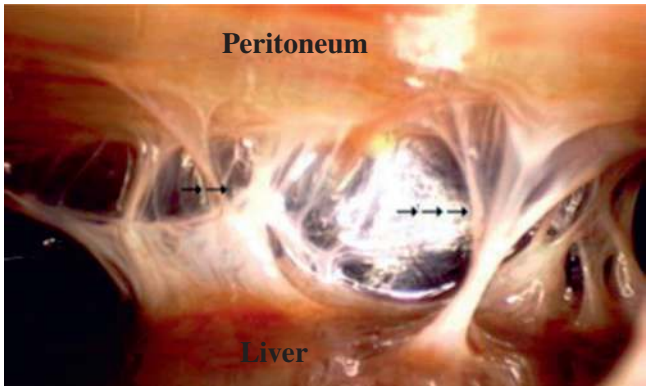


FIG 101.3 Fitz–Hugh–Curtis syndrome. Laparoscopic findings of the violin string between the peritoneum and the liver surface. (From Wilamarta M, Huang K-G, Casanova J, et al. Laparoscopy is the best choice to diagnose Fitz–Hugh–Curtis syndrome. *Gynecol Minim Invas Ther*. 2013;2(4):135–136.)

SIGNS AND SYMPTOMS

Severe pain in the lower abdomen and pelvis is invariably present with fever, nausea, and vomiting often confusing the diagnosis. Vaginal discharge is observed in most patients suffering from PID. The pain of PID is constant and characterized as aching or cramping in nature. Symptoms more likely occur toward the end of menses with gonorrhea and chlamydia-associated infections tend to be of more sudden onset and fulminant evolution.

On physical examination, patients with PID will exhibit pain on movement of the cervix with associated uterine and adnexal tenderness. Mucopurulent cervical and vaginal discharge is invariably present. With more fulminant and more advanced cases, peritoneal signs, including rebound tenderness and guarding, may be noted. Adnexal fullness or mass may suggest tubo-ovarian abscess.

TESTING

The diagnosis of PID is primarily based on the history and physical examination with the presence of fever, cervicovaginal discharge, pain on cervical motion, and abdominal findings improving the diagnostic specificity. All patients suspected of suffering from PID should undergo laboratory testing consisting of complete blood cell count, sedimentation rate, C-reactive protein, and automated blood chemistries. Blood and urine cultures should be immediately obtained in all febrile patients thought to have PID to allow immediate implementation of antibiotic therapy while the workup is in progress. Gram stains and cultures of the cervicovaginal discharge material should also be obtained and nucleic acid amplification testing should be undertaken if available, but antibiotic treatment should not be delayed while waiting for this information. Pregnancy testing to rule out ectopic pregnancy and to guide antibiotic selection is mandatory. Given the patient population most like to suffer from PID, rapid protein regain testing for syphilis, hepatitis, and human immunodeficiency virus is strongly indicated. Analysis of fluid obtained from the cul-de-sac via culdocentesis may help differentiate infectious processes as characterized by purulent fluid from the straw-colored fluid of inflammatory disease or the blood of ectopic pregnancy.

Transvaginal ultrasound will provide additional diagnostic specificity and sensitivity when diagnosing PID and also help diagnose other pathologic processes that may account for the patient's symptomatology. Ultrasound findings in patients suffering from PID include thickened, fluid-filled fallopian tubes with thickened cilia often present. Pyosalpinx and tubo-ovarian abscess may be present in more fulminant cases (Figs. 101.4 and 101.5). Free pelvic fluid and fluid in the adnexa and cul-de-sac are often present, but not diagnostic of PID. Endometritis and oophoritis may also be identified as may abscesses involving adjacent organs. Computerized tomography (CT) may also provide confirmatory information, especially if there is a mass, abdominal spread, hemorrhagic cyst, or endometriosis present (Fig. 101.6). CT is also useful if there is a question of acute appendicitis. Magnetic resonance imaging is also highly specific and sensitive when used in the diagnosis of PID and is superior to CT when diagnosing hydro- or pyosalpinx (Fig. 101.7). Laparoscopy may be required to confirm a clinical diagnosis of PID with hydro- or pyosalpinx, tubo-ovarian abscess, fallopian tube edema and hyperemia, pelvic abscess, perihepatic infection, ectopic pregnancy, ovarian cysts, endometriosis, and tumor are easily visualized (Figs. 101.8 and 101.9)

DIFFERENTIAL DIAGNOSIS

Any acute pathologic process of the pelvis and lower abdomen may mimic the clinical presentation of PID with appendicitis, urinary tract infection, ectopic pregnancy, ovarian

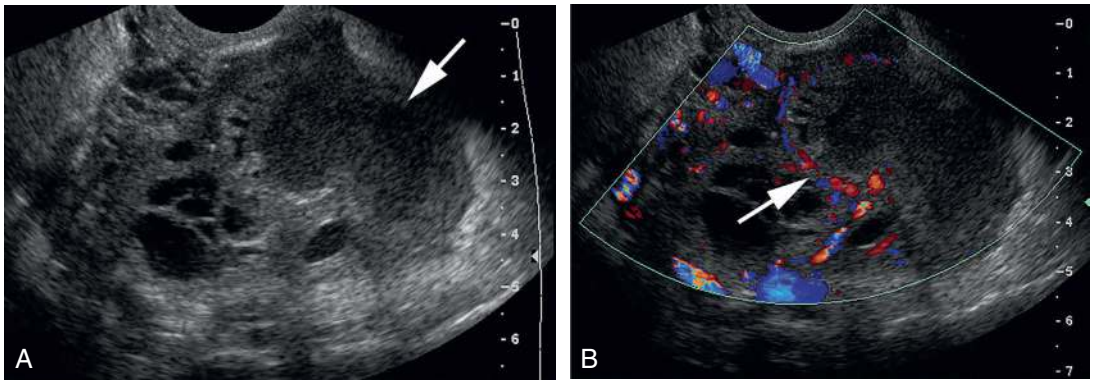


FIG 101.4 Tubo-ovarian abscess. **A,** Gray-scale sagittal ultrasound (US) image shows a heterogeneous thick-walled mass with low-level internal echoes (*arrow*) intimately associated with the ovary. **B,** Color Doppler sagittal US image shows increased vascularity along the periphery of the tubo-ovarian abscess (*arrow*). (From Chu LC, Coquia SF, Hamper UM. Ultrasonography evaluation of pelvic masses. *Radiol Clin North Am.* 2014;52(6):1237–1252.)

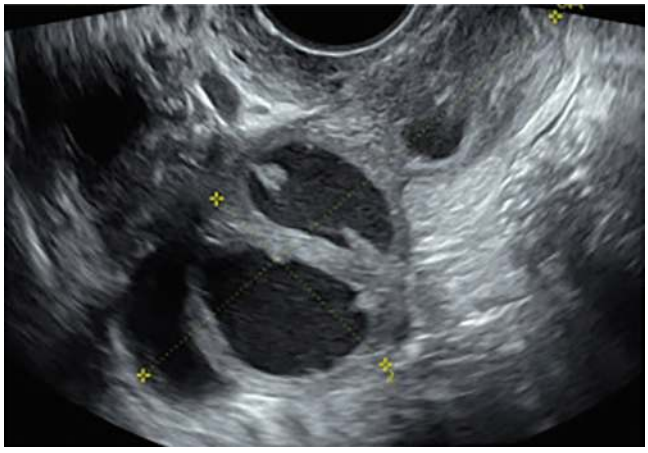


FIG 101.5 Ultrasound image of right tubo-ovarian abscess measuring 40 × 70 mm. (From Eloy C-G, Elena F-P, Berta U-T, Mireia G-C, Pelvic inflammatory disease presenting 16 months after vaginal hysterectomy: a case report and literature review. *Case Rep Women Health.* 2021;31:e00335.)

torsion, endometriosis, cervicitis, ruptured ovarian cysts, interstitial cystitis, and adnexal tumors at the top of the list.

TREATMENT

Initial treatment of the PID is focused at treating the acute infection, relieving pain, and reducing long-term sequelae of the disease, including chronic pelvic pain, ectopic pregnancy, and infertility. Because PID is usually the result of a sexually transmitted disease, identification of current and recent partners for evaluation and treatment is mandatory. Immediate antibiotic therapy with broad-spectrum coverage that is effective against *Chlamydia* and *Neisseria* as well as Gram-negative bacteria, anaerobes, and streptococci is the mainstay of treatment of PID. Monitoring for compliance and microbial resistance is important to improve the therapeutic outcome. Intrauterine devices should be removed and alternative forms of birth control provided. Symptomatic pain relief should be provided.

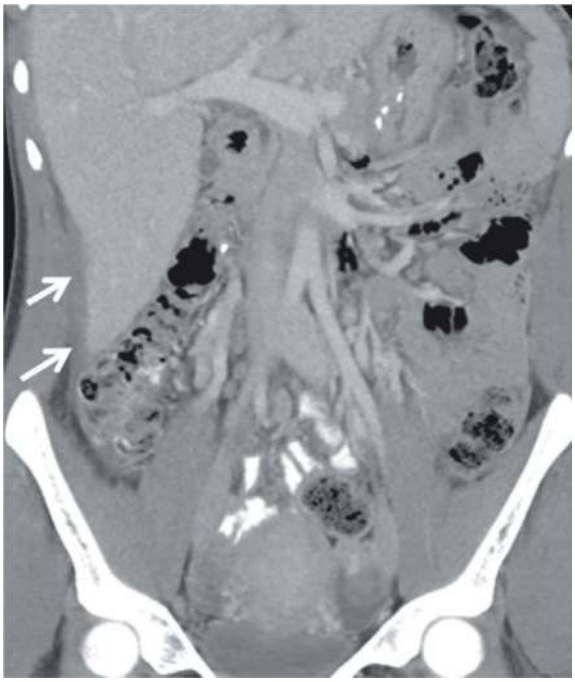


FIG 101.6 Computerized tomographic scan of a patient with Fitz–Hugh–Curtis syndrome, pelvic inflammatory disease, and right upper quadrant pain. There is spread of inflammation along the surface of the right hepatic lobe (*arrows*). (From Bennett GL. Evaluating patients with right upper quadrant pain. *Radiol Clin North Am.* 2015;53(6):10101–1130.)

COMPLICATIONS AND PITFALLS

Untreated, undertreated, or improperly treated PID can result in scarring and abscess formation that may permanently damage the fallopian tubes, ovaries, and uterus and may result in chronic pelvic and lower abdominal pain, ectopic pregnancy, and infertility. If infection spreads to the abdomen, perihepatic infection with subsequent painful adhesions and scarring may occur.

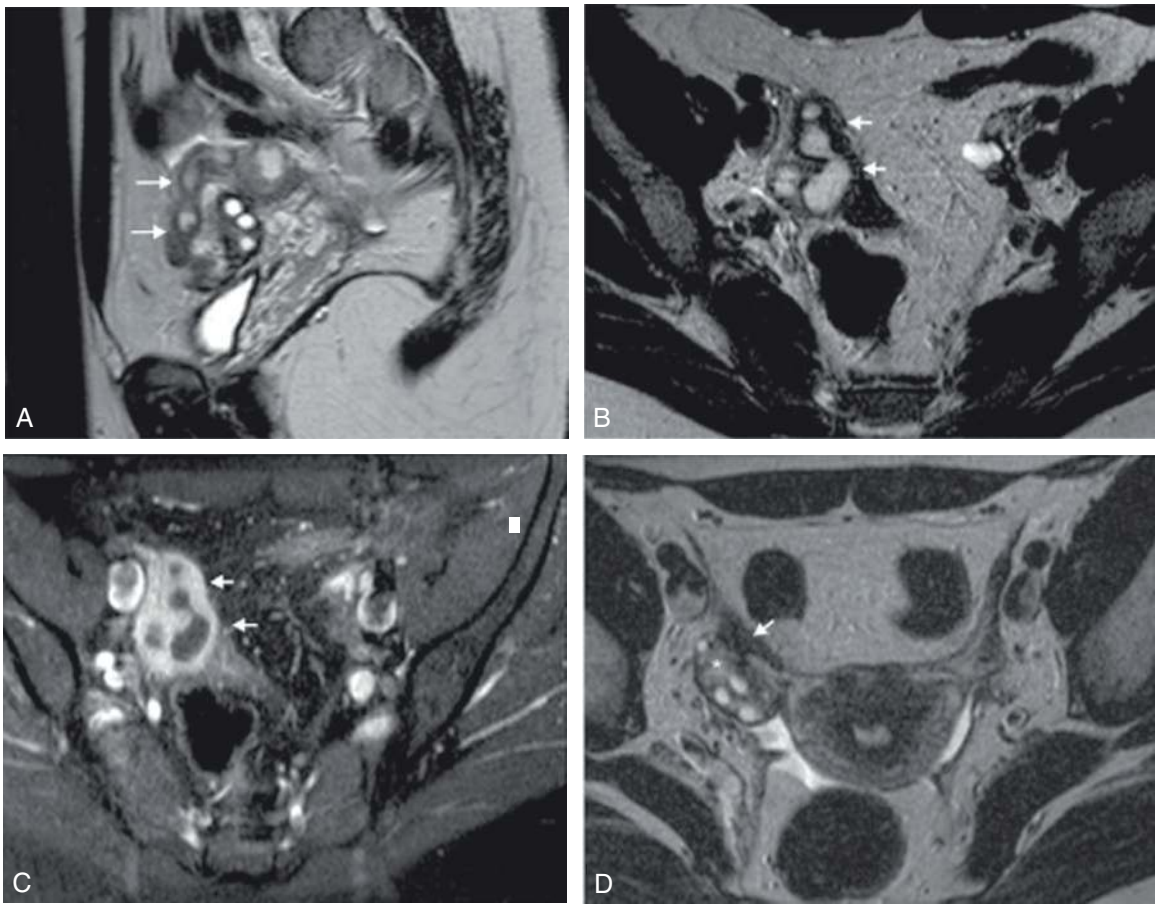


FIG 101.7 Magnetic resonance (MR) imaging of pelvic inflammatory disease, unilateral salpingitis: **A,** sagittal T2 TSE, **B,** axial T2 TSE, and **C,** fat-saturated axial T1 TSE and intravenous (IV) contrast. Asymptomatic 25-year-old woman diagnosed with right adnexal mass with transvaginal ultrasound. Elevated CA 125. On MR there is a right tube dilation (*arrows*) with an irregular-looking thickened wall enhanced by contrast. Tubaric content is liquid with a signal lower than water on T2 and greater on T1 with saturated fat, compatible with pyosalpinx. Both left tube and ovaries are normal (*left one is not shown*). Control after antibiotic therapy: **D,** axial T2 TSE. Resolution of process with normal-looking tubes (*arrow*). CA 125 was normal too. (From La Parra Casado C, Molina Fàbrega R, Forment Navarro M, et al. Fallopian tube disease on magnetic resonance imaging. *Radiología (Engl Ed)*. 2013;55(5):385–397.)

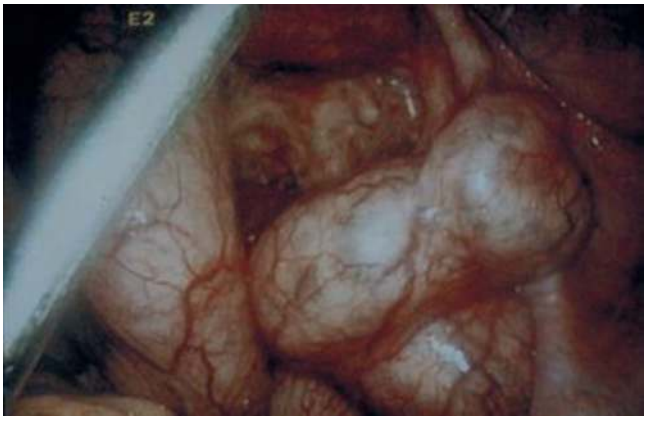


FIG 101.8 Laparoscopic view of severe pelvic inflammatory disease. (From Ross J. Pelvic inflammatory disease. *Medicine*. 2010;38(5):255–259.)

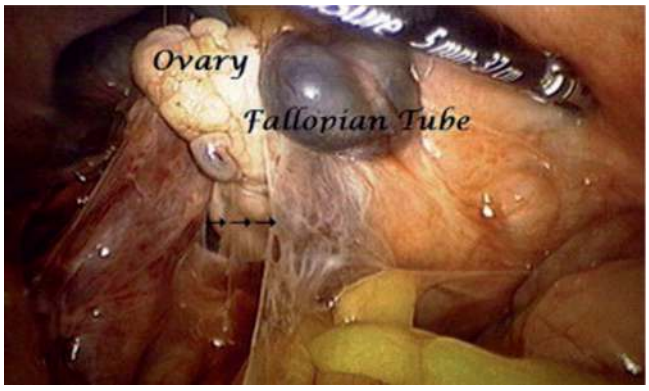


FIG 101.9 Pelvic inflammatory disease. Laparoscopic view of fibrous adhesion in all pelvic organs, especially the ovaries and the fallopian tubes. (From Wilamarta M, Huang K-G, Casanova J, et al. Laparoscopy is the best choice to diagnose Fitz–Hugh–Curtis syndrome. *Gynecol Minimal Invas Ther*. 2013;2(4):135–136.)

CLINICAL PEARLS

Initial treatment of the PID is focused at treating the acute infection, relieving pain, and reducing the long-term sequelae of the disease, including chronic pelvic pain, ectopic pregnancy, and infertility. Identification of current and recent sexual partners to allow prompt evaluation and treatment is an important public health concern.

SUGGESTED READINGS

Greydanus DE, Cabral MD, Patel DR. Pelvic inflammatory disease in the adolescent and young adult: an update. *Disease-a-Month*. 2022;68(3):101287. https://doi.org/10.1016/j.disamonth.2021.101287.

Gupta S, Kasule SN, Seville MT. Cerebral abscess with *Streptococcus intermedius* as a complication of pelvic inflammatory disease in the setting of intrauterine device use. *IDCases*. 2022;27:e01454. https://doi.org/10.1016/j.idcr.2022.e01454.

Jivraj S, Farkas A. Gynaecological causes of abdominal pain. *Surgery (Oxford)*. 2015;33(5):226–230.

Kielly M, Jamieson MA. Pelvic inflammatory disease in virginal adolescent females without tubo-ovarian abscess. *J Pediatr Adolesc Gynecol*. 2014;27(1):e5–e7.

Marcinkowski KA, Mehta V, Mercier R, Berghella V. Pelvic inflammatory disease in pregnancy: a systematic review focusing on perinatal outcomes. *Am J Obstetrics Gynecol*. 2022;4(4):100643. https://doi.org/10.1016/j.ajogmf.2022.100643.

Ravel J, Moreno I, Simón C. Bacterial vaginosis and its association with infertility, endometritis, and pelvic inflammatory disease. *Am J Obstetrics Gynecol*. 2021;224(3):251–257. https://doi.org/10.1016/j.ajog.2020.10.019.

Ross J. Pelvic inflammatory disease. *Medicine*. 2010;38(5):255–259.

Safrai M, Rottenstreich A, Shushan A, et al. Risk factors for recurrent pelvic inflammatory disease. *Eur J Obstet Gynecol Reprod Biol*. 2020;244:40–44. https://doi.org/10.1016/j.ejogrb.2019.11.004.

Sordia-Hernández LH, Serrano Castro LG, Sordia-Piñeyro MO, et al. Comparative study of the clinical features of patients with a tubo-ovarian abscess and patients with severe pelvic inflammatory disease. *Int J Gynecol Obstet*. 2016;132(1):17–19.

Yagur Y, Weitzner O, Tiosano LB, et al. Characteristics of pelvic inflammatory disease caused by sexually transmitted disease – an epidemiologic study. *J Gynecol Obs Hum Reprod*. 2021;50(9):102176. https://doi.org/10.1016/j.jogoh.2021.102176.

Interstitial Cystitis

ICD-10 CODE N30.10

THE CLINICAL SYNDROME

First described in 1887, interstitial cystitis is a bladder disorder of unknown etiology characterized by a constellation of irritative lower urinary tract symptoms including:

- dysuria
- pelvic pressure or pain
- urinary urgency, frequency, and a compulsion to constantly void
- sensation of incomplete bladder emptying
- associated sexual dysfunction

Interstitial cystitis, which is also known as bladder pain syndrome, occurs nine times more frequently in women than in men and is characterized by exacerbations and remission with rare asymptomatic periods (Fig. 102.1). The intensity of symptoms often varies from day to day, and in females this waxing and waning may correlate with the patient's ovulatory cycle. In men, complaints of concomitant groin, perineal, penile, and scrotal pain are common. Patients suffering from interstitial cystitis often also suffer from irritable bowel syndrome, fibromyalgia, and focal vulvitis. Sleep disturbance is common as the symptoms of interstitial cystitis are often worse at night. The diagnosis of interstitial cystitis is often one of exclusion when no other demonstrable pathology can be found to account for the patient's symptomatology.

FIG 102.1 Interstitial cystitis is a bladder disorder of unknown etiology characterized by a constellation of irritative lower urinary tract symptoms, including dysuria, associated pelvic pressure or pain, urinary urgency, urinary frequency, a compulsion to constantly void, a sensation of incomplete bladder emptying, and associated sexual dysfunction.

SIGNS AND SYMPTOMS

Physical examination is often unrevealing in patients with interstitial cystitis. Careful bimanual pelvic examination, palpation of the full bladder, rectal examination, and neurologic examination are mandatory to identify other causes of lower urinary tract dysfunction. Examination of females suffering from interstitial cystitis may reveal increased pain on palpation of the urethra and the base of the bladder. In male patients, careful digital examination of the prostate is mandatory to rule out prostatitis and prostadynia, which may confuse the diagnosis. Anxiety may also be noted on physical examination.

TESTING

Urinalysis is invariably normal in patients with interstitial cystitis as is urine cytology. Cystoscopy with hydrodistention of the bladder is indicated in all patients suspected of suffering from interstitial cystitis primarily to rule out other diseases that may account for the patient's irritative bladder symptoms, as there are no pathognomonic cystoscopic findings associated with cystoscopy other than the Hunner ulcer. The Hunner ulcer occurs in approximately 5% of patients with interstitial cystitis. Also known as the Hunner lesion, the Hunner ulcer appears as a friable patch or ulcer of the bladder mucosa. Scarring may occur with small vessels radiating outward from the central lesion (Fig. 102.2). Biopsy of the Hunner ulcer is mandatory to rule out occult malignancy including carcinoma in situ. Cystoscopic findings following bladder overdistention which are suggestive, but not diagnostic of interstitial cystitis, are small, petechial, raspberry-like hemorrhages within the bladder known as glomerulations (Fig. 102.3). These glomerulations are invariably present in at least three quadrants of the bladder with sparing of the trigone. They are frequently distributed in a unique lattice or checkerboard appearance. It should be noted that

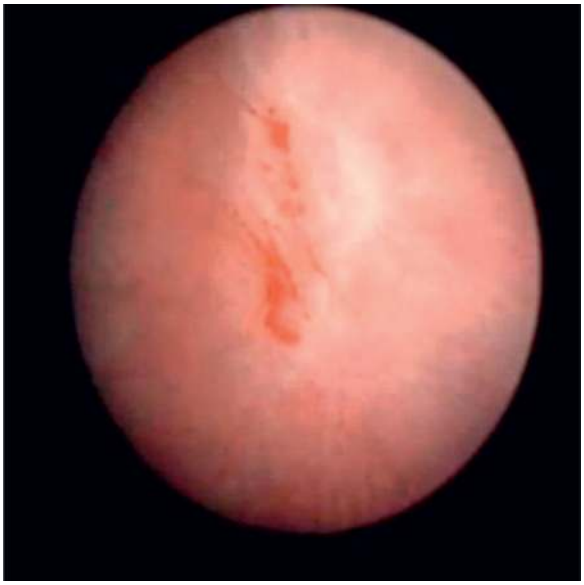


FIG 102.2 Hunner ulcer. (From Hammett J, Krupski TL, Corbett ST. Adolescent pelvic pain: interstitial cystitis. *J Pediatr Urol*. 2013;9(3):e134–e137.)

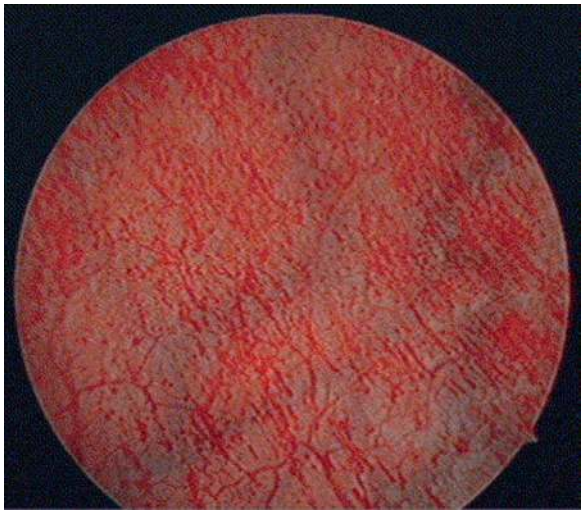


FIG 102.3 Bladder scar with mucosal tethering in a patient with interstitial cystitis. (From Rosamilia A. Painful bladder syndrome/interstitial cystitis. *Best Pract Res Clin Obstet Gynaecol*. 2005;19(6)843–859.)

glomerulations are also seen in patients suffering from bladder tumors, radiation cystitis, infection, chemical cystitis, and bladders that are chronically underfilled owing to renal failure or urinary diversion surgery (Fig. 102.4).

Many patients with interstitial cystitis have a dysfunctional bladder uroepithelium that allows increased urothelial permeability to the intravesicular instillation of potassium chloride. Intravesicular instillation of potassium chloride in a concentration of 40 mEq/100 mL of water will cause minimal symptoms in patients with normal bladders, but elicit immediate severe irritative bladder symptoms in patients suffering from interstitial cystitis.

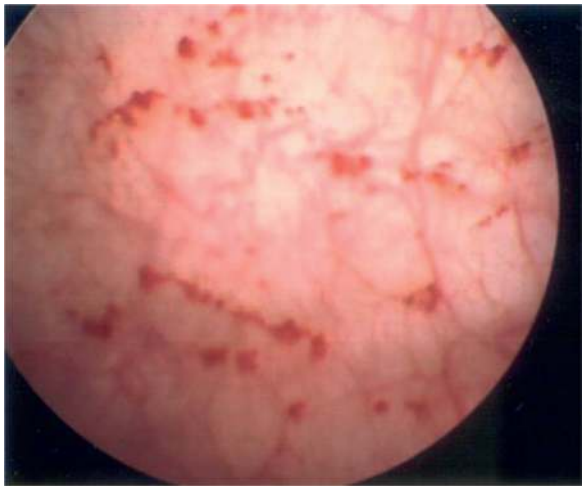


FIG 102.4 Glomerulations in a patient with interstitial cystitis. (From Barr S. Diagnosis and management of interstitial cystitis. *Obstet Gynecol Clin N Am*. 2014;41(3):397–407.)

Urodynamic studies may be abnormal in patients suffering from interstitial cystitis, but there are no specific findings to point to the diagnosis, although pain on bladder filling suggests the diagnosis of interstitial cystitis in the absence of other obvious urodynamic findings. Computerized tomography may also aid in the diagnosis of interstitial cystitis in complex cases (Fig. 102.5).

DIFFERENTIAL DIAGNOSIS

The diagnosis of interstitial cystitis is made on the basis of history and absence of clinical findings rather than specific laboratory, cystoscopic, urodynamic, or radiographic testing. For this reason, a careful targeted history and physical examination, with a systematic search for other causes of irritative lower urinary tract symptoms, must be carried out in every patient suspected of suffering from interstitial cystitis. The clinician must rule out other coexisting disease processes that may mimic interstitial cystitis including urinary tract infection, inflammatory vulvovestibulitis, pelvic malignancy, endometriosis, prostate disease, bladder outlet obstruction with overflow incontinence, and urethral diverticulum (Fig. 102.6). Neurologic disorders, including Parkinson disease, spinal tumor, spinal stenosis, and multiple sclerosis, must also be ruled out (Table 102.1). The clinician must also identify coexisting psychological and behavioral abnormalities that may exacerbate the symptoms associated with interstitial cystitis.

TREATMENT

Treatment of interstitial cystitis is focused on providing symptomatic relief and managing associated stress. A good starting point in the treatment of interstitial cystitis is to help patients identify foods and beverages that may exacerbate the symptoms and remove them from their diet. Frequent

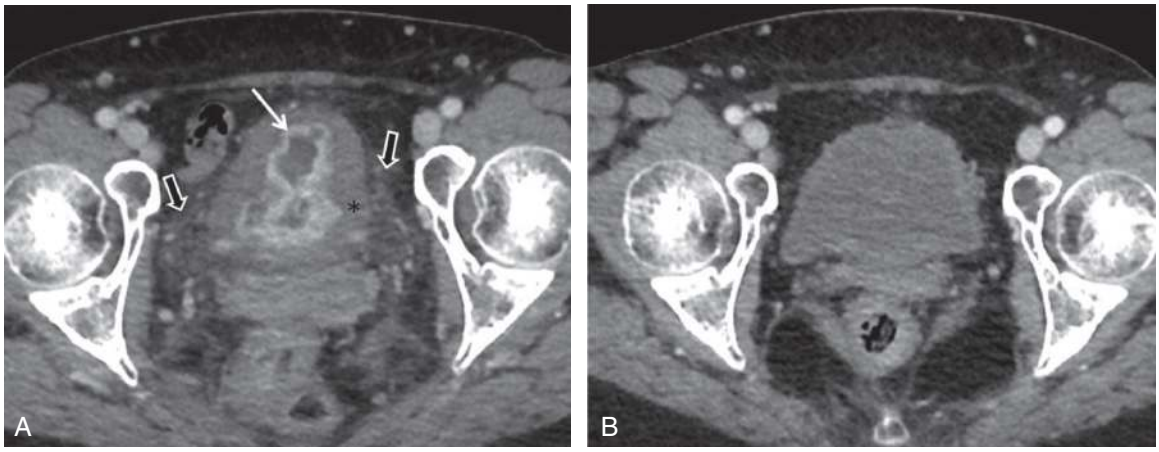


FIG 102.5 Axial transverse computerized tomography (CT) image of the bladder at a portal phase after intravenous administration of iodinated contrast medium. **A,** CT at initial presentation. The bladder shows a low level of repletion, an edematous submucosal wall thickening (asterisk), and a marked enhancement of the mucosa (*white arrow*). Infiltration of the surrounding fat (*black arrows*) is also denoted. **B,** Follow-up CT after systemic steroid treatment, showing normalization of the bladder wall. (From Kinan El H, Hélène L, Audrey M-L, et al. A case of severe interstitial cystitis associated with pembrolizumab. *Curr Prob Cancer*. 2021;4:100–101.)

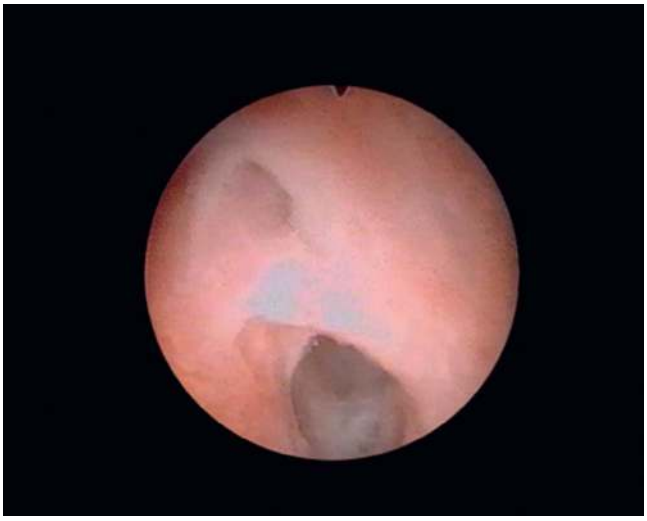


FIG 102.6 Urethral diverticulum. Endoscopic aspect of multiple urethral diverticula. (From Geavlete P, Drăguţescu M, Mulţescu R, et al. Endoscopic management of urethral abnormalities. In: *Endoscopic diagnosis and treatment in urethral pathology*. San Diego: Academic Press; 2016:189–198.)

offenders include caffeinated beverages, alcohol, carbonated beverages, acidic foods including citrus and tomatoes, vinegar, spicy foods, chocolate, and cranberry juice. Foods and beverages that may provide amelioration of symptoms include increased water intake, milk, rice, mushrooms, bananas, melon, blueberries, chicken, and eggs.

The addition of oral polysulfated xylan and pentosan polysulfate may provide symptomatic relief in some patients suffering from interstitial cystitis. The mechanism of action of this drug is thought to be owing to its protective action on the bladder wall in a manner analogous to the naturally occurring glycosaminoglycan coating of the inner bladder in healthy humans. Improvement in the irritative bladder symptoms following intravesicular instillation of potassium chloride supports this theory. This drug may also decrease inflammation by preventing mast cell influx into the bladder lining. A trial of 6–9 months is required before significant improvement may be observed.

The addition of anticholinergic agents, such as oxybutynin and tolterodine, may also be considered in patients with significant irritative symptoms. Amitriptyline given at night may help with sleep disturbance and treat underlying depression. Immune modulation with cyclosporine, a calcineurin, may reduce irritative symptoms, especially in those patients in whom Hunner ulcer is observed on cystoscopy. Intravesicular instillation of dimethyl sulfoxide with or without the addition of steroids, bicarbonate, and heparin may also provide symptomatic relief as may bladder hydrodistention. Recent clinical experience suggests that intradetrussor muscle injection of type A botulinum as well as implantation of spinal cord stimulator and sacral nerve root and pudendal nerve neurostimulators may help in carefully selected patients.

COMPLICATIONS AND PITFALLS

The lack of pathognomonic findings in the diagnosis of interstitial cystitis makes the possibility of misdiagnosis significant. Given the serious nature of many of the diseases that mimic interstitial cystitis, a diligent search for diagnosable causes of the patient's irritative lower urinary tract symptomatology is of the utmost importance.

TABLE 102.1 Diseases That May Mimic Interstitial Cystitis

Urologic	Infectious/Inflammatory	Gynecologic	Neurologic
Bladder malignancy	Urinary tract infection	Pelvic malignancy	Spinal tumor
Bladder outlet obstruction	Infected Bartholin or Skene gland	Pelvic inflammatory disease	Cauda equine syndrome
Radiation cystitis	Tuberculous cystitis	Fibroid tumors	Spinal stenosis
Chemical cystitis	Schistosomiasis	Endometriosis	Parkinson disease
Urethrtitis	Vulvovestibulitis	Low estrogen atrophy	Multiple sclerosis
Prostate disease	Vaginitis	Surgical adhesions	Stroke
Urolithiasis	Diverticulitis	Vulvadynia	Neuropathic bladder
Urethral diverticulum	Inflammatory bowel disease		

CLINICAL PEARLS

Although interstitial cystitis is a common disorder, it is often misdiagnosed. Therefore, in patients suspected of suffering from interstitial cystitis, a careful evaluation to identify underlying disease processes is mandatory. The disease commonly coexists with various somatic and psychological disorders which must be treated concurrently.

SUGGESTED READINGS

Bharucha AE, Lee TH. Anorectal and pelvic pain. *Mayo Clin Proc*. 2016;91(10):1471–1486.

Chen Y, Ying Z, Xiao Y, et al. The diagnostic and therapeutic efficacy of cystoscopy with hydrodistension and random biopsies in clinically suspected interstitial cystitis/bladder pain syndrome. *Eur J Obstet Gynecol Reprod Biol*. 2021;265:156–161. https://doi.org/10.1016/j.ejogrb.2021.08.025.

Chermansky CJ, Guirguis MO. Pharmacologic management of interstitial cystitis/bladder pain syndrome. *Urol Clin N Am*. 2022;49(2):273–282. https://doi.org/10.1016/j.ucl.2022.01.003.

Jhang JF, Kuo HC. Clinical characteristics and immunochemical study of urothelial dysfunction in the patients with interstitial cystitis/bladder pain syndrome with and without Hunner's lesion. *Urol Sci*. 2015;26(2 suppl 1):S24.

Moufarrij N, Huebner M. Relief of interstitial cystitis/bladder pain syndrome by spinal cord stimulation. *Interdiscip Neurosurg*. 2022;28:101517. https://doi.org/10.1016/j.inat0.2022.101517.

Vasudevan V, Moldwin R. Addressing quality of life in the patient with interstitial cystitis/bladder pain syndrome. *Asian J Urol*. 2017;4(1):50–54.

Testicular Torsion

ICD-10 CODE N44.00

THE CLINICAL SYNDROME

Testicular torsion is a painful urologic emergency caused by twisting of the spermatic cord causing compromise of the blood supply to the affected testis (Fig. 103.1). The extent of torsion is variable, ranging from 90 to 720 degrees, with complete torsion occurring with twisting of 360 degrees or greater. Although testicular torsion is primarily a disease of neonates and adolescents, it is occasionally seen in 40- to 50-year-olds as well as in patients who suffer testicular trauma. In adolescents and adults, the tunica vaginalis is attached to the posterolateral aspect of the testes, effectively tethering the testes in place. In approximately 17% of patients this attachment is higher than normal, allowing the spermatic cord to rotate within the tunica vaginalis (Fig. 103.2). This anatomic variation, known as the bell-clapper deformity, is associated with an increased incidence of intravaginal testicular torsion in this age group (Fig. 103.3). In neonates, because the tunica vaginalis is not yet attached to the gubernaculum, both the spermatic cord and tunica vaginalis twist as a unit. This type of testicular torsion is called extravaginal torsion and can occur pre- or postnatally.

Testicular torsion is a true urologic emergency and must be treated within 6 hours of symptom onset if the testicle is to remain viable. In almost all patients, testicular necrosis occurs after 24 hours of vascular compromise (Fig. 103.4). Testicular torsion is associated with testicular malignancy.

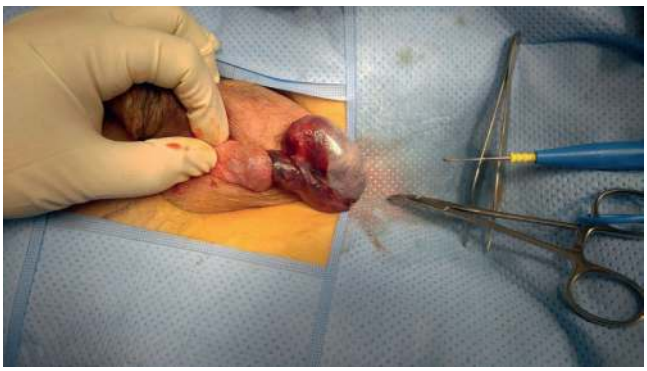


FIG 103.1 Intraoperative finding: dusty looking and congested right testis. (From Tang YH, Yeung VHW, Chu PSK, et al. A 55-year-old man with right testicular pain: too old for torsion? *Urol Case Rep*. 2017;11:74–75.)

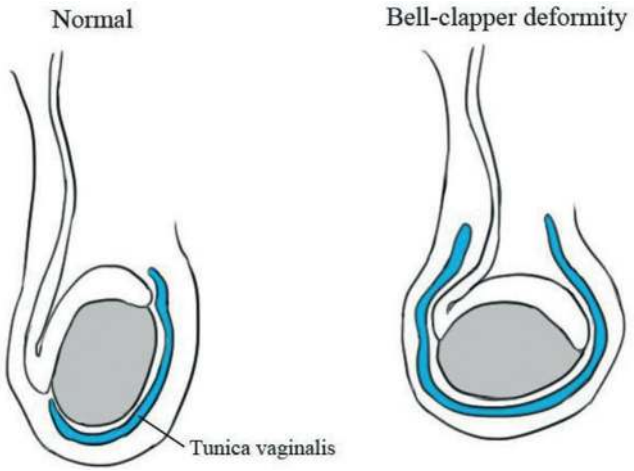


FIG 103.2 Computer drawing illustrating high attachment of tunica vaginalis. (From Tang YH, Yeung VHW, Chu PSK, et al. A 55-year-old man with right testicular pain: too old for torsion? *Urol Case Rep*. 2017;11:74–75.)

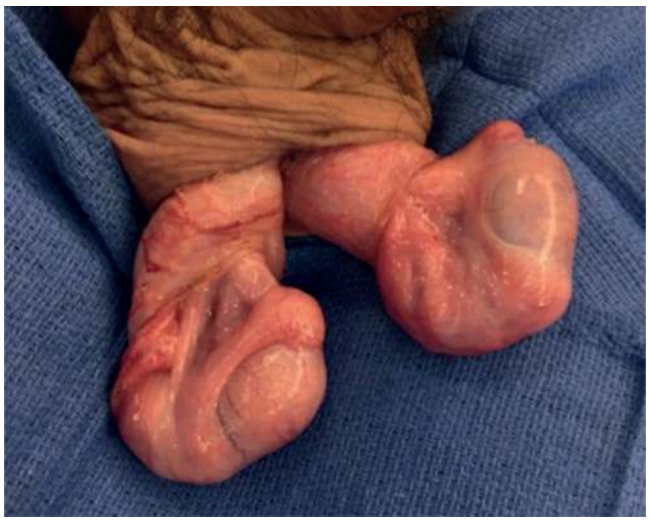


FIG 103.3 Bilateral "bell-clapper" configuration of the testicles. Note the transverse testicular lie of the testicle relative to the spermatic cord. This patient underwent bilateral orchidopexy with complete resolution of his previously intermittent bilateral testicular pain. (From Bowlin PR, Gatti JM, Murphy JP. Pediatric testicular torsion. *Surg Clin North Am*. 2017;97(1):161–172.)

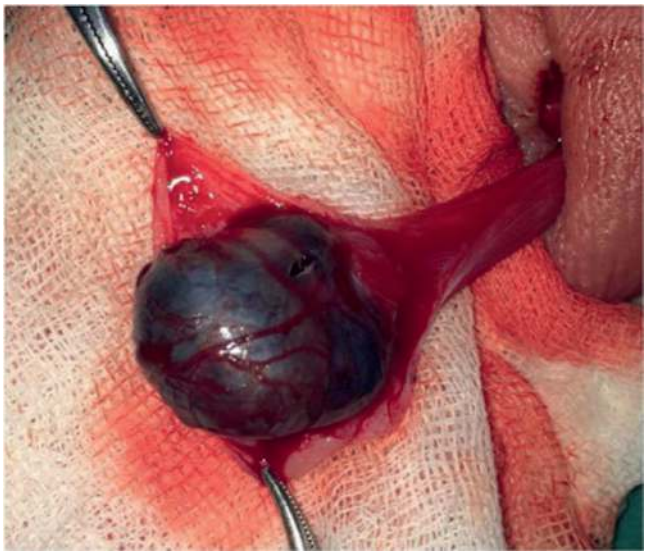


FIG 103.4 Photographic view of the left testis during operation shows left testicular necrosis. (From Acar T, Efe D. Is contrast-enhanced MRI efficient in testicular infarction mimicking testicular tumor on scrotal ultrasound? *Turk J Emerg Med.* 2015;15(4):192–193.)

Sequelae of testicular torsion include testicular infarction, infection, infertility, pathologic alterations of the retained injured testis, and postsurgical cosmetic deformity.

SIGNS AND SYMPTOMS

The pain of testicular torsion occurs suddenly and in the absence of antecedent trauma, without warning. The pain is severe and is associated with nausea and vomiting in a third of patients. Physical examination will reveal a tender, high-riding testis with an abnormal transverse position and loss of the cremasteric reflex. Scrotal edema is invariably present, which complicates the examination. Lifting the affected torsed testis will not relieve the pain as is seen with acute epididymitis. Torsion of the testicular appendix will produce a characteristic "blue-dot" sign which is caused by the cyanotic testicular appendix (Fig. 103.5).

TESTING

Routine laboratory testing and urinalysis will provide limited diagnostic information but may help point the clinician toward other diseases that may be mimicking the clinical presentation of testicular torsion (e.g., urinary tract infection, epididymitis). The Testicular Workup for Ischemia and Suspected Torsion (TWIST) scoring system was devised to increase the accuracy of clinical diagnosis of testicular torsion (Box 103.1). The specificity of this scoring system is such that high-risk patients with a score of five or greater can proceed directly to surgery without color Doppler sonography, with intermediate-risk patients with a score of three or four undergoing urgent color Doppler sonography to determine if surgery is required.

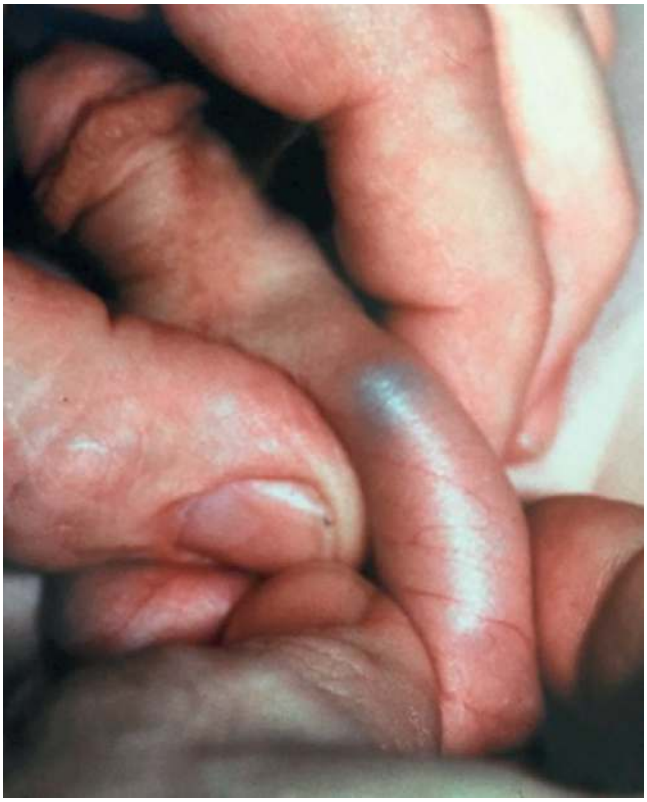


FIG 103.5 Physical examination of the scrotum demonstrating a classic "blue-dot" sign.

BOX 103.1 The TWIST (Testicular Workup for Ischemia and Suspected Torsion) Scoring System for the Diagnosis of Testicular Torsion

Testis swelling	2 points
Hard testis	2 points
Absent cremasteric reflex	1 point
Nausea and vomiting	1 point
High-riding testis	1 point

Score of >5 = high risk.
Score of 3–4 = intermediate risk.
Score of 2 or below = low risk.

Color Doppler sonography is highly accurate in diagnosing testicular torsion (Fig. 103.6). Findings consistent with the diagnosis include absent or significantly decreased blood flow to the affected testis, decreased flow velocity within the intratesticular arteries, increased resistive indices within the intratesticular arteries, and low resistance hypervascularity after partial detorsion of the affected testis. Magnetic resonance imaging with contrast is also highly accurate when whirlpool and torsion knot patterns are identified (Fig. 103.7). Radionuclide studies are also highly accurate in identifying alterations in testicular blood flow. Recent clinical experience with near-infrared spectroscopy suggests that it may prove to be a highly efficient and accurate diagnostic modality in adults with suspected testicular torsion.

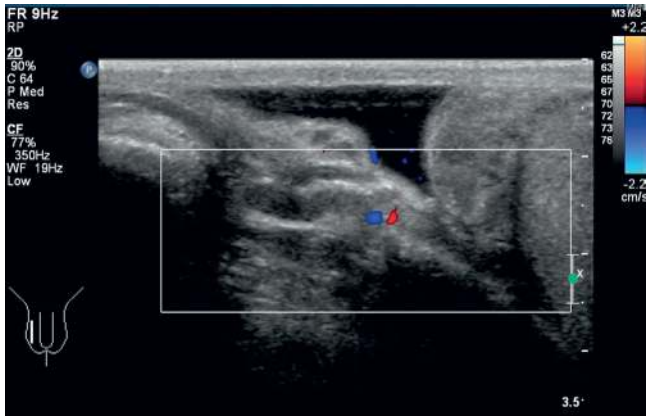


FIG 103.6 The ultrasound image of our patient demonstrated a twisted cord, reduced echogenicity of right testis and diffusely enlarged epididymis with near absent color Doppler flow. (From Tang YH, Yeung VHW, Chu PSK, et al. A 55-year-old man with right testicular pain: too old for torsion? *Urol Case Rep*. 2017;11:74–75, Fig. 1.)

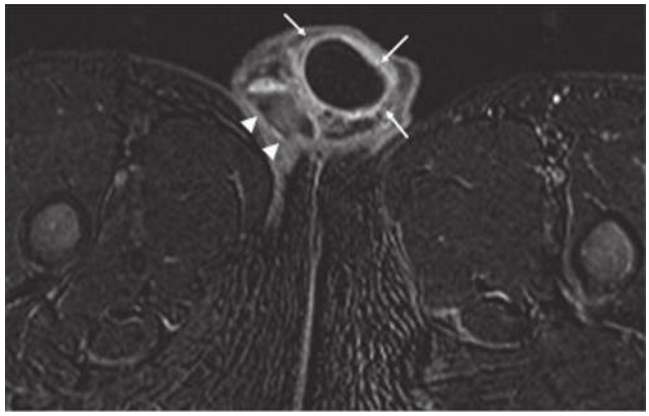


FIG 103.7 Postcontrast subtraction axial magnetic resonance image reveals left testicular enlargement and left paratesticular enhancement. Note that the affected left testis is hypointense, and no contrast enhancement is observed that is diagnostic for testicular torsion *(arrows)*. However, right unaffected testis is preserved and shows normal contrast enhancement *(arrowheads)*. (From Acar T, Efe D. Is contrast-enhanced MRI efficient in testicular infarction mimicking testicular tumor on scrotal ultrasound? *Turk J Emerg Med*. 2015;15(4):192–193.)

DIFFERENTIAL DIAGNOSIS

Testicular torsion is a clinical diagnosis supported by a combination of clinical history, physical examination, and color Doppler sonography. Pain syndromes that may mimic testicular torsion include epididymitis, orchitis, epididymo-orchitis, torsion of the epididymal appendage, testicular malignancy, hydrocele, idiopathic testicular infarction, and traumatic injuries, including hematoma and rupture.

TREATMENT

Urgent surgical detorsion is the definitive treatment for testicular torsion and must be carried out within 6 hours of symptom onset. In some patients, manual detorsion may help improve blood flow to the ischemic testis, but definitive surgical treatment with orchiopexy is ultimately required to prevent recurrence. If testicular necrosis has occurred and orchiectomy is required, a testicular prosthesis may be placed after complete healing of the orchiectomy.

Preoperatively, opioid analgesics will decrease ischemic pain. Antiemetic and anxiolytics may also be used for symptomatic relief, with care being taken not to potentiate the respiratory side effects of administered opioids.

COMPLICATIONS AND PITFALLS

Failure to timely diagnose and treat testicular torsion will result in testicular necrosis. Given the incidence of malignancy that is associated with testicular torsion, a high index of suspicion is indicated. It should be remembered that torsion of an undiagnosed undescended testis can occur and is an extremely uncommon cause of the acute abdomen (Fig. 103.8).

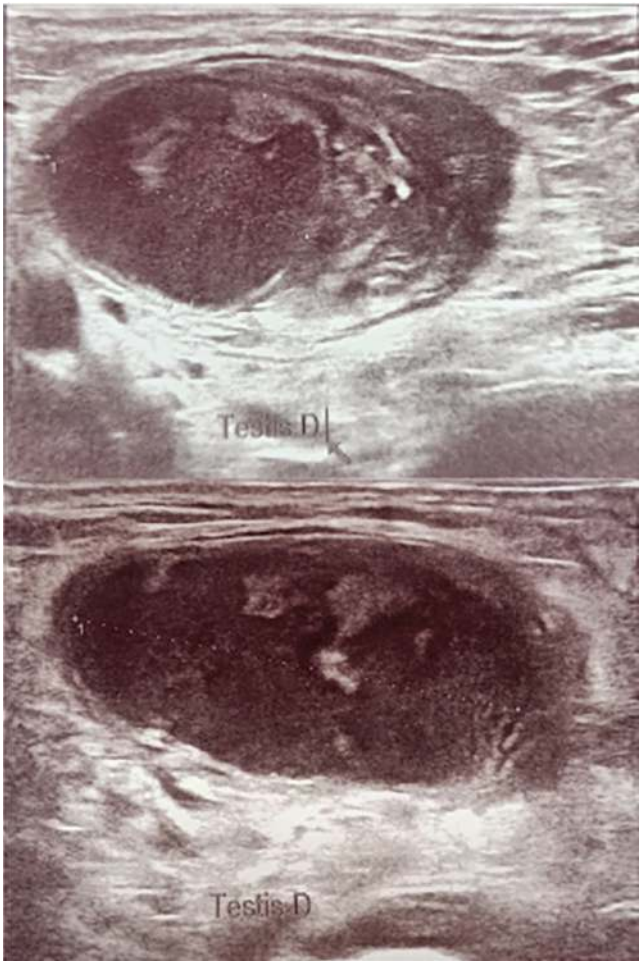


FIG 103.8 Ultrasound images showing torsion of a right undescended inguinal testicle measuring 35×21×17 mm, heterogeneous hypoechoic containing hyperechoic areas, not vascularized on Doppler. (From Anouar El M, Abdelghani O, Mohamed M, et al. Acute abdomen revealing a testicular torsion of an undescended testis "case report". *Urol Case Rep*. 2022;42:102035. https://doi.org/10.1016/j.eucr.2022.102035.)

CLINICAL PEARLS

The use of the TWIST scoring test will increase the accuracy of clinical diagnosis in patients suspected of suffering from testicular torsion. Patients who score in the high-risk category should be taken directly to surgery as time is of the essence.

SUGGESTED READINGS

Aihole JA. Testicular torsion; clinical diagnosis or imaging diagnosis? *Radiol Case Rep*. 2022;17(8):2665–2667. https://doi.org/10.1016/j.radcr.2022.05.010.

El Moudane A, Ouraghi A, Mokhtari M, et al. Acute abdomen revealing a testicular torsion of an undescended testis "case report". *Urol Case Rep*. 2022;42:102035. https://doi.org/10.1016/j.eucr.2022.102035.

Fehér AM, Bajory Z. A review of main controversial aspects of acute testicular torsion. *J Acute Dis*. 2016;5(1):1–8.

Gielchinsky I, Suraqui E, Hidas G, et al. Pregnancy rates after testicular torsion. *J Urol*. 2016;196(3):852–855.

Ludvigson AE, Beaule LT. Urologic emergencies. *Surg Clin North Am*. 2016;96(3):407–424.

Pogorelić Z, Anand S, Artuković L, Krishnan N. Comparison of the outcomes of testicular torsion among children presenting during the Coronavirus Disease 2019 (COVID-19) pandemic versus the pre-pandemic period: a systematic review and meta-analysis. *Journal of Pediatric Urology*. 2022;18(2):202–209. https://doi.org/10.1016/j.jpurol.2022.01.005.

Sheth KR, Keays M, Grimsby GM, et al. Diagnosing testicular torsion before urological consultation and imaging: validation of the twist score. *J Urol*. 2016;1103(6):1870–1876.

Tang YH, Yeung VH, Chu PS, et al. A 55-year-old man with right testicular pain: too old for torsion? *Urol Case Rep*. 2017;11:74–75.

Levator Ani Syndrome

ICD-10 CODE M79.7

THE CLINICAL SYNDROME

The levator ani muscle is susceptible to the development of myofascial pain syndrome. Such pain is often the result of repetitive microtrauma to the muscle during such activities as mountain biking and horseback riding (Fig. 104.1). Injury to the muscle during childbirth or blunt trauma to the muscle may also incite levator ani myofascial pain syndrome (Fig. 104.2).

Myofascial pain syndrome is a chronic pain syndrome that affects a focal or regional portion of the body. The sine qua non of myofascial pain syndrome is the finding of myofascial trigger points on physical examination. Although these trigger points are generally localized to the part of the body affected, the pain is often referred to other areas. This referred pain may be misdiagnosed or attributed to other organ systems, thus leading to extensive evaluation and ineffective treatment. Patients with myofascial pain syndrome involving the levator ani muscle have primary pain in the pelvic floor that may be referred to the posterior buttocks and posterior lower extremity (Fig. 104.3).

The trigger point, a pathognomonic lesion of myofascial pain syndrome, is characterized by a local point of exquisite tenderness in the affected muscle. Mechanical stimulation of the trigger point by palpation or stretching produces not only intense local pain but also referred pain. In addition, involuntary withdrawal of the stimulated muscle, called a jump sign, often occurs and is characteristic of myofascial pain syndrome. Patients with levator ani syndrome have a trigger point along the rectum or perineum.

Taut bands of muscle fibers are often identified when myofascial trigger points are palpated. Despite this consistent

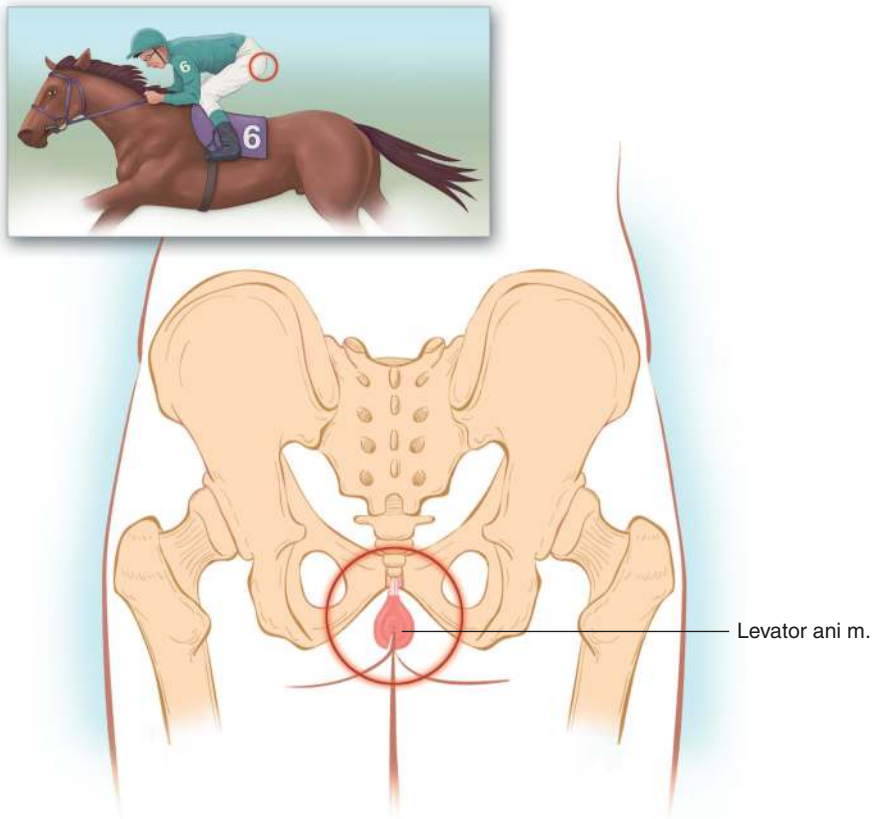


FIG 104.1 Levator ani syndrome may be caused by repetitive microtrauma to the muscle during such activities as mountain biking and horseback riding.

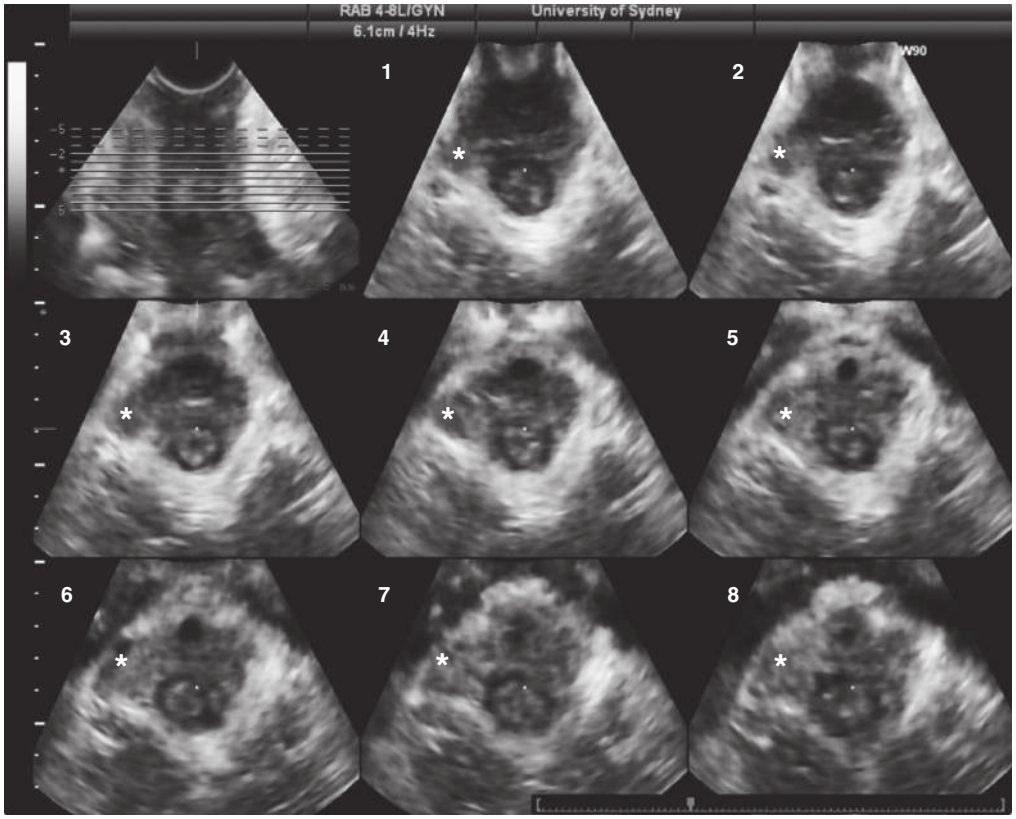


FIG 104.2 Tomographic ultrasound imaging showing right-sided avulsion. The *asterisks* indicate a right-sided levator avulsion. (From Shek KL, Dietz HP. Can levator avulsion be predicted antenatally? *Am J Obstet Gynecol*. 2010;202(6):5106.)

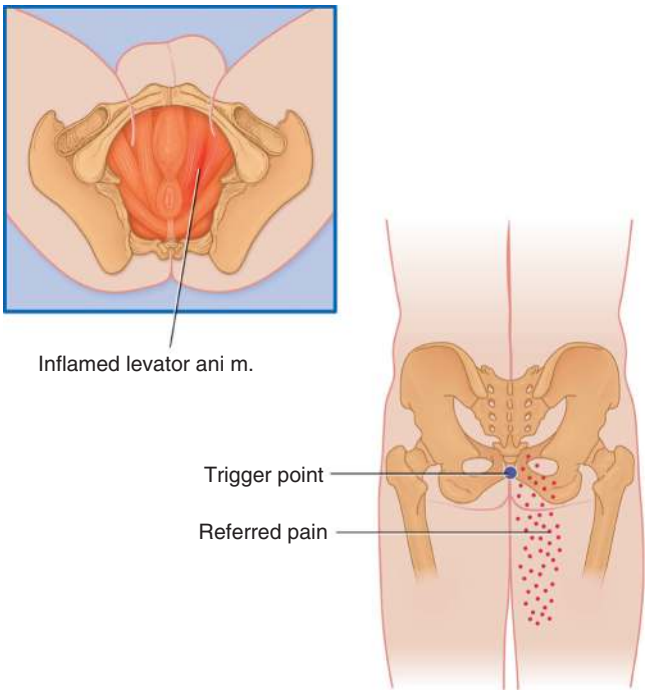


FIG 104.3 Patients with myofascial pain syndrome involving the levator ani muscle have primary pain in the pelvic floor that may be referred to the posterior buttocks and posterior lower extremity. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:385.)

physical finding, the pathophysiology of the myofascial trigger point remains elusive, although trigger points are believed to be caused by microtrauma to the affected muscle. This trauma may result from a single injury, repetitive microtrauma, or chronic deconditioning of the agonist and antagonist muscle unit.

In addition to muscle trauma, various other factors seem to predispose patients to develop myofascial pain syndrome. For instance, a weekend athlete who subjects his or her body to unaccustomed physical activity may develop myofascial pain syndrome. Poor posture while sitting at a computer or while watching television has also been implicated as a predisposing factor. Previous injuries may result in abnormal muscle function and lead to the development of myofascial pain syndrome. All these predisposing factors may be intensified if the patient also suffers from poor nutritional status or coexisting psychological or behavioral abnormalities, including chronic stress and depression. The levator ani muscle seems to be particularly susceptible to stress-induced myofascial pain syndrome.

Stiffness and fatigue often coexist with pain, and they increase the functional disability associated with this disease and complicate its treatment. Myofascial pain syndrome may occur as a primary disease state or in conjunction with other painful conditions, including radiculopathy and chronic regional pain syndromes. Psychological or behavioral abnormalities, including depression, frequently

coexist with the muscle abnormalities, and management of these psychological disorders is an integral part of any successful treatment plan.

SIGNS AND SYMPTOMS

The trigger point, the pathognomonic lesion of levator ani syndrome, is characterized by a local point of exquisite tenderness in the levator ani muscle. Mechanical stimulation of the trigger point by palpation or stretching produces primary pain in the pelvic floor and referred pain in the posterior buttocks and posterior lower extremity (see Fig. 104.3). In addition, the jump sign is often present.

TESTING

Biopsies of clinically identified trigger points have not revealed consistently abnormal histologic features. The muscle hosting the trigger point has been described either as "moth-eaten" or as containing "waxy degeneration." Increased plasma myoglobin has been reported in some patients with levator ani syndrome, but this finding has not been corroborated by other investigators. Electrodiagnostic testing has revealed an increase in muscle tension in some patients, but again, this finding has not been reproducible. Because of the lack of objective diagnostic testing, the clinician must rule out other coexisting disease processes that may mimic levator ani syndrome (see "Differential Diagnosis"). Imaging modalities, including computed tomography, magnetic resonance imaging, and ultrasound, should be considered if the diagnosis of levator ani syndrome is in question (see Figs. 104.2 and 104.4).

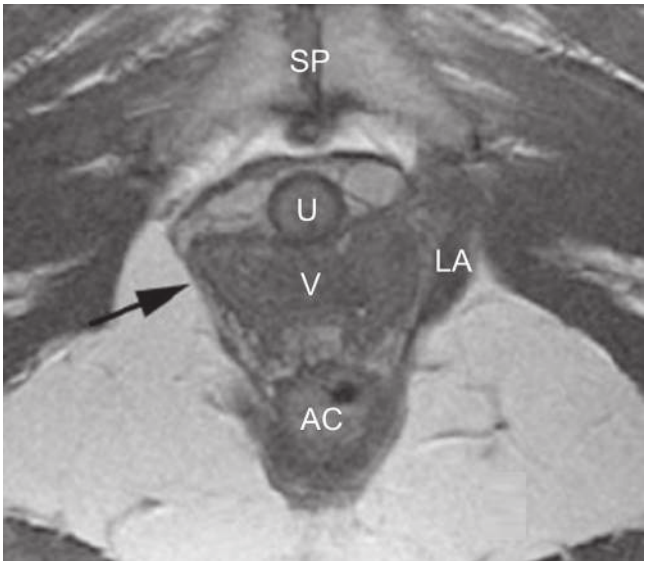


FIG 104.4 Pelvic magnetic resonance image with external phased array. The levator ani (LA) is damaged on the right side (*arrow*), with a complete detachment from the inferior pubic rami. *AC*, Anal canal; *SP*, symphysis pubis; *U*, urethra; *V*, vagina. (From Santoro GA, Sultan AH. Pelvic floor anatomy and imaging. *Semin Colon Rectal Surg.* 2016;27(1):5–14.)

DIFFERENTIAL DIAGNOSIS

The diagnosis of levator ani syndrome is based on clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for trigger points and identification of a positive jump sign, must be carried out in every patient suspected of suffering from levator ani syndrome. The clinician must rule out other coexisting disease processes that may mimic levator ani syndrome, including primary inflammatory muscle disease, primary hip disorders, rectal and pelvic tumors, gluteal bursitis, and gluteal nerve entrapment (Fig. 104.5). The use of electrodiagnostic and radiographic testing can identify coexisting disorders such as rectal and pelvic tumors and lumbosacral nerve lesions. The clinician must also identify coexisting psychological and behavioral abnormalities that may mask or exacerbate the symptoms of levator ani syndrome.

TREATMENT

Treatment is focused on blocking the myofascial trigger point and achieving prolonged relaxation of the affected muscle. Because the mechanism of action is poorly understood, an element of trial and error is often required when developing a treatment plan. Conservative therapy consisting of trigger point injections with local anesthetic or saline solution is the starting point. Because underlying depression and anxiety are present in many patients suffering from fibromyalgia of the cervical spine, the administration of antidepressants is an integral part of most treatment plans. Pregabalin and gabapentin have also been shown to provide some palliation of the symptoms associated with fibromyalgia. Milnacipran, a serotonin–norepinephrine reuptake inhibitor has also been shown to be effective in the management of fibromyalgia. The synthetic cannabinoid nabilone has also been used to manage fibromyalgia in selected patients who have failed to respond to other treatment modalities.

In addition, several adjuvant methods are available for the treatment of fibromyalgia of the cervical spine. The therapeutic use of heat and cold is often combined with trigger point injections, antidepressants, and physical therapy to achieve pain relief. Some patients experience decreased pain with the application of transcutaneous nerve stimulation or electrical stimulation to fatigue the affected muscles. Exercise may also provide some palliation of symptoms and reduce the fatigue associated with this disease. The injection of minute quantities of botulinum toxin type A directly into trigger points has been used with success in patients who have not responded to traditional treatment modalities.

COMPLICATIONS AND PITFALLS

Trigger point injections are extremely safe if careful attention is paid to the clinically relevant anatomy. Sterile technique must be used to avoid infection, along with universal

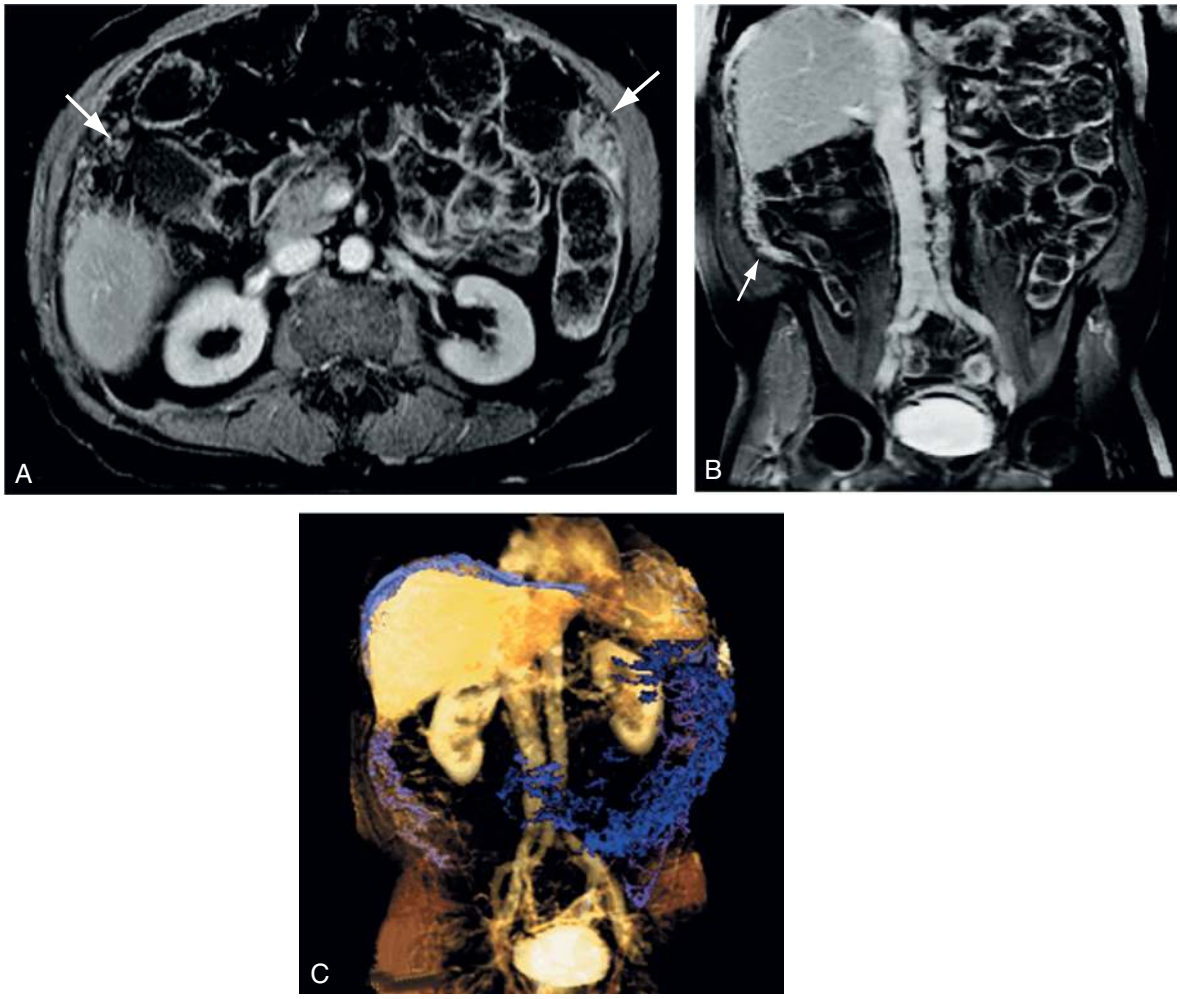


FIG 104.5 Recurrent rectal cancer with carcinomatosis. Axial **(A)** and coronal **(B)** gadolinium-enhanced spoiled gradient-echo magnetic resonance images show heterogeneously enhancing peritoneal and omental metastases (*arrows*) from recurrent rectal cancer. **C,** Three-dimensional color model generated from the coronal gadolinium-enhanced images shows the distribution of the peritoneal and omental tumor in purple. (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:2780.)

precautions to minimize any risk to the operator. Most complications of trigger point injection are related to needle-induced trauma at the injection site and in underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. The avoidance of overly long needles can decrease the incidence of trauma to underlying structures. Special care must be taken to avoid trauma to the sciatic nerve.

CLINICAL PEARLS

Although levator ani syndrome is a common disorder, it is often misdiagnosed. Therefore, in patients suspected of suffering from levator ani syndrome, a careful evaluation to identify underlying disease processes is mandatory. Levator ani syndrome often coexists with various somatic and psychological disorders.

SUGGESTED READINGS

Arnold LM. The pathophysiology, diagnosis and treatment of fibromyalgia. *Psychiatr Clin North Am*. 2010;33(2):375–408.

Bharucha AE, Lee TH. Anorectal and pelvic pain. *Mayo Clin Proc*. 2016;91(10):1471–1486.

Bradley LA. Pathophysiology of fibromyalgia. *Am J Med*. 2009;122(12 suppl 1):S22–S30.

Corton MM. Anatomy of pelvic floor dysfunction. *Obstet Gynecol Clin North Am*. 2009;36(3):401–419.

Di Tella M, Ghiggia A, Tesio V, et al. Pain experience in Fibromyalgia Syndrome: the role of alexithymia and psychological distress. *J Affect Disord*. 2017;208:87–93.

Farré M, Farré A, Fiz J, et al. Cannabis use in fibromyalgia. In: Preedy VR, ed. *Handbook of cannabis and related pathologies*. San Diego: Academic Press; 2017:e158–e167.

Imamura M, Cassius DA, Fregni F. Fibromyalgia: from treatment to rehabilitation. *Eur J Pain Suppl*. 2009;3(2):117–122.

Rao SSC, Bharucha AE, Chiarioni G, et al. Anorectal disorders. *Gastroenterology*. 2016;150(6):1430–1442.e4.

Santoro GA, Sultan AH. Pelvic floor anatomy and imaging. *Semin Colon Rectal Surg*. 2016;27(1):5–14.

Waldman SD. Levator ani syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017: 441–444.

Wong J, McClurg AB, Carey ET. Levator ani trigger point injections and chemodenervation with onabotulinum toxin a: an introduction and how-to guide. *J Minimal Invas Gynecol*. 2021;28(suppl 11):S27–S28. https://doi.org/10.1016/j.jmig.2021.09.349.

105

Coccydynia

ICD-10 CODE M53.3

THE CLINICAL SYNDROME

Coccydynia is a common syndrome characterized by pain localized to the tailbone that radiates into the lower sacrum and perineum. Coccydynia affects female patients more frequently than male patients. It occurs most commonly after direct trauma from a kick or a fall directly onto the coccyx. Coccydynia can also occur after a difficult vaginal delivery. The pain of coccydynia is thought to be the result of strain of the sacrococcygeal ligament or, occasionally, fracture of the coccyx (Box 105.1). Less commonly, arthritis of the sacrococcygeal joint can cause coccydynia. A recent clinical report attributed the onset of coccydynia to use of an intravaginal contraceptive ring.

SIGNS AND SYMPTOMS

On physical examination, patients exhibit point tenderness over the coccyx; the pain increases with movement of the coccyx. Movement of the coccyx may also cause sharp paresthesias into the rectum, which patients find quite distressing. On rectal examination, the levator ani, piriformis, and coccygeus muscles may feel indurated, and palpation of these muscles may induce severe spasm. Sitting exacerbates the pain of coccydynia, and patients often attempt to sit on one buttock to avoid pressure on the coccyx (Fig. 105.1).

TESTING

Plain radiography is indicated in all patients who present with pain thought to be emanating from the coccyx to rule out occult bony disease and tumor. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the pelvis, including the sacrococcygeal joint and ligament, are indicated if occult mass or tumor is suspected (Fig. 105.2). Radionuclide bone scanning may be useful to exclude stress fractures not visible on plain radiographs. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

BOX 105.1 Etiology of Coccydynia

- Postpartum
- Traumatic
- Degenerative
- Idiopathic
- Psychosomatic

DIFFERENTIAL DIAGNOSIS

Primary disease of the rectum and anus is occasionally confused with the pain of coccydynia. Primary tumors or metastatic lesions of the sacrum or coccyx may also manifest as coccydynia (Fig. 105.3, Box 105.2). Proctalgia fugax can be distinguished from coccydynia because movement of the coccyx does not reproduce the pain. Insufficiency fractures of the pelvis or sacrum and disorders of the sacroiliac joints may on occasion mimic coccydynia.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors, and a foam donut to prevent further irritation to the sacrococcygeal ligament is a reasonable first step in the treatment of coccydynia. If the patient does not experience rapid improvement, injection is a reasonable next step.

To treat the pain of coccydynia, the patient is placed in the prone position. The legs and heels are abducted to prevent tightening of the gluteal muscles, which can make identification of the sacrococcygeal joint difficult. A wide area of skin is prepared with antiseptic solution so that all the landmarks can be palpated aseptically. A fenestrated sterile drape is placed to avoid contamination by the palpating finger. The middle finger of the operator's nondominant hand is placed over the sterile drape into the natal cleft, with the fingertip palpating the sacrococcygeal joint at the base of the sacrum. After locating the sacrococcygeal joint, a 1½-inch, 25-gauge needle is inserted through the skin at a 45-degree angle into the region of the sacrococcygeal joint and ligament. If the ligament is penetrated, a "pop" will be felt, and the needle should be withdrawn through the ligament. If contact with the bony wall of the sacrum occurs, the needle should be withdrawn slightly to disengage the needle tip from the periosteum. When the needle is satisfactorily positioned, a syringe containing 5 mL of 1% preservative-free lidocaine and 40-mg methylprednisolone is attached to the needle. Gentle aspiration is carried out to identify cerebrospinal fluid

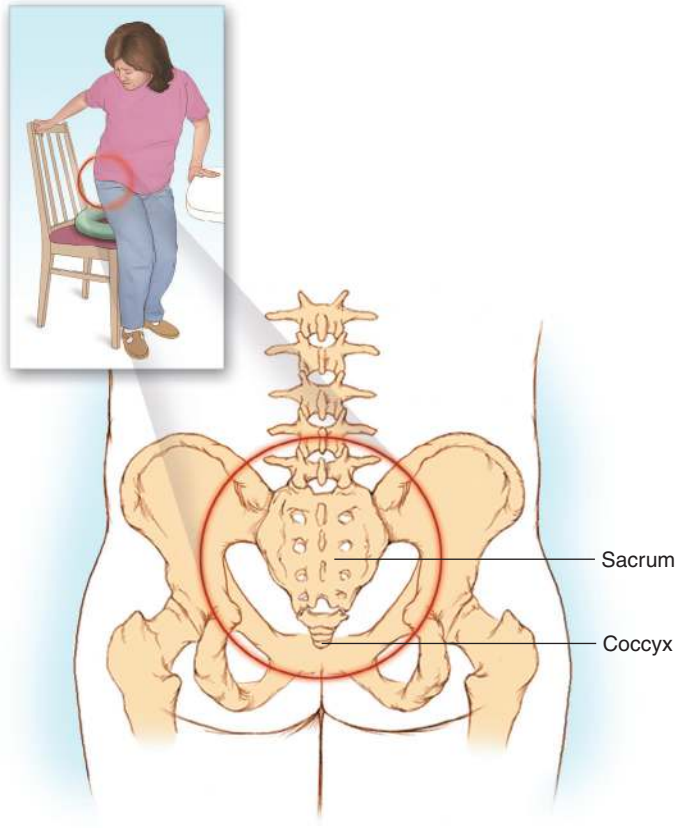


FIG 105.1 The pain of coccydynia is localized to the coccyx and is made worse by sitting.

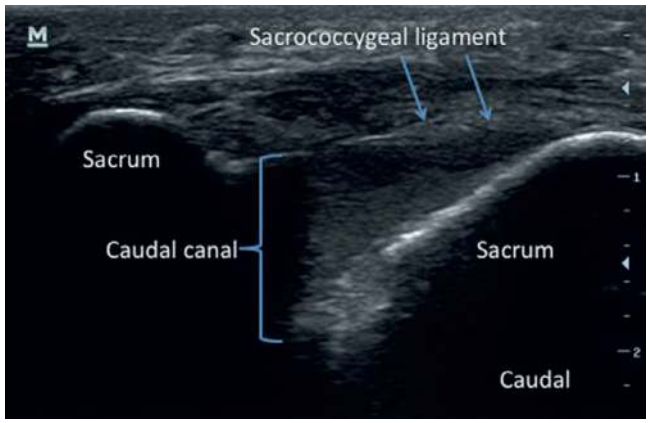


FIG 105.2 Longitudinal ultrasound image demonstrating the sacrococcygeal ligament.

or blood. If the aspiration test result is negative, the contents of the syringe are slowly injected. Little resistance to injection should be felt. Any significant pain or sudden increase in resistance during injection suggests incorrect needle placement, and the clinician should stop injecting immediately and reassess the needle position. After injection, the needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance may improve the accuracy of needle placement and decrease the incidence of needle-related complications. If prolonged pain relief is not obtained with the technique, blockade of the ganglion impar should be considered (Fig. 105.4). Clinical reports suggest that the use of extracorporeal shock wave therapy may provide symptomatic relief in patients suffering from coccydynia.

Physical modalities, including local heat, gentle range-of-motion exercises, and rectal massage of the affected muscles, including levator ani stretching, should be introduced several days after the patient undergoes injection for coccygeal pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with the injection technique.

COMPLICATIONS AND PITFALLS

Coccydynia should be considered a diagnosis of exclusion in the absence of trauma to the coccyx and its ligaments, because failure to diagnose underlying tumor can have disastrous consequences. The injection technique is safe if careful attention is paid to clinically relevant anatomy. The major complication of injection is infection, given the proximity to the rectum. This complication should be exceedingly rare if strict aseptic technique is followed, as well as universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

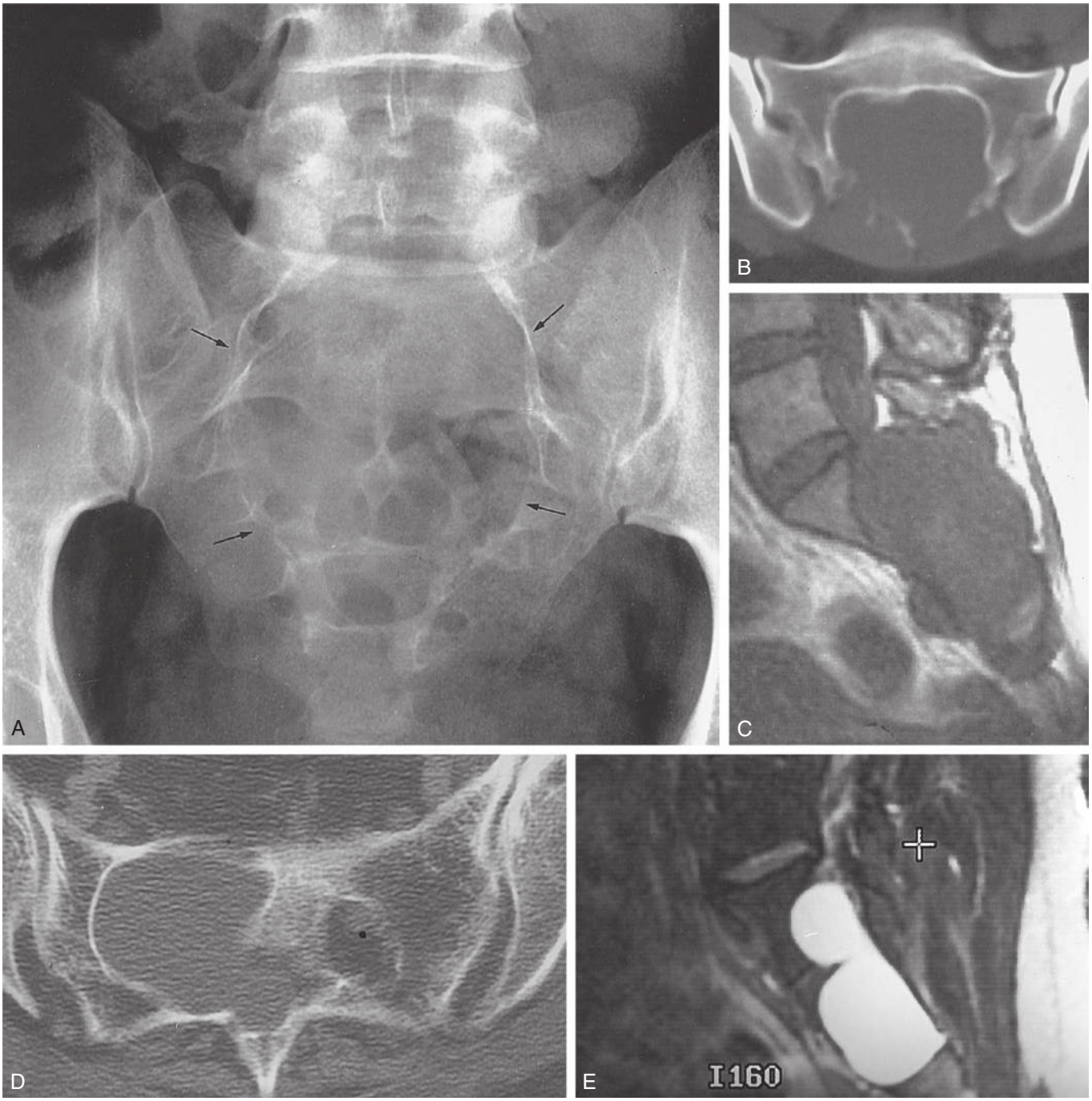


FIG 105.3 Differential diagnosis of coccydynia. **A–C,** Ependymoma. In this 28-year-old woman with low back pain of several years' duration and a normal neurologic examination, routine radiography **(A)** reveals an osteolytic lesion (*arrows*) in the sacrum. Transaxial computed tomography (CT) scan **(B)** confirms its central location and posterior extension. Sagittal, T1-weighted, spin-echo magnetic resonance imaging (MRI) **(C)** shows its large size, posterior extension, and low signal intensity. The tumor was of high signal intensity on T2-weighted spin-echo MRI (not shown). Histologic analysis confirmed a myxopapillary ependymoma that did not communicate with the dural sac. **D** and **E,** Meningocele. Transaxial CT scan **(D)** shows a right-sided sacral lesion distorting the neural foramen. It is sharply delineated, with a sclerotic margin. Sagittal fast spin-echo MRI **(E)** reveals a lesion of high signal intensity with bone erosion. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:4018.)

BOX 105.2 Pathologic Processes That May Cause Coccydynia

- Arthritis
- Crystal deposition disease
- Ligamentous abnormality
- Abnormal coccygeal morphology
- Infection
- Discogenic disease
- Tumor
- Pelvic muscle dysfunction
- Referred pain

CLINICAL PEARLS

The use of a foam donut when sitting, along with the other treatment modalities discussed, may provide symptomatic relief and allow the sacrococcygeal ligament to heal. The injection technique described is extremely effective in the treatment of coccydynia. Coexistent sacroiliitis may contribute to coccygeal pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

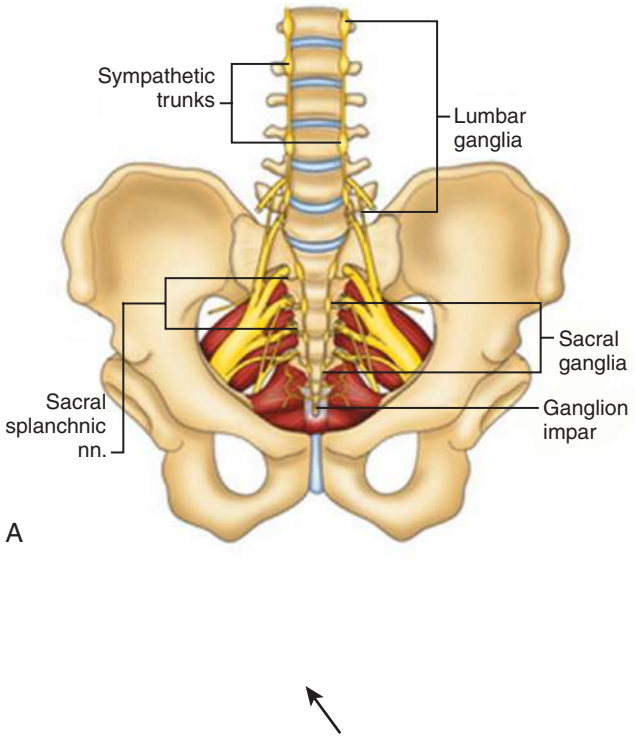


FIG 105.4 **A,** Anatomic location of the ganglion impar. Ganglion impar represents the termination of the paravertebral sympathetic chains, converging at the sacrococcygeal level. **B,** Sagittal, T2-weighted magnetic resonance imaging showing the ganglion impar as a small, isointense signal structure anterior to the sacrococcygeal level (*white arrow*). **C,** Contrast medium outlining the ganglion impar, seen as a filling defect (*black arrow*) in the pool of contrast. (From Datir A, Connell D. CT-guided injection for ganglion impar blockade: a radiological approach to the management of coccydynia. *Clin Radiol*. 2010;65(1):21–25.)

SUGGESTED READINGS

Bharucha AE, Lee TH. Anorectal and pelvic pain. *Mayo Clin Proc*. 2016;91(10):1471–1486.

Hodges SD, Eck JC, Humphreys SC. A treatment and outcomes analysis of patients with coccydynia. *Spine J*. 2004;4(2):138–140.

Marwan Y, Husain W, Alhajii W, et al. Extracorporeal shock wave therapy relieved pain in patients with coccydynia: a report of two cases. *Spine J*. 2014;14(1):e1–e4.

Rao SSC, Bharucha AE, Chiarioni G, et al. Anorectal disorders. *Gastroenterology*. 2016;150(6):1430–1442.e4.

Tejón P, Belmonte MA, Lerma JJ, et al. Coccydynia related to the use of a contraceptive vaginal ring. *Reumatol Clín*. 2017;13(1):42–43.

Trouvin AP, Goeb V, Vandhuick T, et al. Role for magnetic resonance imaging in coccydynia with sacrococcygeal dislocation. *Joint Bone Spine*. 2013;80(2):214–216.

Waldman SD. Coccydynia and other abnormalities of the sacrococcygeal joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:703–709.

Waldman SD. Sacrococcygeal joint injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:482–486.4.

106

Arthritis Pain of the Hip

ICD-10 CODE M16.9

THE CLINICAL SYNDROME

Arthritis of the hip is commonly encountered in clinical practice. The hip joint is susceptible to the development of arthritis from various conditions that have the ability to damage the joint cartilage. Osteoarthritis is the most common form of arthritis that results in hip joint pain; rheumatoid arthritis and posttraumatic arthritis are also common causes of hip pain. Less frequent causes of arthritis-induced hip pain include the collagen vascular diseases, infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the hip joint, although hip pain secondary to collagen vascular disease responds exceedingly well to the treatment modalities described here.

SIGNS AND SYMPTOMS

Most patients presenting with hip pain secondary to arthritis complain of pain localized around the hip and upper leg (Fig. 106.1). Most patients with intrinsic hip disorders have a positive Patrick-FABERE (flexion, abduction, external rotation, extension) test result (Fig. 106.2). Patients may initially present with ill-defined pain in the groin; occasionally, the pain is localized to the buttocks. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping

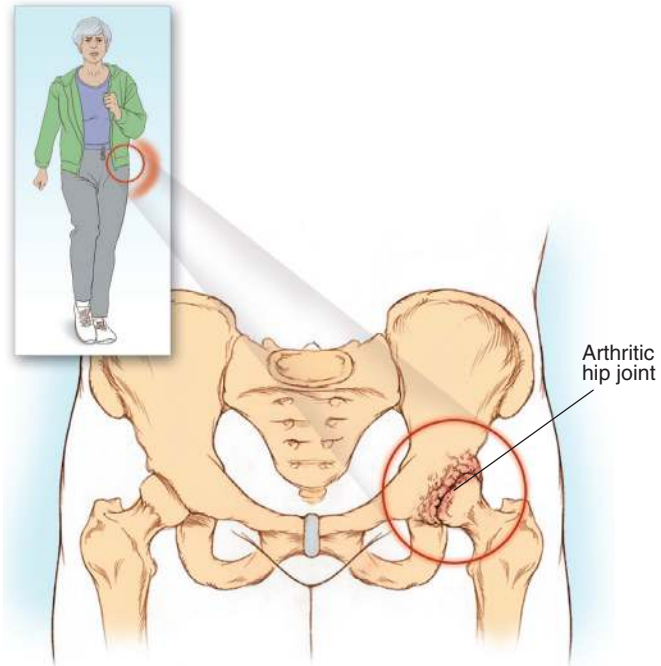


FIG 106.1 The pain of arthritis of the hip is localized to the hip, groin, and upper leg; it is made worse by weight-bearing exercise.

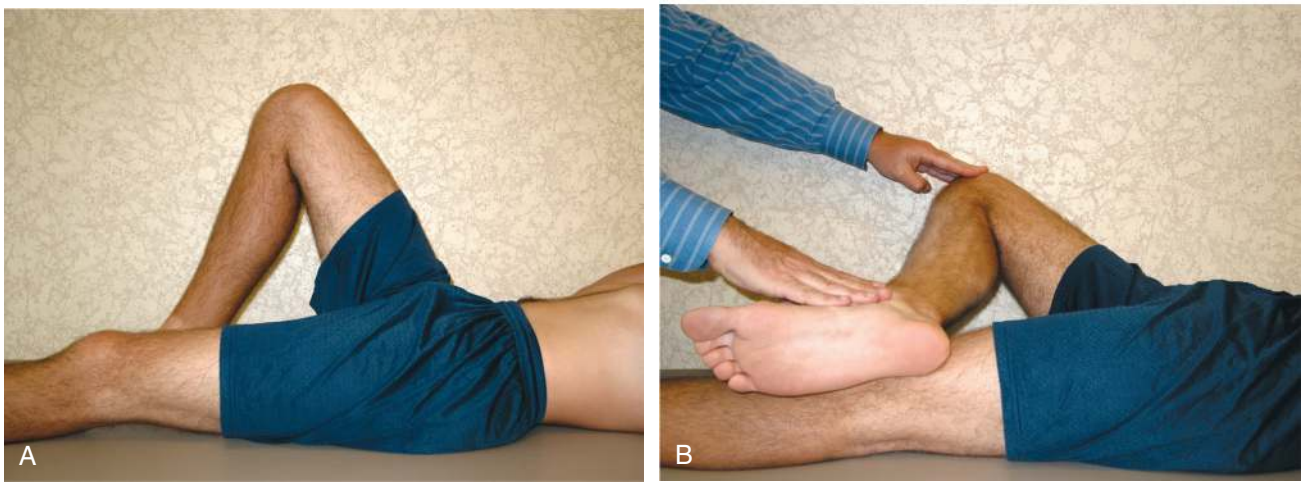


FIG 106.2 A, B, Performing the Patrick-FABERE (flexion, abduction, external rotation, extension) test. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:304.)

sensation with use of the joint, and crepitus may be noted on physical examination.

In addition to pain, patients often experience a gradual decrease in functional ability caused by reduced hip range of motion that makes simple everyday tasks such as walking, climbing stairs, and getting into and out of a car quite difficult. With continued disuse, muscle wasting may occur, and a frozen hip secondary to adhesive capsulitis may develop.

TESTING

Plain radiography is indicated in all patients who present with hip pain. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the hip are indicated if aseptic necrosis or an occult mass or tumor is suspected or if the diagnosis is in question (Figs. 106.3, 106.4, and 106.5).

DIFFERENTIAL DIAGNOSIS

Many diseases can cause hip pain (Table 106.1). Lumbar radiculopathy may mimic the pain and disability associated with arthritis of the hip; however, in such patients, hip examination results should be negative. Entrapment neuropathies, such as meralgia paresthetica and trochanteric bursitis, may confuse the diagnosis; both these conditions can coexist with arthritis of the hip. Primary and metastatic tumors of the hip and spine may also manifest similarly to arthritis of the hip.

TREATMENT

Initial treatment of the pain and functional disability of arthritis of the hip includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

Intraarticular injection of the hip is performed by placing the patient in the supine position. The skin overlying the hip, subacromial region, and joint space is prepared with antiseptic solution. A sterile syringe containing 4-mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 2-inch, 25-gauge needle using strict aseptic technique. The femoral artery is identified; then, at a point approximately 2 inches lateral to the femoral artery, just below the inguinal ligament, the hip joint space is identified. The needle is carefully advanced through the skin and subcutaneous tissues through the joint capsule into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly and slightly more medially. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will simplify this procedure, improve the accuracy of needle placement, and decrease the incidence of needle-related complications. Recent clinical experience suggests that the intraarticular injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of patients suffering from osteoarthritis of the hip.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection for hip pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

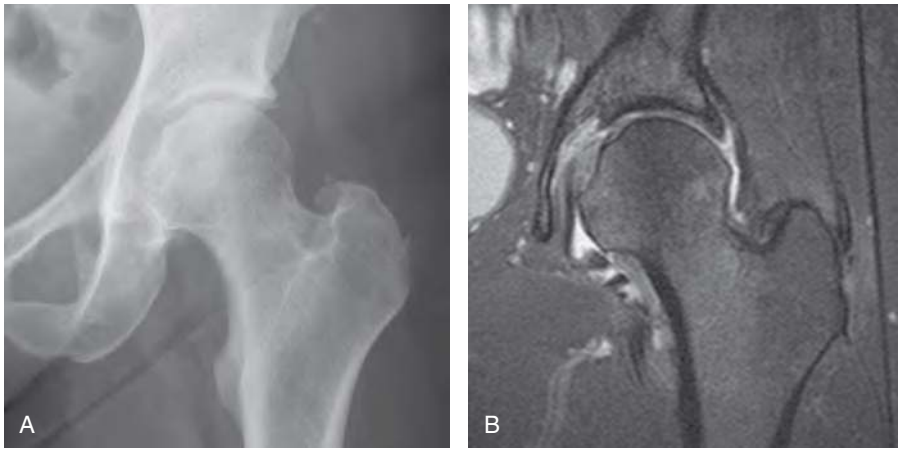


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FIG 106.3 **A,** Anteroposterior radiograph of a 60-year-old patient with hip pain, which shows no significant osteoarthritis (OA) changes. **B,** However, the coronal T2-weighted with fat suppression (FST2W) magnetic resonance image clearly demonstrates a high–signal intensity (SI) hip joint effusion with diffuse areas of cartilage loss across the femoral head, owing to early OA. **C,** Compared with the FST2W magnetic resonance image of a normal hip with intermediate-SI cartilage overlying the low-SI subchondral bone plate (*white arrows*). **D,** The cartilage loss is also seen on the sagittal proton density image (*broken black arrows*), but with some areas of cartilage preservation (*black arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Elsevier; 2011.)

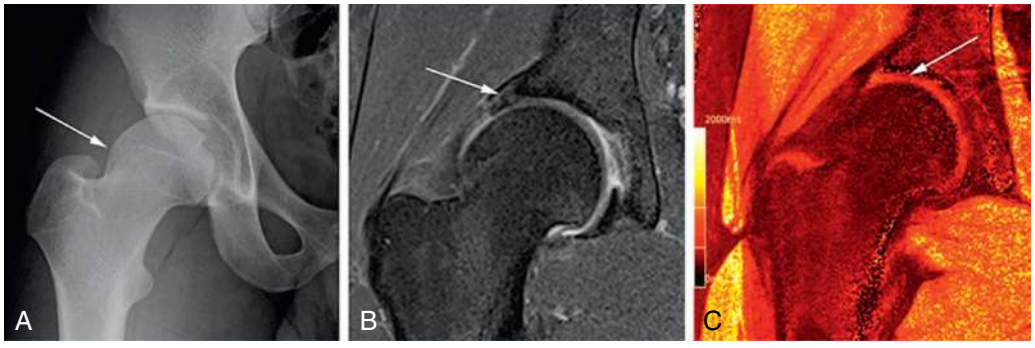

FIG 106.4 Delayed gadolinium-enhanced magnetic resonance image of cartilage (dGEMRIC) performed in an 18-year-old athletic male with clinical features of FAI. **A,** Anteroposterior pelvic radiograph shows cam-type deformity at the superior and lateral femoral head-neck transition (*arrow*). Note the normal joint space width and the absence of marginal osteophytes at the right hip. **B,** Correspondent coronal proton density-weighted fat-suppressed image of the right hip showing normal morphology of the articular cartilage and mild labral degeneration (*arrow*). **C,** Correspondent coronal dGEMRIC color map depicted a focal decreased dGEMRIC index represented by the focal dark line of the acetabular cartilage (*arrow*), indicating potential early cartilage degeneration. (From Vinatier C, Merceron C, Guicheux J. Osteoarthritis: from pathogenic mechanisms and recent clinical developments to novel prospective therapeutic options. *Drug Discovery Today*. 2016;21(12):1932–1937.)

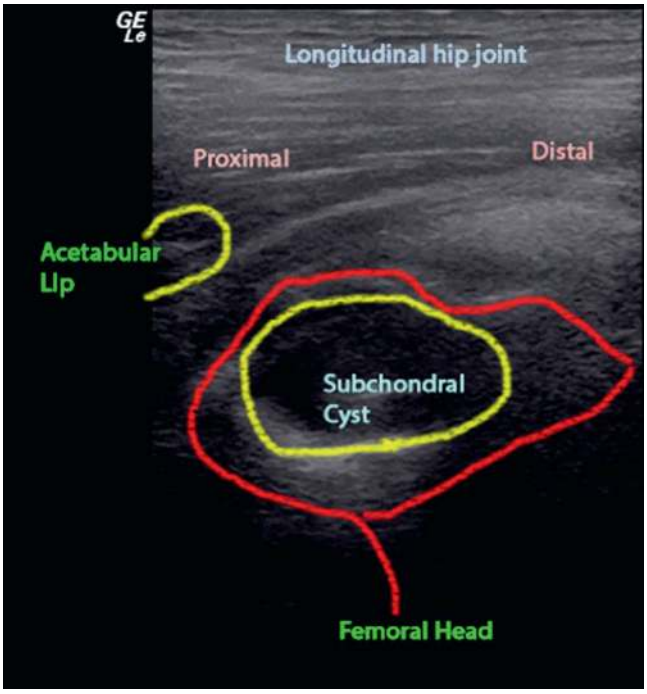

FIG 106.5 Longitudinal ultrasound image demonstrating avascular necrosis of the femoral head.

COMPLICATIONS AND PITFALLS

Failure to identify a primary or metastatic tumor of the hip or spine that is causing the patient's pain can be disastrous. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of intraarticular injection of the hip is infection; however, it should be exceedingly rare if strict aseptic technique is followed, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after intraarticular injection of the hip joint, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to hip pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in the treatment of pain secondary to arthritis of the hip joint.

TABLE 106.1 Causes of Hip Pain and Dysfunction

Localized Bony or Joint Space Pathology	Periarticular Pathology	Systemic Disease	Sympathetically Mediated Pain	Referred From Other Body Areas	Vascular Disease
Fracture	Bursitis	Rheumatoid arthritis	Causalgia	Lumbar plexopathy	Aortoiliac atherosclerosis
Primary bone tumor	Tendinitis	Collagen vascular disease	Reflex sympathetic dystrophy	Lumbar radiculopathy	Internal iliac artery occlusion
Primary synovial tissue tumor	Adhesive capsulitis	Reiter's syndrome	Lumbar spondylosis		
Joint instability	Joint instability	Gout	Fibromyalgia		
Localized arthritis	Muscle strain	Other crystal arthropathies	Myofascial pain syndromes		
Osteophyte formation	Muscle sprain	Charcot's neuropathic arthritis	Inguinal hernia		
Osteonecrosis of femoral head	Periarticular infection not involving joint space			Entrapment neuropathies	
Joint space infection					
Hemarthrosis					
Villonodular synovitis					
Intraarticular foreign body					
Slipped capital femoral epiphysis (Legg's disease)					
Chronic hip dislocation					

From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006.

SUGGESTED READINGS

Caughran AT, Giangarra CE. The arthritic hip. In: Giangarra CE, Manske RC, eds. *Clinical orthopaedic rehabilitation: a team approach*. 4th ed. Philadelphia: Elsevier; 2018: chap 65, 432–435.

Crema MD, Watts GJ, Guermazi A, et al. A narrative overview of the current status of MRI of the hip and its relevance for osteoarthritis research—what we know, what has changed and where are we going? *Osteoarthr Cartil*. 2017;25(1):1–13.

El-Bakoury A, Williams M. Management of hip pain in young adults. *Surgery (Oxford)*. 2020;38(2):74–78.

Foye PM, Stitik TP, Shah VP, et al. Hip osteoarthritis. In: Frontera WR, Silver Julie K, Rizzo Thomas D, eds. *Essentials of physical medicine and rehabilitation*. 4th ed. Philadelphia: Elsevier; 2020: chap 55, 307–314.

Vinatier C, Merceron C, Guicheux J. Osteoarthritis: from pathogenic mechanisms and recent clinical developments to novel prospective therapeutic options. *Drug Discov Today*. 2016;21(12):1932–1937.

Waldman SD. Arthritis and other abnormalities of the hip joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:613–622.

Waldman SD. *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kuwer; 2016:613–621.

Waldman SD. Arthritis pain of the hip. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:299–300.

Waldman SD. Functional anatomy of the hip. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:134–137.

Waldman SD. Hip joint. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:140–141.

Waldman SD. Intra-articular injection of the hip joint. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:385–390.

Waldman SD. Intra-articular injection of the hip. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:514–517.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the pelvis, hip, and lower extremity pain syndromes. In: *Imaging of pain*. Philadelphia: Saunders; 2011:335–338.

Waldman SD, Campbell RSD. Osteoarthritis of the hip joint. In: *Imaging of pain*. Philadelphia: Saunders; 2011:351–352.

107

Snapping Hip Syndrome

ICD-10 CODE M 65.80

THE CLINICAL SYNDROME

Patients with snapping hip syndrome experience a snapping sensation in the lateral hip associated with sudden, sharp pain in the area of the greater trochanter. The snapping sensation and pain are the result of the iliopsoas tendon subluxating over the greater trochanter or iliopectineal eminence (Fig. 107.1). The trochanteric bursa lies between the greater trochanter and the tendon of the gluteus medius muscle and the iliotibial tract. The gluteus medius muscle originates from the outer surface of the ilium, and its fibers pass downward and laterally to attach on the lateral surface of the greater trochanter. The gluteus medius muscle locks the pelvis in place during walking and running; this muscle is innervated by the superior gluteal nerve. The iliopectineal eminence is the point at which the ilium and the pubis bone merge. The psoas and iliacus muscles join at the lateral side of the psoas, and the combined fibers are referred to as the iliopsoas muscle. Like the psoas muscle, the iliacus flexes the thigh on the trunk or, if the thigh is fixed, flexes the trunk on the thigh, such as when moving from a supine to a sitting position.

The symptoms of snapping hip syndrome, which is also known as coxa saltans, occur most commonly when rising from a sitting to a standing position or when walking briskly (Fig. 107.2). Often, trochanteric bursitis coexists with snapping hip syndrome and increases the patient's pain and disability.

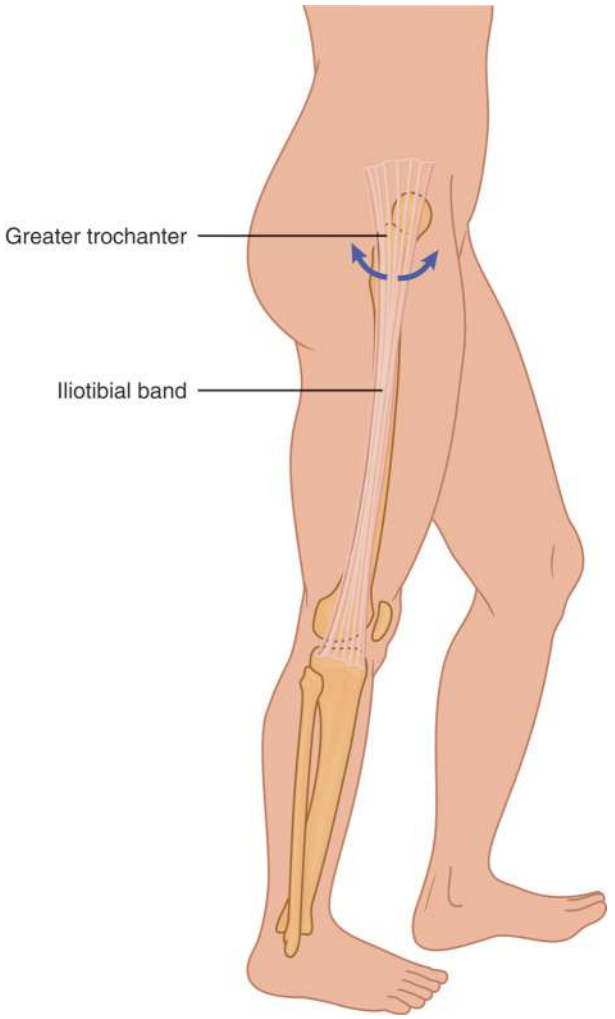

FIG 107.1 The snapping sensation and pain are the result of the iliopsoas tendon subluxating over the greater trochanter or iliopectineal eminence. (From Waldman SD. Snapping hip syndrome. In: *Atlas of pain management injection techniques.* 2nd ed. Philadelphia: Saunders; 2007:368.)

SIGNS AND SYMPTOMS

Physical examination reveals that patients can recreate the snapping and pain by moving from a sitting to a standing position and adducting the hip. This positive snap sign is considered diagnostic for snapping hip syndrome (Fig. 107.3). Point tenderness over the trochanteric bursa, indicative of trochanteric bursitis, is often present.

TESTING

Plain radiographs are indicated in all patients who present with pain thought to be emanating from the hip, to rule out occult bony disorders and tumor. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance and ultrasound imaging of the affected hip are indicated if an occult mass or aseptic necrosis is suspected and to aid in confirmation of the diagnosis (Figs. 107.4 and 107.5). Electrodiagnostic and radiographic testing can identify coexisting diseases such as internal derangement of the hip joint and lumbosacral nerve lesions. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

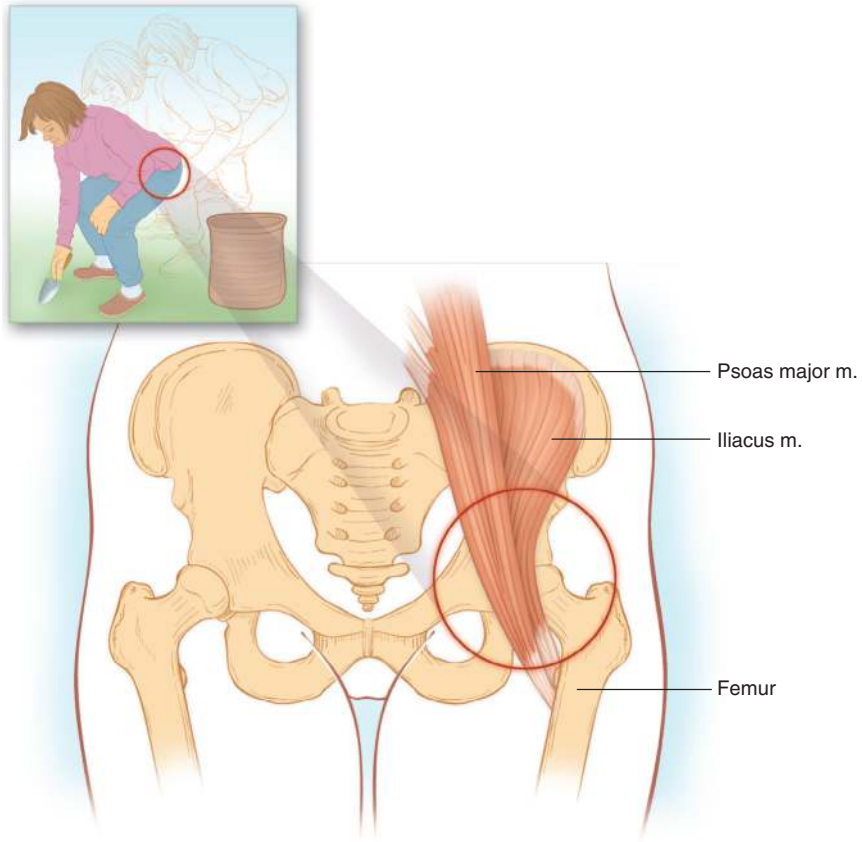

FIG 107.2 The symptoms of snapping hip syndrome commonly occur when rising from a sitting to a standing position or when walking briskly.

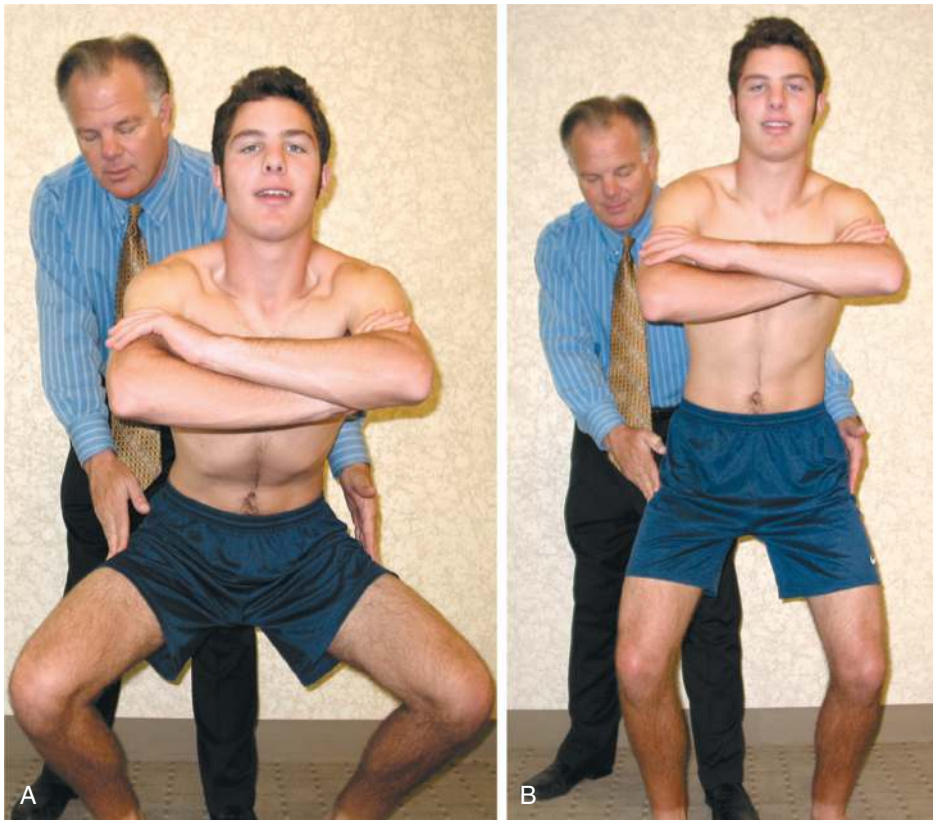

FIG 107.3 A, B, Eliciting the snap sign. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:320.)

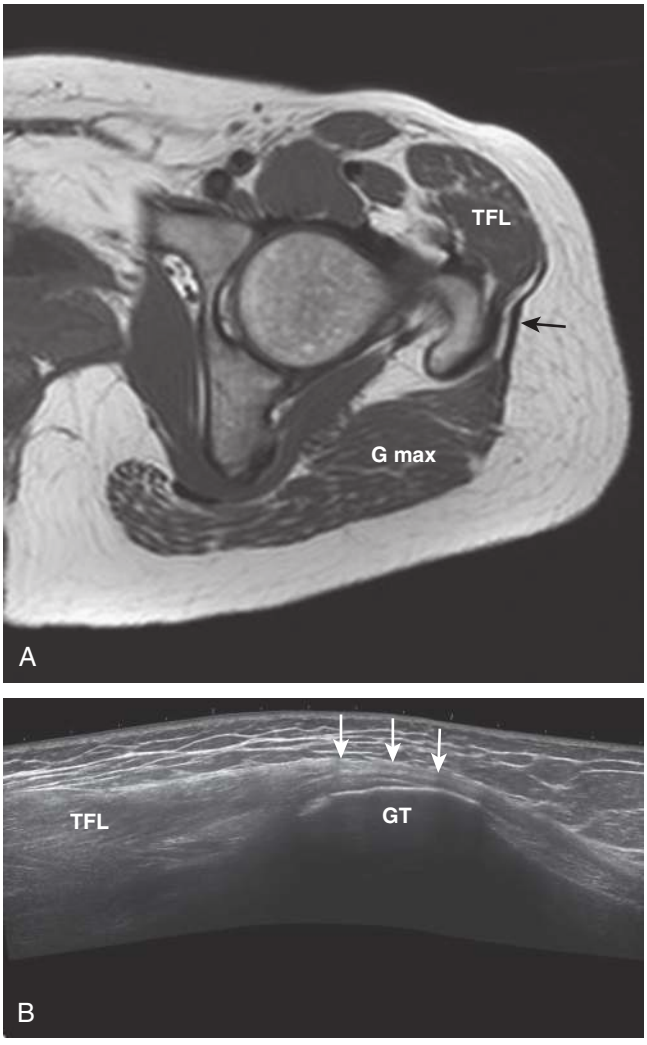

FIG 107.4 A, Normal axial T1W magnetic resonance image showing the relationships of the low signal intensity (SI), thin iliotibial band (ITB) (*black arrow*) which is continuous with the tensor fascia lata (TFL) anteriorly and the gluteus maximus (G max) posteriorly. B, A longitudinal ultrasound image shows the echo-bright ITB (*white arrow*) lying immediately superficial to the greater trochanter and its relationship with the TFL muscle proximally. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011.)

DIFFERENTIAL DIAGNOSIS

The diagnosis of snapping hip syndrome is based on clinical findings rather than specific laboratory, electrodiagnostic, or radiographic testing. For this reason, a targeted history and physical examination, with a systematic search for other causes of hip pain, should be performed. The clinician must rule out other coexisting disease processes that may mimic snapping hip syndrome, including primary inflammatory muscle disease, primary hip disorders, and rectal and pelvic tumors.

TREATMENT

Initial treatment of the pain and functional disability associated with snapping hip syndrome includes a combination

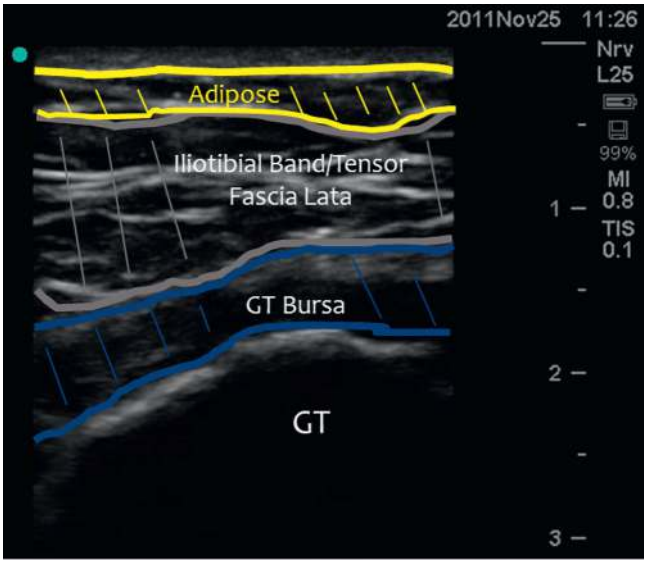

FIG 107.5 Ultrasound image of the hip joint demonstrating the greater trochanter and overlying trochanteric bursa and iliotibial band.

of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To perform the injection, the patient is placed in the lateral decubitus position with the affected side upward. The midpoint of the greater trochanter is identified, and the skin overlying this point is prepared with antiseptic solution. A syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 3½-inch, 25-gauge needle. Before needle placement, the patient should be instructed to say "There!" as soon as a paresthesia is felt in the lower extremity, thus indicating that the needle has impinged on the sciatic nerve. Should a paresthesia occur, the needle is immediately withdrawn and repositioned more laterally. The needle is slowly advanced through the previously identified point at a right angle to the skin, directly toward the center of the greater trochanter, until the needle hits bone; the needle is then withdrawn out of the periosteum. After careful aspiration for blood, and if no paresthesia is present, the contents of the syringe are gently injected. Resistance to injection should be minimal. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy, particularly the sciatic nerve. The proximity to the sciatic nerve makes it imperative that this procedure be performed only by those familiar with the regional anatomy and experienced in the technique.

Although infection is rare, sterile technique must be used, along with universal precautions to minimize any risk to the operator. Most complications of the injection technique are related to needle-induced trauma at the injection site and in the underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Many patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Snapping hip syndrome is a common disorder that often coexists with trochanteric bursitis. Because snapping hip syndrome is often misdiagnosed, a careful evaluation to identify underlying disease processes is mandatory. The injection technique described is extremely effective in the treatment of snapping hip syndrome.

SUGGESTED READINGS

Anderson CN. Iliopsoas: pathology, diagnosis, and treatment. *Clin Sports Med*. 2016;35(3):419–433.

Gopisankar Balaji G, Patil NK, Menon J. Coxa saltans caused by extraarticular synovial chondromatosis overlying an isolated osteochondroma of the greater trochanter: a rare aetiology. *J Clin Orthop Trauma*. 2015;6(2):126–130.

Maldonado DR, Glein RM, Lee MS, et al. Patients with concomitant painful external snapping hip and femoroacetabular impingement syndromes reported complete snapping resolution with release of the gluteus maximus and iliotibial band, and comparable minimum 2-year outcomes to a propensity-matched control group. *Arthroscopy*. 2022;38(6):1890–1899. https://doi.org/10.1016/j.arthro.2021.11.048.

Spencer-Gardner LS, Pourcho AM, Smith J, et al. Atypical coxa saltans due to partial proximal hamstring avulsion: a case presentation highlighting the role for dynamic sonography. *PMR*. 2015;7(10):1102–1105.

Waldman SD. External snapping hip syndrome. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:717–724.

Waldman SD. Arthritis pain of the hip. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:365–366.

Waldman SD. Functional anatomy of the hip. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:134–137.

Waldman SD. Snapping hip syndrome. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:424–428.

Waldman SD. Chapter 7 - Jimmie Kaline: a 27-year-old male with a snapping hip. In: Waldman Steven D, ed. *The hip and pelvis*. Philadelphia: Elsevier; 2022:80–93. https://doi.org/10.1016/B978-0-323-76297-7.00007-1.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the pelvis, hip, and lower extremity pain syndromes. In: *Imaging of pain*. Philadelphia: Saunders; 2011:335–338.

Waldman SD, Campbell RSD. Snapping hip syndrome. In: *Imaging of pain*. Philadelphia: Saunders; 2011:351–352.

108

Iliopectineal Bursitis

ICD-10 CODE M70.70

THE CLINICAL SYNDROME

Bursae are formed from synovial sacs, whose purpose is to allow the easy sliding of muscles and tendons across one another at areas of repetitive movement. Lining these synovial sacs is a synovial membrane invested with a network of blood vessels that secrete synovial fluid. With overuse or misuse, the bursa may become inflamed or, rarely, infected; inflammation of the bursa results in an increase in the production of synovial fluid that causes swelling of the bursal sac. Although significant interpatient variability exists in the number, size, and location of bursae, the iliopectineal bursa generally lies between the psoas and iliacus muscles and the iliopectineal eminence. This bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs.

The iliopectineal bursa, which is also known as the limbo dancer's bursa, is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries often involve direct trauma to the bursa through hip injuries (Fig. 108.1); overuse injuries may also occur, such as the use of exercise equipment for lower extremity strengthening. If inflammation of the iliopectineal bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Patients with iliopectineal bursitis frequently complain of pain in the anterior hip and groin. The pain is localized to the area just below the crease of the groin anteriorly, with referred pain noted in the hip joint and anterior pelvis. Often,

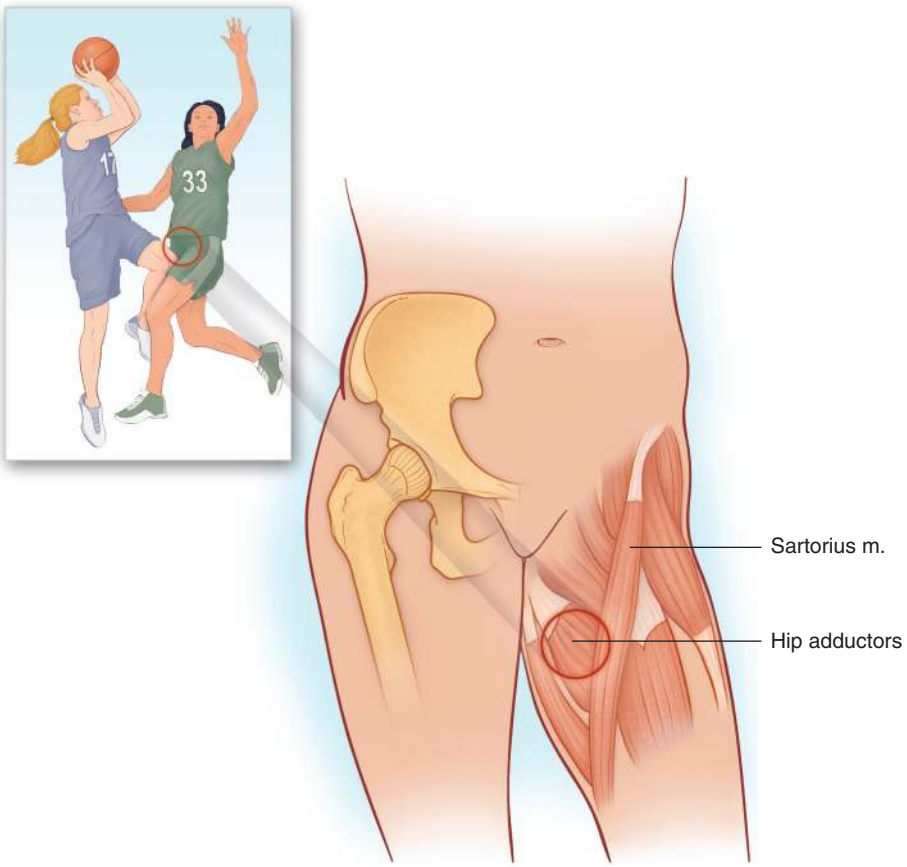

FIG 108.1 The iliopectineal bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries may be caused by direct trauma to the bursa through hip injuries.

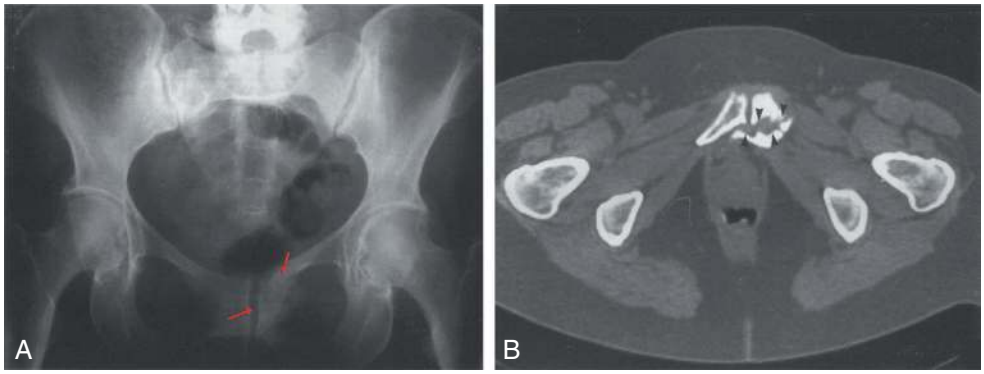

FIG 108.2 Pubic insufficiency fracture simulating a neoplasm in a 61-year-old woman. **A,** Pelvic radiograph shows changes in the symphysis on the left with sclerosis that suggest a chondroid- or osteoid-producing lesion (*red arrows*). **B,** Computed tomography reveals a linear fracture plane and surrounding callus (*red arrowheads*) resulting from a healing fracture; no evidence of a soft tissue mass is present. (From Haaga JR, Lanzieri CF, Gilkeson RC (eds.). *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:1924.)

patients are unable to sleep on the affected hip and may complain of a sharp "catching" sensation with range of motion of the hip. Iliopectineal bursitis often coexists with arthritis of the hip joint.

Physical examination may reveal point tenderness in the upper thigh just below the crease of the groin. Passive flexion, adduction, and abduction, as well as active resisted flexion and adduction of the affected lower extremity, can reproduce the pain. Sudden release of resistance during this maneuver causes a marked increase in pain.

TESTING

Plain radiographs or computed tomography scanning may reveal calcification of the bursa and associated structures that is consistent with chronic inflammation (Fig. 108.2). Magnetic resonance and ultrasound imaging of the hip and pelvis are indicated if tendinitis, partial disruption of the ligaments, stress fracture, internal derangement of the hip, or pelvic mass is suspected as well as to confirm the diagnosis (Fig. 108.3). Ultrasonography may confirm the cystic nature of the structures (Fig. 108.4). Radionuclide bone scanning is indicated if occult fracture, metastatic disease, or primary tumor involving the hip or pelvis is being considered. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Iliopectineal bursitis is a common cause of hip and groin pain. Osteoarthritis, rheumatoid arthritis, posttraumatic arthritis, and, less frequently, aseptic necrosis of the femoral head are also common causes of hip and groin pain that may coexist with iliopectineal bursitis. Less common causes of arthritis-induced pain include the collagen vascular diseases,

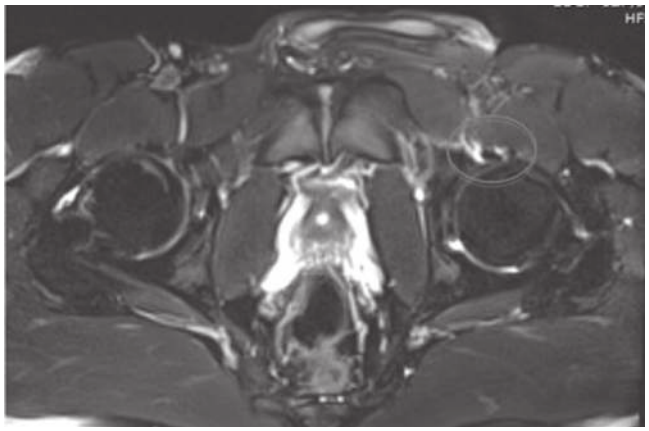

FIG 108.3 Axial proton density fat sat. Right iliopectineal bursitis. No evidence of underlying hip disease. (From Brunot S, Dubeau S, Laumonier H, et al. Acute inguinal pain associated with iliopectineal bursitis in four professional soccer players. *Diagn Interv Imaging*. 2013;94(1):91–94.)

infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the hip joint, although pain secondary to collagen vascular disease responds exceedingly well to the injection technique described here.

TREATMENT

Initial treatment of the pain and functional disability associated with iliopectineal bursitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection of

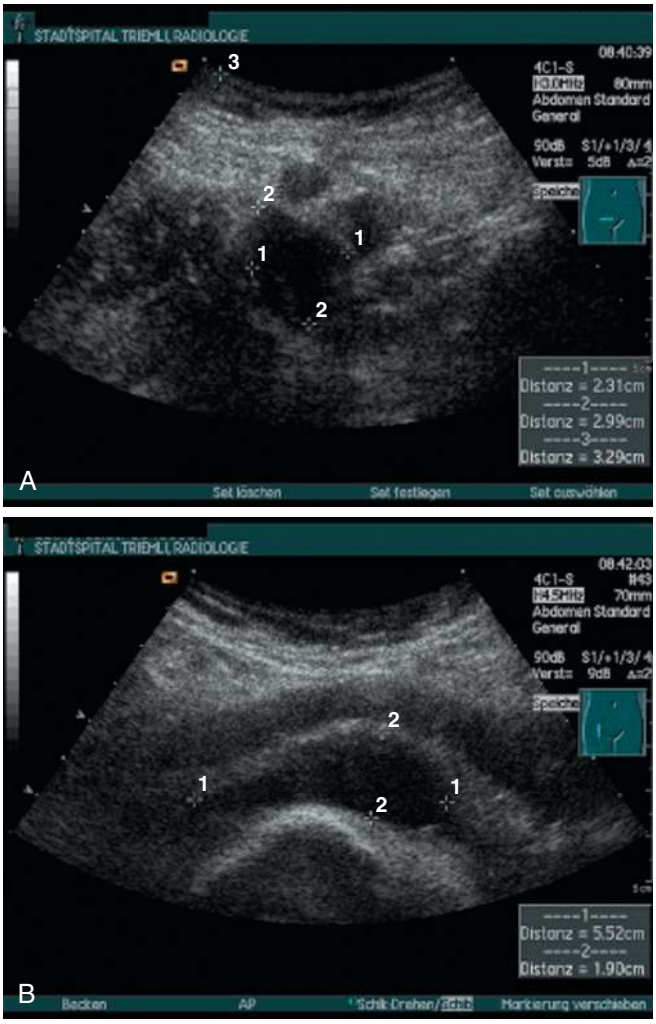

FIG 108.4 (A and B) Longitudinal and transverse sonograms of the right groin. (From Weber M, Prim P, Lüthy R. Inguinal pain with limping: iliopectineal bursitis as first sign of polymyalgia rheumatica. *Joint Bone Spine*. 2008;75(3):332–333.)

local anesthetic and steroid into the iliopectineal bursa is a reasonable next step.

Injection into the iliopectineal bursa is performed with the patient in the supine position. The pulsation of the femoral artery at the midpoint of the inguinal ligament is identified. At a point 2½-inches below and 3½-inches lateral to this pulsation lies the entry point of the needle; this point should be at the lateral edge of the sartorius muscle. The skin overlying this point is prepared with antiseptic solution. A syringe containing 9 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 3½-inch, 25-gauge needle. Before needle placement, the patient should be instructed to say "There!" as soon as a paresthesia is felt in the lower extremity, thus indicating that the needle has impinged on the femoral nerve. Should a paresthesia occur, the needle is immediately withdrawn and is repositioned more laterally. The needle is then carefully advanced through the previously identified point at a 45-degree angle cephalad to allow the needle to pass safely beneath the femoral artery, vein, and nerve. The needle is advanced very slowly to avoid trauma to

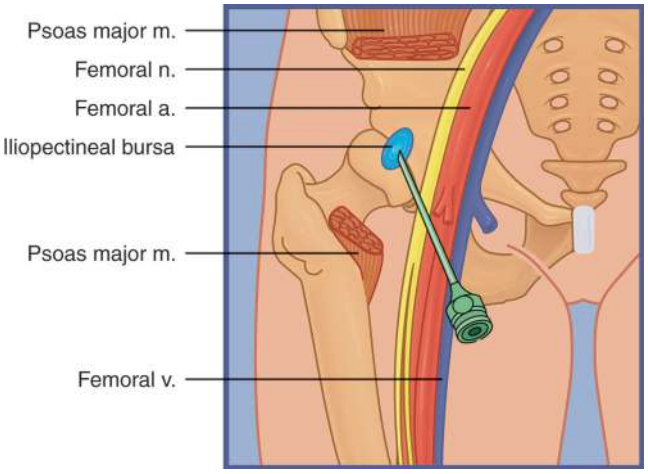

FIG 108.5 Correct needle placement for injection of the iliopectineal bursa. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:359.)

the femoral nerve until the needle hits bone at the point where the ilium and pubic bones merge (Fig. 108.5); the needle is then withdrawn out of the periosteum. After careful aspiration for blood, and if no paresthesia is present, the contents of the syringe are gently injected into the bursa. Resistance to injection should be minimal. Ultrasound needle guidance will increase the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. Specifically, care must be taken to avoid trauma to the femoral nerve. The major complication of injection of the iliopectineal bursa is infection, although it should be exceedingly rare if strict aseptic technique is followed, along with universal precautions to minimize any risk to the operator. Other complications are related to needle-induced trauma at the injection site and in the underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of iliopectineal bursitis. Iliopectineal bursitis frequently coexists with arthritis of the hip, which may require specific treatment to achieve pain relief and return of function.

SUGGESTED READINGS

Brunot S, Dubeau S, Laumonier H, et al. Acute inguinal pain associated with iliopectineal bursitis in four professional soccer players. *Diagn Interv Imaging*. 2013;94(1):91–94.

Mayer SW, Flemig AD, Pearce SS. Hip disorders in the female athlete, Chapter 11. In: Frank RM, ed. *The female athlete*. Philadelphia: Elsevier; 2022:139–160. https://doi.org/10.1016/B978-0-323-75985-4.00015-5.

Tibor LM, Sekiya JK. Differential diagnosis of pain around the hip joint. *Arthroscopy*. 2008;24(12):1407–1421.

Waldman SD. Iliopectineal bursitis. In: *'Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:666–670.

Waldman SD. Arthritis pain of the hip. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:299–300.

Waldman SD. Iliopectineal bursa injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:549–552.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the pelvis, hip, and lower extremity pain syndromes. In: *Imaging of pain*. Philadelphia: Saunders; 2011: 335–338.

Ischial Bursitis

ICD-10 CODE M70.70

THE CLINICAL SYNDROME

Bursae are formed from synovial sacs, whose purpose is to allow the easy sliding of muscles and tendons across one another at areas of repetitive movement. Lining these synovial sacs is a synovial membrane invested with a network of blood vessels that secrete synovial fluid. With overuse or misuse, the bursa may become inflamed or, rarely, infected; inflammation of the bursa results in an increase in the production of synovial fluid that causes swelling of the bursal sac. Although significant interpatient variability exists in the number, size, and location of bursae, the ischial bursa generally lies between the gluteus maximus muscle and the bone of the ischial tuberosity. It may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs.

The ischial bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are often caused by direct trauma to the bursa from falls onto the buttocks and from overuse, such as prolonged riding of horses or bicycles (Fig. 109.1). Running on uneven or soft surfaces such as sand also may cause ischial bursitis. If inflammation of the ischial bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Patients suffering from ischial bursitis frequently complain of pain at the base of the buttock with resisted extension of the lower extremity. The pain is localized to the area over the ischial tuberosity; referred pain is noted in the hamstring muscle, which may develop coexistent tendinitis. Often, patients are unable to

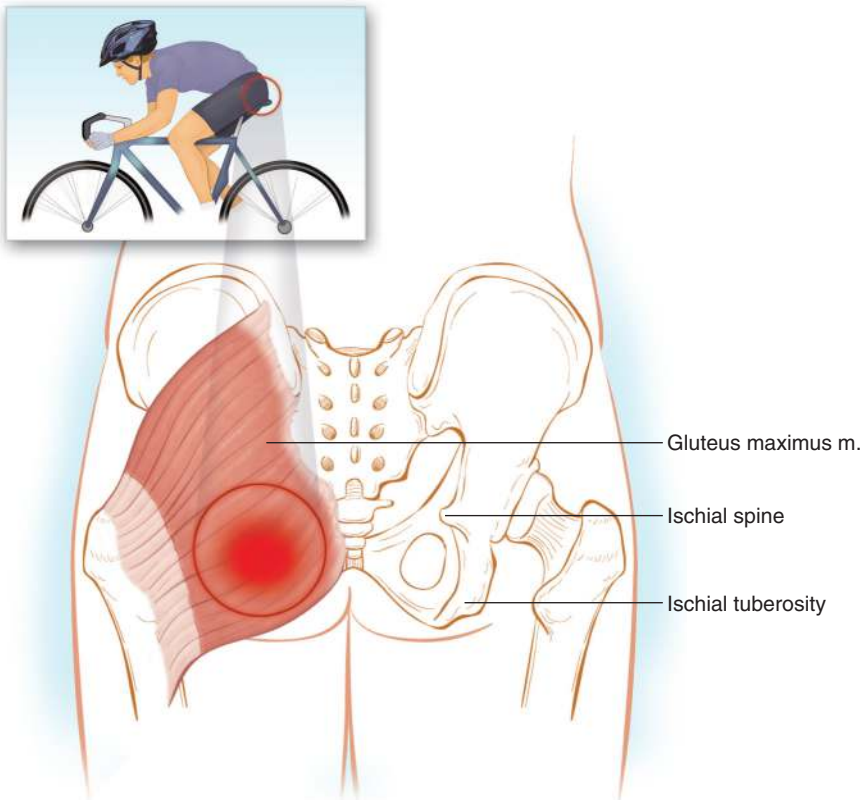

FIG 109.1 The ischial bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are caused by direct trauma to the bursa from falls onto the buttocks and from overuse, such as prolonged riding of horses or bicycles.

FIG 109.2 Physical examination of the patient suffering from ischial bursitis will reveal point tenderness over the ischial tuberosity.

FIG 109.3 The resisted hip extension test for ischial bursitis. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:309.)

sleep on the affected hip and may complain of a sharp "catching" sensation when extending and flexing the hip, especially on first awakening. Physical examination may reveal point tenderness over the ischial tuberosity (Fig. 109.2). Passive straight leg raising and active resisted extension of the affected lower extremity reproduce the pain. Sudden release of resistance during this maneuver causes a marked increase in pain; this increase in pain is considered a positive resisted hip extension test, a finding supporting the diagnosis of ischial bursitis (Fig. 109.3).

TESTING

Plain radiographs may reveal calcification of the bursa and associated structures that is consistent with chronic inflammation. Magnetic resonance and ultrasound imaging of the hip and pelvis are indicated if tendinitis, partial disruption of the ligaments, stress fracture, internal derangement of the hip, or hip or pelvic mass is suspected and to confirm the diagnosis (Figs. 109.4 and 109.5). Radionuclide bone scanning is indicated if occult fracture, metastatic disease, or primary tumor involving the hip or pelvis is being considered.

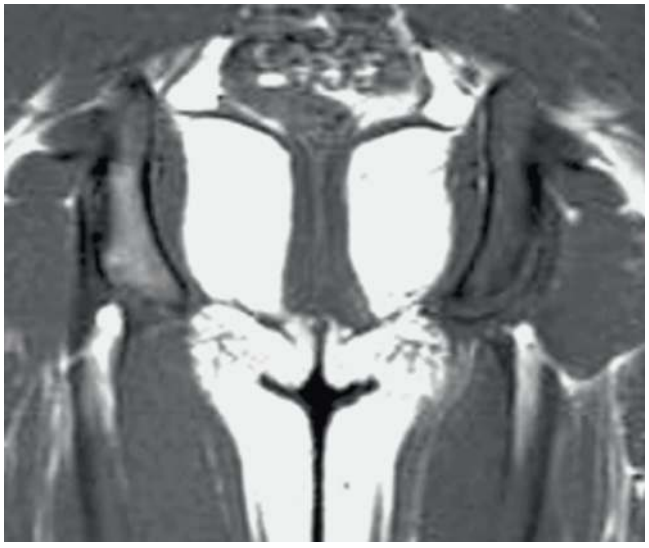

FIG 109.4 Ischial stress injury in a 16-year-old female athlete. Coronal T1-weighted magnetic resonance imaging shows asymmetric decreased marrow signal intensity involving the left ischium. (From Edelman RR, Hesselink JR, Zlatkin MB, Crues JV, eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3385.)

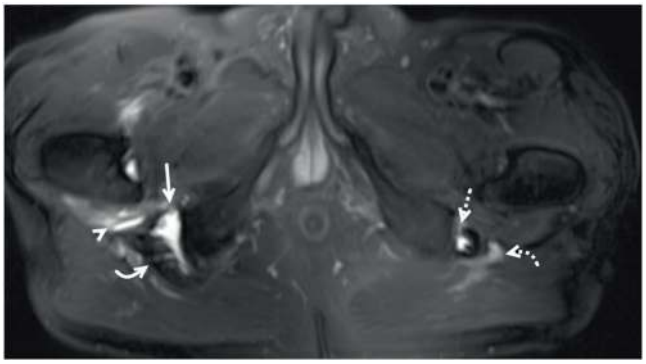

FIG 109.5 A 77-year-old with 5 years of progressive buttock pain. Axial T2-weighted fat-saturated magnetic resonance image demonstrates ischial bursal distention (*arrow*) between the tendinopathic hamstrings origin (*curved arrow*) and ischial tuberosity. Note mild contralateral ischial (*dotted arrow*), ischiogluteal (*curved dotted arrow*), and ipsilateral ischiofemoral (*arrowhead*) bursitis. (From Friedman MV, Stensby JD, Long JR, Currie SA, Hillen TJ. Beyond the greater trochanter: a pictorial review of the pelvic bursae. *Clin Imaging*. 2017;41:37–41.)

Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Ischial bursitis is a common cause of hip and groin pain. Osteoarthritis, rheumatoid arthritis, posttraumatic arthritis, and, less frequently, aseptic necrosis of the femoral head are

also common causes of hip and groin pain that may coexist with ischial bursitis. Hamstring tendinitis or tears of the hamstring muscles may also be present. Less common causes of arthritis-induced pain include the collagen vascular diseases, infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the hip joint, although pain secondary to collagen vascular disease responds exceedingly well to the injection technique described here.

TREATMENT

Initial treatment of the pain and functional disability associated with ischial bursitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, injection of local anesthetic and steroid into the ischial bursa is a reasonable next step.

To inject the ischial bursa, the patient is placed in the lateral position with the affected side upward and the affected leg flexed at the knee. The skin overlying the ischial tuberosity is prepared with antiseptic solution. A syringe containing 4 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle. The ischial tuberosity is identified with a sterilely gloved finger. Before needle placement, the patient should be instructed to say "There!" as soon as a paresthesia is felt in the lower extremity, thus indicating that the needle has impinged on the sciatic nerve. Should a paresthesia occur, the needle is immediately withdrawn and is repositioned more medially. The needle is then carefully advanced through the skin, subcutaneous tissues, muscle, and tendon until it impinges on the bone of the ischial tuberosity (Fig. 109.6). Care must be taken to keep the needle in the midline and not to advance it laterally, or it could contact the sciatic nerve. After careful aspiration, and if no paresthesia is present, the contents of the syringe are gently injected into the bursa. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related injuries (Fig. 109.7).

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

The injection technique is safe if careful attention is paid to the clinically relevant anatomy. Special care must be taken to avoid trauma to the sciatic nerve. The major complication of injection of the ischial bursa is infection, although it should be exceedingly rare if strict aseptic technique is followed, along with universal precautions to minimize any risk to the operator. Other complications are related to needle-induced trauma at the injection site and in the underlying tissues. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after injection. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

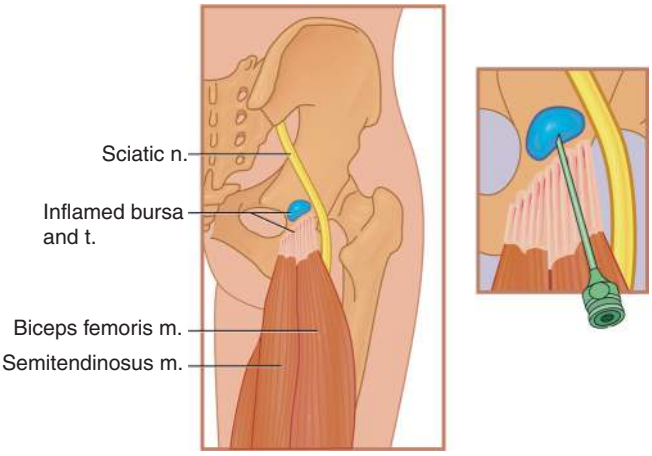

FIG 109.6 Correct needle placement for injection of the ischial bursa. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007.)

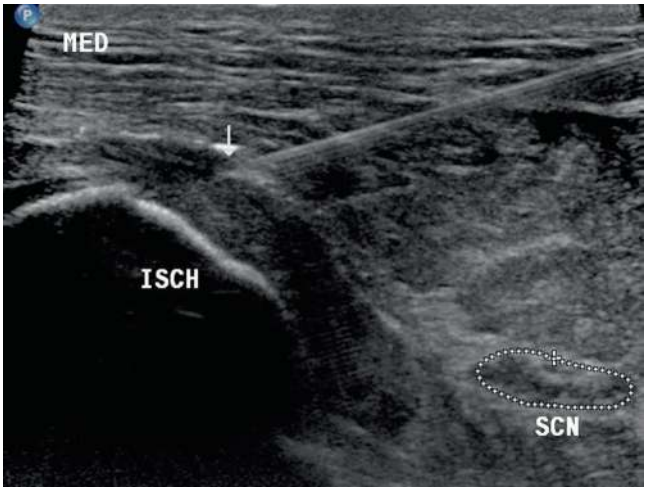

FIG 109.7 Ultrasound image, demonstrating a needle within the ischial bursa. The gluteus maximus muscle is above the needle tip, and the hamstring tendon origin is below the needle tip. The *arrow* indicates the needle tip. The top is superficial; the bottom is deep; the left is medial (MED); and the right is lateral. *ISCH*, Ischium; *SCN*, sciatic nerve. (From Wisniewski SJ, Hurdle M, Erickson JM, Finnoff JT, Smith J. Ultrasound-guided ischial bursa injection: technique and positioning considerations. *PMR*. 2014;6(1):56–60.)

CLINICAL PEARLS

The injection technique described is extremely effective in the treatment of ischial bursitis. Ischial bursitis frequently coexists with arthritis of the hip, which may require specific treatment to achieve pain relief and return of function.

SUGGESTED READINGS

Hodnett PA, Shelly MJ, MacMahon PJ, et al. MR imaging of overuse injuries of the hip. *Magn Reson Imaging C*. 2009;17(4):667–679.

Sando JP, McCambridge TM. Nontraumatic sports injuries to the lower extremity. *Clin Pediatr Emerg Med*. 2013;14(4):327–339.

Waldman SD. Ischial bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:655–659.

Waldman SD. Ischial bursa injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023: 534–538.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the pelvis, hip, and lower extremity pain syndromes. In: *Imaging of pain*. Philadelphia: Saunders; 2011: 335–338.

Wisniewski SJ, Hurdle M, Erickson JM, et al. Ultrasound-guided ischial bursa injection: technique and positioning considerations. *PMR*. 2014;6(1):56–60.

Meralgia Paresthetica

ICD-10 CODE G57.10

THE CLINICAL SYNDROME

Meralgia paresthetica is caused by compression of the lateral femoral cutaneous nerve by the inguinal ligament. This entrapment neuropathy manifests as pain, numbness, and dysesthesias in the distribution of the lateral femoral cutaneous nerve. The symptoms often begin as a burning pain in the lateral thigh, with associated cutaneous sensitivity. Patients suffering from meralgia paresthetica note that sitting, squatting, or wearing low-cut trousers (taille basse) and/or wide belts causes the symptoms to worsen (Fig. 110.1). Although traumatic lesions to the lateral femoral cutaneous nerve have been implicated in meralgia paresthetica, in most patients, no obvious antecedent trauma can be identified.

SIGNS AND SYMPTOMS

Physical findings include tenderness over the lateral femoral cutaneous nerve at the origin of the inguinal ligament at the anterior superior iliac spine. A positive Tinel sign over the lateral femoral cutaneous nerve as it passes beneath the inguinal ligament may be present. Patients may complain of burning dysesthesias in the nerve's distribution (Fig. 110.2). Careful sensory examination of the lateral thigh reveals a sensory deficit in the distribution of the lateral femoral cutaneous nerve; no motor deficit should be present. Sitting or wearing low-cut trousers (taille basse), tight waistbands, or wide belts can compress the nerve and exacerbate the symptoms of meralgia paresthetica.

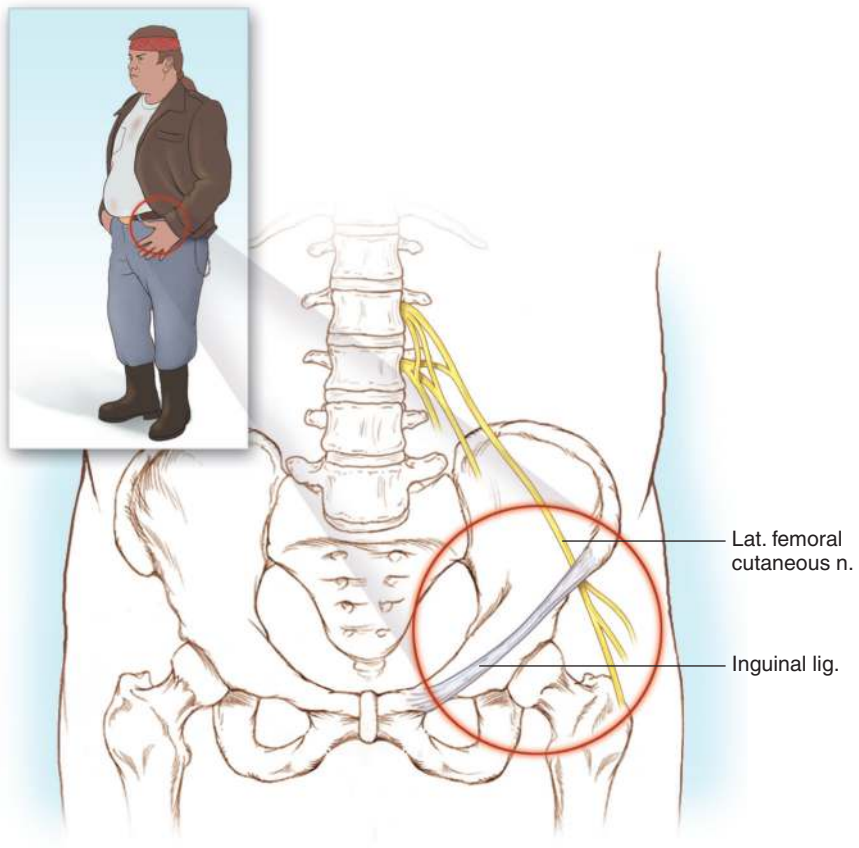

FIG 110.1 Obesity and the wearing of wide belts may compress the lateral femoral cutaneous nerve, thus resulting in meralgia paresthetica.

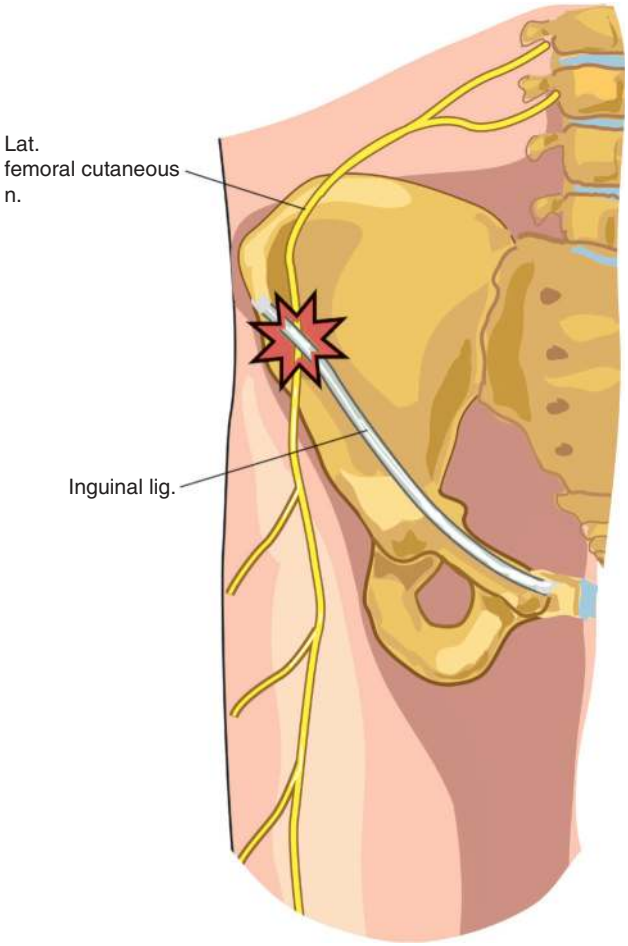

FIG 110.2 Burning pain in the lateral thigh is indicative of meralgia paresthetica. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:279.)

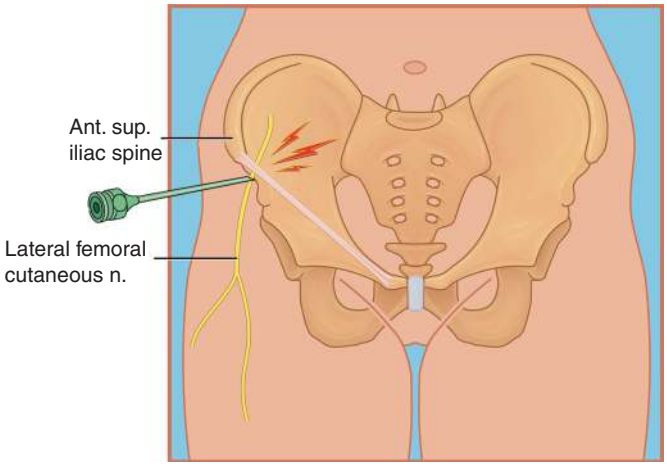

FIG 110.3 Correct needle placement for injection of the lateral femoral cutaneous nerve to treat meralgia paresthetica. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

TESTING

Electromyography (EMG) can distinguish lumbar radiculopathy and diabetic femoral neuropathy from meralgia paresthetica. Plain radiographs of the back, hip, and pelvis are indicated in all patients who present with meralgia paresthetica to rule out occult bony disease. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) and ultrasound imaging of the back are indicated if a herniated disk, spinal stenosis, or space-occupying lesion is suspected. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Meralgia paresthetica is often misdiagnosed as lumbar radiculopathy, trochanteric bursitis, or primary hip disease. Radiographs of the hip and EMG can distinguish meralgia paresthetica from radiculopathy or pain emanating from the hip. In addition, most patients suffering from lumbar radiculopathy have back pain associated with reflex, motor, and sensory changes, whereas patients with meralgia paresthetica have no back pain and no motor or reflex changes; the sensory changes of meralgia paresthetica are limited to the distribution of the lateral femoral cutaneous nerve and should not extend below the knee. Lumbar radiculopathy and lateral femoral cutaneous nerve entrapment may coexist as the double-crush syndrome. Occasionally, diabetic femoral neuropathy produces anterior thigh pain, which may confuse the diagnosis.

TREATMENT

Patients suffering from meralgia paresthetica should be instructed in avoidance techniques to reduce the symptoms and pain associated with this entrapment neuropathy. A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors is a reasonable first step in the treatment of meralgia paresthetica. If patients do not experience rapid improvement, injection is the next step.

To treat the pain of meralgia paresthetica, the patient is placed in the supine position with a pillow under the knees if lying with the legs extended increases the pain because of traction on the nerve. The anterior superior iliac spine is identified by palpation. A point 1-inch medial to the anterior superior iliac spine and just inferior to the inguinal ligament is identified and prepared with antiseptic solution. A 1½-inch, 25-gauge needle is slowly advanced perpendicular to the skin until the needle is felt to pop through the fascia. A paresthesia is often elicited. After careful aspiration, a solution of 5–7 mL of 1% preservative-free lidocaine and 40-mg methylprednisolone is injected in a fanlike pattern as the needle

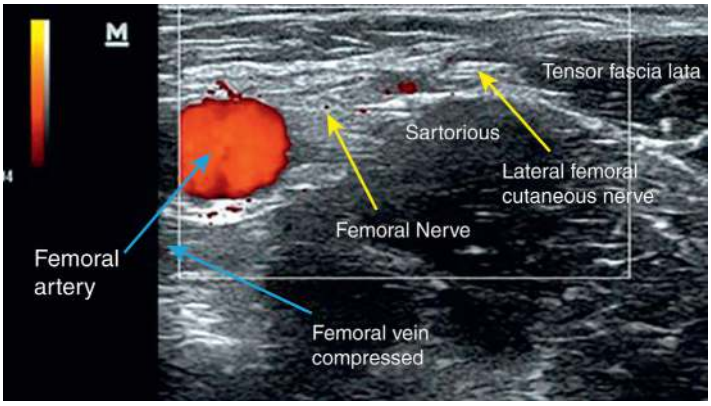

FIG 110.4 Oblique color Doppler image demonstrating the femoral nerve artery and vein that lie medial to the lateral femoral cutaneous nerve.

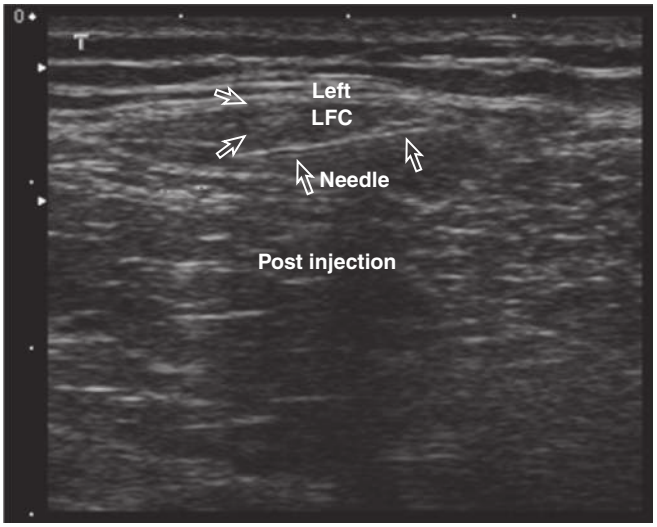

FIG 110.5 Ultrasound image of the lateral femoral cutaneous (LFC) nerve following the injection of 2 mL of local anesthetic. Perineural spread of the local anesthetic can be visualized. The needle is visualized as a linear hyperechoic structure deep into the nerve. (From Hurdle MF, Weingarten TN, Crisostomo RA, et al. Ultrasound-guided blockade of the lateral femoral cutaneous nerve: technical description and review of 10 cases. *Arch Phys Med Rehabil*. 2007;88(10):1362–1364.)

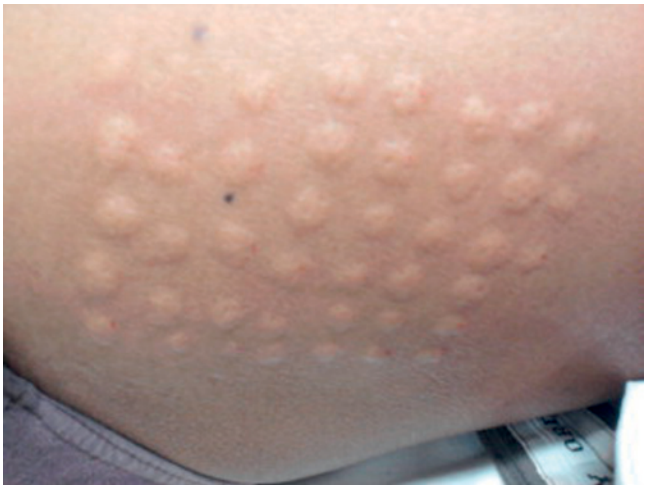

FIG 110.6 Botulinum toxin injection in a grid distribution over the thigh. (From Dhull P, Tewari AK, Upreti V, Prakash MS, Hari Kumar KVS. Botulinum toxin for meralgia paresthetica in type 2 diabetes. *Diabetes Metab Syndr*. 2013;7(1)1–2.)

pierces the fascia of the external oblique muscle. Care must be taken not to place the needle deep enough to enter the peritoneal cavity and perforate the abdominal viscera (Fig. 110.3). After injection of the solution, pressure is applied to the injection site to decrease the incidence of ecchymosis and hematoma formation, which can be quite dramatic, especially in anticoagulated patients. If anatomic landmarks are difficult to identify, the use of fluoroscopic or ultrasound guidance should be considered (Figs. 110.4 and 110.5). Clinical reports suggest that the local infiltration of type A botulinum toxin in the area of sensory deficit may provide symptomatic relief in patients with type 2 diabetes with meralgia paresthetica (Fig. 110.6).

COMPLICATIONS AND PITFALLS

Care must be taken to rule out other conditions that may mimic the pain of meralgia paresthetica. The main complications of the injection technique are ecchymosis and hematoma. If the needle is placed too deep and it enters the peritoneal cavity, perforation of the colon may result in the formation of an intraabdominal abscess and fistula. Early detection of infection is crucial to avoid potentially life-threatening sequelae. If the needle is placed too medially, blockade of the femoral nerve may occur, thus making ambulation difficult.

CLINICAL PEARLS

Meralgia paresthetica is a common complaint that is often misdiagnosed as lumbar radiculopathy. The injection technique described can produce dramatic pain relief. If a patient presents with pain suggestive of meralgia paresthetica but does not respond to lateral femoral cutaneous nerve block, however, a lesion more proximal in the lumbar plexus or an L2–L3 radiculopathy should be considered. Such patients often respond to epidural block with steroid. EMG and MRI of the lumbar plexus are indicated in this patient population to rule out other causes of their pain, including malignant disease invading the lumbar plexus or epidural or vertebral metastatic disease at L2–L3.

SUGGESTED READINGS

Dhull P, Tewari AK, Upreti V, et al. Botulinum toxin for meralgia paresthetica in type 2 diabetes. *Diabetes Metab Syndr*. 2013;7(1):1–2.

Moucharafieh R, Wehbe J, Maalouf G. Meralgia paresthetica: a result of tight new trendy low cut trousers ("taille basse"). *Int J Surg*. 2008;6(2):164–168.

Ramírez Huaranga MA, Ariza Hernández A, Ramos Rodríguez CC, et al. What meralgia paresthetica can hide: renal tumor as an infrequent cause. *Reumatol Clín*. 2013;9(5):319–321.

Stewert JD. Meralgia paresthetica: topography of the sensory deficit. *J Neurol Sci*. 2013;333(suppl 1):e450–e451.

Suh DH, Kim DH, Park JW, et al. Sonographic and electrophysiologic findings in patients with meralgia paresthetica. *Clin Neurophysiol*. 2013;124(7):1460–1464.

Tagliafico AS, Torri L, Signori A. Treatment of meralgia paresthetica (lateral femoral cutaneous neuropathy): a meta-analysis of ultrasound-guided injection versus surgery. *Eur J Radiol*. 2021;139:109736. https://doi.org/10.1016/j.ejrad.2021.109736.

Trummer M, Flaschka G, Under F, et al. Lumbar disc herniation mimicking meralgia paresthetica: case report. *Surg Neurol*. 2000;54(1):80–81.

Waldman SD. Abnormalities of the lateral femoral cutaneous nerve. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:629–633.

Waldman SD. *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:268–269.

Waldman SD. Lateral femoral cutaneous nerve block. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:596–600.

Phantom Limb Pain

ICD-10 CODE M54.6

THE CLINICAL SYNDROME

Almost all patients who undergo amputation experience the often painful and distressing sensation that the absent body part is still present (Fig. 111.1). The cause of this phenomenon is not fully understood, but it is thought to be mediated in large part at the spinal cord level, although recent research points to structural and functional asymmetry of the motor cortex due to deafferentation (Fig. 111.2). Congenitally absent limbs do not seem to be subject to the same phenomenon. Patients may be able to describe the limb in vivid detail, although it is often distorted or in an abnormal position. In many patients, the sensation of the phantom limb fades with time, but in some patients, phantom pain remains a distressing part of daily life. Phantom limb pain is often described as a constant, unpleasant, dysesthetic pain that may be exacerbated by movement or stimulation of the affected cutaneous regions; a sharp, shooting neuritic pain may be superimposed on the constant dysesthetic symptoms, and some patients also note a burning component reminiscent of reflex sympathetic dystrophy. Some investigators reported that severe limb pain before amputation increases the incidence of phantom limb pain, but other investigators failed to find this correlation.

SIGNS AND SYMPTOMS

Phantom limb pain can take multiple forms, but it usually consists of dysesthetic pain. Additionally, patients may experience abnormal kinesthetic sensations (i.e., that the limb is in an abnormal position) or abnormal kinetic sensations

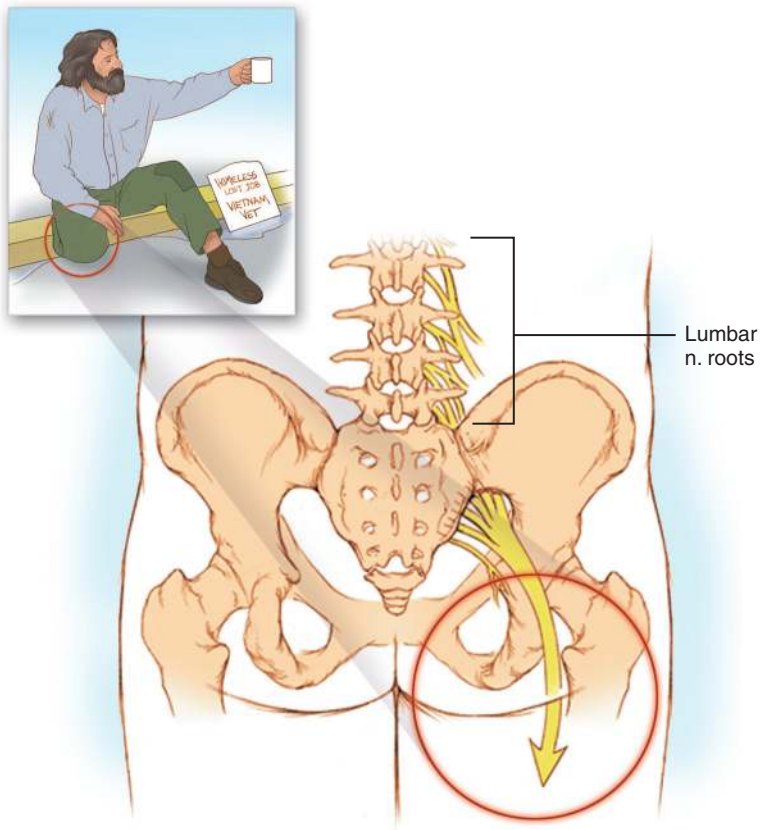

FIG 111.1 Phantom limb pain occurs with varying degrees of intensity in almost all patients who undergo amputation.

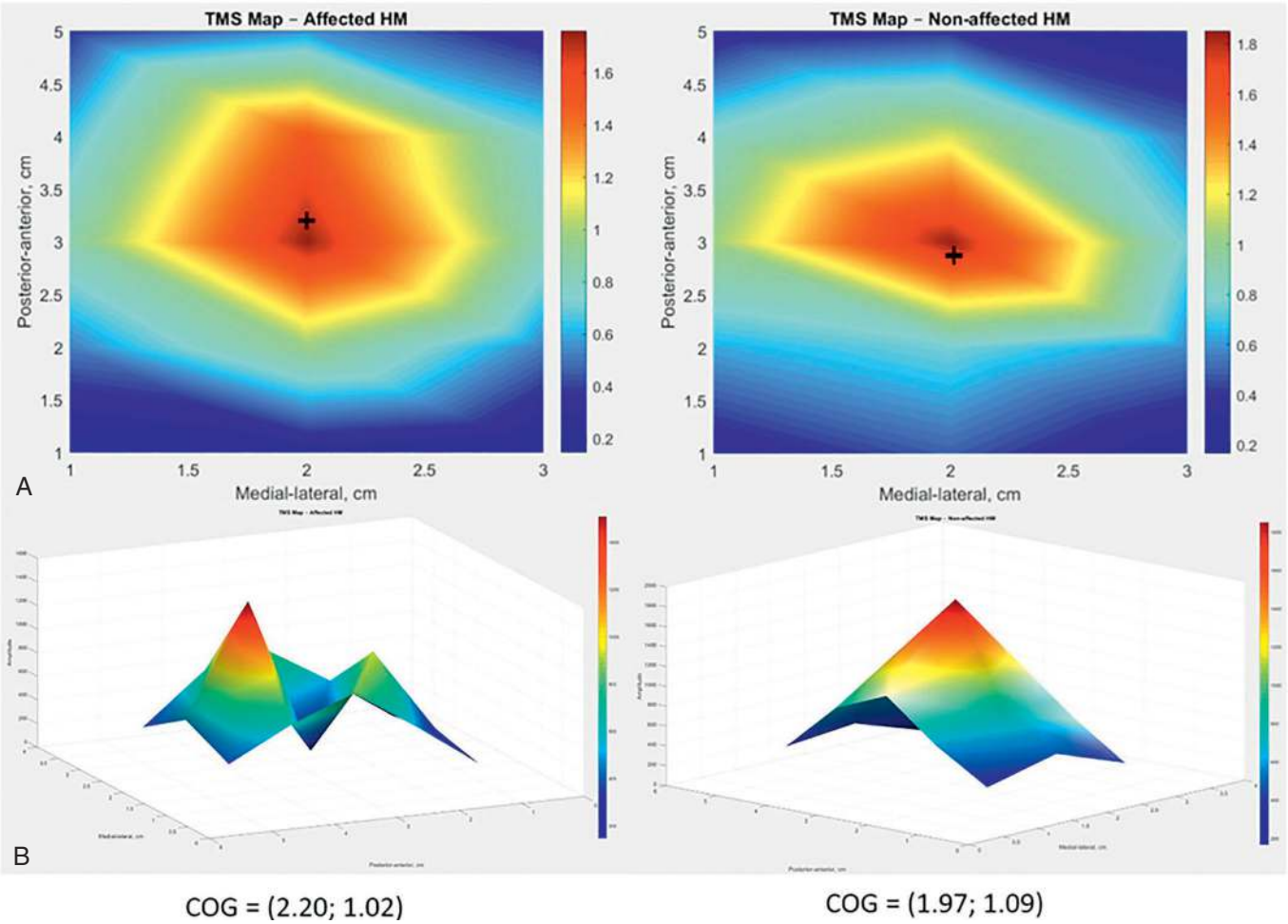

FIG 111.2 Center of gravity (CoG) shift and disorganization of the excitable area. Transcranial magnetic stimulation (TMS) motor cortical mapping asymmetry on the hand area between affected and nonaffected hemispheres. **A,** Anterior shift of the map CoG in the affected hemisphere. **B,** Spread out activation in the affected hemisphere, also disorganization in the peak's activation (multiple peaks activations in the map) compared with the nonaffected hemisphere. *Note:* Colors represent motor evoked potential amplitudes (red = high amplitude). (From Pacheco-Barrios K, Pinto CB, Saleh Velez FG., et al. Structural and functional motor cortex asymmetry in unilateral lower limb amputation with phantom limb pain. *Clin Neurophysiol.* 2020;131(10):2375–2382. https://doi.org/10.1016/j.clinph.2020.06.024.)

(i.e., that the limb is moving). Investigators have reported that many patients with phantom limb pain experience a telescoping phenomenon; for example, a patient may report that the phantom hand feels like it is attached directly to the proximal arm (Figs. 111.3 and 111.4). Phantom limb pain may fade over time, and younger patients are more likely to experience this diminution in symptoms. Because of the unusual nature of phantom limb pain, a behavioral component is invariably present.

TESTING

In most cases, the diagnosis of phantom limb pain is easily made on clinical grounds. Testing is generally used to identify other treatable coexisting diseases, such as radiculopathy. Such testing includes the following: basic laboratory tests; examination of the stump for neuroma, tumor, or occult infection; and plain radiographs and radionuclide bone scanning if fracture or osteomyelitis are suspected.

DIFFERENTIAL DIAGNOSIS

A careful initial evaluation, including a thorough history and physical examination, is indicated in all patients suffering from phantom limb pain if infection or fracture is a possibility. If the amputation was necessitated by malignant disease, occult tumor must be excluded. Other causes of pain in the distribution of the innervation of the affected limb, including radiculopathy and peripheral neuropathy, should be considered.

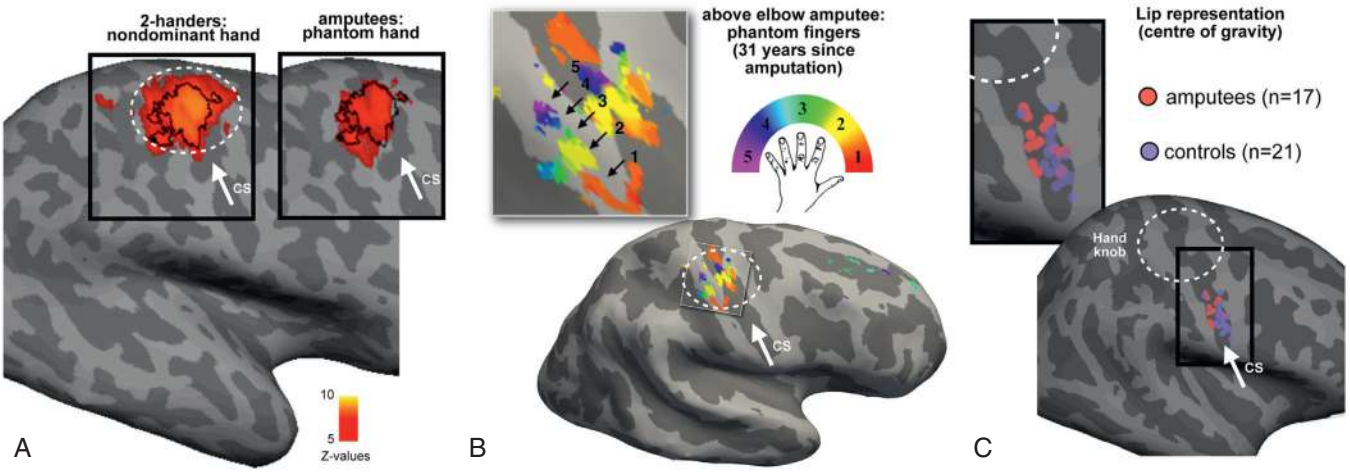

FIG 111.3 Persistent representation of the missing hand. **A,** Activity group maps in controls (*left*) and amputees (*right*) during movements of the nondominant (controls) or phantom hand (amputees). White circle indicates the position of the anatomical hand knob. **B,** A finger-selectivity map (using a traveling wave paradigm) for individual phantom finger movements reveals a complete hand somatotopy in primary somatosensory cortex of an amputee, with specific and adjacent clusters showing selectivity to specific phantom fingers. **C,** Centre of gravity of lip activity clusters in individual participants (amputees, orange; controls, purple) reveals a medial shift in amputees' lip representation, localized to the face area. On average, lips in the deprived hemisphere were shifted medially by 8 mm, compared to the intact hemisphere (note that the hand area is located 63-mm medially to the lips in controls). (**A,** T.R. Makin, J. Scholz, N. Filippini, D. Henderson Slater, I. Tracey, H. Johansen-Berg Phantom pain is associated with preserved structure and function in the former hand area Nat. Commun., 4 (2013), p. 1570, 10.1038/ncomms2571, **B,** S. Kikkert, J. Kolasinski, S. Jbabdi, I. Tracey, C.F. Beckmann, H. Johansen-Berg, T.R. Makin, K.E. Stephan Revealing the neural fingerprints of a missing hand eLife, 5 (2016), Article e15292, 10.7554/eLife.15292, **C,** T.R. Makin, J. Scholz, D. Henderson Slater, H. Johansen-Berg, I. Tracey Reassessing cortical reorganization in the primary sensorimotor cortex following arm amputation Brain, 138 (2015), pp. 2140-2146, 10.1093/brain/awv161.)

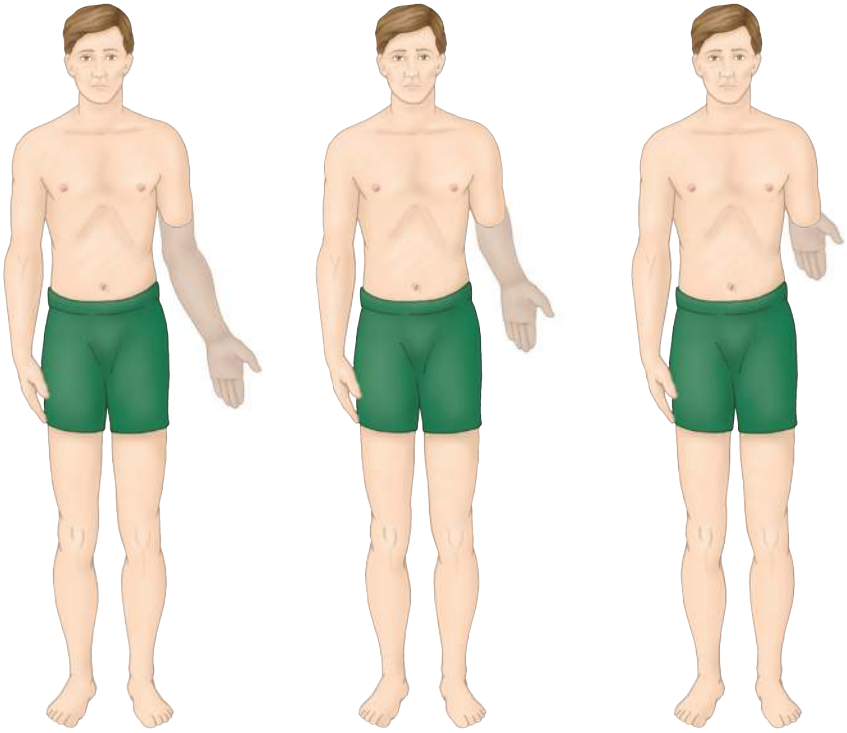

FIG 111.4 The telescoping phenomenon of phantom limb syndrome.

TREATMENT

The first step is to reassure patients that phantom limb pain is normal after the loss of a limb and that these sensations are real, not imagined; this knowledge alone can reduce patients' anxiety and suffering. Many pain specialists agree that preemptive analgesia early in the natural course of a disease that may lead to amputation, such as peripheral vascular insufficiency, can reduce the likelihood that patients will develop phantom limb pain. The following treatments may be useful to relieve phantom limb pain.

Analgesics

The anticonvulsant gabapentin is a first-line treatment in the palliation of phantom limb pain. It should be administered early in the course of the pain syndrome and can be used concurrently with neural blockade, opioid analgesics, and other adjuvant analgesics, including antidepressants, if care is taken to avoid central nervous system side effects. Gabapentin is started at a bedtime dose of 300 mg and is titrated upward in 300-mg increments to a maximum of 3600 mg/day given in divided doses, as side effects allow. Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100-mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine should be considered in patients with severe neuritic pain who do not respond to nerve block and gabapentin. If this drug is used, rigid monitoring of hematologic parameters is indicated, especially in patients receiving chemotherapy or radiation therapy. Phenytoin may also be beneficial in the treatment of neuritic pain, but it should not be used in patients with lymphoma; the drug may induce a pseudolymphoma-like state that is difficult to distinguish from the actual lymphoma.

Antidepressants

Antidepressants may be useful adjuncts in the initial treatment of phantom limb pain. On a short-term basis, these drugs can alleviate the significant sleep disturbance that is common in this setting. In addition, antidepressants may be valuable in ameliorating the neuritic component of the pain, which is treated less effectively with opioid analgesics. After several weeks of treatment, antidepressants may exert a mood-elevating effect, which may be desirable in some patients. Care must be taken to observe closely for central nervous system side effects in this patient population, and these drugs may cause urinary retention and constipation.

Nerve Block

Neural blockade with local anesthetic and steroid by either epidural nerve block or blockade of the sympathetic nerves subserving the painful area is a reasonable next step if the aforementioned pharmacologic modalities fail to control phantom limb pain. The exact mechanism by which neural blockade relieves phantom limb pain is unknown, but it may be related to the modulation of pain transmission at the spinal cord level. In general, neurodestructive procedures have a very low success rate and should be used only after all other treatments have failed, if at all.

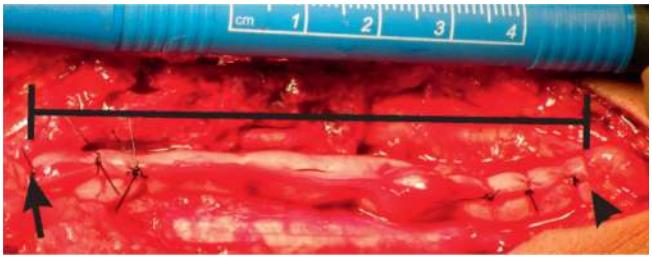

FIG 111.5 Photograph of the treatment of the proximal nerve stump of a radial nerve causing severe chronic neuropathic pain secured within the end of a collagen tube filled with PRP (black line delineates the platelet rich plasma (PRP)-filled collagen tube). The proximal end of the collagen tube is marked by the (*arrowhead*) and the closed distal end with a suture (*arrow*). The excruciating chronic neuropathic pain was eliminated within 2 months. (From Kuffler DP. Can phantom limb pain be reduced/eliminated solely by techniques applied to peripheral nerves? *J Neurorestoratol*. 2019;7(1):26–36.)

Opioid Analgesics

Opioid analgesics have a limited role in the management of phantom limb pain, and they frequently do more harm than good. Careful administration of potent, long-acting opioid analgesics (e.g., oral morphine elixir and methadone) on a time-contingent rather than as-needed basis may be a beneficial adjunct to sympathetic neural blockade. Because many patients suffering from phantom limb pain are older or have severe multisystem disease, close monitoring for the potential side effects of opioid analgesics (e.g., confusion or dizziness, which may cause a patient to fall) is warranted. Daily dietary fiber supplementation and milk of magnesia should be started along with opioid analgesics to prevent constipation.

Adjunctive Treatments

The application of ice packs to the stump may provide relief in some patients with phantom limb pain. The application of heat increases pain in most patients, presumably because of increased conduction of small fibers, but it may be worth trying if the application of cold is ineffective. Transcutaneous electrical nerve stimulation and vibration may also be effective in a limited number of patients. The favorable risk-to-benefit ratio of these modalities makes them reasonable alternatives for patients who cannot or will not undergo sympathetic neural blockade or who cannot tolerate pharmacologic treatment. A trial of spinal cord stimulation is also a reasonable option. The topical application of capsaicin may be beneficial in some patients suffering from phantom limb pain; however, the burning associated with application of this drug often limits its usefulness. Early research in the implanting of painful nerve stumps in collagen tubes containing platelet-rich plasma show promise (Fig. 111.5).

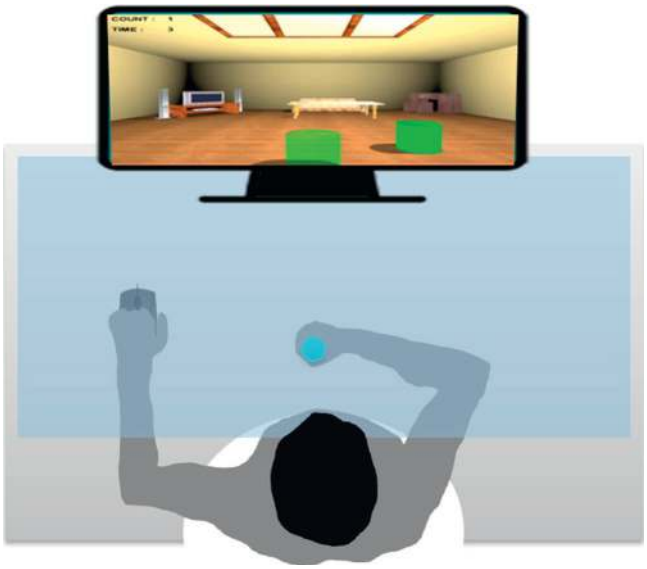

FIG 111.6 Virtual reality-based proprioception training system. The affected hand performed a reaching movement toward the target position (*virtual cylinder*). When the subject believes that the affected hand position corresponds to the target position, the unaffected hand would press the mouse button. (From Cho S, Ku J, Cho et al. Development of virtual reality proprioceptive rehabilitation system for stroke patients. *Comput Methods Programs Biomed.* 2014;113(1):258–265.)

Recent clinical experience with virtual reality multisensory feedback training shows promise in the treatment of the distorted body image issues and pain associated with phantom pain syndrome (Fig. 111.6).

COMPLICATIONS AND PITFALLS

Although no complications are associated with phantom limb pain itself, the consequences of unremitting pain can be devastating. Failure to treat phantom limb pain and the associated symptoms of sleep disturbance and depression aggressively can result in suicide.

CLINICAL PEARLS

Because phantom limb pain can be so severe and have such devastating consequences, the clinician must treat it rapidly and aggressively. Special attention must be paid to the insidious onset of severe depression, which mandates hospitalization with suicide precautions.

SUGGESTED READINGS

Anderson-Barnes VC, McAuliffe C, Swanberg KM, et al. Phantom limb pain: a phenomenon of proprioceptive memory? *Med Hypotheses*. 2009;73(4):555–558.

Boller F, Bogousslavsky J. Paul Wittgenstein's right arm and his phantom: the saga of a famous concert pianist and his amputation. In: Altenmüller E, Finger S, Boller F, eds. *Progress in brain research*. Vol. 216. Philadelphia: Elsevier; 2015:293–303.

Diers M, Krumm B, Fuchs X, et al. The prevalence and characteristics of phantom limb pain and non-painful phantom phenomena in a nationwide survey of 3,374 unilateral limb amputees. *J Pain*. 2022;23(3):411–423. https://doi.org/10.1016/j.jpain.2021.09.003.

Kaur A, Guan Y. Phantom limb pain: a literature review. *Chin J Traumatol*. 2018;21(6):366–368. https://doi.org/10.1016/j.cjtee.2018.04.006.

Kuffler DP. Can phantom limb pain be reduced/eliminated solely by techniques applied to peripheral nerves? *J Neurorestoratol*. 2019;7(1):26–36. https://doi.org/10.26599/JNR.2019.9040002.

Lewis RP, Kriukelyte I. Complex neuropathic pain states. *Anaesth Intens Care Med*. 2016;17(11):571–574.

Makin TR, Flor H. Brain (re)organisation following amputation: implications for phantom limb pain. *NeuroImage*. 2020;218:116943. https://doi.org/10.1016/j.neuroimage.2020.116943.

Nikolajsen L, Springer JS, Haroutiunian S. Phantom limb pain. In: Benzon HT, Rathmell JP, Wu CL, eds. *Practical management of pain*. 5th ed. Philadelphia: Mosby; 2014:369–377.e3.

Pacheco-Barrios K, Pinto CB, Saleh Velez FG, et al. Structural and functional motor cortex asymmetry in unilateral lower limb amputation with phantom limb pain. *Clin Neurophysiol*. 2020;131(10):2375–2382. https://doi.org/10.1016/j.clinph.2020.06.024.

Senkowski D, Heinz A. Chronic pain and distorted body image: implications for multisensory feedback interventions. *Neurosci Biobehav Rev*. 2016;69:252–259.

Wade NJ. Beyond body experiences: phantom limbs, pain and the locus of sensation. *Cortex*. 2009;45(2):243–255.

Waldman SD. Phantom limb pain. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:301–302.

Trochanteric Bursitis

ICD-10 CODE M70.60

THE CLINICAL SYNDROME

Trochanteric bursitis is commonly encountered in clinical practice. Patients suffering from trochanteric bursitis frequently complain of pain in the lateral hip that radiates down the leg and mimics sciatica (Fig. 112.1). The pain is localized to the area over the trochanter. Often, patients are unable to sleep on the affected hip and may complain of a sharp "catching" sensation with range of motion of the hip, especially on first arising. Patients may note that walking upstairs is becoming increasingly difficult. Trochanteric bursitis often coexists with arthritis of the hip, back and sacroiliac joint disease, and gait disturbance.

The trochanteric bursa lies between the greater trochanter and the tendon of the gluteus medius and the iliotibial tract. This bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The trochanteric bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries may be caused by direct trauma to the bursa from falls onto the greater trochanter or previous hip surgery, as well as by overuse injuries, including running on soft or uneven surfaces. If inflammation of the trochanteric bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Physical examination reveals point tenderness in the lateral thigh just over the greater trochanter. Passive adduction and abduction, as well as active resisted abduction, of the affected lower extremity reproduce the pain. Sudden release of resistance during this maneuver causes a marked increase in pain (Fig. 112.2). No sensory deficit should be noted in the distribution of the lateral femoral cutaneous

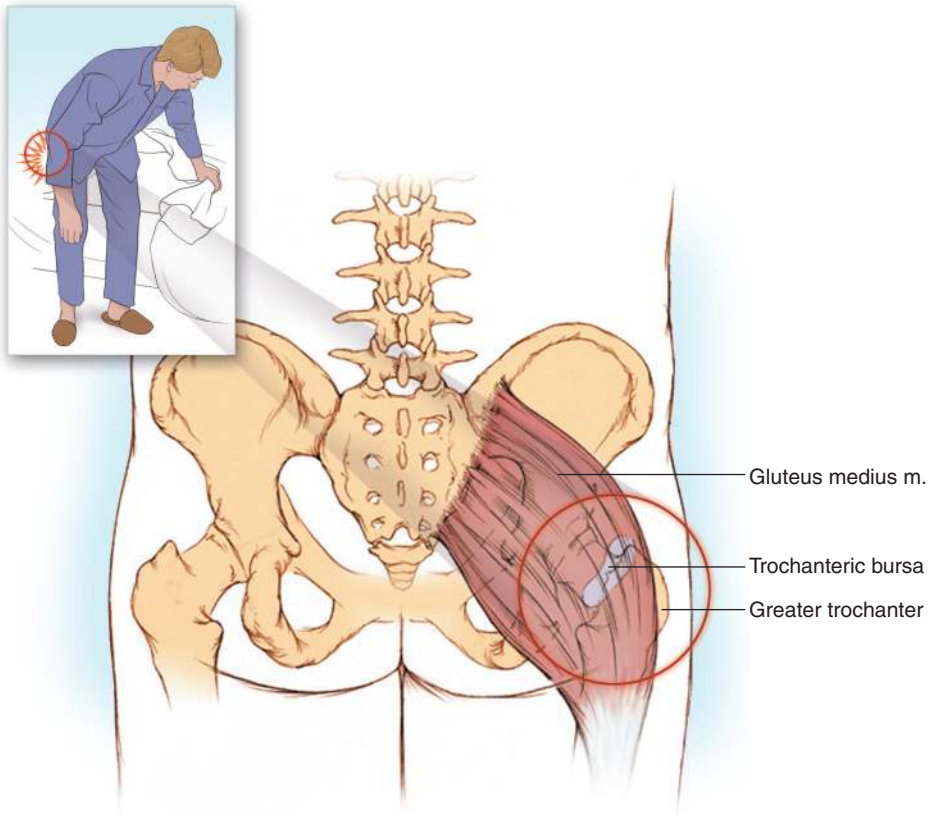

FIG 112.1 The pain of trochanteric bursitis often mimics that of sciatica.

FIG 112.2 Trendelenburg gait sign: **(A)** excessive contralateral hip drop or **(B)** ipsilateral trunk side bending during the stance phase of gait. (From Mulligan EP, Middleton EF, Brunette M. Evaluation and management of greater trochanter pain syndrome. *Phys Ther Sport*. 2015;16(3):205–214.)

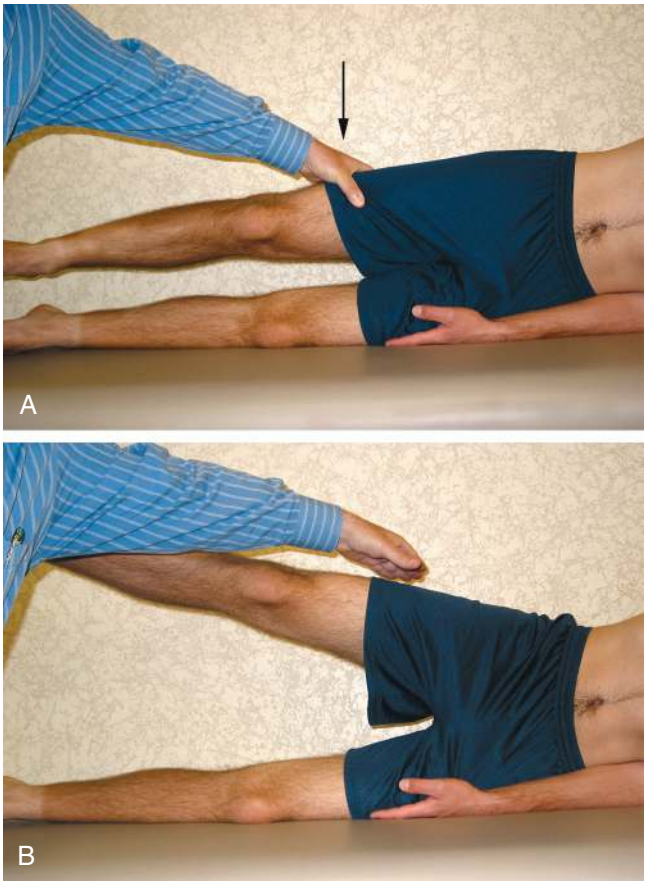

FIG 112.3 A, B, The resisted abduction release test. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:316.)

nerve; this feature distinguishes trochanteric bursitis from meralgia paresthetica. A trendelenburg gait may be present (Fig. 112.3).

TESTING

Plain radiographs of the hip may reveal calcification of the bursa and associated structures, findings consistent with chronic inflammation (Fig. 112.4). Magnetic resonance imaging is indicated if an occult mass or tumor of the hip or groin is suspected or to confirm the diagnosis (Figs. 112.5 and 112.6). A complete blood count and erythrocyte sedimentation rate are useful if infection is suspected. Electromyography (EMG) can distinguish trochanteric bursitis from meralgia paresthetica and sciatica. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Trochanteric bursitis frequently coexists with arthritis of the hip. Occasionally, trochanteric bursitis can be confused with meralgia paresthetica, because both manifest with pain in the lateral thigh. In patients with meralgia paresthetica,

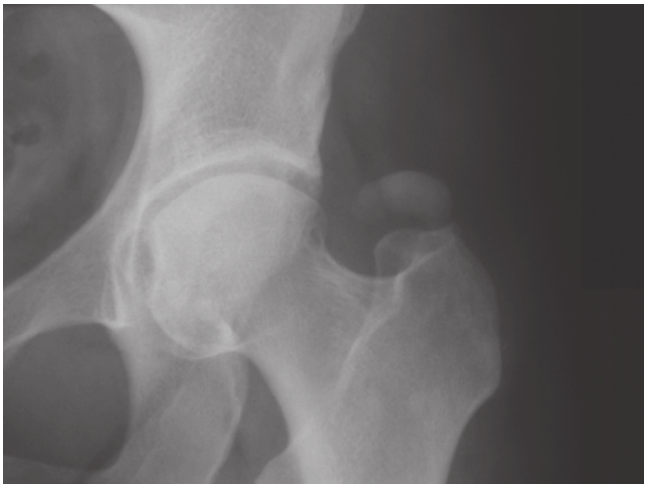

FIG 112.4 Radiograph of the hip of a patient with calcific tendinitis caused by hydroxyapatite crystal deposition; it can be identified as a focal area of calcification in the gluteal tendon superior to the greater trochanter. Radiographs obtained 6 months later showed that the calcification had spontaneously resolved. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

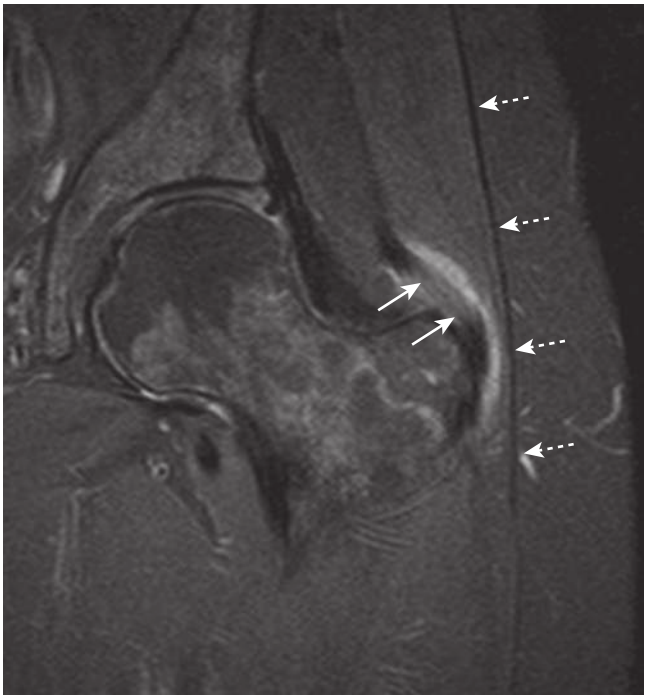

FIG 112.5 Coronal fat-suppressed T2-weighted magnetic resonance image of a patient with lateral hip pain and trochanteric bursitis with high signal intensity (SI) fluid lying between the iliotibial tract (*broken white arrows*) and the gluteus minimus tendon (*white arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

however, palpation over the greater trochanter does not elicit pain. EMG can help sort out confusing clinical presentations. Primary or secondary tumors of the hip must also be considered in the differential diagnosis of trochanteric bursitis (Box 112.1).

BOX 112.1 Differential Diagnosis of Trochanteric Bursitis

- Arthritides of the hip
- Acetabular labral tear
- Avascular necrosis of the femoral head
- Septic arthritis
- Femoroacetabular impingement syndrome
- Fractures of the femoral neck
- Avulsion fractures of the greater trochanter
- Gluteal medius tendinopathy
- Meralgia paresthetica
- Impingement syndromes
- Piriformis tendinopathy
- Piriformis syndrome
- Iliopsoas tendinopathy
- Iliotibial band inflammation
- Snapping hip syndrome
- Leg-Calve-Perthes disease
- Lumbar radiculopathy
- Complex regional pain syndrome

FIG 112.6 Synovial osteochondromatosis or chondromatosis. **A,** Axial, fat-saturated, T2-weighted magnetic resonance imaging (MRI). **B,** Coronal, fat-saturated, T2-weighted MRI. (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3392.)

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs), or cyclooxygenase-2 inhibitors is a reasonable first step in the treatment of trochanteric bursitis. Patients should be instructed to avoid repetitive activities that may be responsible for the development of trochanteric bursitis, such as running on sand. If patients do not experience rapid improvement, injection is a reasonable next step.

Injection of the trochanteric bursa is carried out by placing the patient in the lateral decubitus position with the affected side upward. The midpoint of the greater trochanter is identified, and the skin overlying this point is prepared with antiseptic solution. A syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 3½-inch, 25-gauge needle. Before needle placement, the patient should be instructed to say "There!" as soon as a paresthesia is felt in the lower extremity, thus indicating that the needle has impinged on the sciatic nerve. If paresthesia occurs, the needle is immediately withdrawn and is repositioned more laterally. The needle is then slowly advanced through the previously identified point at a right angle to the skin, directly toward the center of the greater trochanter (Fig. 112.7), until it hits bone; the needle is then withdrawn out of the periosteum. After careful aspiration for blood, and if no paresthesia is present, the contents of the syringe are gently injected into the bursa. Resistance to injection should be minimal. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease needle-induced complications (Fig. 112.8).

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics, NSAIDs, and antimyotonic agents can be used concurrently with this injection technique. Low-energy extracorporeal shock wave therapy has also been shown to decrease the pain associated with trochanteric bursitis as has the injection of platelet-rich plasma and/or stem cells. Rarely, surgical excision of the bursa will be required.

COMPLICATIONS AND PITFALLS

Other conditions that may mimic the pain of trochanteric bursitis must be excluded. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. Special care must be taken to avoid trauma to the sciatic nerve, which makes it imperative that this procedure be performed only by those familiar with the regional anatomy and experienced in the technique. Most complications of the injection technique are related to needle-induced trauma at the injection site and in the underlying tissues. Infection, although rare, can occur, and this possibility makes careful attention to sterile technique mandatory. Many patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

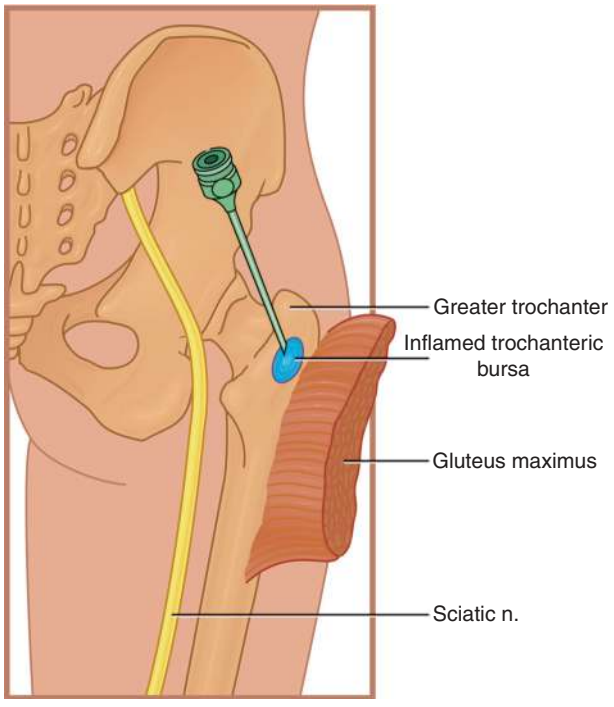

FIG 112.7 Correct needle placement for injection of the trochanteric bursa. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

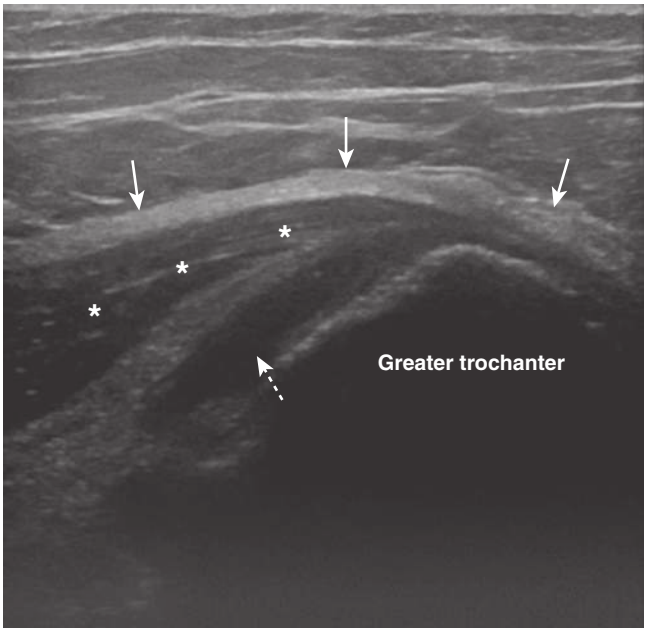

FIG 112.8 Axial ultrasound image of the hip in a patient with lateral hip pain. There is low-echo fluid within the subgluteus medius bursa (*broken white arrow*), which lies between the gluteus medius muscle and tendon (*asterisks*) and the posterolateral facet of the greater trochanter. The iliotibial band is superficial to the gluteus medius (*white arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

CLINICAL PEARLS

Trochanteric bursitis frequently coexists with arthritis of the hip, which may require specific treatment to achieve pain relief and return of function. The injection technique described is extremely effective in the treatment of trochanteric bursitis.

SUGGESTED READINGS

Hodnett PA, Shelly MJ, MacMahon PJ, et al. MR imaging of overuse injuries of the hip. *Magn Reson Imaging Clin N Am*. 2009;17(4):667–679.

Mallow M, Nazarian LN. Greater trochanteric pain syndrome diagnosis and treatment. *Phys Med Rehabil Clin N Am*. 2014;25(2):279–289.

Mulligan EP, Middleton EF, Brunette M. Evaluation and management of greater trochanter pain syndrome. *Phys Ther Sport*. 2015;16(3):205–214.

Reid D. The management of greater trochanteric pain syndrome: a systematic literature review. *J Orthop*. 2016;13(1):15–28.

Shemesh SS, Moucha CS, Keswani A, et al. Trochanteric bursitis following primary total hip arthroplasty: incidence, predictors, and treatment. *J Arthroplast*. 2018;33(4):1205–1209. https://doi.org/10.1016/j.arth.2017.11.016.

Waldman SD. Trochanteric bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:671–676.

Waldman SD. Trochanteric bursitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:303–304.

Waldman SD. Trochanteric bursa injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:553–557.

Waldman SD, Campbell RSD. Trochanteric bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:361–363.

113

Arthritis Pain of the Knee

ICD-10 CODE M17.9

THE CLINICAL SYNDROME

Arthritis of the knee is a common painful condition. The knee joint is susceptible to the development of arthritis from various conditions that can damage the joint cartilage. Osteoarthritis is the most common form of arthritis that results in knee pain; rheumatoid arthritis and posttraumatic arthritis are also common causes of knee pain. Less-frequent causes of arthritis-induced knee pain include the collagen vascular diseases, infection, villonodular synovitis, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the knee joint, although knee pain secondary to collagen vascular disease responds exceedingly well to the treatment modalities described here.

SIGNS AND SYMPTOMS

Most patients with osteoarthritis or posttraumatic arthritis of the knee complain of pain localized around the knee and distal femur. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with use of the joint, and crepitus may be present on the physical examination.

In addition to pain, patients often experience a gradual reduction in functional ability because of decreasing knee range of motion that makes simple everyday tasks such as walking, climbing stairs, and getting in and out of a car quite difficult (Fig. 113.1). With continued disuse, muscle wasting may occur, and a frozen knee resulting from adhesive capsulitis may develop.

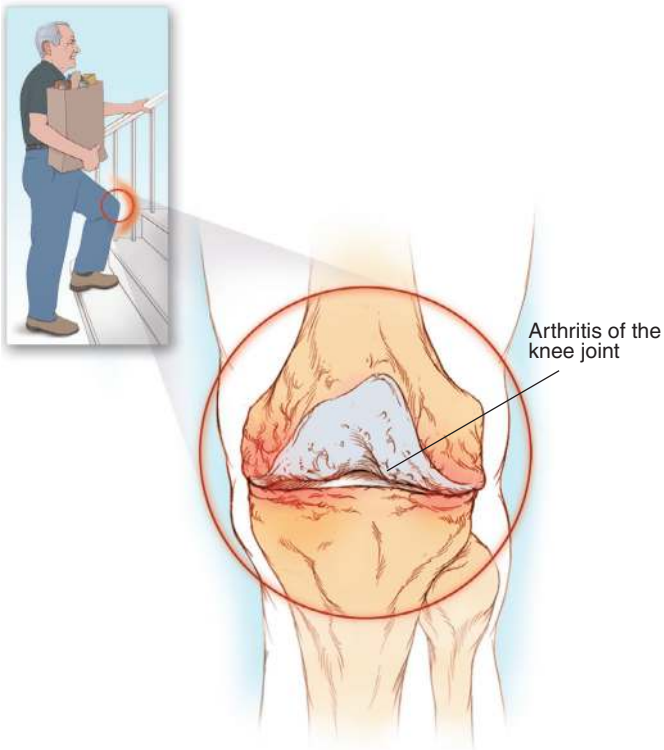

FIG 113.1 The pain of arthritis of the knee is made worse with weight-bearing activities.

TESTING

Plain radiographs, magnetic resonance imaging (MRI), and ultrasound imaging are indicated in all patients who present with knee pain (Figs. 113.2–113.5). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. MRI and ultrasound imaging of the knee are also indicated if the diagnosis is in question, if an occult mass or tumor is suspected, or in the presence of trauma (Fig. 113.6).

DIFFERENTIAL DIAGNOSIS

Lumbar radiculopathy may mimic the pain and disability associated with arthritis of the knee. In such patients, results of the knee examination should be negative. Bursitis of the

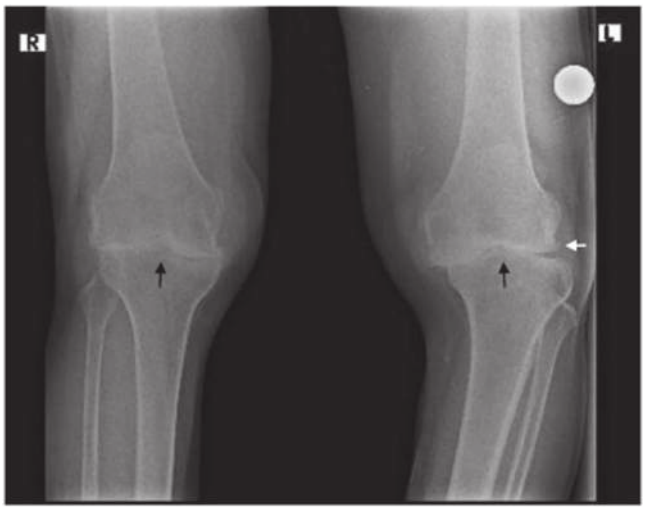

FIG 113.2 X-ray of both knee joints showing narrowed joint space (*black arrow*) with the presence of osteophytes (*white arrow*), consistent with osteoarthritis. (From Das S, Chattopadhyay P, Ray A, Sharma V. Incidental diagnosis of bilateral synovial lipomatosis in long standing knee osteoarthritis. *Human Pathol*. 2015;2(4):103–113.)

knee and entrapment neuropathies, such as meralgia paresthetica, may also confuse the diagnosis; both these conditions may coexist with arthritis of the knee. Primary and metastatic tumors of the femur and spine may also manifest in a manner similar to arthritis of the knee (Box 113.1).

TREATMENT

Initial treatment of the pain and functional disability associated with arthritis of the knee includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

For intraarticular injection of the knee, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the medial joint is prepared with antiseptic solution. A sterile syringe containing 5 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The joint space is identified, and the clinician places his or her thumb on the lateral margin of the patella and pushes it medially. At a point in the middle of the medial edge of the patella, the needle is inserted between the patella and the femoral condyles. The needle is then carefully advanced through the skin and subcutaneous tissues through the joint capsule and into the joint (Fig. 113.7). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection

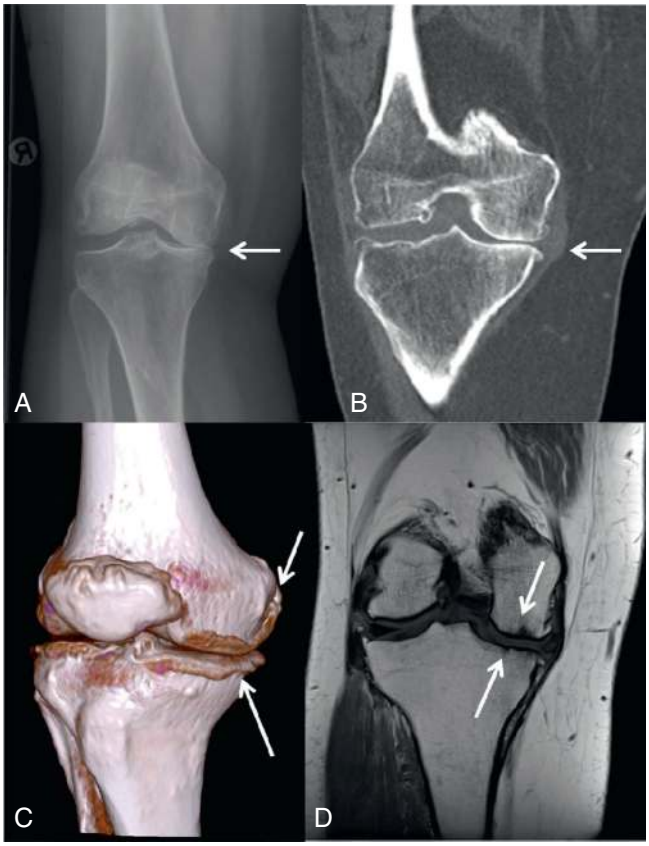

FIG 113.3 Imaging of osteoarthritis of the knee. Examples of various imaging technologies used in osteoarthritis (OA) studies, using imaging results of a 57-year-old female with knee OA and a painful and swollen knee. **(A)** Conventional standing semiflexed radiograph of the right knee, showing severe medial knee OA. **(B)** Computed tomography (CT) scan of the same knee, showing in more detail the kissing bone, osteophytes at the medial and lateral joint margins and the tibial eminentia as well as subchondral sclerosis and meniscal extrusion (*arrow*). **(C)** 3D volume rendering of the CT scan, allowing to study the bony morphology in 3D. It becomes apparent that osteophytes are made up of a bony rim enlargement from the edges of both tibia and femur (*arrows*). **(D)** Coronal T1 weighted magnetic resonance imaging without fat saturation of the same knee, now also showing small hypointense bone marrow lesions in the medial femur and tibia (*arrows*). (From Van Spil WE, Kubassova O, Boesen M, Bay-Jensen AC, Mobasheri A. Osteoarthritis phenotypes and novel therapeutic targets. *Biochemical Pharmacology*. 2019;165:41–48.)

should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-induced complications (Fig. 113.8). The injection of platelet-rich plasma and/or stem cells has been

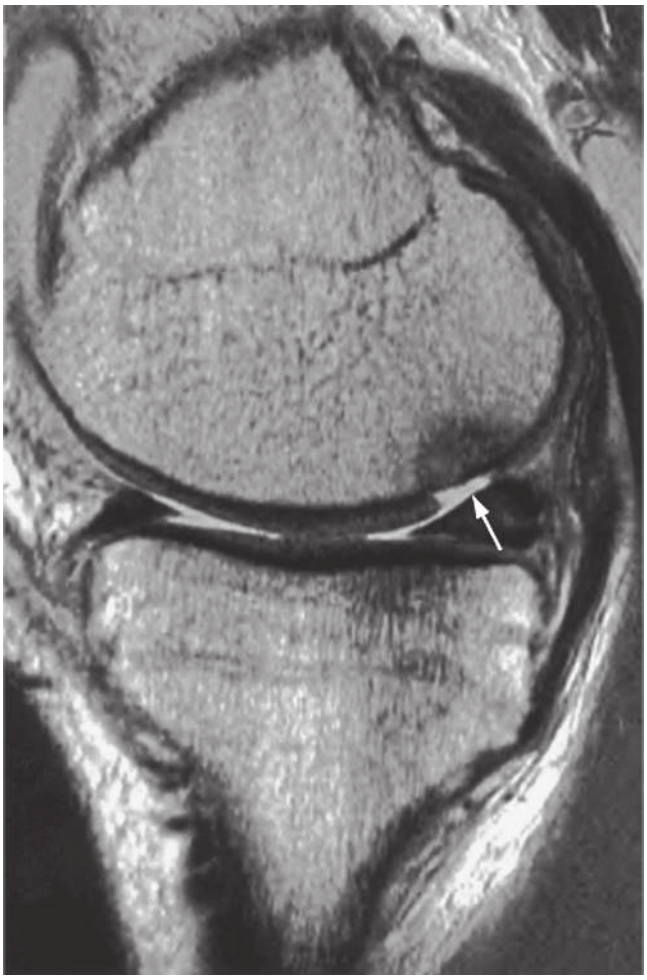

FIG 113.4 Sagittal fast spin-echo image through the medial joint line demonstrates focal full-thickness chondral injury with associated subchondral osseous changes (*arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3425.)

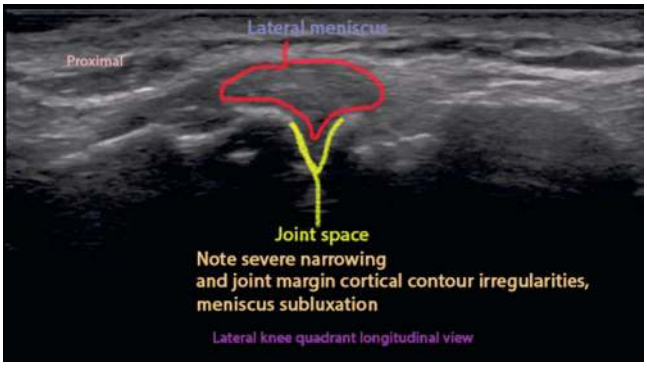

FIG 113.5 Longitudinal ultrasound image demonstrating severe osteoarthritis of the joint.

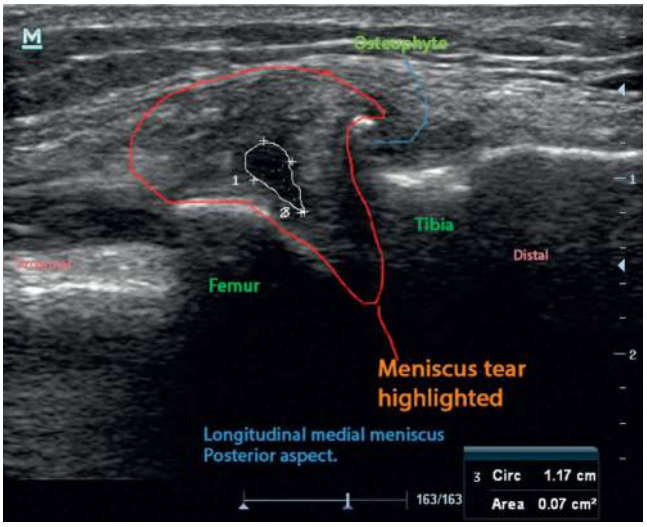

FIG 113.6 Longitudinal ultrasound image demonstrating a flap tear of the medial meniscus.

BOX 113.1 Causes of Knee Pain and Dysfunction

Arthritis
- Osteoarthritis
- Rheumatoid
- Gout
- Pseudogout
- Reactive arthritis
- Septic arthritis

Trauma
- Fractures
- Meniscal injuries
- Tendinitis
- Bursitis
- Ligamentous injuries

Mechanical Abnormalities
- Joint mouse
- Altered gait due to hip, foot, or ankle problems
- Iliotibial band syndrome
- Patellar abnormalities (e.g., patella alta, bipartite patella)

Other Causes
- Avascular necrosis
- Foreign-body synovitis
- Charcot joint
- Neurofibromatosis
- Malignancy
- Pseudorhematism

shown to decrease the pain associated with many pathologic conditions of the knee.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Flat, flexible footwear may help decrease medial knee joint loading and help with symptom relief in selected patients. Vigorous exercises should be avoided because they will exacerbate the patient's symptoms.

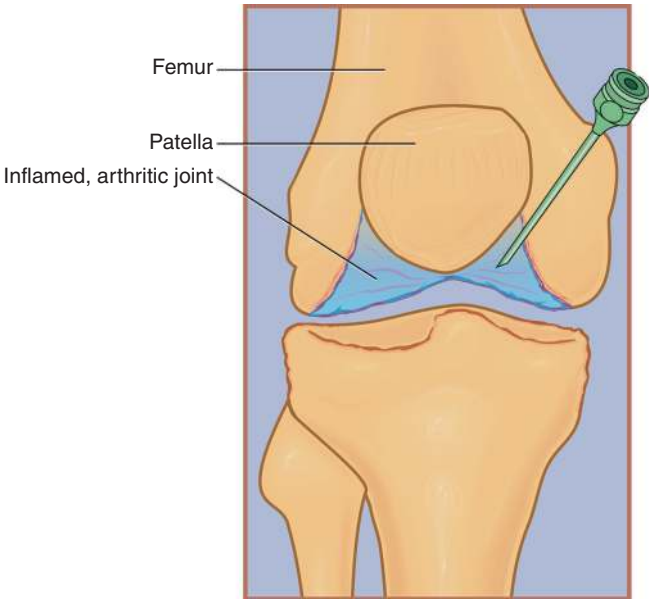

FIG 113.7 Proper needle placement for intraarticular injection of the knee. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

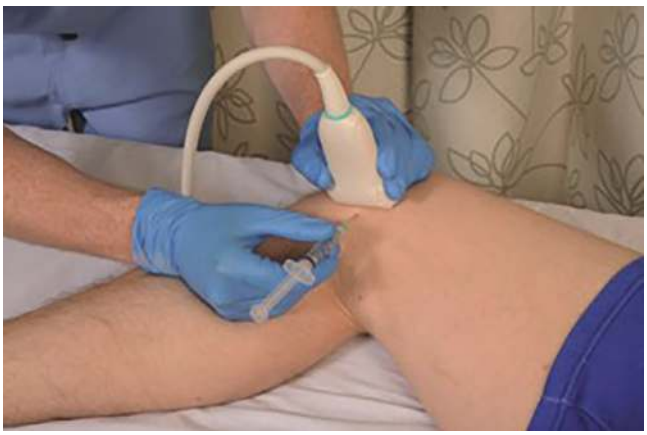

FIG 113.8 Ultrasound-guided injection of the knee. (Courtesy Steven Waldman MD.)

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the knee or spine that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of intraarticular injection of the knee is infection, although it should be exceedingly rare if strict aseptic technique is followed, along with universal precautions to minimize any risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is applied to the injection site immediately after the injection. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to knee pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in the treatment of pain secondary to arthritis of the knee.

SUGGESTED READINGS

Waldman SD. Arthritis and other abnormalities of the knee. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:725–740.

Waldman SD. Functional anatomy of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:142–144.

Waldman SD. Intra-articular injection of the knee joint. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:546–547.

Waldman SD. Intra-articular injection of the knee. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:651–666.

Waldman SD, Campbell RSD. Anatomy, special imaging considerations of the knee. In: *Imaging of pain*. Philadelphia: Saunders Elsevier; 2011:367–368.

Waldman SD, Campbell RSD. Osteonecrosis of the knee. In: *Imaging of pain*. Philadelphia: Saunders Elsevier; 2011:393–396.

Xu C, Peng H, Li R, et al. Risk factors and clinical characteristics of deep knee infection in patients with intra-articular injections: a matched retrospective cohort analysis. *Sem Arthr Rheum*. 2018;47(6):911–916.

114

Lyme Disease

ICD-10 A69.29

THE CLINICAL SYNDROME

With over 30,000 cases annually, Lyme disease is the most common vector-transmitted disease in the United States. This multisystem disease is caused by infection with the spirochete *Borrelia burgdorferi* and is transmitted by painless bites from infected *Ixodes* genus ticks (Figs. 114.1 and 114.2). Lyme disease is associated with significant morbidity, and the severity of this disease is often misunderstood given the mild initial clinical presentation. Because only 50% of patients with laboratory-proven Lyme disease can recall to have been bitten by a tick, the clinician must maintain a high index of suspicion for the disease, especially in those patients presenting with a flulike illness, adenopathy, and rash as they spend significant amounts of time outdoors. The peak occurrence of Lyme disease is seasonal from late spring to early fall.

SIGNS AND SYMPTOMS

The triad of signs and symptoms of early Lyme disease are (1) flulike illness; (2) tender local adenopathy; and (3) the characteristic rash, erythema migrans (Fig. 114.3). Erythema migrans occurs in over two-thirds of patients with Lyme disease (Fig. 114.4). This characteristic rash may occur as a single or multiple lesion which occurs in proximity to the site of the tick bite. The rash usually is apparent within 7 days of the tick bite and tends to expand in concentric rings over the next several days producing the characteristic bulls-eye rash (see Figs. 114.4 and 114.5). Untreated rash may persist

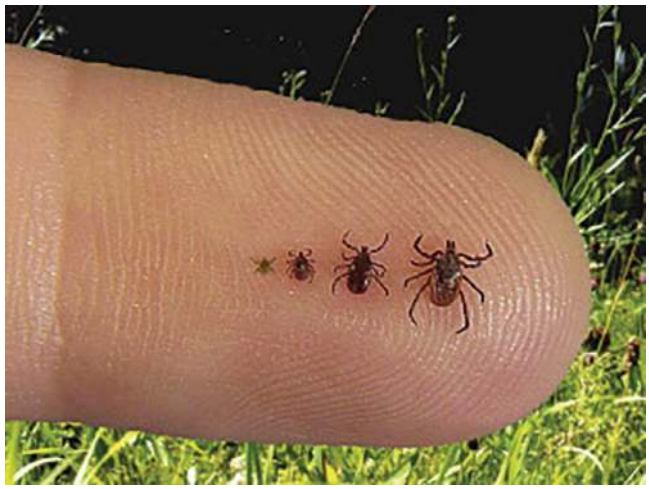

FIG 114.2 Ticks on the fingertip demonstrating their small size. (Courtesy of California Department of Public Health, Sacramento, CA.)

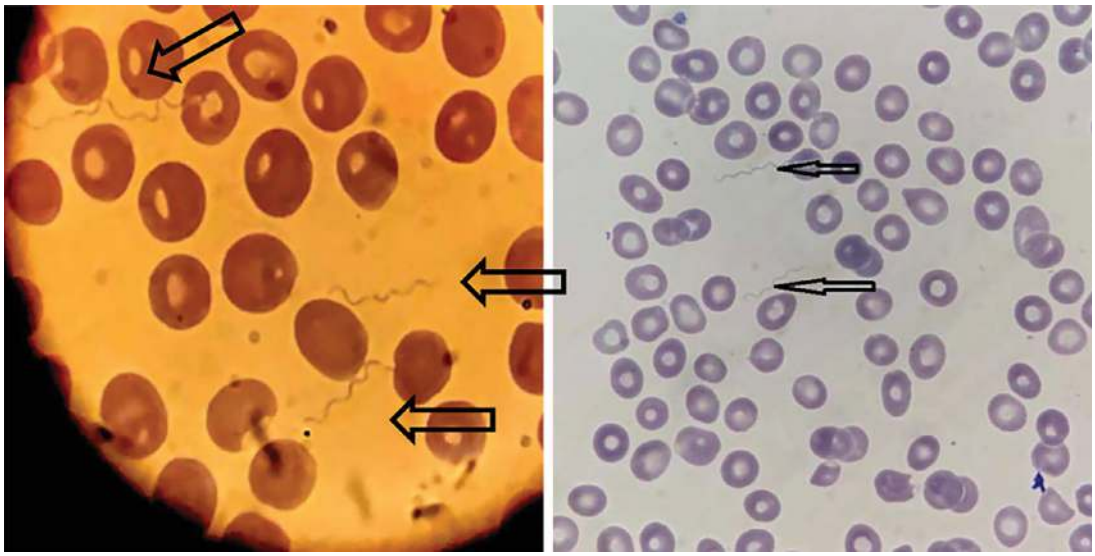

FIG 114.1 Patient blood smear showing *Borrelia* spirochetes. (From Eiferman V, Le Guenno G, Boiret-Dupré N, et al. Atypical *Borrelia garinii* infection in an immunocompromised patient mimicking high-grade lymphoma. *Int J Infect Dis.* 2022;121:102–104.)

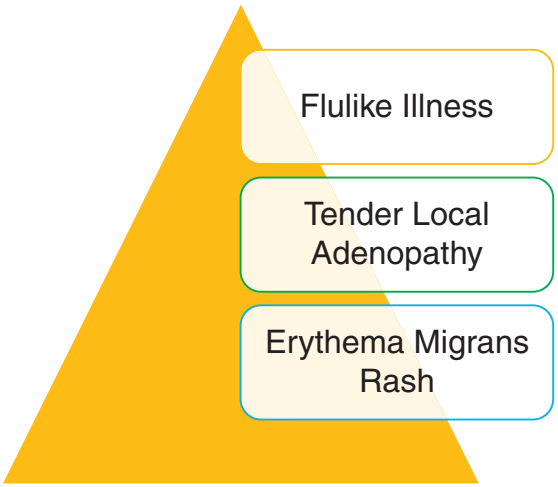

FIG 114.3 The classic triad of signs and symptoms of Lyme disease.

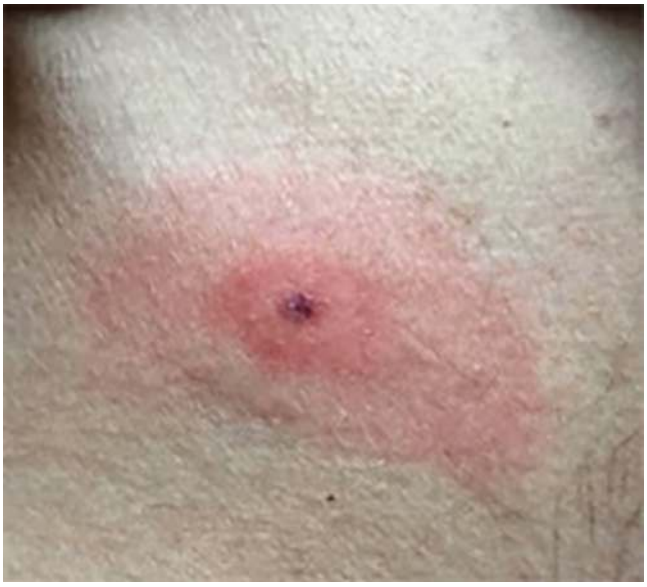

FIG 114.4 The targetoid or bull's eye rash known as erythema migrans, diagnostic of early localized Lyme disease. (From DelBuono N, Saks M. Early localized Lyme disease. *Vis J Emerg Med*. 2022;27:101283.)

for 2–3 weeks with recurrent episodes occasionally observed in untreated patients. As the disease progresses, in addition to initial clinical trial of symptoms, the patient will experience systemic symptoms including headache, fever, myalgia, arthralgia, and a stiff neck.

Over the ensuing 2–3 weeks, two-thirds of patients suffering from Lyme disease will progress to Stage 2 disease. Stage 2 disease is characterized by increasing systemic symptoms including high fever, malaise, cardiac arrythmias, myocarditis, cranial nerve palsies, encephalopathy, stroke, visual disturbance, scleritis, and conjunctivitis (Figs. 114.6–114.8 and Box 114.1). Musculoskeletal complaints including severe migratory polyarthralgias with associated bursitis and tendinitis are common with the knee, ankle, and wrists most commonly affected. Recurrent joint pain is common. Borrelial lymphocytoma is a painless bluish-red lesion that may rarely occur on the ear, breast, or lip (Figs. 114.9 and 114.10).

Stage 3 Lyme disease is also known as late or chronic Lyme disease and occurs months to years after the initial infection. Interestingly, most patients who progress to Stage 3 Lyme disease do not have a history of developing erythema migrans, the identification of which is so crucial to the early diagnosis

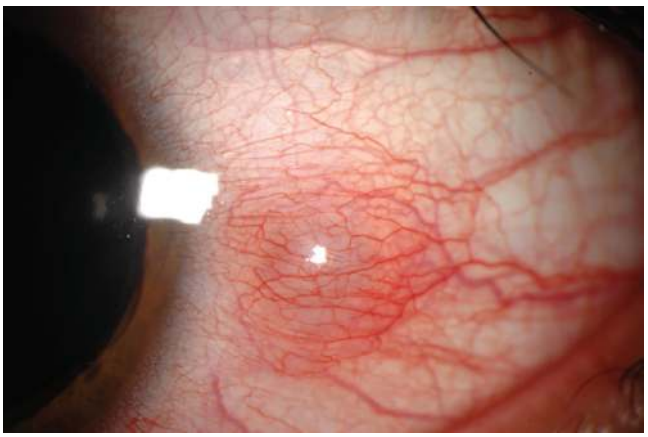

FIG 114.6 Scleritis in patient suffering from Lyme disease. (From Berkenstock MK, Long K, Miller JB, et al. Scleritis in Lyme disease. *Am J Ophthalmol*. 2022:139–144.)

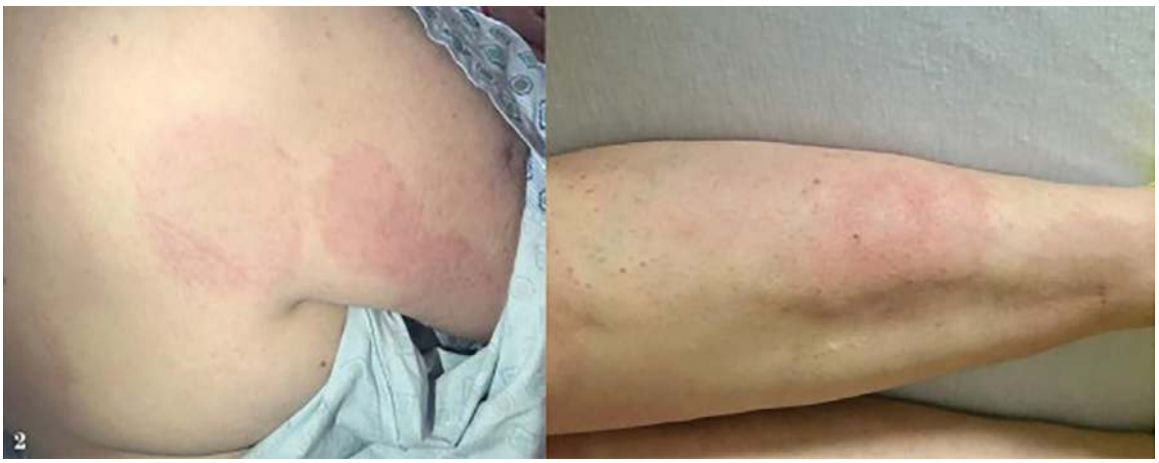

FIG 114.5 Multiple erythema migrans rash. (From Rahangdale R, Alcantara Lima A, Tayal A, et al. Lyme disease presenting as acute ischemic strokes with an embolic pattern. *Interdiscip Neurosurg*. 2020;22:100838.)

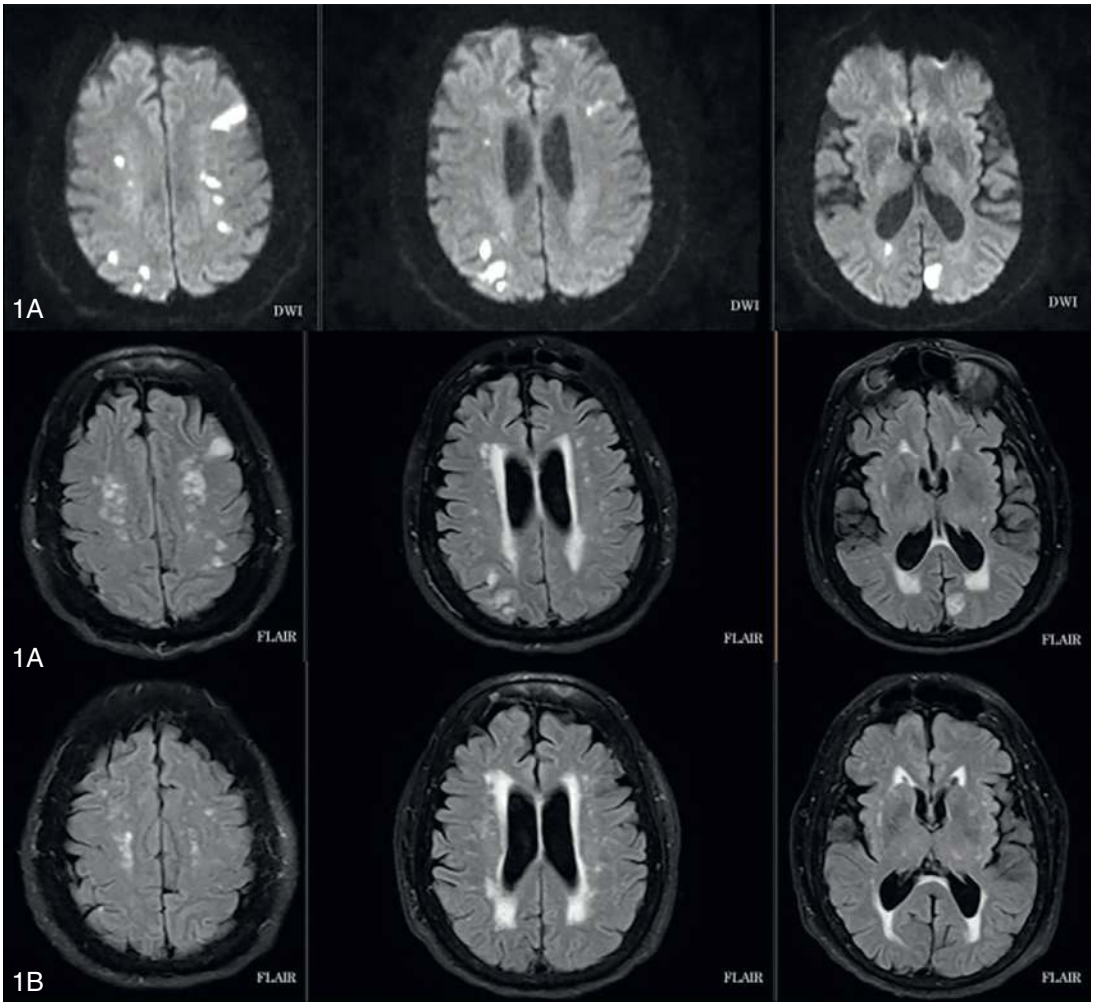

FIG 114.7 Brain magnetic resonance imaging; **(A)** Diffusion weighted images (DWI) and FLAIR images showing acute ischemic strokes in multiple vascular territories at presentation, **(B)** Fluid-attenuated inversion recovery (FLAIR) images showing complete resolution of ischemic stroke lesions six months later. (From Rahangdale R, Alcantara Lima A, Tayal A, et al. Lyme disease presenting as acute ischemic strokes with an embolic pattern. *Interdiscip Neurosurg*. 2020;22:100838.)

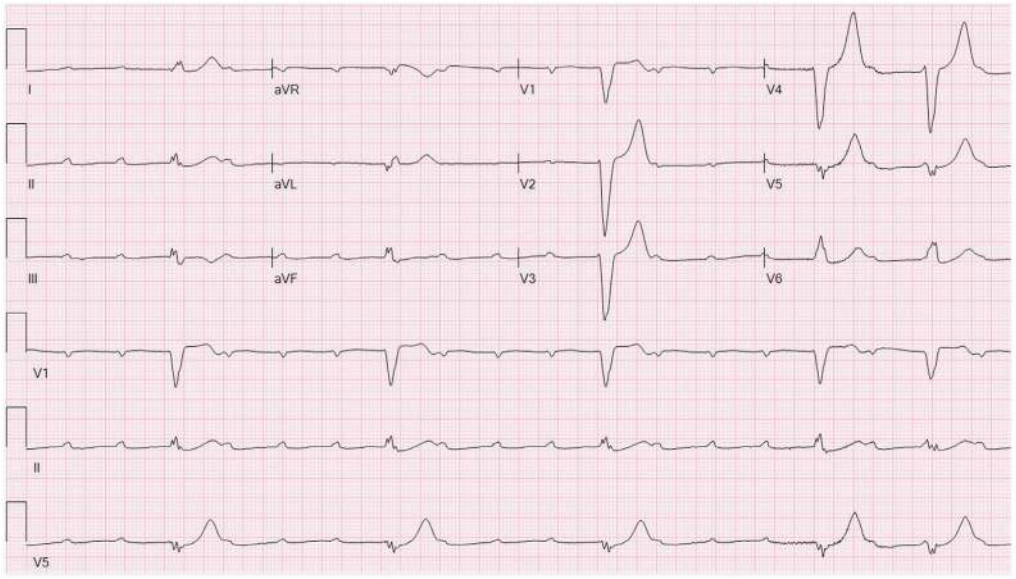

FIG 114.8 Electrocardiogram (ECG) on presentation showing third-degree atrioventricular block (AVB) with ventricular escape rhythm. (From Brissett S, Myint KT, Lopez Y, et al. A curious case of Lyme carditis in an urban hospital. *IDCases*. 2021;25:e01179.)

BOX 114.1 Ocular Manifestations of Lyme Disease

- Cranial nerve palsies
- Keratitis
- Iritis
- Scleritis
- Chorioretinitis
- Retinal vasculitis
- Retinal detachment
- Uveitis
- Pars planitis
- Vitreitis
- Branch artery occlusion
- Orbital myositis

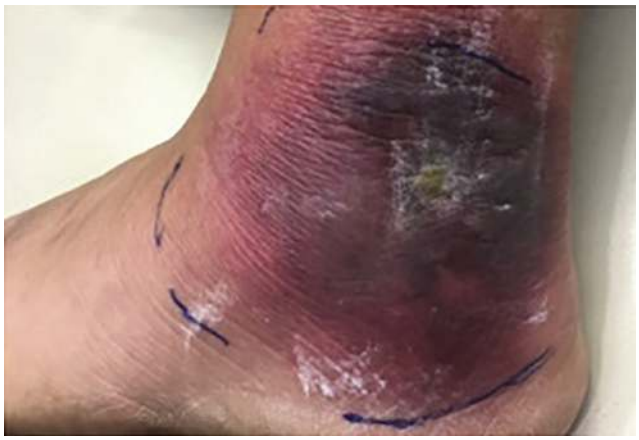

FIG 114.9 Bright red, annular, edematous, vesicular plaque on the lateral aspect of the left malleolus in a patent with Lyme-associated ankle pain. (From Doughty H, O'Hern K, Barton DT, Carter JB. Vesiculobullous Lyme disease: a case series. *JAAD Case Rep*. 2022;24:56–58.)

and treatment of Lyme disease. This means that the clinician must have a high index of suspicion in patients presenting with unexplained rheumatologic and neurologic disease. Patients with Stage 3 Lyme disease may present with autoimmune appearing joint disease with Lyme arthritis involving the knee present in 90% of cases. Superexuberant synovial proliferation is also a common feature of Stage 3 Lyme disease as is unexplained chronic relapsing demyelinating polyneuropathy, chronic pain, and neurocognitive impairment. Fatigue is a common complaint and may lead the clinician to attribute the patient's unexplained symptoms to psychiatric illness. Like borrelial lymphocytoma, acrodermatitis chronica atrophicans occurs primarily in European countries and is associated with Stage 3 Lyme disease.

TESTING

If a patient is suspected of suffering from Lyme disease, accurate diagnosis and rapid treatment with antibiotics are critical if one is to avoid complications. The US Centers for Disease Control and Prevention recommends a two-step testing procedure. The first step typically consists of a screening enzyme immunoassay or enzyme-linked immunosorbent assay; if results are positive or equivocal, a Western immunoblot test is performed to confirm the results. Recent experience with sequential enzyme immunoassay testing may increase the accuracy of the diagnosis of Lyme disease. Polymerase chain reaction (PCR) testing is also becoming more popular in the diagnosis of Lyme disease as when properly performed, PCR testing is highly accurate in the identification of *Borrelia burgdorferi*, in blood, urine, and synovial fluid of patients with Lyme disease. At the present time, biopsy of skin lesions thought to be associated with Lyme disease is not recommended other than to aid in the differential diagnosis of other dermatological diseases. Electrocardiograms should be obtained in all patients suspected of suffering from Lyme disease given the incidence of cardiac abnormalities including high-degree heart block and myocarditis seen in this disease. Synovial and cerebrospinal fluid analysis should

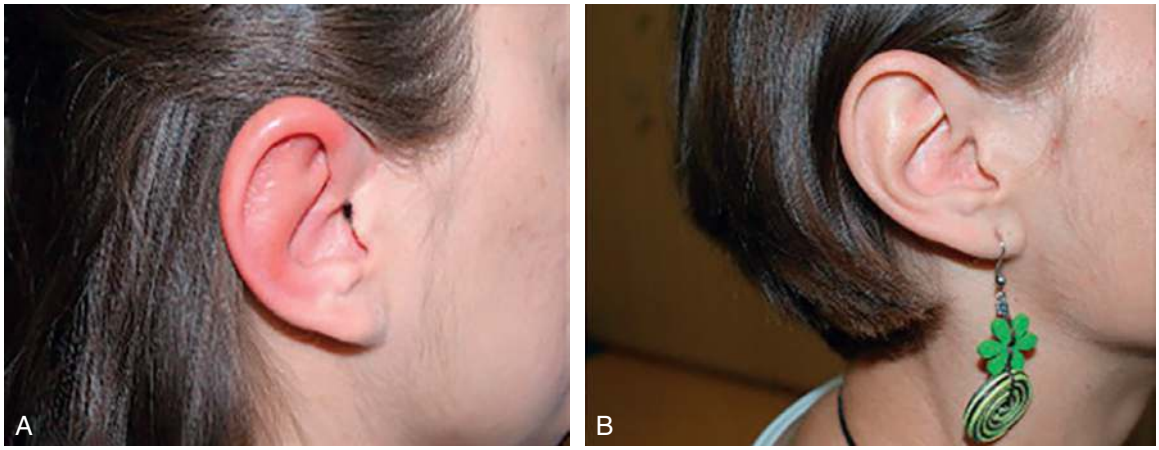

FIG 114.10 Borrelial lymphocytoma of the ear. **A,** Borrelial lymphocytoma before treatment. **B,** Status of skin after treatment of borrelial lymphocytoma. (From Moniuszko A, Czupryna P, Pancewicz S, et al. Borrelial lymphocytoma—a case report of a pregnant woman. *Ticks Tick-borne Dis*. 2012;3(4):257–258.)

be performed in selected patients to aid in the differential diagnosis. It should be remembered that in patients with erythema migrans, treatment with appropriate antibiotics should be initiated regardless of laboratory test results.

DIFFERENTIAL DIAGNOSIS

Clinicians unfamiliar with Lyme disease tend to either overdiagnose or underdiagnose this serious condition (Fig. 114.11). Although most patients present with the characteristic rash of Lyme disease, erythema, and migrans, up to one-third of patients suffering from Lyme disease may present with extradermatologic symptoms leading to diagnostic misadventures. Diseases that commonly mimic the clinical presentation of Lyme disease include collagen vascular diseases, crystal arthropathies including gout and pseudogout, psychiatric illness, cardiac conduction system disease, ankylosing spondylitis, acute infectious arthritis, and a variety of central and peripheral neurologic disorders including Bell's palsy, polyneuropathy, and cognitive diseases (Fig. 114.12 and Box 114.2).

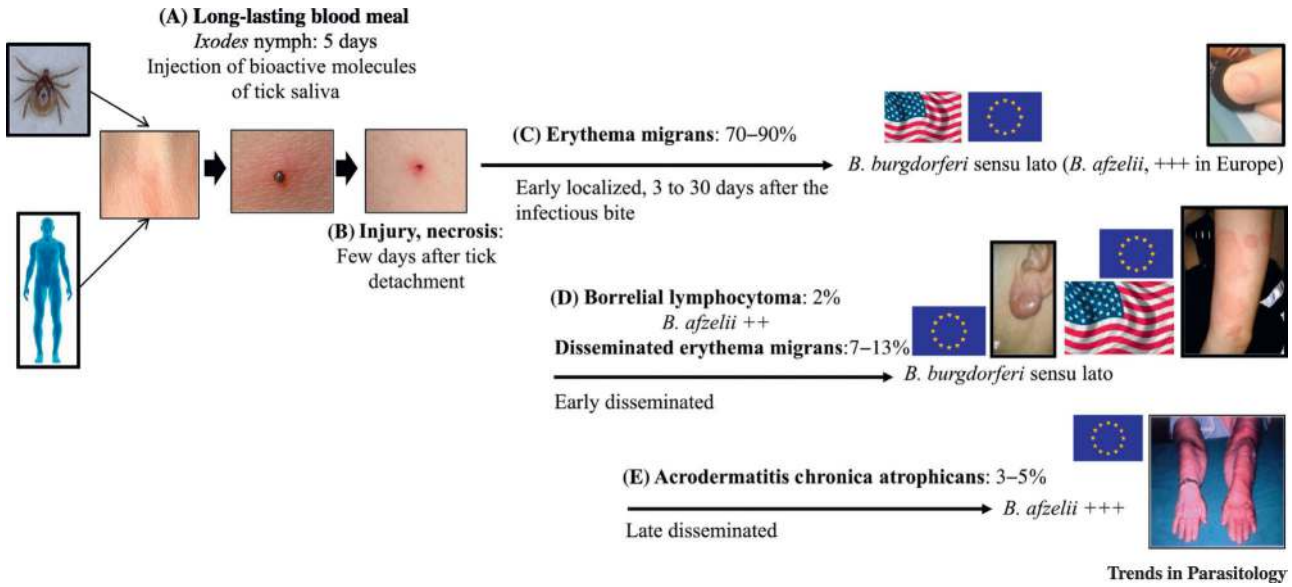

FIG 114.11 The clinical presentation of Lyme disease is driven in part by host skin immunity. Various Steps of an Infecting Tick Bite and Potential Skin Manifestations. **(A)** Ixodes tick bite and saliva inoculation. **(B)** Necrosis after tick detachment. **(C)** Appearance of erythema migrans if the tick is infected with Borrelia 3 to 30 days after the infecting blood meal. **(D)** Borrelial lymphocytoma in Europe only and disseminated erythema migrans mainly in the USA. **(E)** Acrodermatitis chronica atrophicans during the late disseminated phase of the disease in Europe only. (From Bernard Q, Grillon A, Lenormand C, Ehret-Sabatier L, Boulanger N. Skin Interface, a Key Player for Borrelia Multiplication and Persistence in Lyme Borreliosis. *Trends in Parasitology*. 2020; 36(3):304–314.)

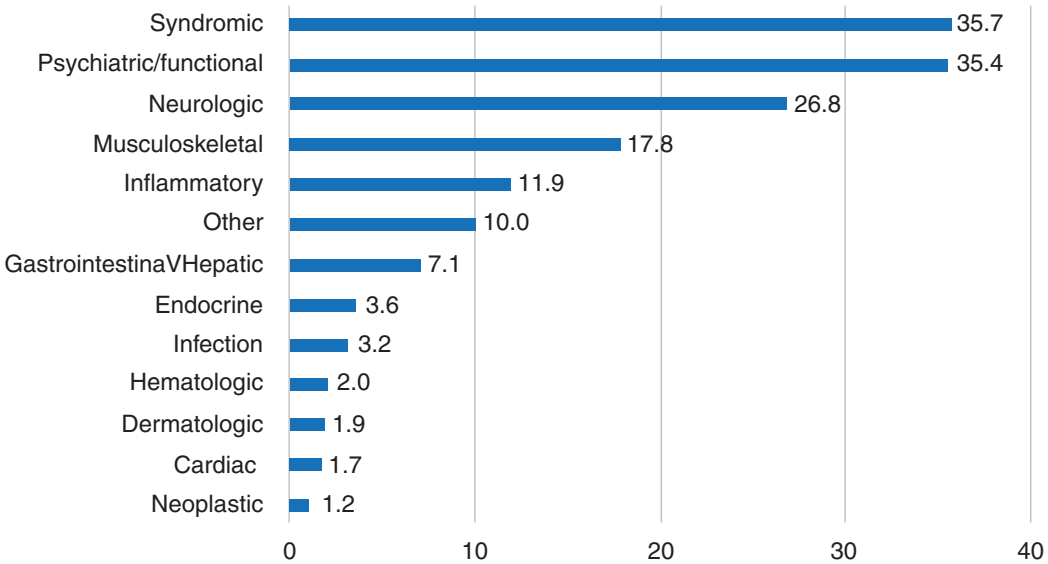

FIG 114.12 The unusual presentation of Lyme disease may lead to missed diagnosis of other significant disease entities. (From Kobayashi T, Higgins Y, Melia MT, Auwaerter PG. Mistaken identity: many diagnoses are frequently misattributed to Lyme disease. *Am J Med*. 2022;135(4):503–511.e5.)

BOX 114.2 Differential Diagnosis of Lyme Disease

- Collagen vascular diseases
- Ankylosing spondylitis
- Depression
- Primary psychiatric disorders
- Diabetes
- Chronic fatigue syndrome
- Fibromyalgia
- Myofascial pain syndrome
- Multiple sclerosis
- Menopausal symptoms
- Vitamin B12 deficiency
- Cranial nerve palsies
- Bell's palsy
- Dementia
- Vasculitis
- Tendinitis
- Syphilis
- Lead poisoning
- Weil's disease
- Meniere's disease
- Migraine
- Lou Gehrig's disease
- Osteoarthritis
- Polymyalgia rheumatica
- Relapsing fever
- Reynaud's syndrome
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Tension headache
- Thyroid disease

TREATMENT

Current clinical guidelines recommend oral doxycycline prophylaxis for all patients within 72 hours of removing a tick, especially when the tick bite occurs in areas endemic for Lyme disease and if the tick is engorged. The recommended dosage is 200 mg for adults and 4.4 mg/kg, not to exceed 200 mg for children. In patients who present with erythema migrans, a 14–21 days course of doxycycline, amoxicillin, or cefuroxime axetil is recommended. In patients with additional symptoms thought to be associated with Lyme disease, longer courses of oral or parenteral antibiotics are indicated. Many clinicians recommend immediate parenteral antibiotics for all pregnant women suspected of having Lyme disease. Hospitalization and close cardiac monitoring are indicated in all patients suffering from Lyme carditis.

COMPLICATIONS AND PITFALLS

In spite of appropriate diagnosis and treatment, some patients may develop posttreatment Lyme disease syndrome (PTLDS). PTLDS is characterized by a chronic fatigue syndrome-like illness with associated arthralgias and pain. It is unclear whether PTLDS is a persistent infection or immune response to Lyme disease or if it is a psychiatric comorbidity that is the result of the chronic illness.

CLINICAL PEARLS

It should be remembered that co-infection with other tick-borne illnesses may occasionally occur in patients suffering from Lyme disease. Examples include babesiosis, ehrlichiosis, flavivirus infection, and Powassan encephalitis. Efforts to prevent or at least mitigate the risk of tick-borne illnesses include promptly removing tick, clearing of underbrush, and the spraying of acaricides in the spring. Antibiotic prophylaxis after a tick bite is not accepted by all clinicians but is gaining popularity. Currently, there is no effective vaccination for Lyme disease in humans.

SUGGESTED READINGS

Baez J, Suffoletto H. Migrating swollen joint and Lyme disease: a case report. *J Emerg Nurs*. 2021;47(4):543–550.

Berkenstock MK, Long K, Miller JB, et al. Scleritis in Lyme disease. *Am J Ophthalmol*. 2022:139–144.

Berkenstock MK, Long K, Miller JB, Burkholder BB, Aucott JN, Jabs DA. Scleritis in Lyme Disease. *Am J Ophthalmol*. 2022 Sep;241:139–144. https://doi:10.1016/j.ajo.2022.04.017. Epub 2022 May 2. PMID: 35513033.

Coiffier G, Tattevin P. Lyme disease: "End of the debate?". *Jt Bone Spine*. 2021;88(4):105181.

DelBuono N, Saks M. Early localized lyme disease. *Vis J Emerg Med*. 2022;27:101283.

Kobayashi T, Higgins Y, Melia MT, Auwaerter PG. Mistaken identity: many diagnoses are frequently misattributed to Lyme disease. *Am J Med*. 2022;135(4):503–511.e5.

Muhammad S, Simonelli RJ. Lyme carditis: a case report and review of management. *Hosp Pharm*. 2018;53(4):263–265.

Neville DN, Alexander ME, Bennett JE, et al. Electrocardiogram as a Lyme disease screening test. *J Pediatr*. 2021;238:228–232.e1.

Rahangdale R, Alcantara Lima A, Tayal A, et al. Lyme disease presenting as acute ischemic strokes with an embolic pattern. *Interdiscip Neurosurg*. 2020;22:100838. https://doi.org/10.1016/j.inat.2020.100838.

Schrestha K, Kadkhoda K. Early Lyme disease-associated Guillain Barre syndrome: a case report. *IDCases*. 2022;27:e01432.

Snydman DR, Hu L. Lyme disease. *Medicine*. 2021;49(12):747–750.

115

Avascular Necrosis of the Knee

ICD-10 CODE M87.059

THE CLINICAL SYNDROME

Avascular necrosis of the knee joint is an often missed diagnosis. Like the scaphoid, the knee joint is extremely susceptible to this disease because of the tenuous blood supply of the articular cartilage. This blood supply is easily disrupted, often leaving the proximal portion of the bone without nutrition, leading to osteonecrosis (Fig. 115.1). A disease of the fourth and fifth decades, with the exception of patients suffering from avascular necrosis of the knee joint secondary to collagen vascular disease, avascular necrosis of the knee joint is more common in men (Fig. 115.2). The disease is bilateral in 45%–50% of cases.

Predisposing factors to avascular necrosis of the knee joint are listed in Box 115.1. They include trauma to the joint, corticosteroid use, Cushing disease, alcohol abuse, connective tissue diseases especially systemic lupus erythematosus, osteomyelitis, human immunodeficiency virus infection, organ transplantation, hemoglobinopathies including sickle cell disease, hyperlipidemia, gout, renal failure, pregnancy, and radiation therapy involving the femoral head.

The patient with avascular necrosis of the knee joint complains of pain over the affected knee joint or knee joints that may radiate into the proximal lower extremity. The pain is deep and aching, and the patient often complains of a "catching" sensation with range of motion of the affected knee joint or knee joints. Range of motion decreases as the disease progresses.

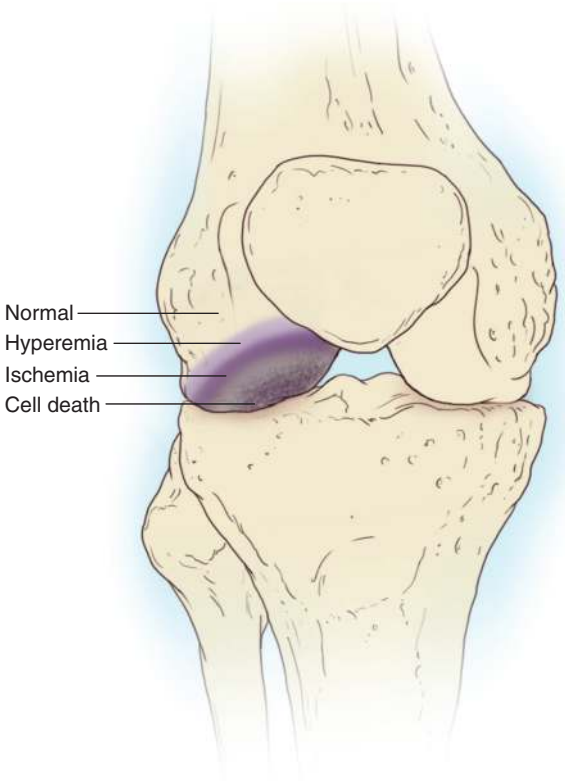

FIG 115.1 The pain of avascular necrosis of the knee joint is worsened by passive and active range of motion.

SIGNS AND SYMPTOMS

Physical examination of patients suffering from avascular necrosis of the knee joint reveals pain to deep palpation of the knee joint. The pain can worsen by passive and active range of motion. A click or crepitus may also be appreciated by the examiner when the knee joint is put through range of motion. The range of motion is invariably decreased.

TESTING

Plain radiographs are indicated in all patients who present with avascular necrosis of the knee joint, to rule out underlying occult bony disease and to identify sclerosis and fragmentation of the osseous support of the articular surface. Early in the course of the disease, however, plain radiographs can be notoriously unreliable, and magnetic resonance imaging (MRI) reveals articular changes before significant changes are evident on plain radiographs (Figs. 115.3–115.6). Based on the patient's clinical presentation, additional testing including complete blood cell count, uric acid, sedimentation rate, screening for coagulopathies, and antinuclear antibody testing may also be indicated. Sickle cell testing is indicated in those of African American descent. MRI of the knee joint is indicated in all patients suspected of suffering from avascular necrosis of the knee joint or if other causes of joint instability, infection, or tumor is suspected, or if the plain radiographs are nondiagnostic (see Fig. 115.5). Administration of gadolinium followed by postcontrast imaging may help delineate the adequacy of blood

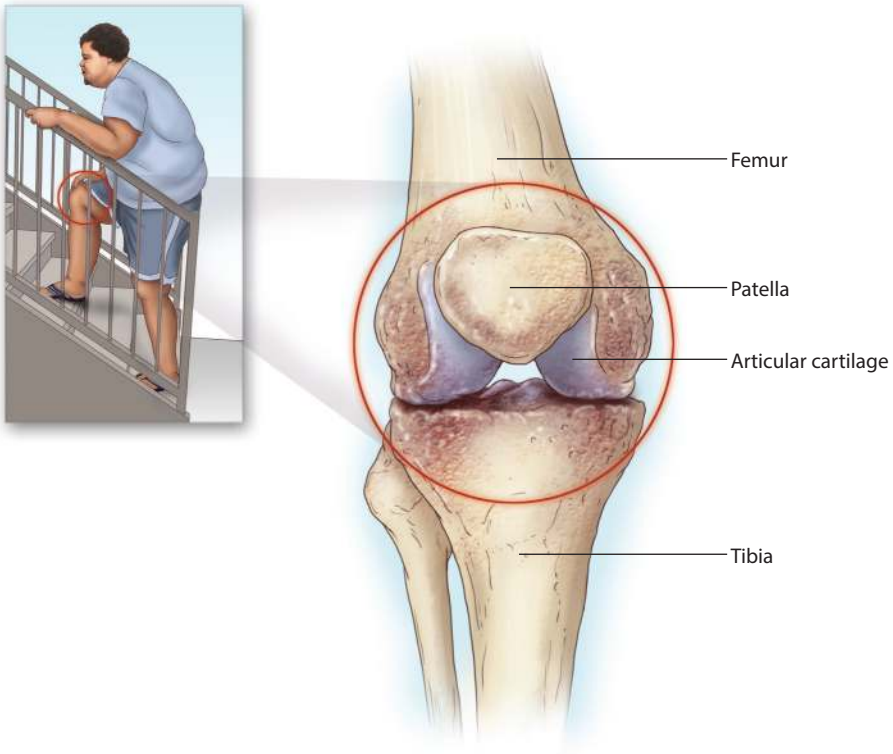

FIG 115.2 Physical examination of patients suffering from avascular necrosis of the knee joint reveals pain to deep palpation of the knee joint. The pain is worsened by passive and active range of motion, and a click or crepitus, as well as decreased range of motion, may also be present.

BOX 115.1 Predisposing Factors for Avascular Necrosis of the Knee Joint

- Trauma to the knee joint
- Steroids
- Cushing disease
- Alcohol abuse
- Connective tissue diseases, especially systemic lupus erythematosus
- Osteomyelitis
- Human immunodeficiency virus infection
- Organ transplantation
- Hemoglobinopathies, including sickle cell disease
- Hyperlipidemia
- Gout
- Renal failure
- Pregnancy
- Radiation therapy

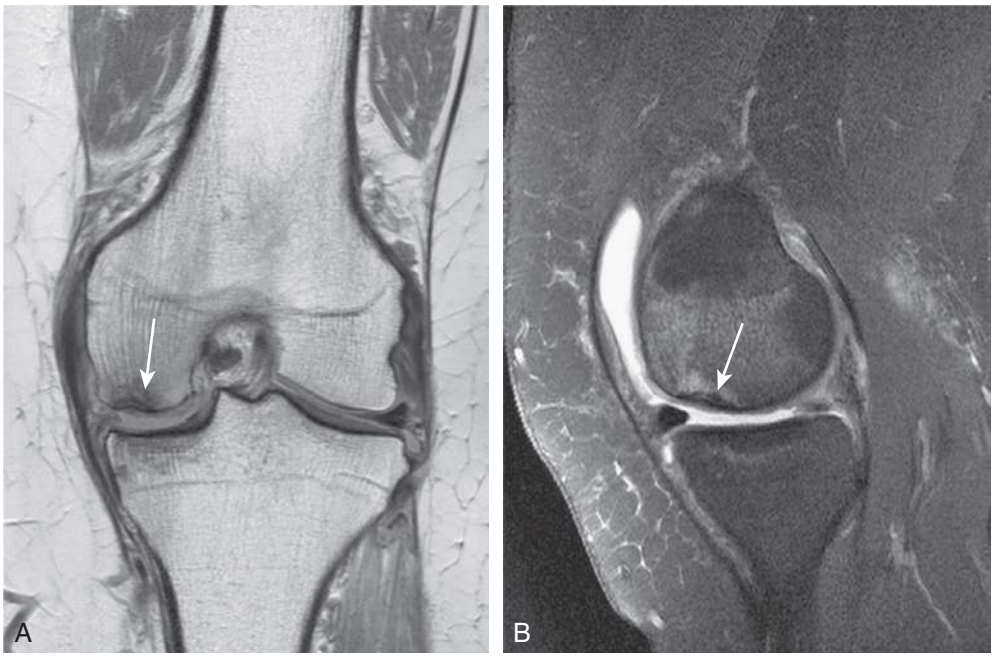

FIG 115.3 **A,** Anteroposterior (AP) radiograph of a 14-year-old patient with knee pain. There is apparent fragmentation of the lateral femoral condyle. **B,** The sagittal gradient echo magnetic resonance image demonstrates the osseous fragmentation (*white arrows*), but with intact overlying articular cartilage. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

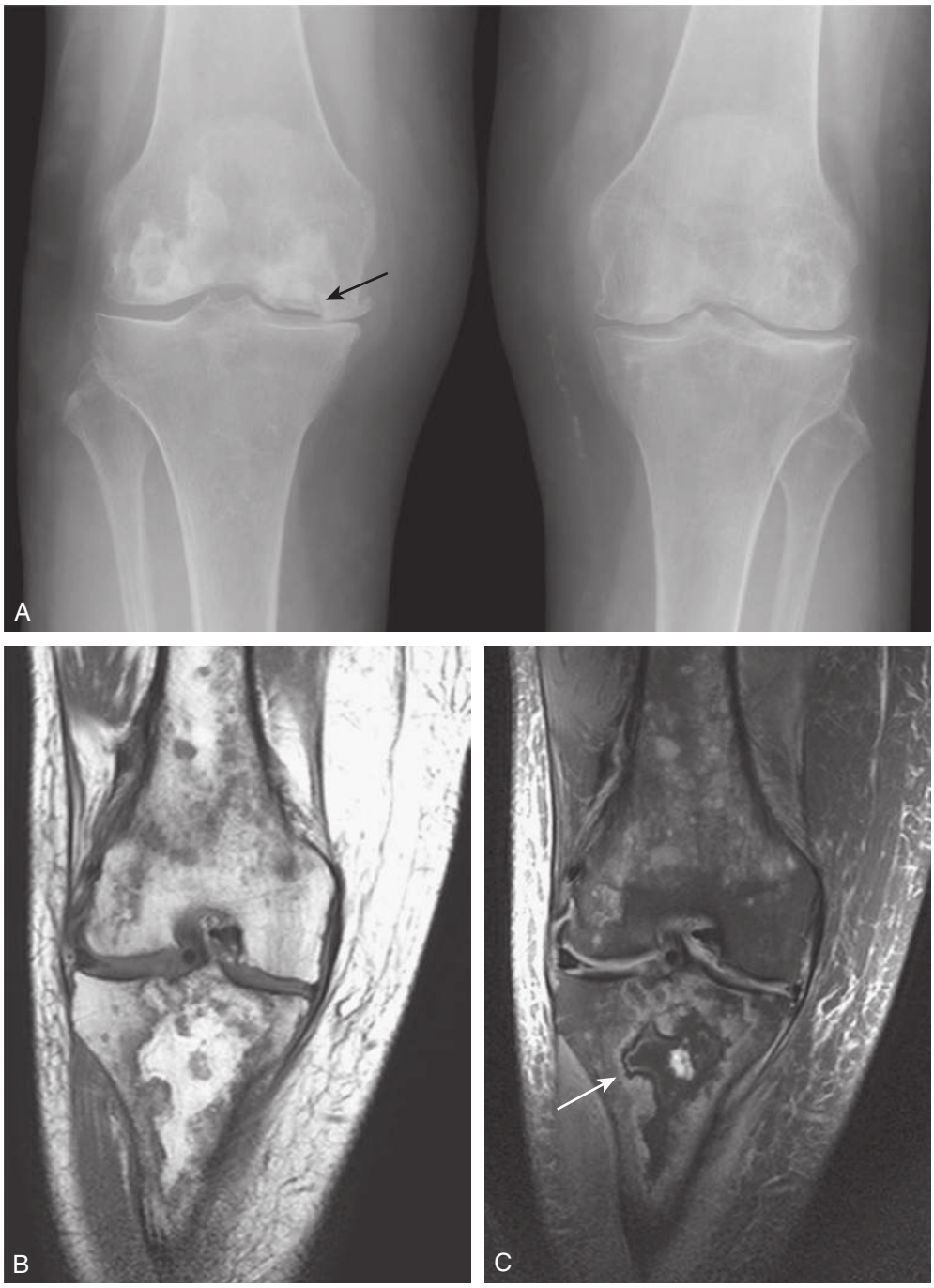

FIG 115.4 **A,** Anterioposterior radiograph of the knee demonstrating a focal area of osteochondritis dissecans (OCD) of the medical femoral condyle with a subchondral lucency (*black arrow*). **B,** The corresponding coronal proton density magnetic resonance image shows a osteochondral fragment with high signal intensity at the base of the lesion and a focal breach in the articular cartilage. **C,** T2-weighted image with fat suppression more clearly defines the image. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

supply; contrast enhancement of the knee joint is a good prognostic sign. Electromyography is indicated if coexistent cervical radiculopathy or brachial plexopathy is suspected. A very gentle intraarticular injection of the knee joint with small volumes of local anesthetic provides immediate reduction of the pain and helps demonstrate that the nidus of the patient's pain is in fact the knee joint. Ultimately, total joint replacement is required in most patients suffering from avascular necrosis of the knee joint, although newer joint preservation techniques are becoming more popular in younger, more active patients, given the short life expectancy of total knee prosthesis.

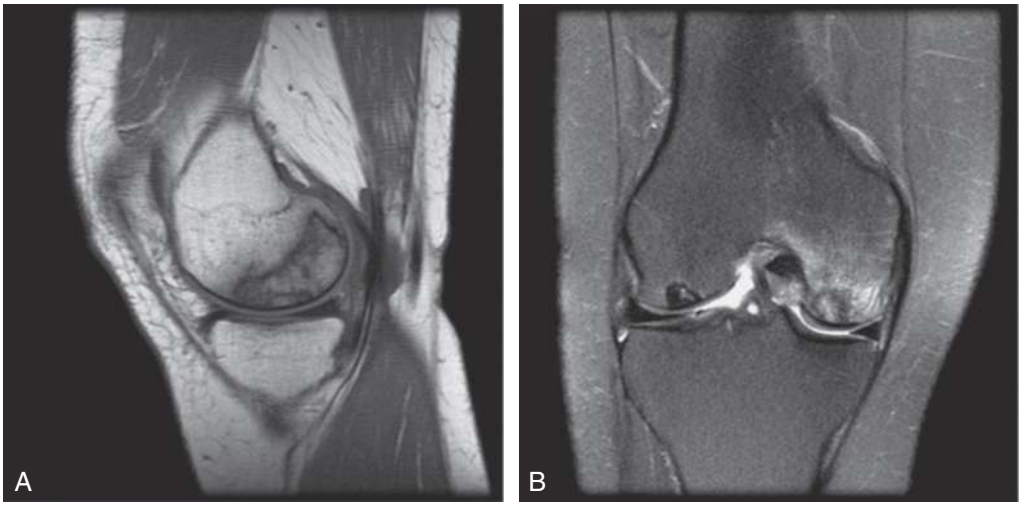

FIG 115.5 T1-weighted (A) and T2-weighted (B) magnetic resonance images taken 3 months before the plain radiograph in Fig. 115.3 show subchondral lesions in both the medial and lateral femoral condyle. (From Zywiel MG, Armocida FM, McGrath MS, et al. Bicondylar spontaneous osteonecrosis of the knee: a case report. *Knee*. 2010;17(2):167–171.)

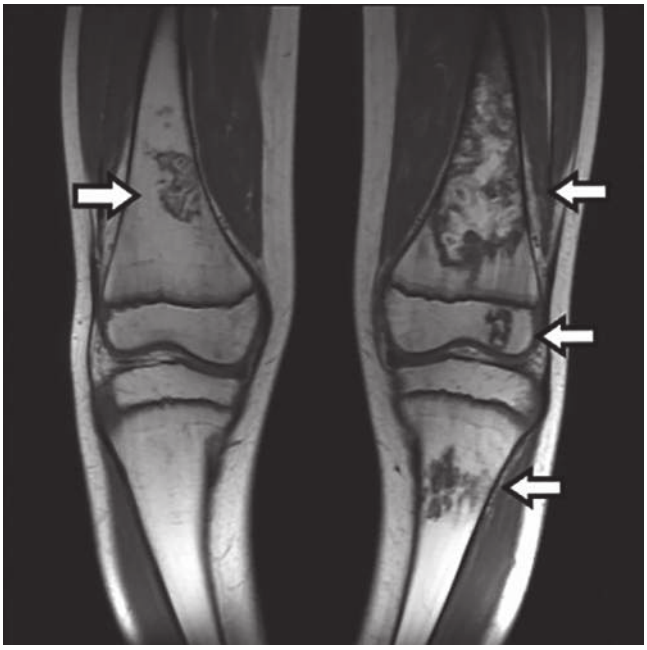

FIG 115.6 Magnetic resonance imaging showing bone infarction with "map"-like appearance of bilateral femurs and tibias in an adolescent who underwent high dose steroid therapy for malignancy (*arrows*; coronal view of T1-weighted images). (From Huang TH, Liu HC, Yeh TC, Hou JY, Lin CY. Osteonecrosis (avascular necrosis) of knee and tibia. *J Pediatr*. 2020;217:210–210.e1.)

DIFFERENTIAL DIAGNOSIS

Coexistent arthritis and gout of the knee joints, bursitis, and tendinitis may also coexist with avascular necrosis of the knee joints and exacerbate the pain and disability of the patient. Tears of the ligaments, bone cysts, bone contusions, and fractures may also mimic the pain of avascular necrosis of the knee joint, as can occult metastatic disease.

TREATMENT

Initial treatment of the pain and functional disability associated with avascular necrosis of the knee joint should include a combination of the nonsteroidal antiinflammatory agents (NSAIDs) or cyclooxygenase-2 inhibitors and decreased weight bearing of the affected knee joint or knee joints. Treatment of underlying diseases responsible for the osteonecrosis, such as lipid-lowering agents, should be initiated. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, an injection of a local anesthetic into the knee joint may be a reasonable next step to provide palliation of acute pain. Clinical experience suggests that pulsed electromagnetic field therapy may provide symptomatic relief of knee pain associated with avascular necrosis of the knee. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Ultimately, surgical repair in the form of total joint arthroplasty is the treatment of choice.

COMPLICATIONS AND PITFALLS

Failure to treat significant avascular necrosis of the knee joint surgically usually results in continued pain and disability and usually leads to ongoing damage to the knee joint (see Fig. 115.3). Injection of the joint with local anesthetic is a relatively safe technique if the clinician is attentive to detail, specifically uses small amounts of local anesthetic, and avoids high injection pressures that may further damage the joint. Another complication of this injection technique is infection. This complication should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after this injection technique, and patients should be warned of this possibility.

CLINICAL PEARLS

Avascular necrosis of the knee joint is a diagnosis that is often missed, thus leading to much unnecessary pain and disability. The clinician should include avascular necrosis of the knee joint in the differential diagnosis in all patients complaining of knee joint pain, especially if any of the predisposing factors listed in Box 115.1 are present. Coexistent arthritis, tendinitis, and gout may also contribute to the pain and may require additional treatment. The use of physical modalities, including local heat and cold, as well as decreased weight bearing, may provide symptomatic relief. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms and may cause further damage to the knee. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

SUGGESTED READINGS

Huang TH, Liu HC, Yeh TC, Hou JY, Lin CY. Osteonecrosis (avascular necrosis) of knee and tibia. *J Pediatr*. 2020;217, 210–210.e1. https://doi.org/10.1016/j.jpeds.2019.10.009.

Jordan RW, Aparajit P, Docker C, et al. The importance of early diagnosis in spontaneous osteonecrosis of the knee—a case series with six year follow-up. *Knee*. 2016;23(4):702–707.

Lieberman JR, Varthi AG, Polkowski GG II. Osteonecrosis of the knee—which joint preservation procedures work? *J Arthroplast*. 2014;29(1):52–56.

Muccioli M, Grassi A, Setti S, et al. Conservative treatment of spontaneous osteonecrosis of the knee in the early stage: pulsed electromagnetic fields therapy. *Eur J Radiol*. 2013;82(3):530–537.

Waldman SD, Campbell RSD. Osteonecrosis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:393–395.

Medial Collateral Ligament Syndrome

ICD-10 CODE M23.50

THE CLINICAL SYNDROME

Medial collateral ligament syndrome is characterized by pain at the medial aspect of the knee joint. This syndrome is usually the result of trauma to the medial collateral ligament from falls with the leg in valgus and externally rotated, typically during snow skiing accidents or football clipping injuries (Fig. 116.1). The medial collateral ligament, which is also known as the tibial collateral ligament, is a broad, flat, band-like ligament that runs from the medial condyle of the femur to the medial aspect of the shaft of the tibia, where it attaches just above the groove where the semimembranosus muscle attaches (Fig. 116.2). It also attaches to the edge of the medial semilunar cartilage. The ligament is susceptible to strain at the joint line or avulsion at its origin or insertion.

SIGNS AND SYMPTOMS

Patients with medial collateral ligament syndrome present with pain over the medial joint and increased pain on passive valgus and external rotation of the knee. Activity, especially flexion and external rotation of the knee, worsens the pain, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Patients with injury to the medial collateral ligament may complain of locking or popping with flexion of the affected knee. Coexistent bursitis, tendinitis, arthritis, or internal derangement of the knee may confuse the clinical picture after trauma to the knee joint.

On physical examination, patients with injury to the medial collateral ligament exhibit tenderness along the course of the ligament from the medial femoral condyle to its tibial insertion. If the ligament is avulsed from its bony insertions, tenderness may be localized to the proximal or distal ligament, whereas patients suffering from strain of the ligament have more diffuse tenderness. Patients with severe injury to the ligament may exhibit joint laxity when valgus and varus stress is placed on the affected knee (Fig. 116.3). The Swain test for medial collateral ligament injury may also be positive with significant injuries to the

FIG 116.1 Medial collateral ligament syndrome is characterized by medial joint pain that is made worse with flexion or external rotation of the knee.

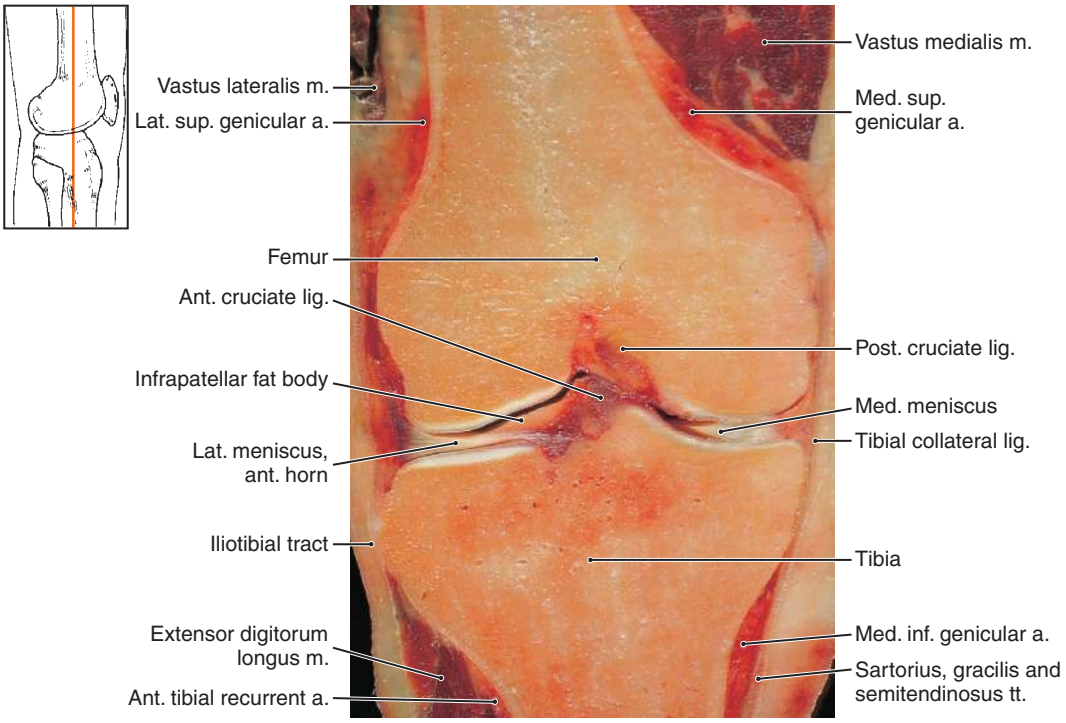

FIG 116.2 The medial collateral ligament is also known as the tibial collateral ligament. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas*. 2nd ed. Philadelphia: Saunders; 2002.)

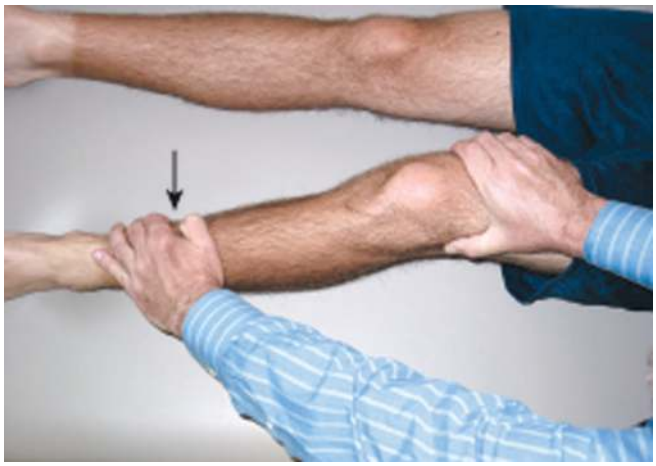

FIG 116.3 The valgus stress test for medial collateral ligament integrity. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:291.)

ligament (Fig. 116.4). Because pain may produce muscle guarding, magnetic resonance imaging (MRI) of the knee may be necessary to confirm the clinical impression. Joint effusion and swelling may be present with injury to the medial collateral ligament, but these findings are also suggestive of intraarticular damage. Again, MRI can confirm the diagnosis.

TESTING

MRI and ultrasound imaging are indicated in all patients who present with medial collateral ligament pain, particularly if internal derangement or an occult mass or tumor is suspected (Figs. 116.5–116.7). In addition, MRI and ultrasound imaging should be performed in all patients with injury to the medial collateral ligament who fail to respond to conservative therapy or who exhibit joint instability on clinical examination (Fig. 116.8). Bone scan may be useful to identify occult stress fractures involving the joint, especially if trauma has occurred. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Any condition affecting the medial compartment of the knee joint may mimic the pain of medial collateral ligament syndrome. Bursitis, arthritis, and entrapment neuropathies may also confuse the diagnosis, as may primary tumors of the knee and spine.

TREATMENT

Initial treatment of the pain and functional disability associated with injury to the medial collateral ligament includes a combination of nonsteroidal antiinflammatory drugs or

FIG 116.4 The Swain test for medial collateral ligament (MCL) injury. With the knee flexed to 90 degrees, the tibia is externally rotated when the knee is externally rotating in flexion, the collateral ligaments are tightened while the cruciates are relatively lax. Pain along the medial side of the joint indicates injury to the MCL complex. Many patients with chronic medial-sided laxity after injury have pain along the medial joint line with this maneuver. Pain along the medial side of the joint indicates medial collateral ligament complex injury.

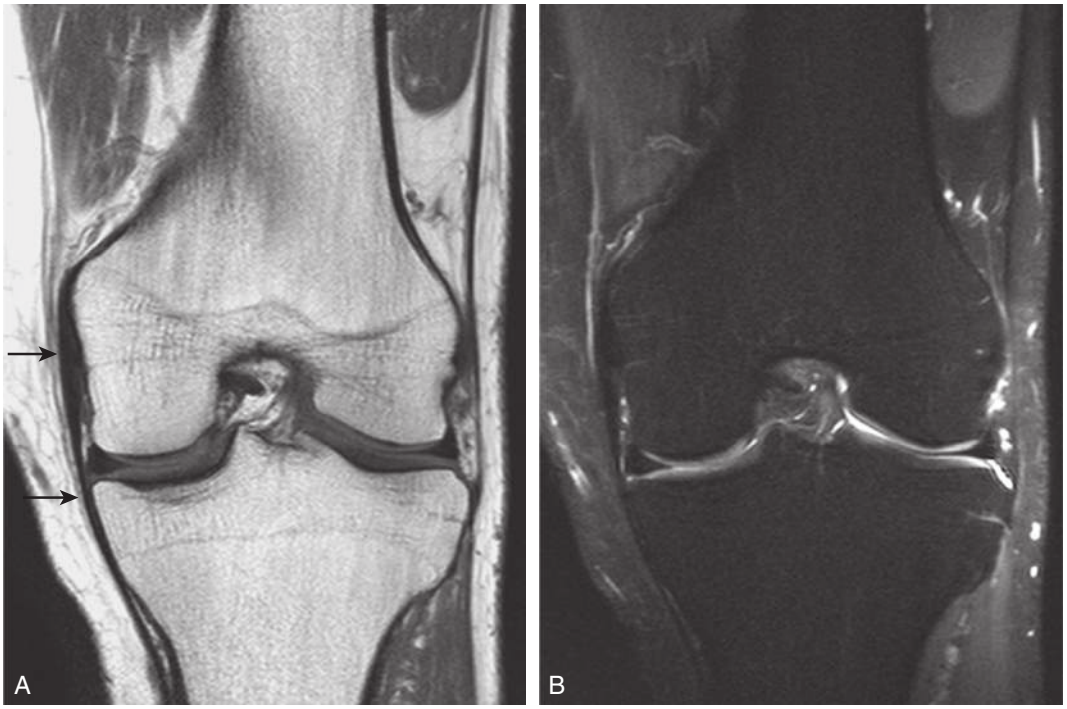

FIG 116.5 A, Coronal proton density magnetic resonance (MR) image of a normal low signal intensity (SI) medial collateral ligament (MCL) (*black arrows*), extending from the medial epicondyle of the femur to the proximal tibial metaphysis. B, The fat-suppressed T2-weighted MR image also shows the low-SI MCL. The adjacent soft tissues are normal, with no soft tissue edema. (From Waldman SD, Campbell RSD. *Imaging of pain.* Philadelphia: Elsevier; 2011.)

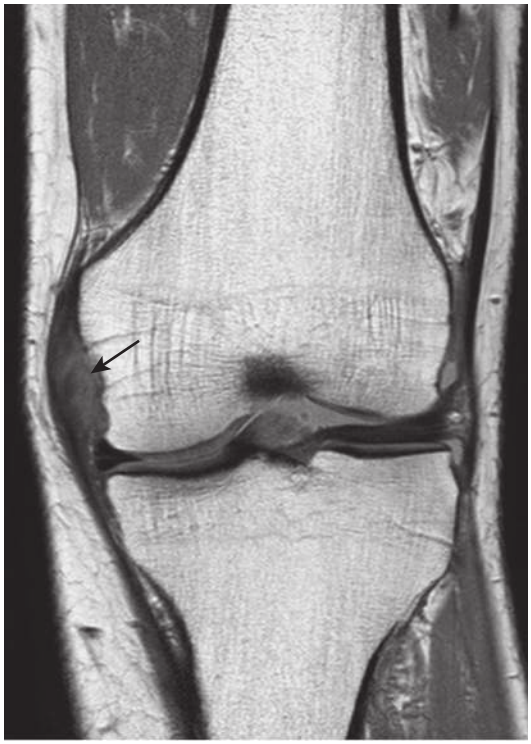

FIG 116.6 Subacute partial tear of the proximal medial collateral ligament (*black arrow*), with thickening and increased signal intensity (SI) of the deep fibers of the ligament on a coronal proton density magnetic resonance image. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

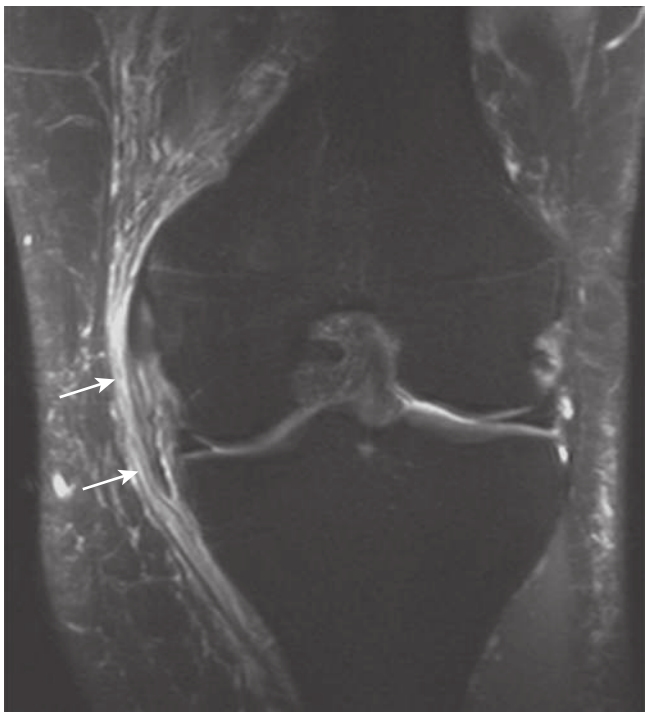

FIG 116.7 Coronal fat-suppressed T2-weighted magnetic resonance image of an acute grade II tear of the medial collateral ligament with poorly defined ligament fibers and surrounding soft tissue edema (*white arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

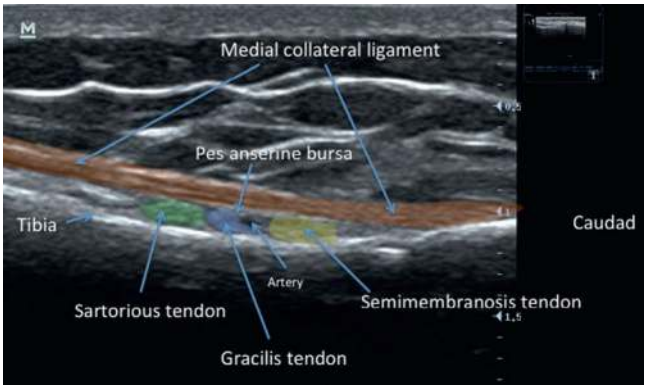

FIG 116.8 Longitudinal ultrasound image of the medial knee joint demonstrating the pes anserine bursa lying beneath the pes anserine tendon.

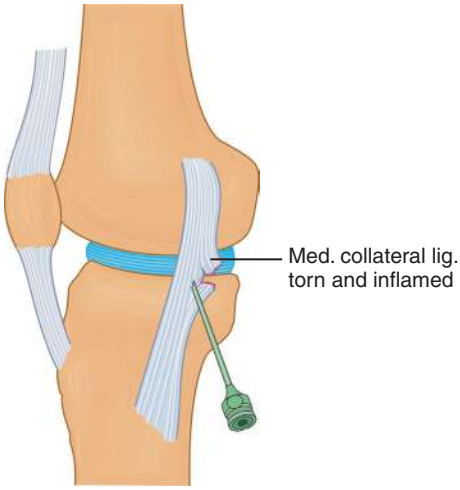

FIG 116.9 Injection technique for medial collateral ligament syndrome. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007.)

cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Any repetitive activity that exacerbates the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities and who do not have lesions that require surgical repair, injection is a reasonable next step.

Injection of the medial collateral ligament is carried out with the patient in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the lateral aspect of the knee joint is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The most tender portion of the ligament is identified, and the needle is inserted at this point at a 45-degree angle to the skin. The needle is carefully advanced through the skin and subcutaneous tissues into proximity with the medial collateral ligament (Fig. 116.9). If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. The contents of the

syringe are then gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-induced complications. The injection of platelet-rich plasma and/or stem cells may provide symptomatic relief in patients with medical collateral ligament injuries.

COMPLICATIONS AND PITFALLS

The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection of the medial collateral ligament, and patients should be warned of this possibility.

CLINICAL PEARLS

Patients with injury to the medial collateral ligament are best examined with the knee in the slightly flexed position. The clinician may want to examine the nonpainful knee first to reduce the patient's anxiety and to ascertain the findings of a normal examination. The injection technique described is extremely effective in the treatment of pain secondary to medial collateral ligament syndrome. Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Dale KM, Bailey JR, Moorman CT. Surgical management and treatment of the anterior cruciate ligament/medial collateral ligament injured knee. *Clin Sports Med*. 2017;36(1):87–103. https://doi.org/10.1016/j.csm.2016.08.005.

Geeslin AG, Lemos DF, Geeslin MG. Knee ligament imaging: preoperative and postoperative evaluation. *Clin Sports Med*. 2021;40(4):657–675. https://doi.org/10.1016/j.csm.2021.05.005.

Kim C, Chasse PM, Taylor DC. Return to play after medial collateral ligament injury. *Clin Sports Med*. 2016;35(4):679–696.

Memarzadeh A, Melton JTK. Medial collateral ligament of the knee: anatomy, management and surgical techniques for reconstruction. *Orthopaed Trauma*. 2019;33(2):91–99. https://doi.org/10.1016/j.mporth.2019.01.004.

Smidt K, Dubose M, Shahrdar C. Traumatic lateral knee dislocation of a well-functioning total knee arthroplasty: a case of medial collateral ligament rupture. *Arthroplast Today*. 2021;11:168–172. https://doi.org/10.1016/j.artd.2021.08.012.

Waldman SD. Abnormalities of the medial collateral ligament. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:754–760.

Waldman SD. Medical collateral ligament injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:687–690.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the knee. In: *Imaging of pain*. Philadelphia: Saunders; 2011:267–269.

Waldman SD, Campbell RSD. Medial collateral ligament tear. In: *Imaging of pain*. Philadelphia: Saunders; 2011:381–383.

117

Medial Meniscal Tear

ICD-10 CODE M23.219

THE CLINICAL SYNDROME

The meniscus is a unique anatomic structure that fulfills various functions to allow ambulation in the upright position (Box 117.1). The meniscus is susceptible to both acute injury from trauma and degenerative tears, which are more chronic. Tears of the medial meniscus are classified by their orientation and shape (Box 117.2).

Acute medial meniscal injury is the most commonly encountered cause of significant knee pain secondary to trauma that is seen in clinical practice. The incidence of acute tear is approximately 60 cases per 100,000 individuals. Acute tears often result from sudden twisting or squatting with weight bearing (Fig. 117.1). The male predominance is more than 2:1. Acute tears often result in pain and fuctional disability for the patient.

A medial meniscal tear is characterized by pain at the medial aspect of the knee joint line. The medial meniscus is a triangular structure on cross-section that is approximately

BOX 117.1 Functions of the Medial Meniscus

- Load bearing
- Conversion of compressive forces to tensile forces
- Load distribution
- Stabilization of the joint
- Lubrication of the joint
- Proprioception

BOX 117.2 Classification of Medial Meniscus Tears

- Longitudinal
- Bucket handle
- Parrot beak–shaped oblique
- Horizontal
- Radial
- Complex combination

FIG 117.1 Sagittal proton density **(A)** and T2W with fat-saturated T2-weighted (FST2W) **(B)** magnetic resonance images of the medial meniscus. There is linear increased signal intensity (SI) within the posterior horn of the meniscus (*white arrows*) that does not extend to the meniscal surface and is not representative of a meniscal tear. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

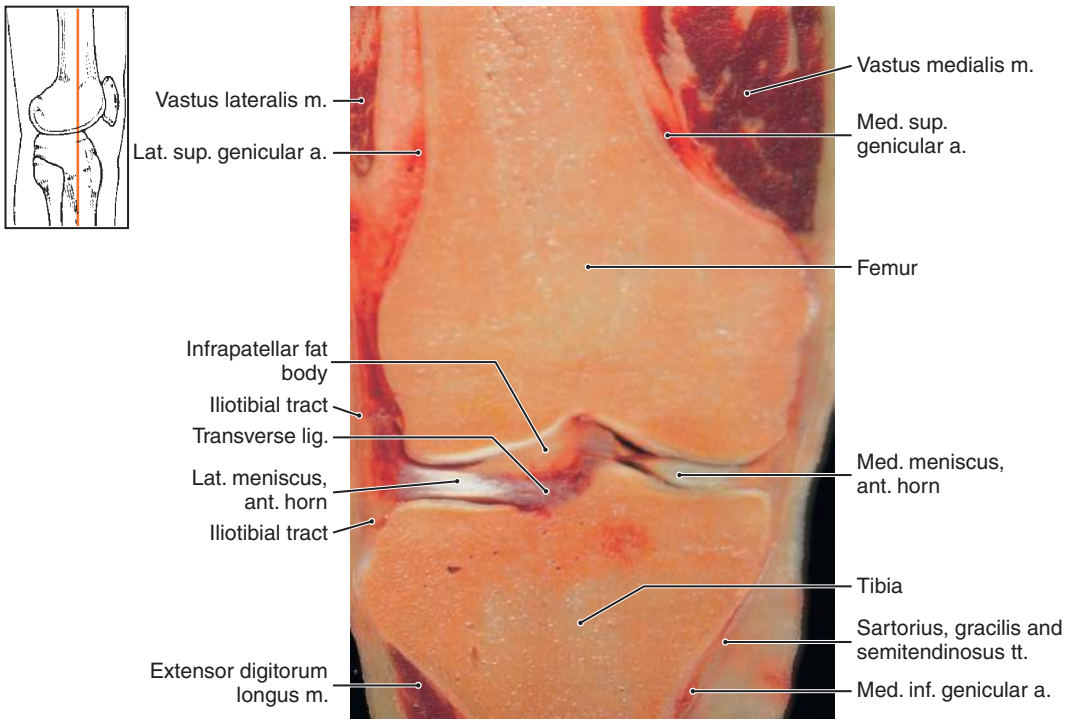

FIG 117.2 The medial meniscus is subject to degenerative changes, as well as tearing secondary to acute trauma. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas.* 2nd ed. Philadelphia: Saunders; 2002:305.)

3.5 cm in length from anterior to posterior (Fig. 117.2). This structure is wider posteriorly and is attached to the tibia by the coronary ligaments, which are also susceptible to trauma, as are the fibrous connections from the joint capsule and the medial collateral ligament.

SIGNS AND SYMPTOMS

Patients with medial meniscal tear present with pain over the medial joint space and increased pain on the McMurray, squat, and Apley grinding tests (Fig. 117.3). Activity, especially flexion and external rotation of the knee, worsens the pain, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Patients with injury to the medial meniscus may complain of locking or popping with flexion of the affected knee. An effusion is often present and can be quite pronounced in some patients. Coexistent bursitis, tendinitis, arthritis, or other internal derangements of the knee may confuse the clinical picture after trauma to the knee joint.

On physical examination, patients with injury to the medial collateral ligament exhibit tenderness along the medial joint line. Patients with tear of the medial meniscus may exhibit a positive McMurray, squat, and Apley test result. Because pain may produce muscle guarding that makes accurate joint examination difficult, magnetic resonance imaging (MRI) of the knee may be necessary to confirm the clinical impression.

TESTING

Plain radiographs, ultrasound imaging, and MRI are indicated in all patients who present with knee pain, particularly if internal derangement or an occult mass or tumor is suspected (Fig. 117.4). In addition, MRI and ultrasound imaging should be performed in all patients with injury to the medial meniscus who fail to respond to conservative therapy or who exhibit joint instability on clinical examination (Figs. 117.5–117.8). Bone scan may be useful to identify occult stress fractures involving the joint, especially if trauma has occurred. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing. Arthroscopy of the affected joint may serve as both a diagnostic and therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Any condition affecting the medial compartment of the knee joint may mimic the pain of medial meniscal tear (Table 117.1). Bursitis, arthritis, and entrapment neuropathies may also confuse the diagnosis, as may primary tumors of the knee and spine.

TREATMENT

Initial treatment of the pain and functional disability associated with injury to the medial collateral ligament includes a combination of nonsteroidal antiinflammatory drugs or

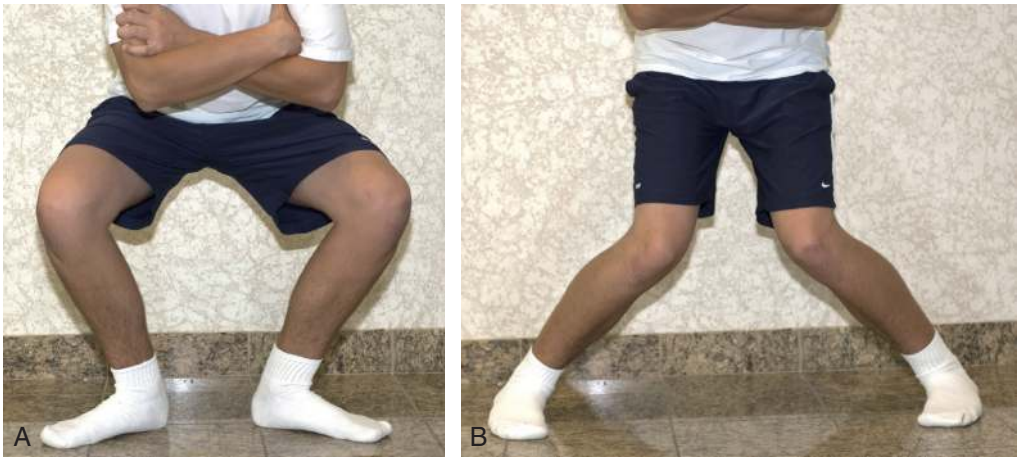

FIG 117.3 The squat test for meniscal tear. **A,** The patient is asked first to perform a full squat with the feet and legs fully externally rotated. **B,** The patient is then asked to perform a full squat with the feet and legs fully internally rotated. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:308.)

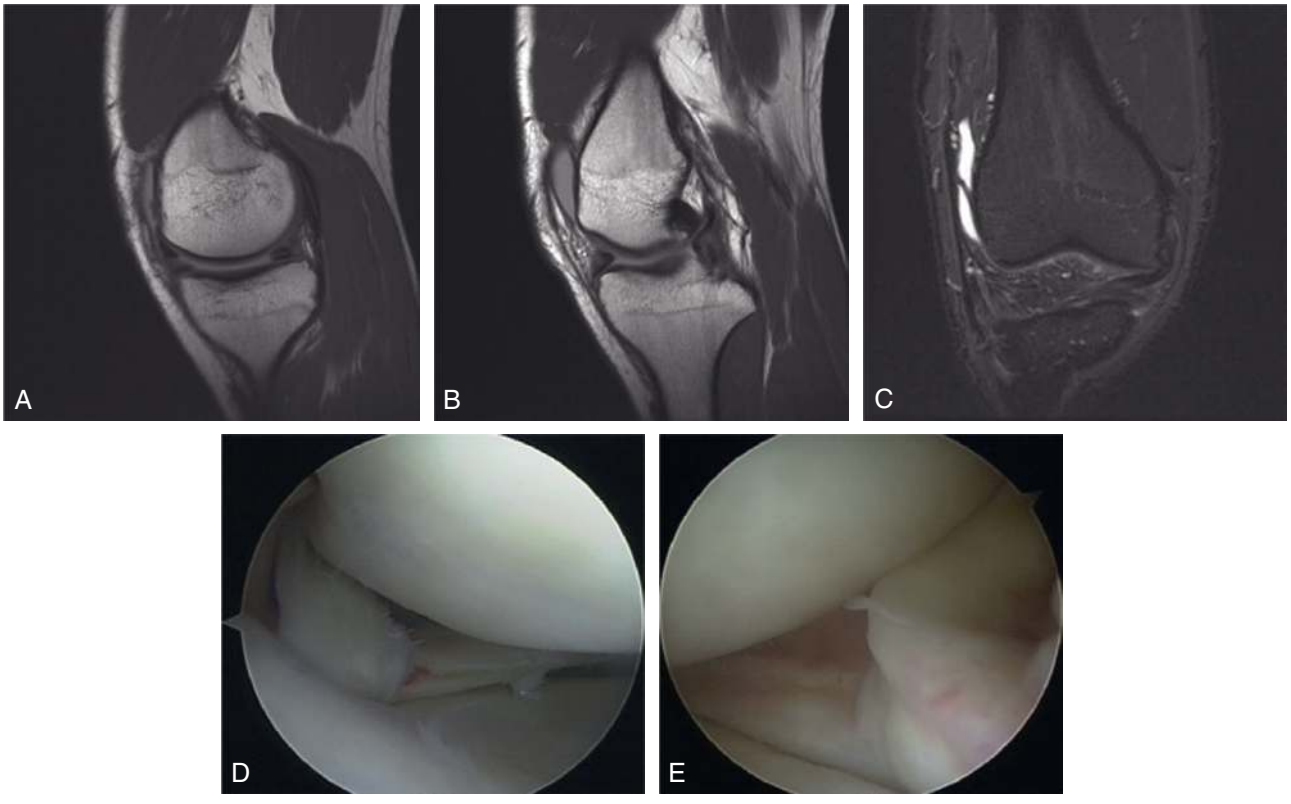

FIG 117.4 A–C, Sagittal proton density magnetic resonance imaging (MRI) demonstrating a horizontal tear of the medial meniscal body with an anterior horn fragment displaced anterior to the medial femoral condyle **(A)** and a posterior horn fragment displaced into the intercondylar notch adjacent to the posterior cruciate ligament **(B)**. Both displaced fragments were missed on MRI. **C,** Coronal short tau inversion recovery MRI also demonstrating the displaced anterior horn meniscal fragment mentioned in **A**. This fragment was palpable to the patient, who brought it to the attention of the surgeon before arthroscopy. **D** and **E,** Arthroscopic images of the same case demonstrating the horizontal tear of the meniscal body and the displaced posterior horn **(D)** and anterior horn fragments **(E)**. (From Sampson MJ, Jackson MP, Moran CJ, et al. Three Tesla MRI for the diagnosis of meniscal and anterior cruciate ligament pathology: a comparison to arthroscopic findings. *Clin Radiol*. 2008;63(10):1106–1111.)

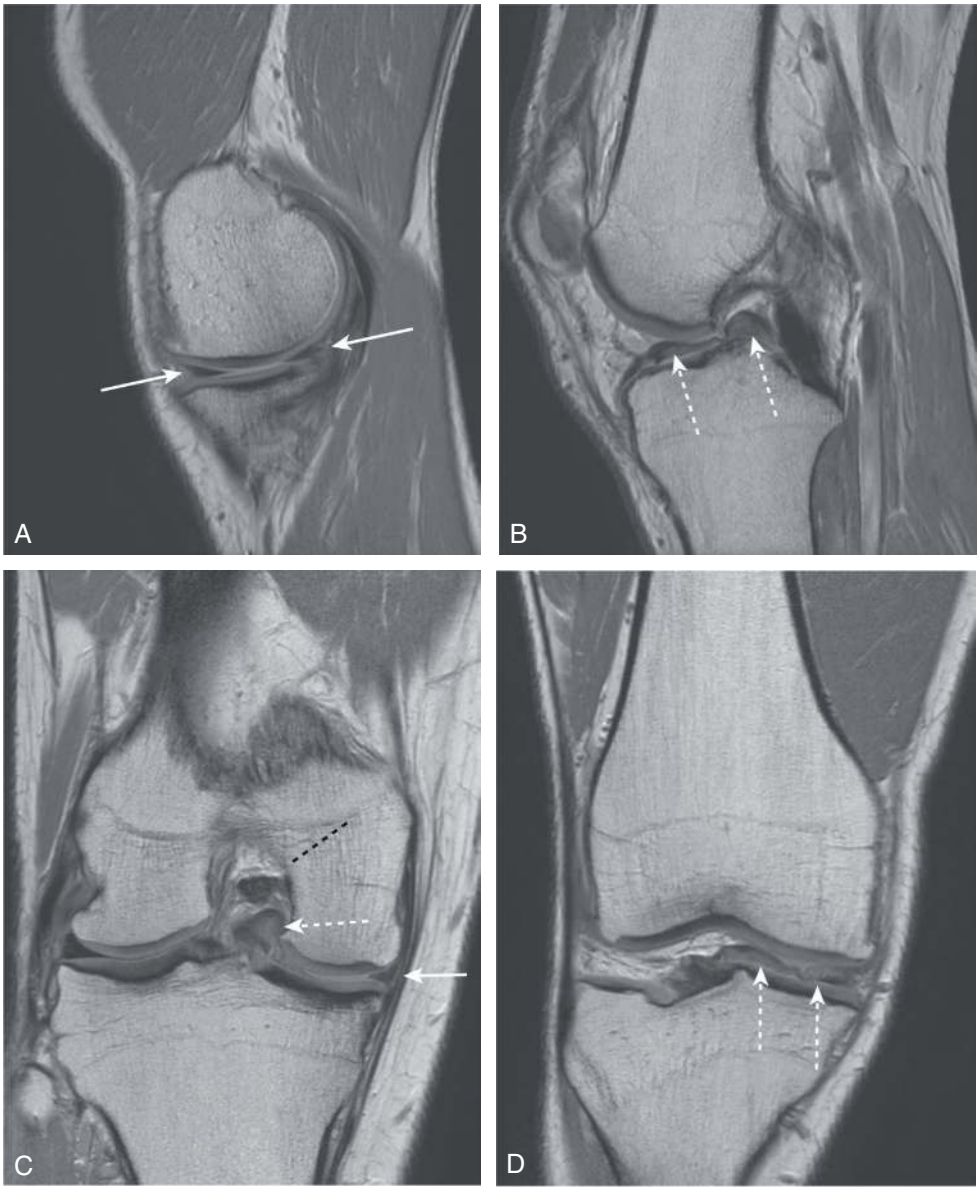

FIG 117.5 Magnetic resonance imaging (MRI) reveals a bucket handle tear of the medial meniscus. **A,** The parasagittal proton density MRI indicates a small meniscus (*white arrows*). **B,** The sagittal proton density MRI through the level of the intercondylar notch shows a displaced bucket handle fragment of the medial meniscus (*broken white arrows*) lying inferior and anterior to the posterior collateral ligament. **C,** Full thickness tear of the supraspinatus tendon as well as **(D)** more chronic tendinopathy of the infraspinatus tendon as evidenced by thickening and high signal intensity of the tendon. (From Waldman S, Campbell R. *Imaging of pain*. Philadelphia: Saunders; 2011.)

cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Any repetitive activity that exacerbates the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities and who do not have lesions that require surgical repair, injection is a reasonable next step.

Injection of the medial meniscus is carried out by injecting the intraarticular space of the affected knee with local anesthetic and steroid. To perform intraarticular injection of the knee, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the medial joint is prepared with antiseptic solution. A sterile syringe containing 5 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The joint space is identified, and the clinician places his or her thumb on the lateral margin of the patella and pushes it medially. At a point in the middle of the medial edge of the patella, the needle is inserted between the patella and the femoral condyles. The needle is then

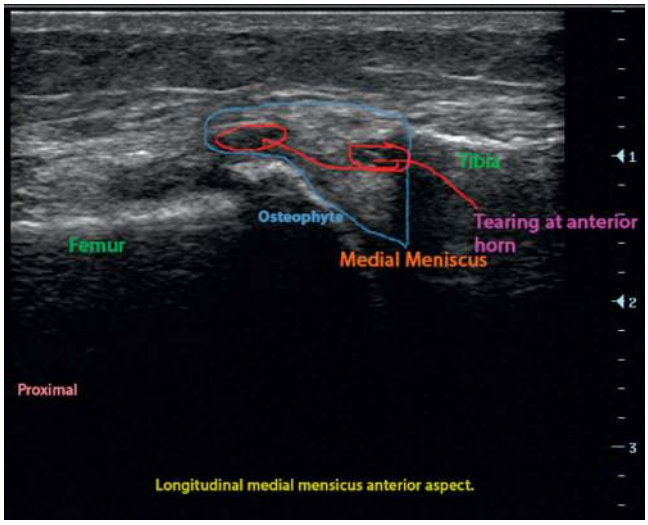

FIG 117.6 Longitudinal ultrasound image demonstrating tearing of the anterior horn of the medial meniscus.

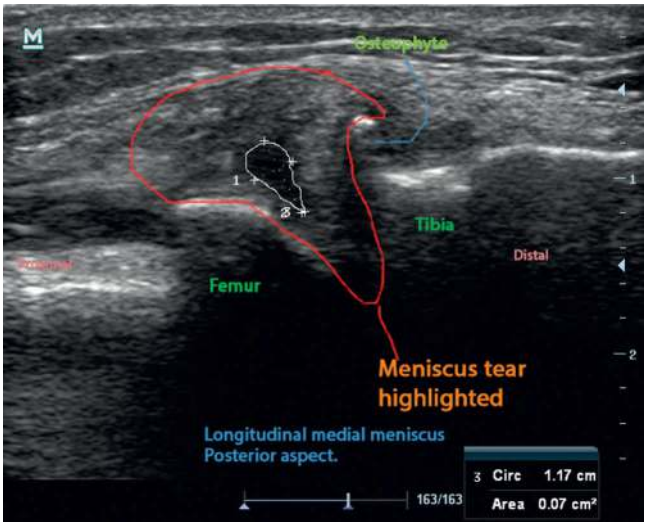

FIG 117.7 Longitudinal ultrasound image demonstrating a flap tear of the medial meniscus.

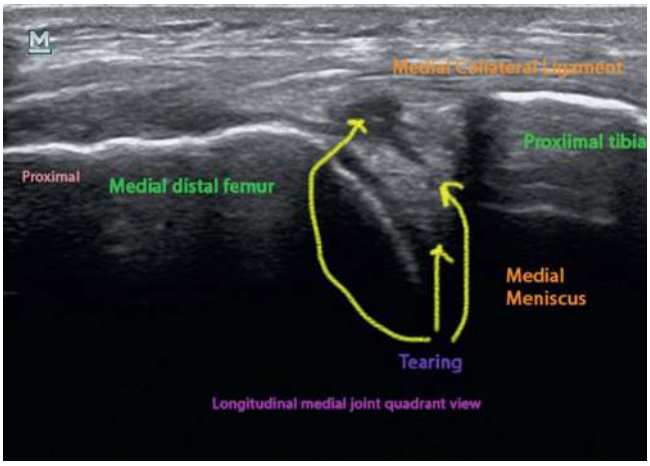

FIG 117.8 Longitudinal ultrasound image revealing complex tearing of the medial meniscus. (Courtesy Steven Waldman, MD.)

TABLE 117.1 Most Common Causes of Knee Pain

Localized Bony or Joint Space Pathology	Periarticular Pathology	Systemic Disease	Sympathetically Mediated Pain	Referred From Other Body Areas
Fracture	Bursitis	Rheumatoid arthritis	Causalgia	Lumbar plexopathy
Primary bone tumor	Tendinitis	Collagen vascular disease	Reflex sympathetic dystrophy	Lumbar radiculopathy
Primary synovial tissue tumor	Adhesive capsulitis	Reiter syndrome		Lumbar spondylosis
Joint instability	Joint instability	Gout		Fibromyalgia
Localized arthritis	Muscle strain	Other crystal arthropathies		Myofascial pain syndromes
Osteophyte formation	Muscle sprain	Charcot neuropathic arthritis		Inguinal hernia
Joint space infection	Periarticular infection not involving joint space			Entrapment neuropathies
Hemarthrosis				Intrapelvic tumors
Villonodular synovitis				Retroperitoneal tumors
Intraarticular foreign body				
Osgood-Schlatter disease				
Chronic dislocation of the patella				
Patellofemoral pain syndrome				
Patella alta				

From Waldman SD. *An overview of painful conditions of the knee.* In: *Physical diagnosis of pain.* 4th ed. Philadelphia: Elsevier; 2021: chap 230, 351–351.

carefully advanced through the skin and subcutaneous tissues through the joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. Clinical experience suggests that the injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of medial meniscal dysfunction.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection of the medial collateral ligament, and patients should be warned of this possibility.

CLINICAL PEARLS

Patients with injury to the medial meniscus collateral ligament are best examined with the knee in the slightly flexed position. The clinician may want to examine the nonpainful knee first to reduce the patient's anxiety and to ascertain the findings of a normal examination. The injection technique described is extremely effective in the treatment of pain secondary to medial meniscal tear. Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Allen H, Chan BY, Davis KW, et al. Overuse injuries of the knee. *Radiol Clin North Am*. 2019;58(5):898909.

Cibulas A, Leyva A, Cibulas G, et al. Acute knee injury. *Radiol Clin North Am*. 2019;58(5):883898.

Englund M, Haugen IK, Guermazi A, et al. Evidence that meniscus damage may be a component of osteoarthritis: the Framingham study. *Osteoarthr Cartil*. 2016;24(2):270–273.

Gimber LH, Hardy JC, Melville DM, et al. Normal magnetic resonance imaging anatomy of the capsular ligamentous supporting structures of the knee. *Can Assoc Radiol J*. 2016;67(4):356–367.

Netter FH. Knee (glenohumeral joint). In: *Atlas of human anatomy*. 4th ed. Philadelphia: Saunders; 2008.

Salati U, Doody O, Munk PL, et al. Evaluation of knee pain in athletes: a radiologist's perspective. *Can Assoc Radiol J*. 2017;68(1):27–40.

Waldman SD. Clinical correlates: diseases of the medial meniscus. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:87–92.

Waldman SD. Medial meniscus disease. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:186–195.

Waldman SD. The Apley grinding test for meniscal tear. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:328.

Waldman SD. The McMurray test for torn meniscus. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:327.

Waldman SD. The squat test for meniscal tear. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:329.

Waldman SD. Arthritis pain of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:304–305.

Waldman SD. *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Saunders; 2018:94–98.

Waldman SD. Clinical correlates: functional anatomy of the knee. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Saunders; 2018.

Waldman SD. Intra-articular injection of the knee. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:651–656.

Waldman SD, Campbell RSD. Anatomy: meniscal degeneration of the knee. In: *Imaging of pain*. Philadelphia: Saunders; 2011:367–388.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the knee. In: *Imaging of pain*. Philadelphia: Saunders; 2011:367–388.

118

Anterior Cruciate Ligament Syndrome

ICD-10 CODE S83.509A

THE CLINICAL SYNDROME

Anterior cruciate ligament syndrome is characterized by pain in the anterior aspect of the knee joint. It is usually the result of trauma to the anterior cruciate ligament from sudden deceleration secondary to planting of the affected lower extremity while extreme twisting or hyperextension forces are placed on the knee, typically during snow skiing accidents, football, and basketball injuries (Fig. 118.1). Unlike many other painful knee syndromes, anterior cruciate ligament syndrome occurs significantly more frequently in female patients.

The anterior cruciate ligament controls the amount of anterior movement or translation of the tibia relative to the femur, as well as providing important proprioceptive information regarding the position of the knee. This ligament is made up of dense fibroblastic fibers that run from the posteromedial surface of the lateral condyle of the distal femur through the intercondylar notch to the anterior surface of the tibia (Fig. 118.2). The anterior cruciate ligament is innervated by the posterior branch of the posterior tibial nerve. The ligament is susceptible to sprain or partial or complete tear.

SIGNS AND SYMPTOMS

Patients with anterior cruciate ligament syndrome present with pain over the anterior knee joint and increased pain on passive valgus stress and range of motion of the knee. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Patients with injury to the anterior cruciate ligament may complain of a sudden popping of the affected knee at the time of acute injury, as well as a sensation that the knee wants to give way or slip

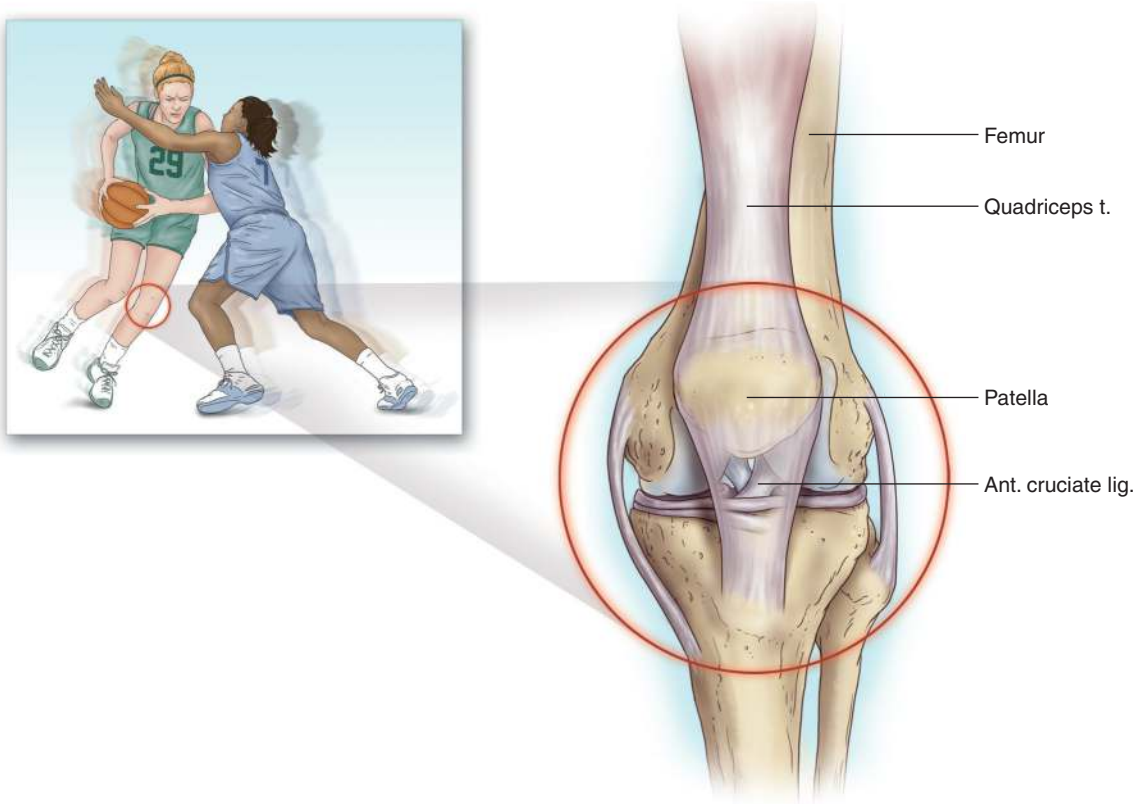

FIG 118.1 Patients with anterior cruciate ligament syndrome present with anterior knee pain and pain that increases with valgus stress applied to the knee.

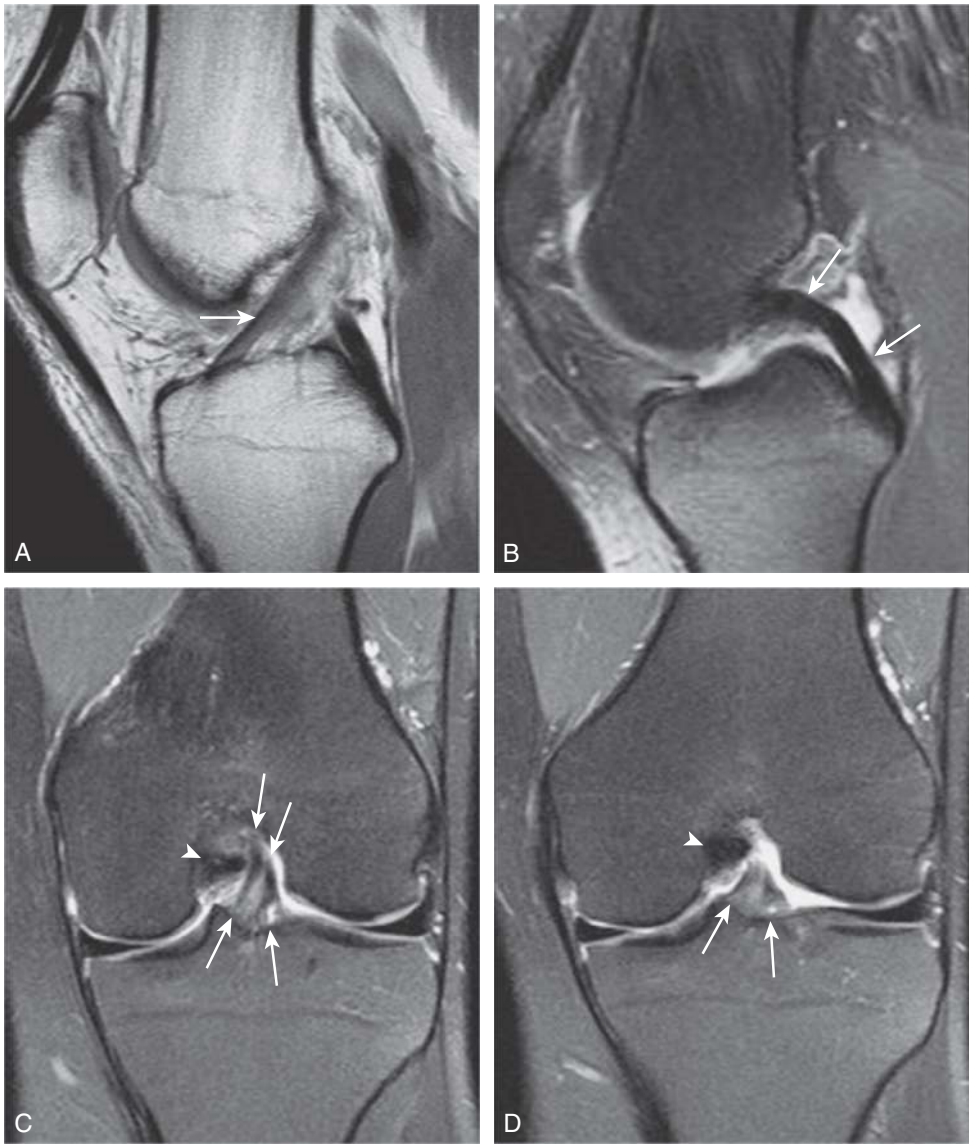

FIG 118.2 Normal magnetic resonance imaging anatomy of the cruciate ligaments. **A,** Sagittal fast spin-echo proton density (FSE PD)–weighted image shows streaky areas of isointense signal within a normal anterior cruciate ligament (ACL). Note the straight anterior border of the ACL (*arrow*), which is parallel to Blumensaat's line. **B,** Sagittal fat-saturated FSE PD–weighted image shows the dark signal intensity of the posterior cruciate ligament (PCL), which has a posterior convex configuration (*arrows*). **C and D,** Contiguous coronal fat-saturated FSE PD–weighted images of a normal ACL and PCL. Note the dark signal intensity of the PCL (*arrowhead*) and relatively high signal intensity of the ACL near its tibial insertion. The anteromedial and posterolateral bundles of the ACL are visualized (*arrows*). (From Kam CK, Chee DW, Peh WC. Magnetic resonance imaging of cruciate ligament injuries of the knee. *Can Assoc Radiol J.* 2010;61(2):80–89.)

backward. Coexistent bursitis, tendinitis, arthritis, or internal derangement of the knee may confuse the clinical picture after trauma to the knee joint. The menisci of the knee are often injured when the patient sustains knee trauma severe enough to disrupt the anterior cruciate ligament.

On physical examination, patients with injury to the anterior cruciate ligament exhibit tenderness to palpation of the anterior knee. If the ligament is avulsed from its bony insertions, tenderness may be localized to the site of insertion, whereas patients suffering from strain of the ligament have more diffuse tenderness. Patients with severe injury to the ligament may exhibit joint laxity when anterior stress is placed on the affected knee. This maneuver is best accomplished by performing an anterior drawer test and Lachman test for anterior cruciate ligament integrity (Figs. 118.3 and 118.4). Other tests to assess the integrity of the anterior cruciate ligament include the flexion-rotation anterior drawer test and the Lachman test.

Because pain may produce muscle guarding, magnetic resonance imaging (MRI) of the knee may be necessary to confirm the clinical impression (Fig. 118.5). Joint effusion

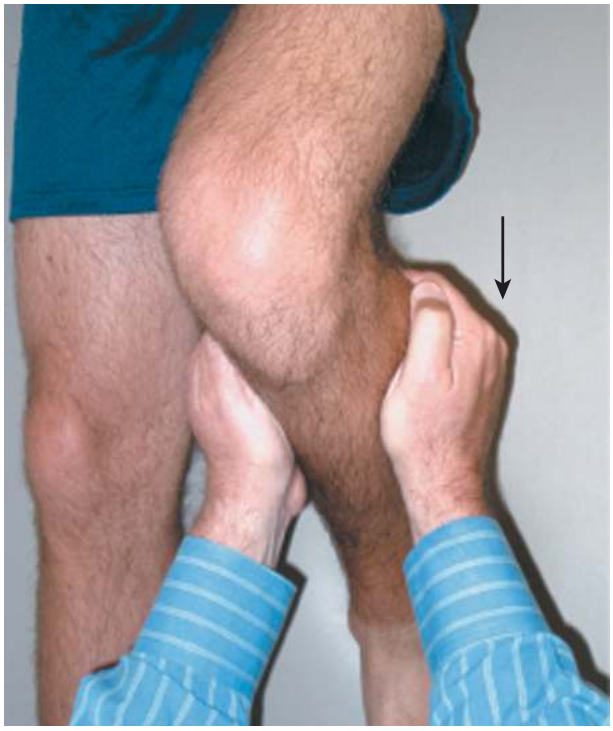

FIG 118.3 The anterior drawer test for anterior cruciate ligament integrity. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:293.)

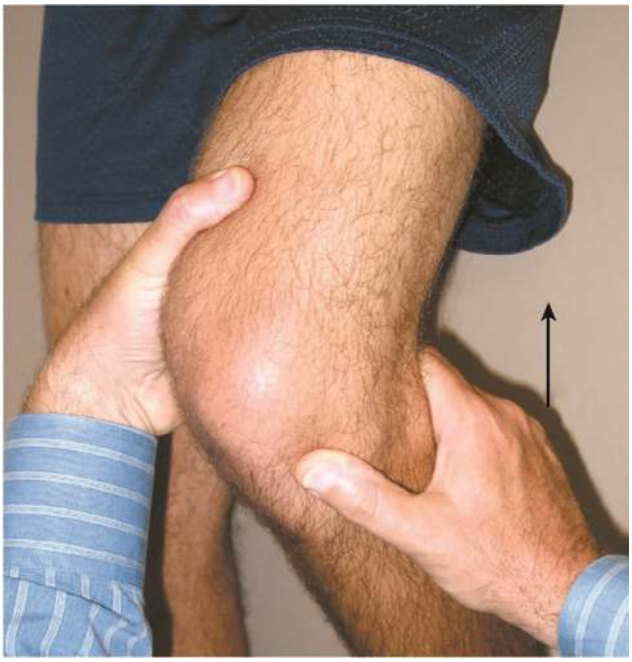

FIG 118.4 The Lachman test for anterior cruciate integrity. (From Waldman S. *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. St Louis: Elsevier; 2016: Fig. 208-1).

and swelling may be present with injury to the ligament, but these findings can also suggest meniscal damage. Again, MRI can confirm the diagnosis.

TESTING

MRI and ultrasound imaging are indicated in all patients who present with anterior cruciate ligament injury, both to rule out coexistent internal derangement, occult mass, or tumor and to confirm the diagnosis (Fig. 118.6). In addition, MRI and ultrasound imaging should be performed in all patients with injury to the anterior cruciate ligament who fail to respond to conservative therapy or who exhibit joint instability on clinical examination (Figs. 118.7 and 118.8). Bone scan may be useful to identify occult stress fractures involving the joint, especially if trauma has occurred. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Any condition affecting the medial compartment of the knee joint may mimic the pain of anterior cruciate ligament syndrome. Bursitis, arthritis, and entrapment neuropathies may also confuse the diagnosis, as may primary tumors of the knee and spine.

TREATMENT

Initial treatment of the pain and functional disability associated with injury to the anterior cruciate ligament includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Any repetitive activity that exacerbates the patient's symptoms should be avoided. For patients who do not respond to these treatment modalities and do not have lesions that require surgical repair, injection is a reasonable next step.

Injection to treat anterior cruciate ligament syndrome is carried out by injecting the interarticular space of the affected knee with local anesthetic and steroid. To perform intraarticular injection of the knee, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the medial joint is prepared with antiseptic solution. A sterile syringe containing 5 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The joint space is identified, and the clinician places his or her thumb on the lateral margin of the patella and pushes it medially. At a point in the middle of the medial edge of the patella, the needle is inserted between the patella and the femoral condyles. The needle is then carefully advanced through the skin and subcutaneous tissues through the joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. After

FIG 118.5 Acute complete anterior cruciate ligament (ACL) tear. **(A)** Mid sagittal fat-saturated fast spin echo (FSE) T2-weighted image shows the distal portion of the ruptured ACL, which is thickened, hyperintense, and horizontally oriented (*arrows*). Its axis is abnormally flattened (away from the Blumensaat's line). **(B)** Lateral parasagittal fat-saturated FSE T2-weighted image shows bone contusions (*arrows*) in the lateral femoral condyle and posterolateral tibial plateau. Deepened lateral femoral sulcus (*arrowhead*) and anterior tibial translation are present. **(C)** Coronal fat-saturated FSE T2-weighted image shows the thickened ACL, with increased signal intensity within its fibers (*arrow*). (Please use fig 2 https://ars.els-cdn.com/content/image/1-s2.0-S0846537109002241-gr3.jpg from Kam CK, Chee DW, Peh WC. Magnetic resonance imaging of cruciate ligament injuries of the knee. *Canadian Association of Radiologists Journal*. 2010;61(2):80–89.)

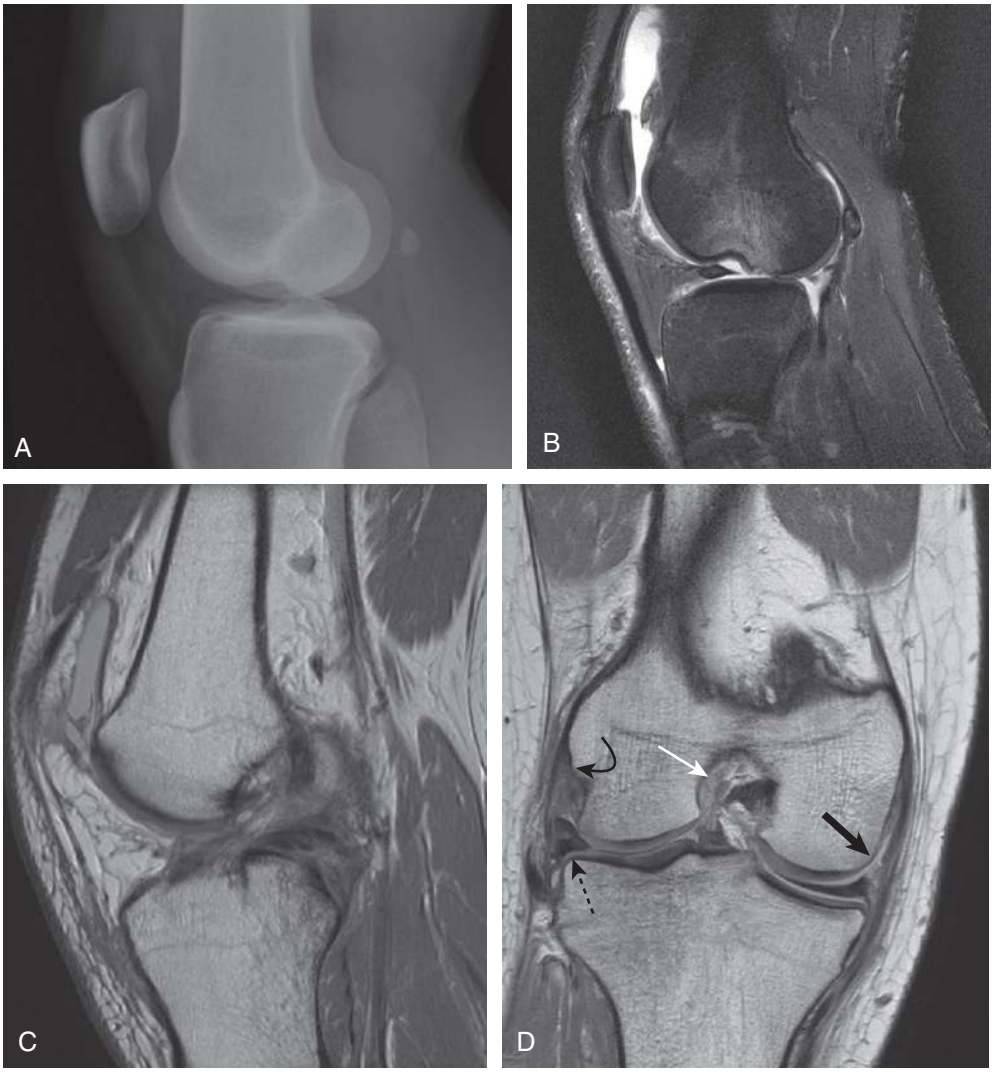

FIG 118.6 Acute complete anterior cruciate ligament (ACL) rupture. **A,** Lateral radiograph shows an impaction fracture of the lateral femoral condyle. **B,** The fracture is clearly seen on the sagittal T2-weighted with fat suppression magnetic resonance (MR) image, with associated trabecular bone bruising in the lateral femoral condyle and a large joint effusion. **C,** On the sagittal proton density (PD) MR image, there is nonvisualization of the ligament. **D,** The coronal PD image demonstrates the "empty notch" sign (*white arrow*). There is also a lateral meniscal tear (*broken black arrow*), a partial tear of the lateral collateral ligament (*curved black arrow*), and a grade II tear of the medial collateral ligament (MCL), including disruption of the meniscofemoral component of the deep fibers of the MCL (*black arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications (Fig. 118.9). Clinical experience suggests that the injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of medial meniscal dysfunction.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Orthotic devices to stabilize the knee may also help improve the patient's functional ability as well as relieve pain.

COMPLICATIONS AND PITFALLS

The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique

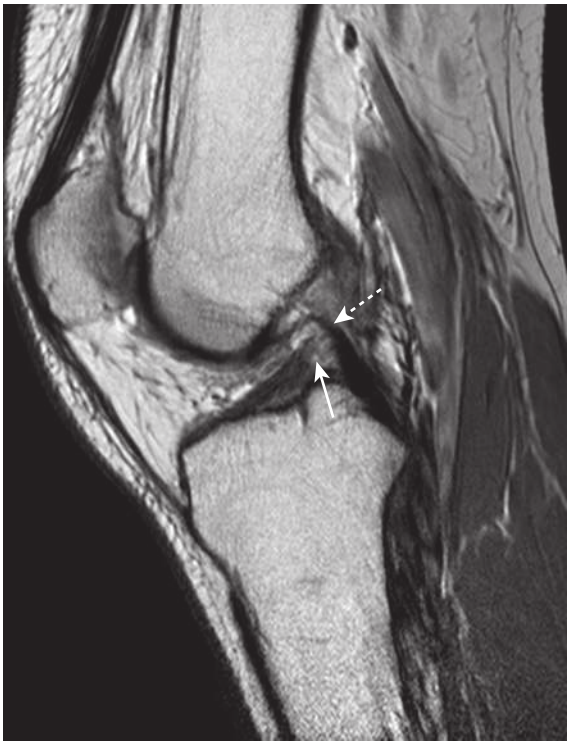

FIG 118.7 Sagittal proton density (PD) magnetic resonance image of a chronic anterior cruciate ligament tear. The torn end of the ligament lies low within the intercondylar notch (*white arrow*), and the posterior cruciate ligament is bowed (*broken white arrow*) because of posterior translation of the tibia on the femur. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

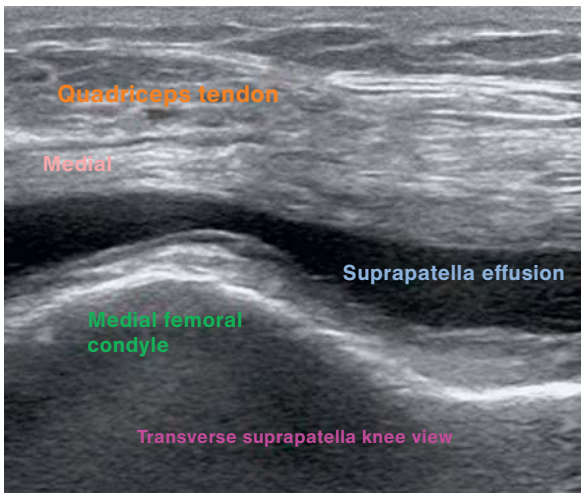

FIG 118.8 Transverse ultrasound image demonstrating a suprapatellar effusion in a patient with an acute knee injury. (Courtesy Dr Steven Waldman MD.)

is followed. Approximately 25% of patients complain of a transient increase in pain after injection of the medial collateral ligament, and patients should be warned of this possibility.

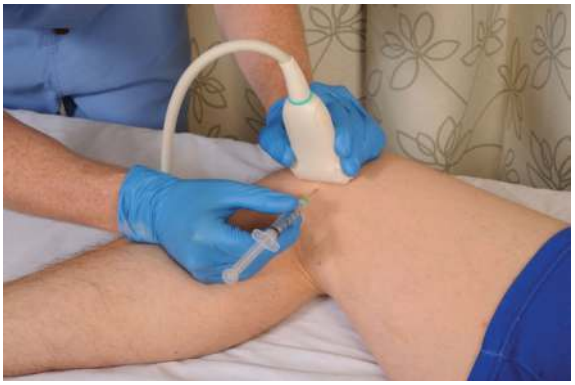

FIG 118.9 Ultrasound-guided intraarticular injection of the knee. (Courtesy Dr Steven Waldman MD.)

CLINICAL PEARLS

Patients with injury to the anterior cruciate ligament are best examined with the knee in the slightly flexed position. The clinician may want to examine the nonpainful knee first to reduce the patient's anxiety and to ascertain the findings of a normal examination. The injection technique described is extremely effective in the treatment of pain secondary to anterior cruciate ligament syndrome. Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Lee SH, Yun SJ. Diagnosis of simultaneous acute ruptures of the anterior cruciate ligament and posterior cruciate ligament using point-of-care ultrasound in the emergency department. *J Emerg Med*. 2018;54(3):335–338. https://doi.org/10.1016/j.jemermed.2017.11.014.

Salati U, Doody O, Munk PL, et al. Evaluation of knee pain in athletes: a radiologist's perspective. *Can Assoc Radiol J*. 2017;68(1):27–40.

Waldman SD. Functional anatomy of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2014:142–144.

Waldman SD. An overview of painful conditions of the knee. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:308.

Waldman SD. Arthritis and other abnormalities of the knee. *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:725–740.

Waldman SD. The anterior drawer test for anterior cruciate ligament instability. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:314.

Waldman SD. The flexion-rotation anterior drawer test for anterior cruciate ligament instability. In: *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016:315.

Waldman SD. Intra-articular injection of the knee. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:651–656.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the knee. In: *Imaging of pain*. Philadelphia: Saunders; 2011:367–388.

Waldman SD, Campbell RSD. Anterior cruciate ligament tear. In: *Imaging of pain*. Philadelphia: Elsevier; 2011:375–377.

119

Jumper's Knee

ICD-10 CODE M77.9

THE CLINICAL SYNDROME

Jumper's knee is characterized by pain at the inferior or superior pole of the patella. It occurs in up to 20% of jumping athletes at some point in their careers. It may affect one or both knees; boys and men are affected twice as commonly as girls and women when just one knee is involved. Jumper's knee is usually the result of overuse or misuse of the knee joint caused by running, jumping, or overtraining on hard surfaces or direct trauma to the quadriceps or patellar tendon, such as from kicks or head butts during football or kickboxing (Fig. 119.1).

The quadriceps tendon is made up of fibers from the four muscles that constitute the quadriceps muscle: vastus lateralis, vastus intermedius, vastus medialis, and rectus femoris. These muscles are the primary extensors of the lower extremity at the knee. The tendons of these muscles converge and unite to form a single, exceedingly strong tendon (Fig. 119.2). The patella functions as a sesamoid bone within the quadriceps tendon, with fibers of the tendon expanding around the patella and forming the medial and lateral patella retinacula, which strengthen the knee joint. The patellar tendon extends from the patella to the tibial tuberosity. Weak or poor quadriceps and hamstring muscle flexibility, congenital variants in knee anatomy (e.g., patella alta or baja), and limb length discrepancies have been implicated as risk factors for the development of jumper's knee. Investigators have postulated that the strong eccentric contraction of the quadriceps muscle to strengthen the knee joint during landing is the inciting factor rather than the jump itself.

SIGNS AND SYMPTOMS

Patients with jumper's knee present with pain over the superior or inferior pole (or both) of the sesamoid. Jumper's knee can affect both the medial and lateral sides of both the quadriceps and the patellar tendons. Patients note increased pain

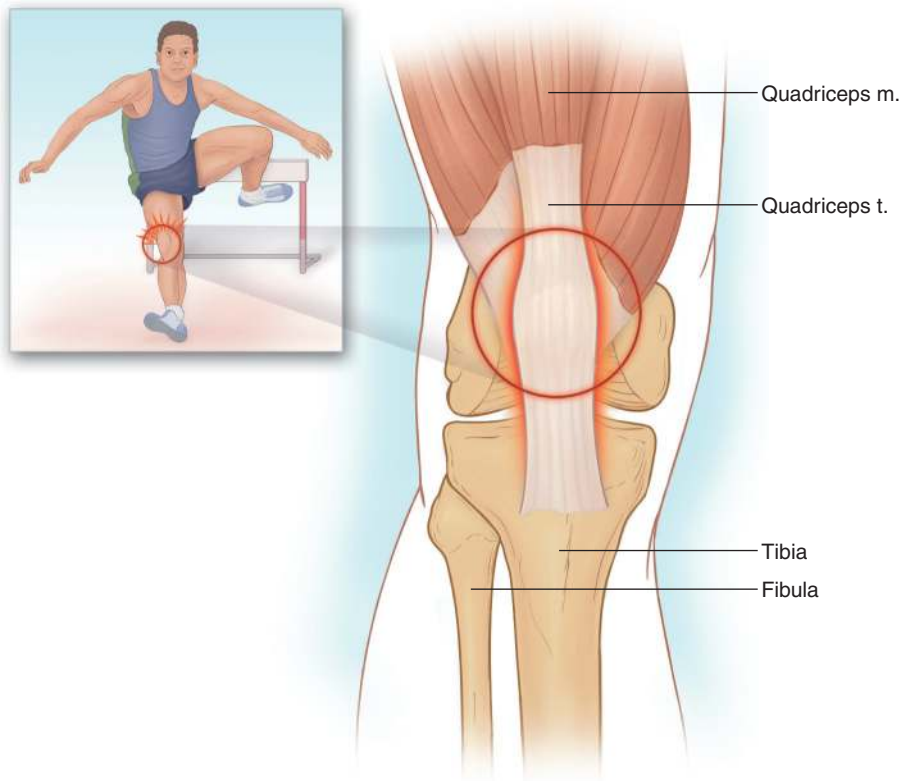

FIG 119.1 Jumper's knee—characterized by pain at the inferior or superior pole of the patella—occurs in up to 20% of jumping athletes at some point in their careers.

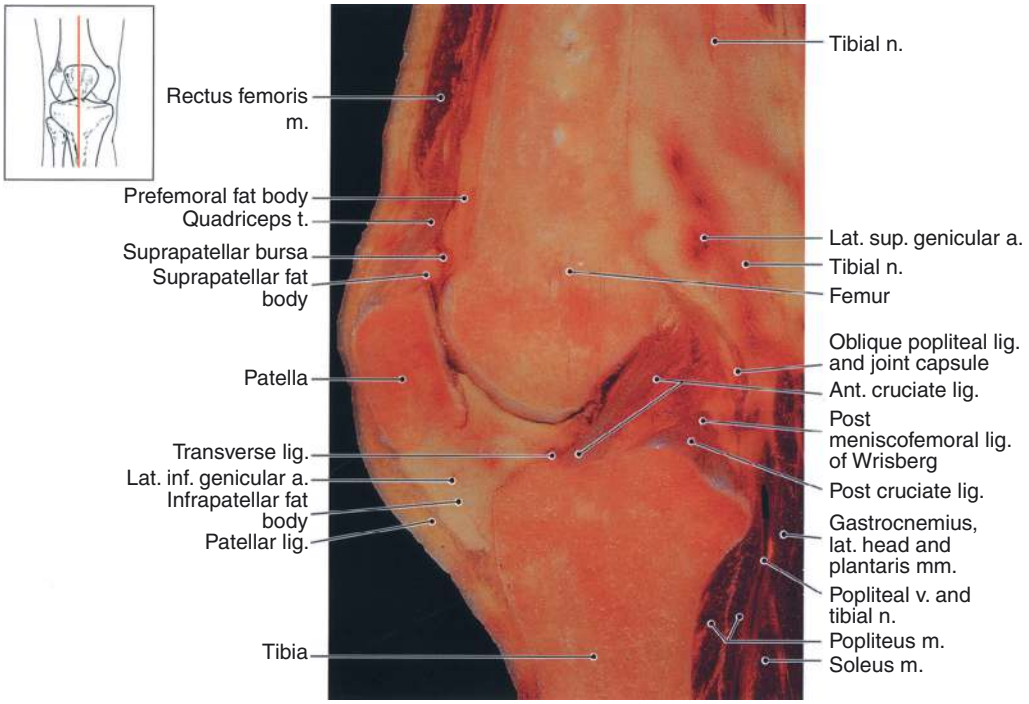

FIG 119.2 Sagittal view of the knee. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas*. 2nd ed. Philadelphia: Saunders; 2002:341.)

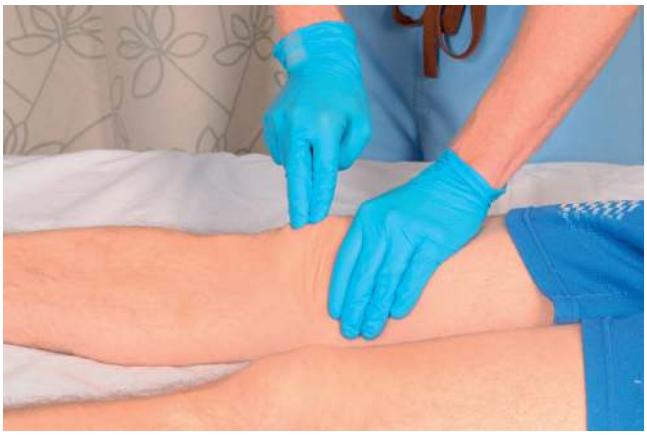

FIG 119.3 Patients suffering from jumper's knee often have large joint effusions and exhibit a positive ballottement test. To perform the ballottement test for knee effusions, the clinician has the patient extend and fully relax the knee. The clinician then grasps the affected knee just above the joint space and applies pressure to displace synovial fluid from the suprapatellar pouch into the joint, which will elevate the patella. The clinician then ballotes the patella. The test is considered positive if the patella ballots easily.

on walking down slopes or down stairs. Activity using the knee, especially jumping, makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. On physical examination, the patient notes tenderness of the quadriceps or patellar tendon (or both), and joint effusion may be present. Actively resisted extension of the knee reproduces the pain. Ballottement test will be positive in patients suffering from jumper's knee (Fig. 119.3). Coexistent suprapatellar and infrapatellar bursitis, tendinitis, arthritis, or internal derangement of the knee may confuse the clinical picture after trauma to the knee.

TESTING

Plain radiographs are indicated in all patients who present with knee pain. Magnetic resonance imaging and ultrasound imaging of the knee are indicated if jumper's knee is suspected, because these imaging modalities readily demonstrate tendinosis of the quadriceps or patellar tendon (Fig. 119.4). Ultrasound imaging may also provide beneficial information regarding the vascularity and integrity of the patellar and quadriceps tendons (Fig. 119.5). Bone scan may be useful to identify occult stress fractures involving the joint, especially if trauma has occurred. Ultrasound tissue characterization testing may also provide useful information regarding the degree of tendinopathy (Fig. 119.6). Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Jumper's knee is a repetitive stress disorder that causes tendinosis of the quadriceps and patellar tendons and is a distinct clinical entity from tendinitis of those tendons or quadriceps expansion syndrome which may coexist with jumper's knee

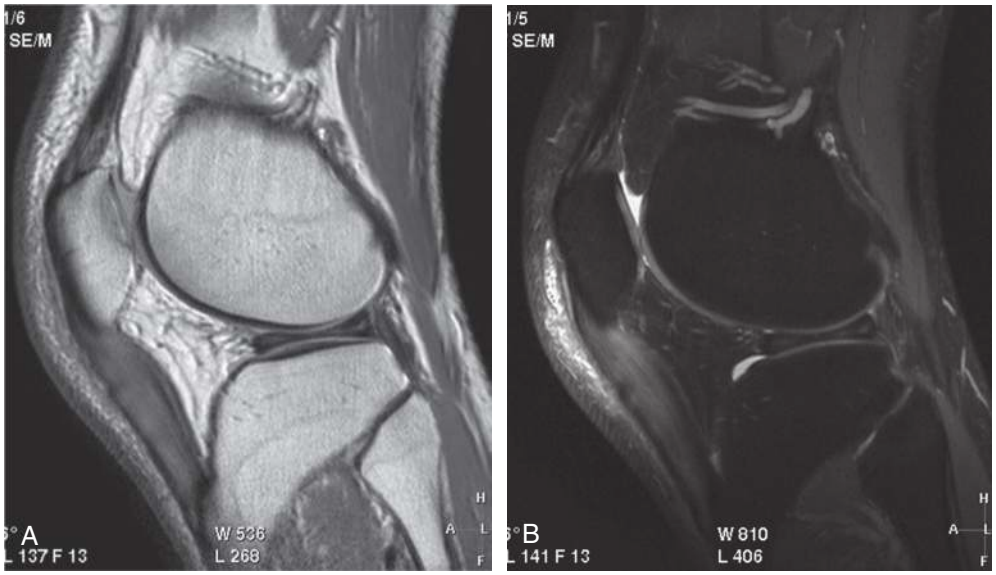

FIG 119.4 Sagittal T1W **(A)** and fat-suppressed T2-weighted (FST2W) **(B)** magnetic resonance (MR) images of a patient with diffuse patellar tendinopathy. The tendon is thickened and of increased signal intensity (SI) on both pulse sequences, but the SI is not as bright as that of fluid on the FST2W MR image. High-SI prepatellar bursitis is also evident on the FST2W MR image. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

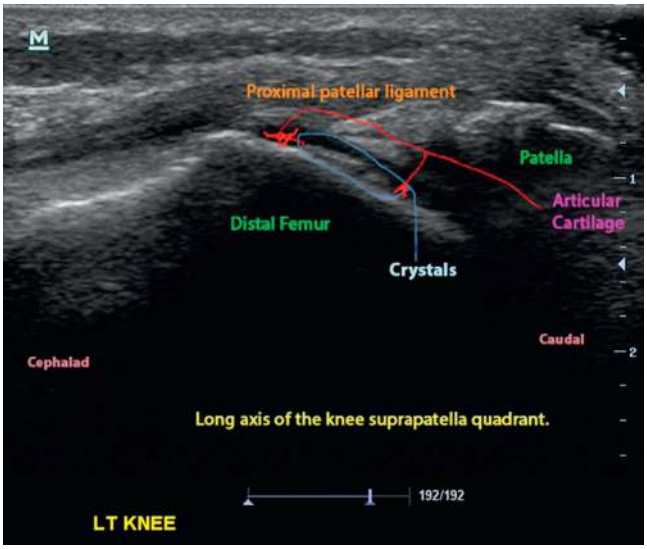

FIG 119.5 Longitudinal ultrasound image demonstrating crystal deposition disease affecting the patellar tendon.

and confuse the clinical picture. Quadriceps expansion syndrome has a predilection for the medial side of the superior pole of the patella. The quadriceps tendon is also subject to acute calcific tendinitis, which may coexist with acute strain injuries and the more chronic changes in jumper's knee. Calcific tendinitis of the quadriceps has a characteristic radiographic appearance of whiskers on the anterosuperior patella. The suprapatellar, infrapatellar, and prepatellar bursae also may become inflamed with dysfunction of the quadriceps tendon.

TREATMENT

Initial treatment of the pain and functional disability associated with jumper's knee includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. A nighttime splint to protect the knee may also help relieve the symptoms. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To perform the injection, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. If only the quadriceps tendon is affected, the skin overlying the medial aspect of the knee joint is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The superior margin of the medial patella is identified (Fig. 119.7). Just above this point, the needle is inserted horizontally to slide just beneath the quadriceps tendon (Fig. 119.8). If the needle strikes the femur, it is withdrawn slightly and is redirected with a more anterior trajectory. When the needle is in position just below the quadriceps tendon, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site.

If only the patellar tendon is affected, the skin overlying the medial portion of the lower margin of the patella is prepared with antiseptic solution. A sterile syringe containing

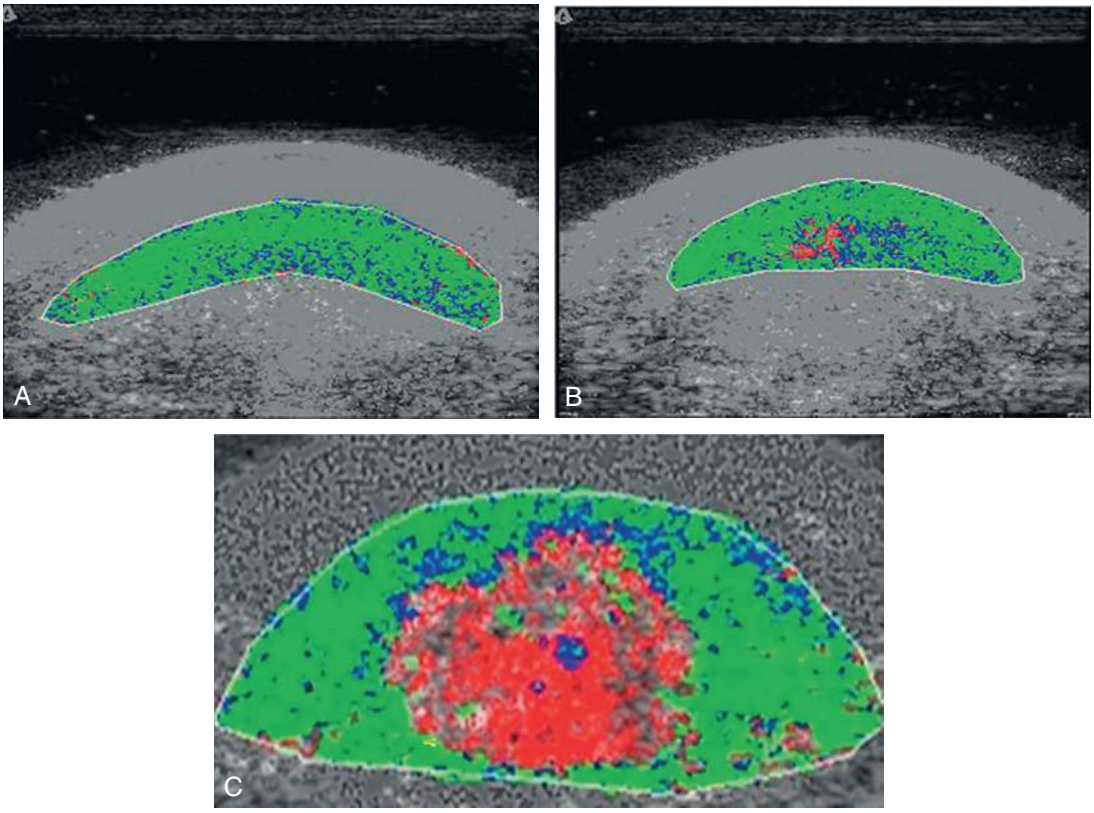

FIG 119.6 Ultrasonic tissue characterization: **(A)** normal patellar tendon appearance, **(B)** mild patellar tendon disorganization, and **(C)** severe patellar tendon disorganization. Note: green color represents good tendon structure; blue, red, and black represent increasing structural disruption. (From Rudavsky A, Cook J. Physiotherapy management of patellar tendinopathy (jumper's knee). *J Physiother*. 2014;60(3):122–129.)

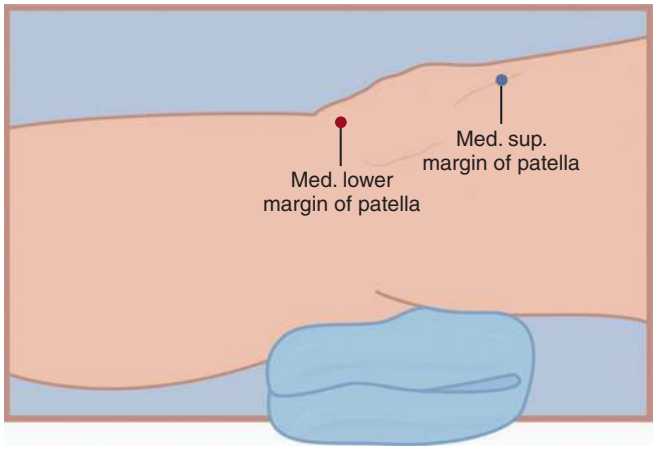

FIG 119.7 Identification of the superior margin of the medial patella (*blue*) and of the medial lower margin of the patella (*red*). (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:269, 278.)

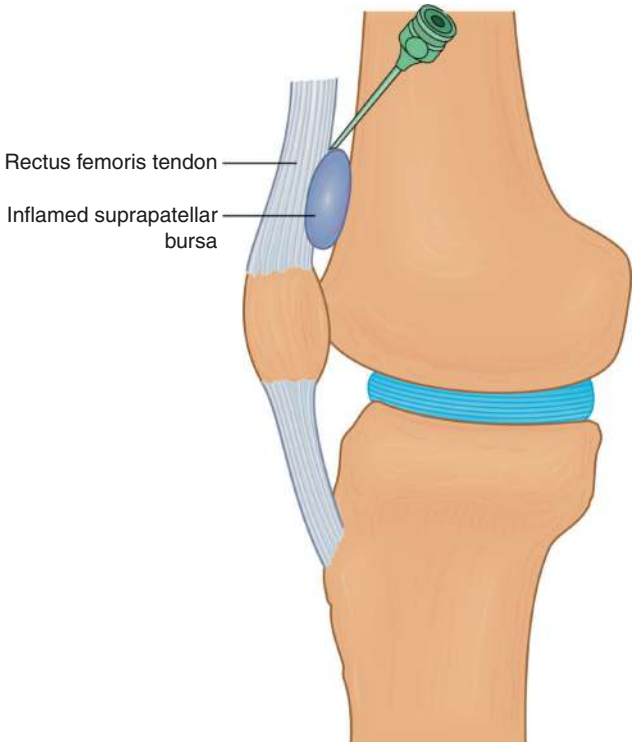

FIG 119.8 Correct needle placement for injection of the suprapatellar bursa. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:269.)

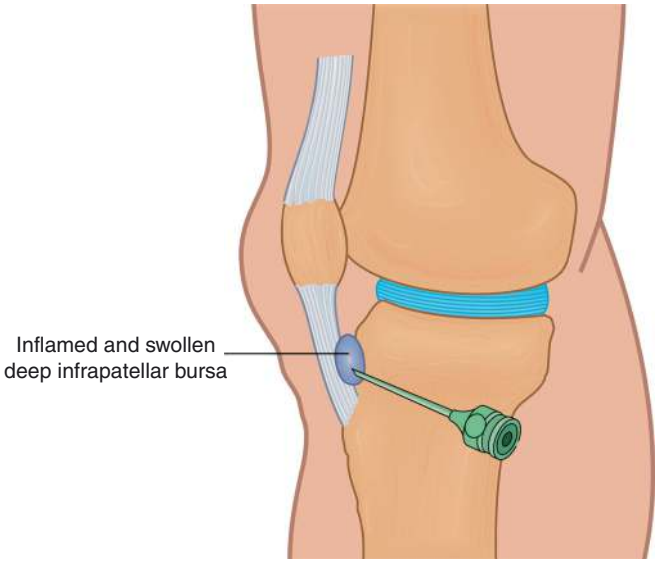

FIG 119.9 Correct needle placement for injection of the deep infrapatellar bursa. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:278.)

2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The medial lower margin of the patella is identified (see Fig. 119.7). Just below this point, the needle is inserted at a right angle to the patella to slide beneath the patellar ligament into the deep infrapatellar bursa (Fig. 119.9). If the needle strikes the patella, it is withdrawn slightly and is redirected with a more inferior trajectory. When the needle is in position in proximity to the deep infrapatellar bursa, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. If both the quadriceps and patellar tendons are affected, both injections should be performed. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. Clinical experience suggests that the injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of jumper's knee.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain in jumper's knee.

SUGGESTED READINGS

Hannington M, Docking S, Cook J, et al. Self-reported jumpers' knee is common in elite basketball athletes—but is it all patellar tendinopathy? *Phys Ther Sport*. 2020;43:58–64. https://doi.org/10.1016/j.ptsp.2020.01.012.

Nuttall C, Winters BA. Understanding anterior knee pain: patellofemoral pain syndrome. *J Nurse Pract*. 2015;11(10):1032–1035.

Rudavsky A, Cook J. Physiotherapy management of patellar tendinopathy (jumper's knee). *J Physiother*. 2014;60(3):122–129.

Waldman SD. Jumper's knee and other abnormalities of the patella. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:771–779.

Waldman SD, Campbell RSD. Patellar tendinopathy. In: *Imaging of pain*. Philadelphia: Saunders; 2011:397–398.

Runner's Knee

ICD-10 CODE M76.899

THE CLINICAL SYNDROME

Runner's knee, also known as iliotibial band friction syndrome, is a common cause of lateral knee pain. The iliotibial band is an extension of the fascia lata, which inserts at the lateral condyle of the tibia. The iliotibial band bursa lies between the iliotibial band and the lateral condyle of the femur. Runner's knee is an overuse syndrome caused by friction injury to the iliotibial band as it rubs back and forth across the lateral epicondyle of the femur during running (Fig. 120.1); this rubbing can also irritate the iliotibial bursa beneath it. If inflammation of the iliotibial band becomes chronic, calcification may occur. Impingement of the iliotibial band against the lateral femoral epicondyle may also contribute to the patient's symptomatology (Fig. 120.2).

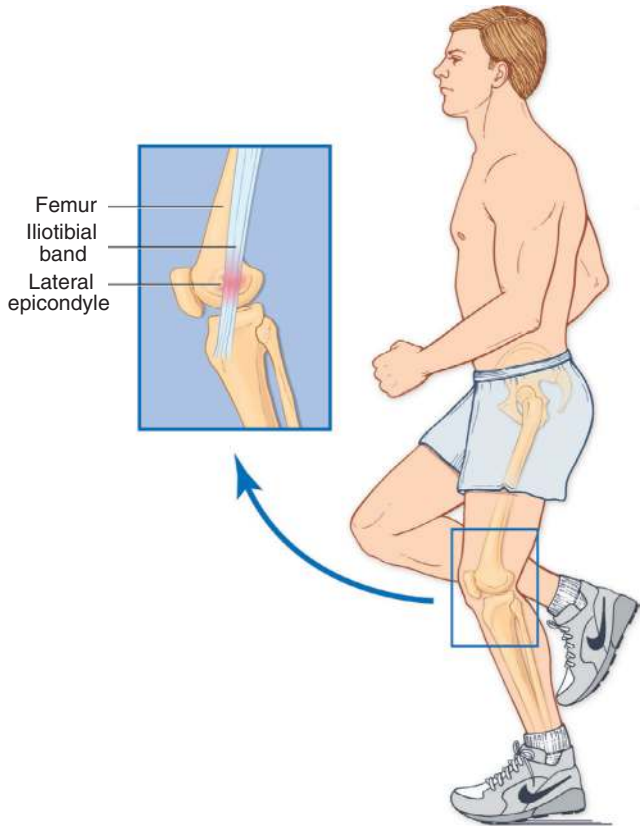

FIG 120.1 Runner's knee is an overuse syndrome caused by friction injury to the iliotibial band as it rubs back and forth across the lateral epicondyle of the femur. (From Waldman SD. *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2000:476.)

Runner's knee is a distinct clinical entity from iliotibial bursitis, although these two painful conditions frequently coexist. Runner's knee occurs more commonly in patients with genu varum and planus feet, although worn-out jogging shoes have also been implicated in the development of this syndrome.

SIGNS AND SYMPTOMS

Patients with runner's knee present with pain over the lateral side of the distal femur just over the lateral femoral epicondyle. Compared with iliotibial bursitis, the pain tends to be a little less localized, and effusion is rare. The onset of runner's knee frequently occurs after long-distance biking or jogging in worn-out shoes that lack proper cushioning (Fig. 120.3). Activity, especially that involving resisted abduction and passive adduction of the lower extremity, makes the pain worse, whereas rest and heat provide some relief. Flexion of the affected knee reproduces the pain in many patients with runner's knee. The modified Noble compression test is usually positive in patients suffering from Runner's knee (Fig.120.4). Often, patients are unable to kneel or walk downstairs. The pain is constant and is characterized as aching; it may interfere with sleep. Coexistent bursitis, tendinitis, arthritis, or internal derangement of the knee may confuse the clinical picture after trauma to the knee.

Physical examination may reveal point tenderness over the lateral epicondyle of the femur just above the tendinous insertion of the iliotibial band (see Fig. 120.1). If iliotibial bursitis is also present, the patient may have swelling and fluid accumulation around the bursa. Palpation of this area while the patient flexes and extends the knee may result in a creaking or "catching" sensation. Actively resisted abduction of the lower extremity reproduces the pain, as does passive adduction. Sudden release of resistance during this maneuver causes a marked increase in pain. Pain is exacerbated when the patient stands with all his or her weight on the affected extremity and then flexes the affected knee 30–40 degrees.

TESTING

Plain radiographs of the knee may reveal calcification of the bursa and associated structures, including the iliotibial band tendon, findings consistent with chronic inflammation. Magnetic resonance imaging is indicated

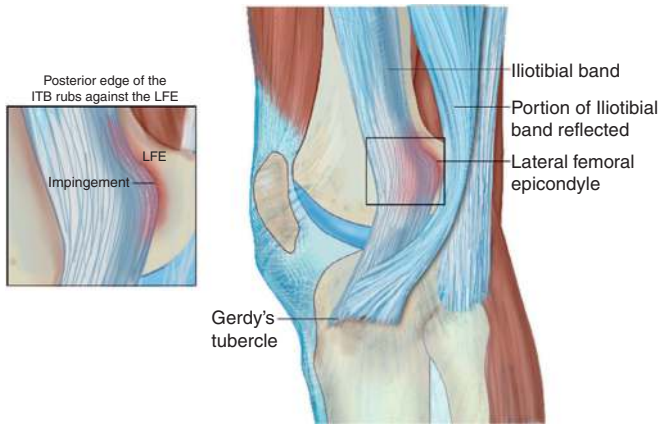

FIG 120.2 Friction and impingement model. *ITB*, Iliotibial band; *LFE*, lateral femoral epicondyle. (From Baker RL, Fredericson M. Iliotibial band syndrome in runners: biomechanical implications and exercise interventions. *Phys Med Rehabil Clin N Am*. 2016;27(1):53–77.)

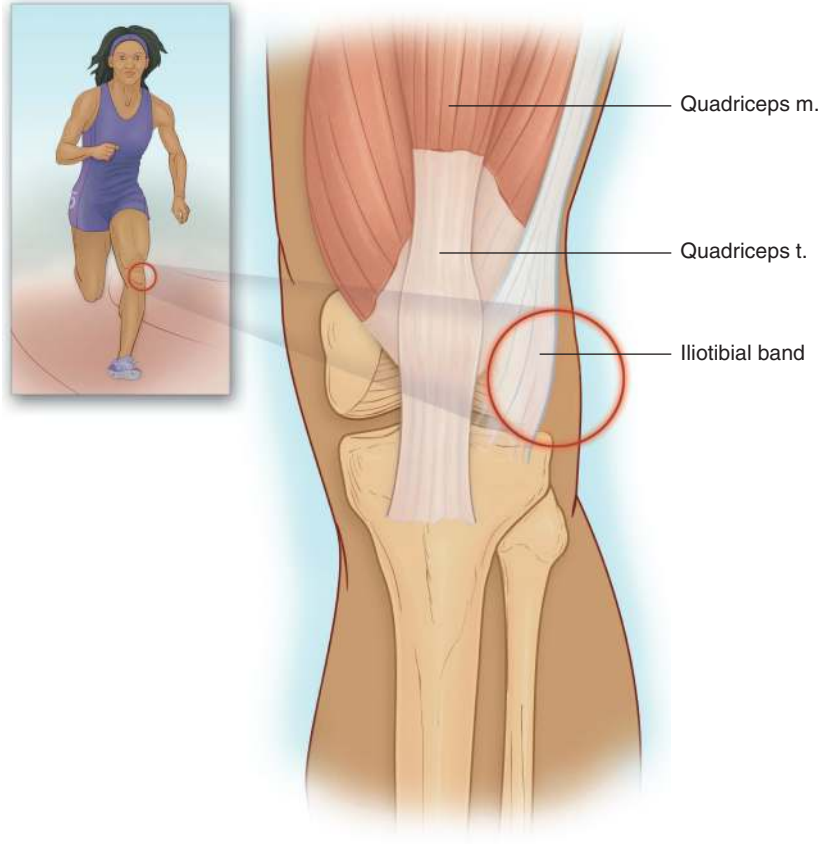

FIG 120.3 Patients with runner's knee present with pain over the lateral side of the distal femur, just over the lateral femoral epicondyle. The condition is often associated with running or jogging in worn-out shoes.

if runner's knee, iliotibial band bursitis, internal derangement, an occult mass, or a tumor of the knee is suspected (Figs. 120.5–120.8). Ultrasound imaging may also help clarify the diagnosis (Fig. 120.9). Electromyography can distinguish iliotibial band bursitis from neuropathy, lumbar radiculopathy, and plexopathy. Bone scan may be useful to identify occult stress fractures involving the joint, especially if trauma has occurred.

DIFFERENTIAL DIAGNOSIS

The iliotibial, suprapatellar, infrapatellar, and prepatellar bursae may become inflamed with dysfunction of the iliotibial band. The patellar tendon, which extends from the patella to the tibial tuberosity, is also subject to tendinitis, which may confuse the clinical picture. Internal derangement of the knee joint may coexist with iliotibial band bursitis.

FIG 120.4 The modified Noble compression test. (From Plastaras CT, Rittenberg JD, Rittenberg KE, et al: Comprehensive functional evaluation of the injured runner. *Phys Med Rehabil Clin N Am.* 16:623–649, 2005.)

TREATMENT

Initial treatment of the pain and functional disability associated with runner's knee includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. A nighttime splint to protect the knee may also relieve the symptoms. For patients who fail to respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To perform the injection, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin over the lateral epicondyle of the femur is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The iliotibial band bursa is identified by locating the point of maximal tenderness over the lateral condyle of the femur. At this point, the needle is inserted at a 45-degree angle to the femoral condyle, and the needle passes through the skin, subcutaneous tissues, and iliotibial band into the iliotibial band bursa. If the needle strikes the femur, it is withdrawn slightly into the substance of the bursa. When the needle is positioned in proximity to the iliotibial band bursa, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

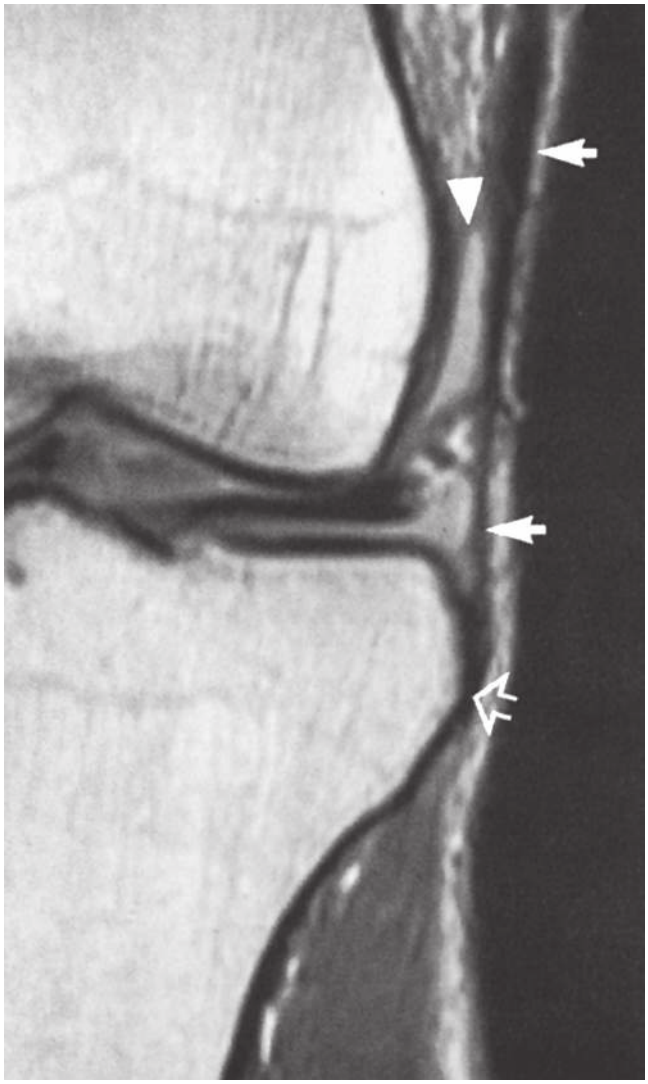

FIG 120.5 Normal iliotibial tract. Coronal, intermediate-weighted, spin-echo magnetic resonance image shows the iliotibial tract (*solid arrows*) attaching to Gerdy's tubercle (*open arrow*) in the tibia. A small joint effusion is evident just medial to the iliotibial tract (*arrowhead*). (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3231.)

Clinical experience suggests that the injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability associated with runner's knee.

Physical modalities, including local heat, iliotibial band stretching exercises, and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection (Fig. 120.10). Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can

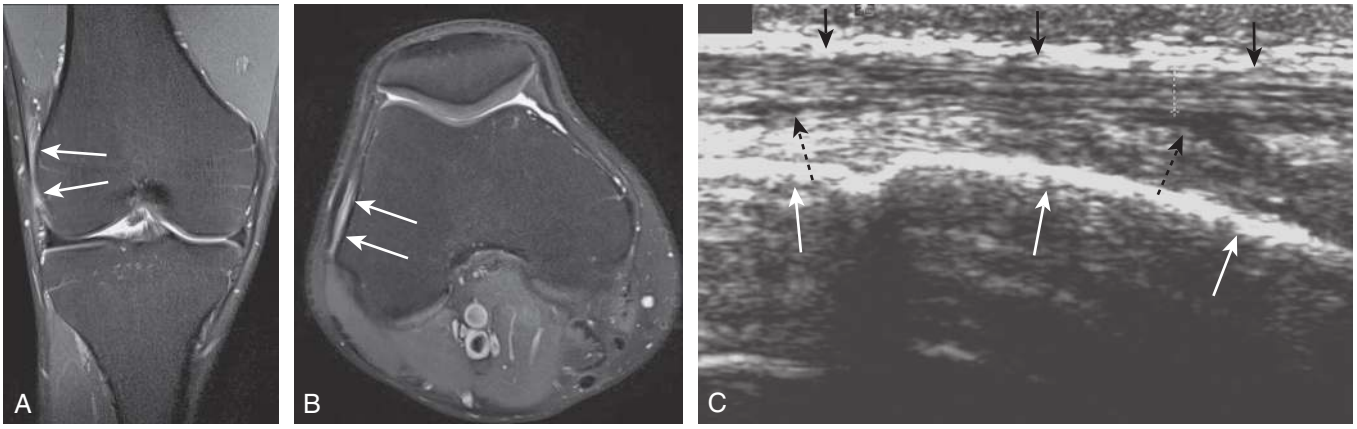

FIG 120.6 Coronal **(A)** and axial **(B)** T2-weighted with fat suppression magnetic resonance images of a runner with lateral joint pain. There is high–signal intensity (SI) fluid (*white arrows*) lying between the femoral epicondyle and the iliotibial band; it is caused by iliotibial band friction syndrome. (*Note:* It is important not to mistake joint fluid in the parapatellar recess for iliotibial bursitis.) **C,** The correlative transverse ultrasound image acquired during imaging-guided injection shows the echo-bright epicondyle (*white arrows*) and the iliotibial band (*black arrows*) with a thin layer of intervening fluid (*broken black arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

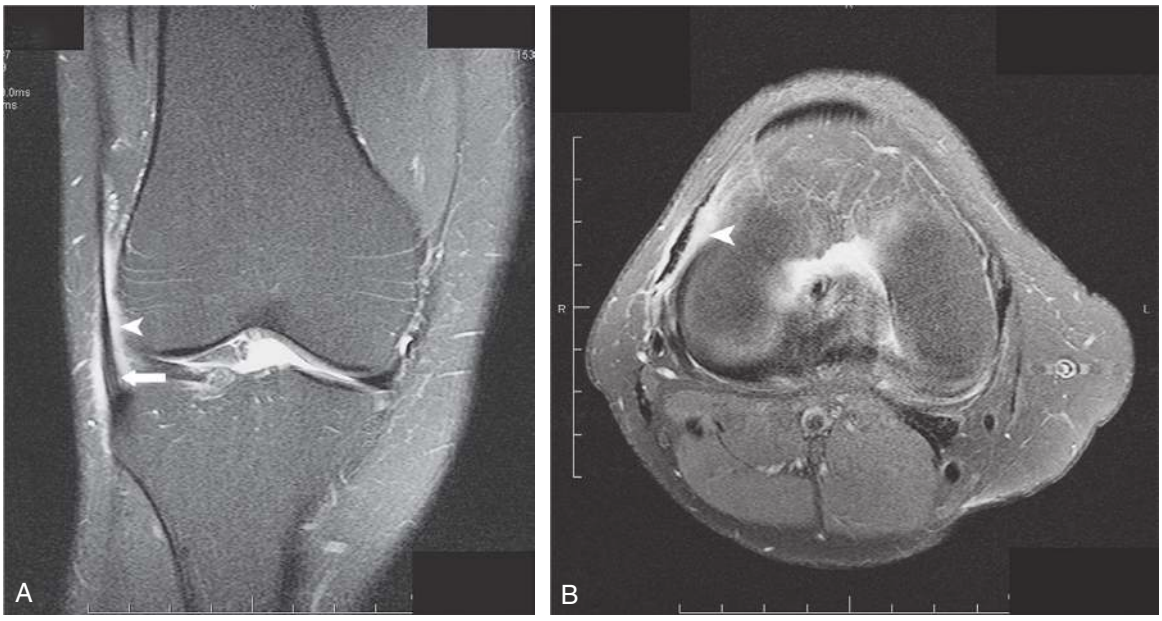

FIG 120.7 Iliotibial band friction syndrome. **A,** Coronal T2 fat-saturated image demonstrates high signal intensity in the fatty tissue deep to the iliotibial band (*arrowhead*) with loss of definition of the normally low signal intensity band (*arrow*). **B,** Axial T2 fat-saturated image demonstrates high signal intensity in the fatty tissue deep to the iliotibial band consistent with replacement by inflammatory tissue (*arrowhead*). (From O'Keeffe SA, Hogan BA, Eustace SL, et al. Overuse injuries of the knee. *MRI Clin N Am*. 2009;17(4):725–739.)

have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of runner's knee.

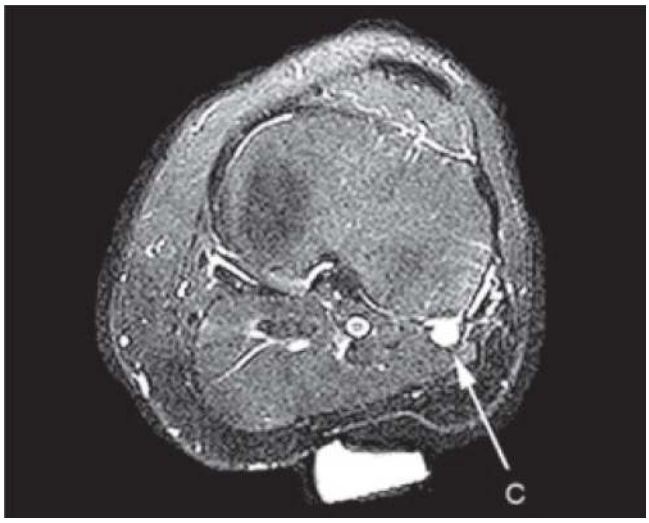

FIG 120.8 Magnetic resonance imaging of a patient with the clinical diagnosis of runner's knee. Fluid collection (C) posterior to the lateral femoral condyle is consistent with a synovial cyst with no fluid deep into the iliotibial band. (From Costa ML, Marshall T, Donell ST, et al. Knee synovial cyst presenting as iliotibial band friction syndrome. *Knee*. 2004;11(3):247–248, Fig. 1.)

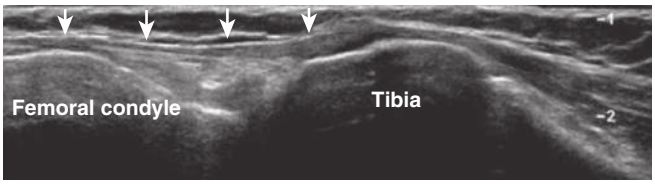

FIG 120.9 Iliotibial band syndrome. Wide longitudinal ultrasound scan along the iliotibial band (*arrows*): edematous swelling of the soft tissues deep to the iliotibial band, whose fibers do not show any alteration. (From Draghi F, Danesino GM, Coscia D, et al. Overload syndromes of the knee in adolescents: sonographic findings. *J Ultrasound*. 2008;11(4):151–157.)

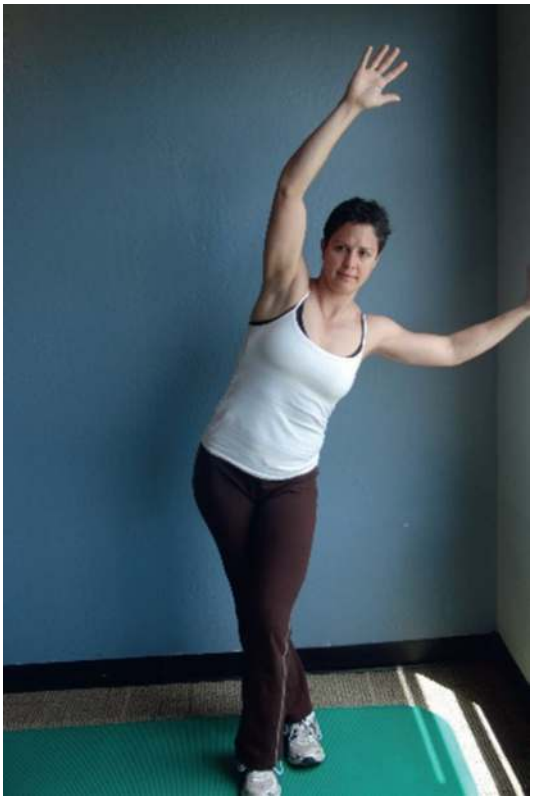

FIG 120.10 Iliotibial band stretch. This iliotibial band stretch outperformed two other stretches using 30-second holds at three stretches. (From Baker RL, Fredericson M. Iliotibial band syndrome in runners: biomechanical implications and exercise interventions. *Phys Med Rehabil Clin N Am*. 2016;27(1):53–77.)

SUGGESTED READINGS

Baker RL, Fredericson M. Iliotibial band syndrome in runners: biomechanical implications and exercise interventions. *Phys Med Rehabil Clin N Am*. 2016;27(1):53–77.

Draghi F, Danesino GM, Coscia D, et al. Overload syndromes of the knee in adolescents: sonographic findings. *J Ultrasound*. 2008;11(4):151–157.

Ellis R, Hing W, Reid D. Iliotibial band friction syndrome: a systematic review. *Man Ther*. 2007;12(3):200–208.

Fairclough J, Hayashi K, Toumi H, et al. Is iliotibial band syndrome really a friction syndrome? *J Sci Med Sport*. 2007;10(2):74–76.

Hamill J, Miller R, Noehren B, et al. A prospective study of iliotibial band strain in runners. *Clin Biomech (Bristol, Avon)*. 2008;23(8):1018–1025.

Waldman SD. Runner's knee. In: *Atlas of pain management injection techniques*. 2nd ed. Philadelphia: Saunders; 2007:475–480.

Waldman SD. The iliotibial band bursa. In: *Pain review*. Philadelphia: Saunders; 2009:154–155.

Waldman SD. Arthritis and other abnormalities of the knee. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:726–760.

Waldman SD Functional anatomy of the knee. In: *Pain review*. 2nd ed. Philadelphia: 2016.

Waldman SD. The iliotibial band bursa. In: Waldman SD, ed. *Pain review*. 2nd ed. Philadelphia: Elsevier; 2016:158–167.

Waldman SD, ed. Atlas of pain management injection techniques. 4th ed. Philadelphia: Elsevier; 2017:292–294.

Waldman SD, Campbell RSD. Anatomy, special imaging considerations for knee. In: *Imaging of pain*. Philadelphia: Saunders Elsevier; 2011:367–368.

121

Suprapatellar Bursitis

ICD-10 CODE M70.50

THE CLINICAL SYNDROME

The suprapatellar bursa extends superiorly from beneath the patella under the quadriceps femoris muscle and its tendon. The bursa is held in place by a small portion of the vastus intermedius muscle called the articularis genus muscle. The suprapatellar bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The suprapatellar bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries may be caused by direct trauma to the bursa during falls onto the knee or patellar fractures. Overuse injuries may result from running on soft or uneven surfaces or from jobs that require crawling on the knees, such as carpet lying. If inflammation of the suprapatellar bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Patients suffering from suprapatellar bursitis complain of pain in the anterior knee above the patella that may radiate superiorly into the distal anterior thigh. Often, patients are unable to kneel or walk downstairs (Fig. 121.1). Patients may also complain of a sharp "catching" sensation with range of motion of the knee, especially on first arising. Suprapatellar bursitis often coexists with arthritis and tendinitis of the knee, thus confusing the clinical picture.

Physical examination may reveal point tenderness in the anterior knee just above the patella. Passive flexion and actively resisted extension of the knee reproduce the pain. Sudden release of resistance during this maneuver causes a marked increase in pain. The patient may have swelling in the suprapatellar region, with a boggy feeling on palpation.

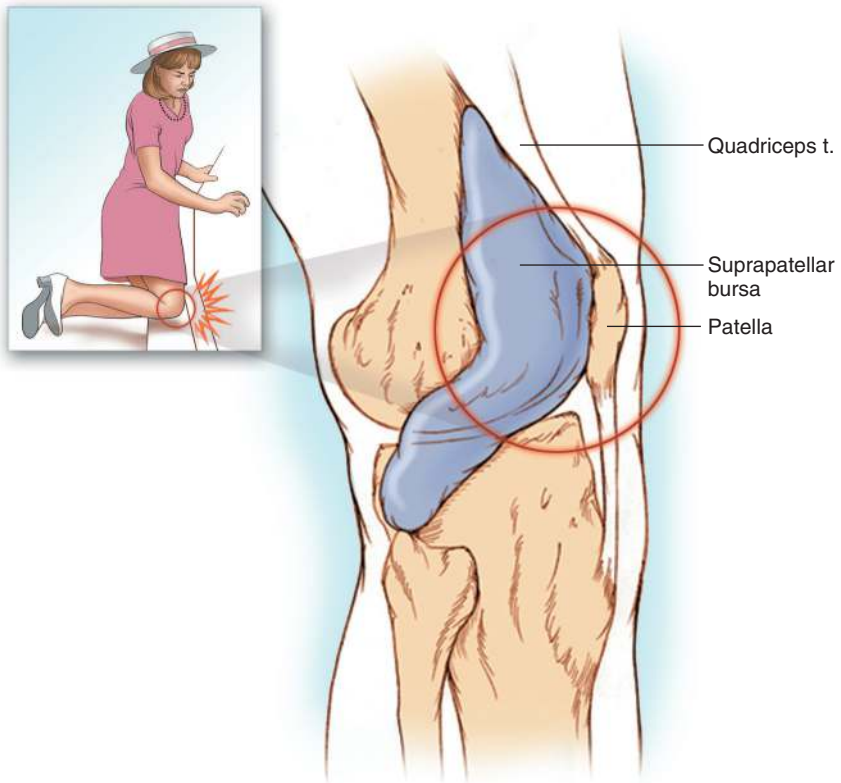

FIG 121.1 Suprapatellar bursitis is usually the result of direct trauma from either acute injury or repeated microtrauma, such as prolonged kneeling.

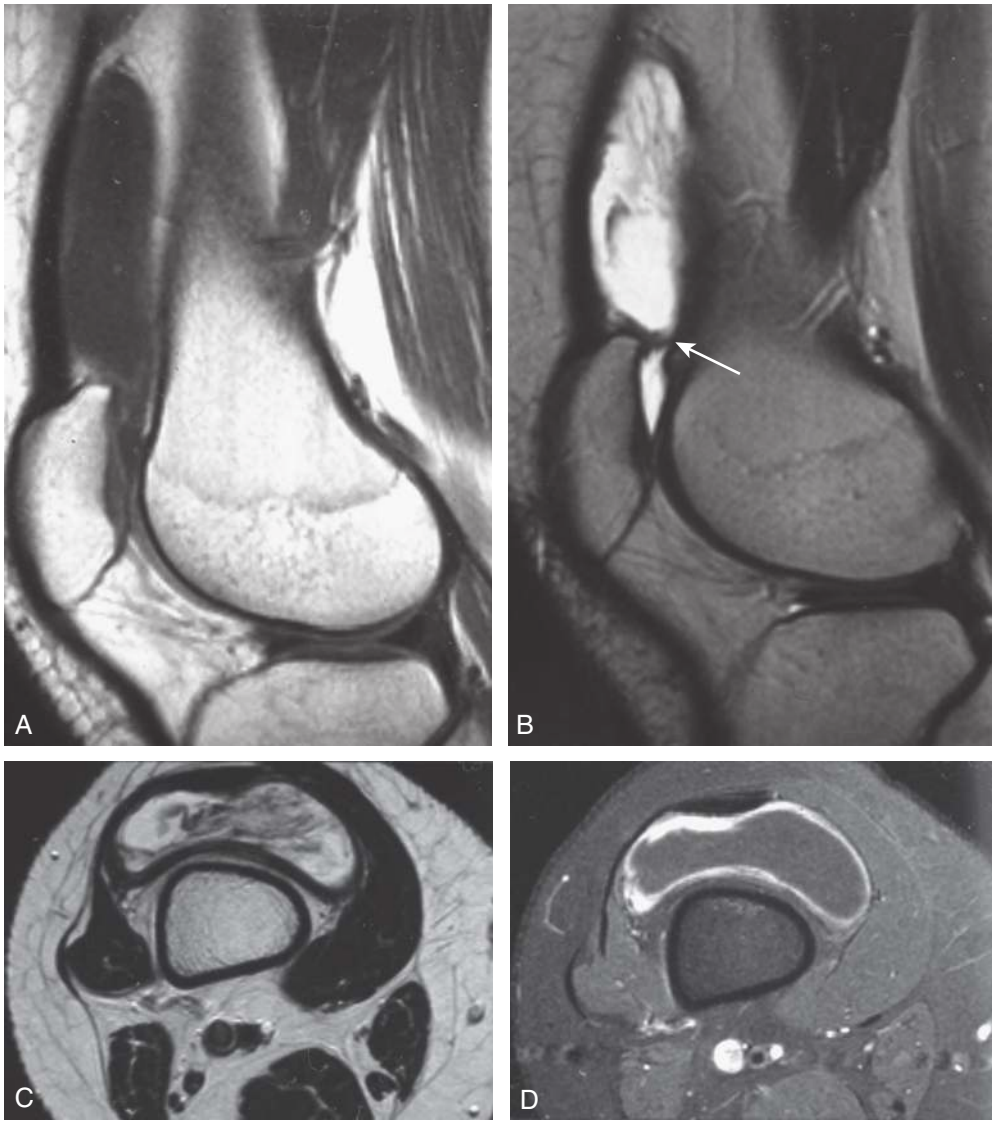

FIG 121.2 Sagittal T1-weighted (T1W) **(A)** and T2-weighted (T2W) **(B)** magnetic resonance (MR) images of a patient with an imperforate superior plica (*white arrow*). There is a loculated effusion within the suprapatellar bursa but no significant effusion in the other recesses of the knee joint. A few low-SI fronds of synovium within the bursa can be seen on the sagittal T2W MR image and on an axial T2W MR image **(C)**. **D,** An axial T1W with fat suppression MR image obtained after administration of a contrast agent shows minor enhancement of the synovial lining of the bursa. (From Waldman SD, Campbell RSD. *Imaging of pain.* Philadelphia: Elsevier; 2011.)

Occasionally, the suprapatellar bursa becomes infected, with systemic symptoms such as fever and malaise, as well as local symptoms such as rubor, color, and dolor.

TESTING

Plain radiographs, ultrasound imaging, and magnetic resonance imaging (MRI) of the knee may reveal calcification of the bursa and associated structures, including the quadriceps tendon, findings consistent with chronic inflammation (Figs. 121.2–121.4). MRI and ultrasound imaging are indicated if internal derangement, an occult mass, or a tumor of the knee is suspected (Fig. 121.5). Positron emission tomography may help identify infection of the suprapatellar bursa (Fig. 121.6). Electromyography can distinguish suprapatellar bursitis from femoral neuropathy, lumbar radiculopathy, and plexopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver. A complete blood count, and automated chemistry profile including uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing are indicated if collagen vascular disease is suspected. If infection is a possibility, aspiration, Gram stain, and culture of bursal fluid should be performed on an emergency basis (Fig. 121.7).

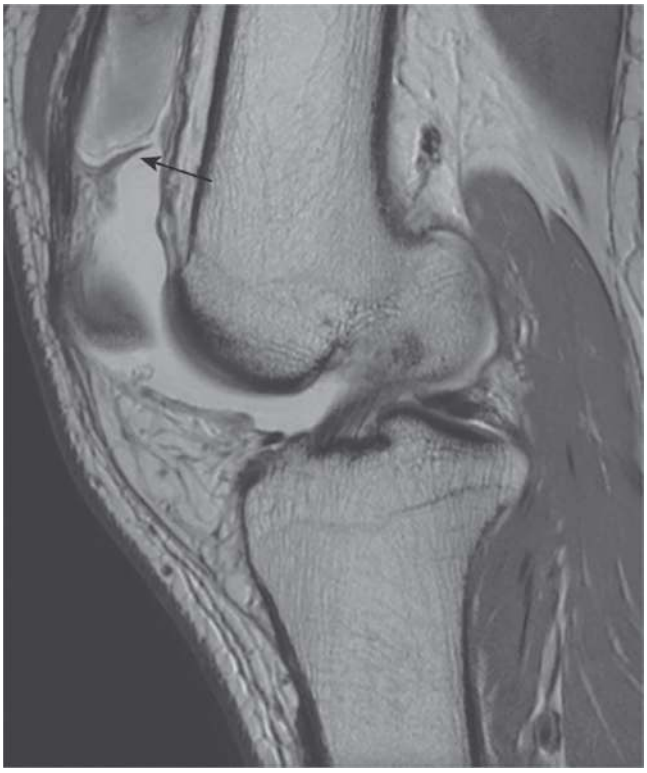

FIG 121.3 Sagittal T2-weighted magnetic resonance image of an imperforate plica (*black arrow*) in a patient with a loculated hematoma in the suprapatellar bursa (*white arrow*) following an acute injury. (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Saunders; 2011: Fig. 156.2.)

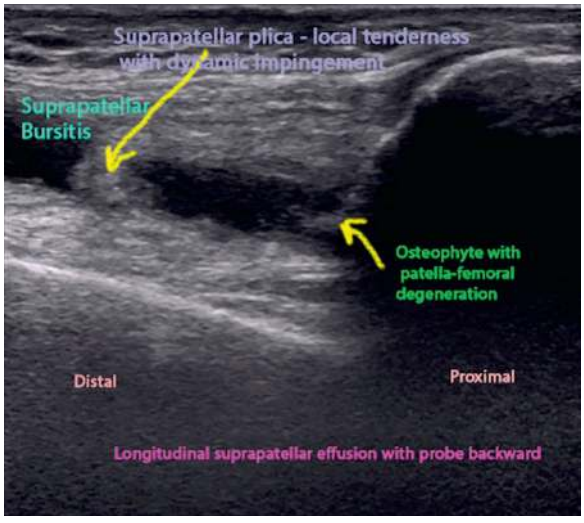

FIG 121.4 Longitudinal ultrasound image demonstrating suprapatellar bursitis and plica formation. Note the osteophyte and patella-femoral degenerative changes.

DIFFERENTIAL DIAGNOSIS

Because of the anatomy of the region, the associated tendons and other bursae of the knee can become inflamed along with the suprapatellar bursa, thus confusing the diagnosis. Both the quadriceps tendon and the suprapatellar bursa

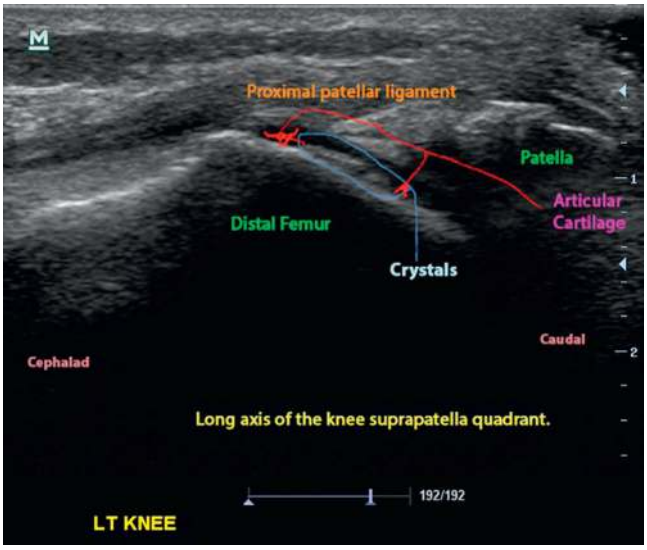

FIG 121.5 Ultrasound image demonstrating crystal deposition disease of the knee.

are subject to inflammation from overuse, misuse, or direct trauma. The tendon fibers, called expansions, are vulnerable to strain, and the tendon proper is subject to the development of tendinitis. The suprapatellar, infrapatellar, and prepatellar bursae may also become inflamed with dysfunction of the quadriceps tendon. Anything that alters the normal biomechanics of the knee can result in inflammation of the suprapatellar bursa.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs), or cyclooxygenase-2 inhibitors, and a knee brace to prevent further trauma is the first step in the treatment of suprapatellar bursitis. If patients do not experience rapid improvement, injection is a reasonable next step.

To perform the injection, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the medial aspect of the knee joint is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The superior margin of the medial patella is identified. Just above this point, the needle is inserted horizontally to slide beneath the quadriceps tendon. If the needle strikes the femur, it is withdrawn slightly and is redirected with a more anterior trajectory. When the needle is in position just below the quadriceps tendon, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site.

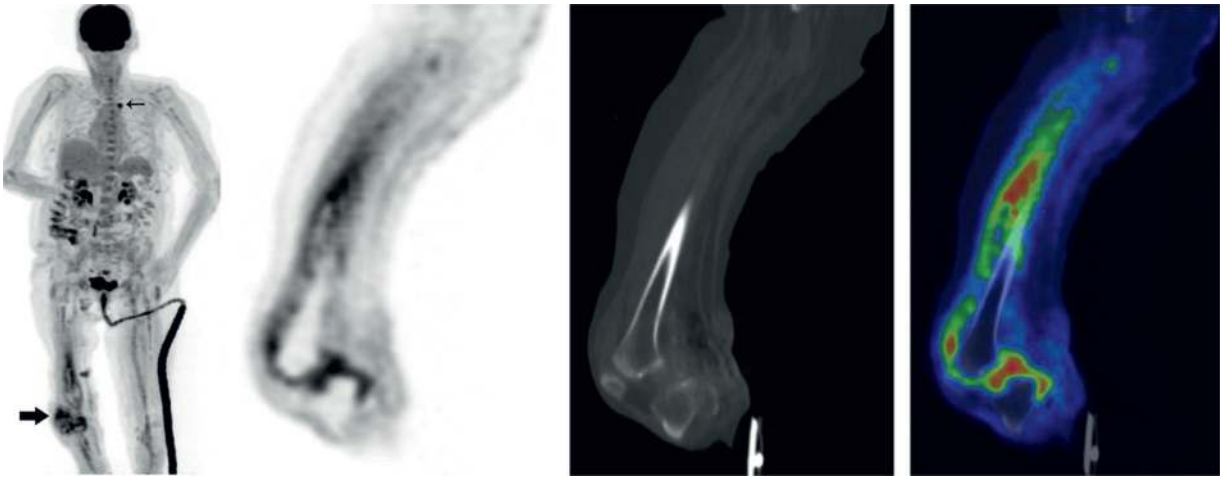

FIG 121.6 Fever of unknown origin (FUO) in a 75-year-old woman with negative blood cultures and normal magnetic resonance image (MRI) of the spine. The maximum intensity projection (MIP) positron emission tomography (PET) image on the left showed abnormal fluorodeoxyglucose (FDG) uptake at the right knee and thigh (*black thick arrow*). Note the focus of increased uptake related to the thoracic aortic arch (*thin arrow*). Sagittal PET (center) and noncontrast computed tomography (CT) (*left*) show increased FDG uptake in the knee joint, extending cranially through the suprapatellar bursa, deep to the quadriceps muscle (*arrow*). This was clinically confirmed as a septic arthritis and infective bursitis. This highlights the advantage of PET/CT in terms of whole-body coverage over CT and MRI. The focal uptake at the aortic arch was further investigated with CT and confirmed as a focal atheromatous plaque. (From Vaidyanathan S, Patel CN, Scarsbrook AF, Chowdhury FU. FDG PET/CT in infection and inflammation—current and emerging clinical applications. *Clin Radiol*. 2015;70(7):787–800.)

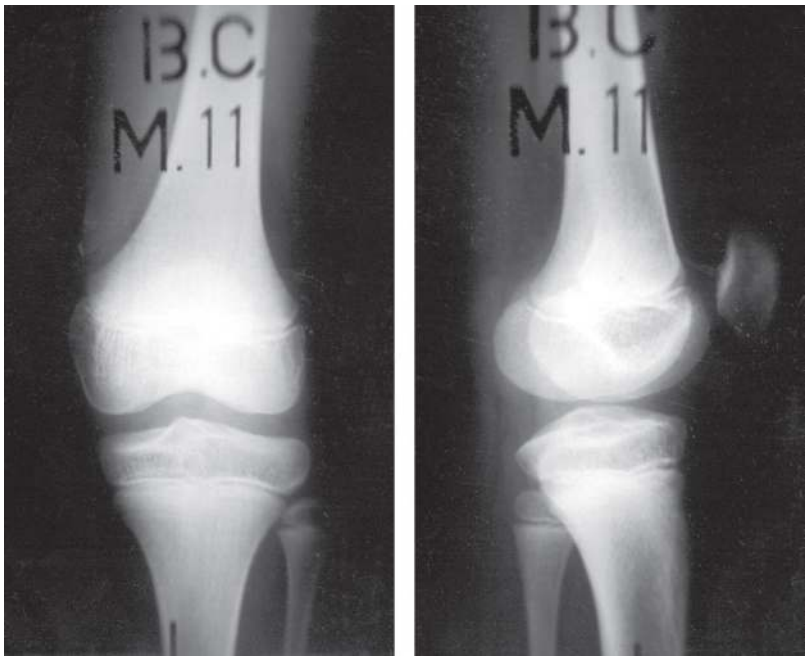

FIG 121.7 Arthroscopic views of rice bodies within the intraarticular space of a septic knee. (From Aşik M, Eralp L, Çetik O, et al. Rice bodies of synovial origin in the knee joint. *Arthrosc J Arthrosc Relat Surg*. 2001;17(5):14, Fig. 2.)

Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-induced complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of suprapatellar bursitis.

SUGGESTED READINGS

O'Keeffe SA, Hogan BA, Eustace SJ, et al. Overuse injuries of the knee. *Magn Reson Imaging Clin N Am*. 2009;17(4):725–739.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. Philadelphia: Saunders; 2009:318–322.

Waldman SD. Injection technique for suprapatellar bursitis. In: *Pain review*. Philadelphia: Saunders; 2009:584–585.

Waldman SD. Suprapatellar bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:780–785.

Waldman SD. Ultrasound-guided injection technique for suprapatellar bursitis. In: *Comprehensive atlas of ultrasound guided pain management injection techniques*. Philadelphia: Lippincott; 2014:810–816.

Waldman SD. *Comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:780–785.

Waldman SD. Suprapatellar bursitis and other disorders of the suprapatellar bursa. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:206–217.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308, 318–322.

Waldman SD. Injection technique for suprapatellar bursitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017.

Waldman SD. Suprapatellar bursa injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2017:523–526, 703–707.

Waldman SD. The suprapatellar bursa. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:303–304.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the knee. In: *Imaging of Pain*. Philadelphia, PA: Saunders; 2011:367–388.

Waldman SD, Campbell RSD. Anatomy: suprapatellar bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:401–402.

Prepatellar Bursitis

ICD-10 CODE M70.40

THE CLINICAL SYNDROME

The prepatellar bursa lies between the subcutaneous tissues and the patella. This bursa is held in place by the patellar ligament. The prepatellar bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs (Fig. 122.1). The prepatellar bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are caused by direct trauma to the bursa during falls onto the knee or patellar fracture. Overuse injuries may be caused by running on soft or uneven surfaces or jobs that require crawling or kneeling, such as carpet laying or scrubbing floors, hence the other name for prepatellar bursitis: housemaid's knee (Fig. 122.2). If inflammation of the prepatellar bursa becomes chronic, calcification may occur.

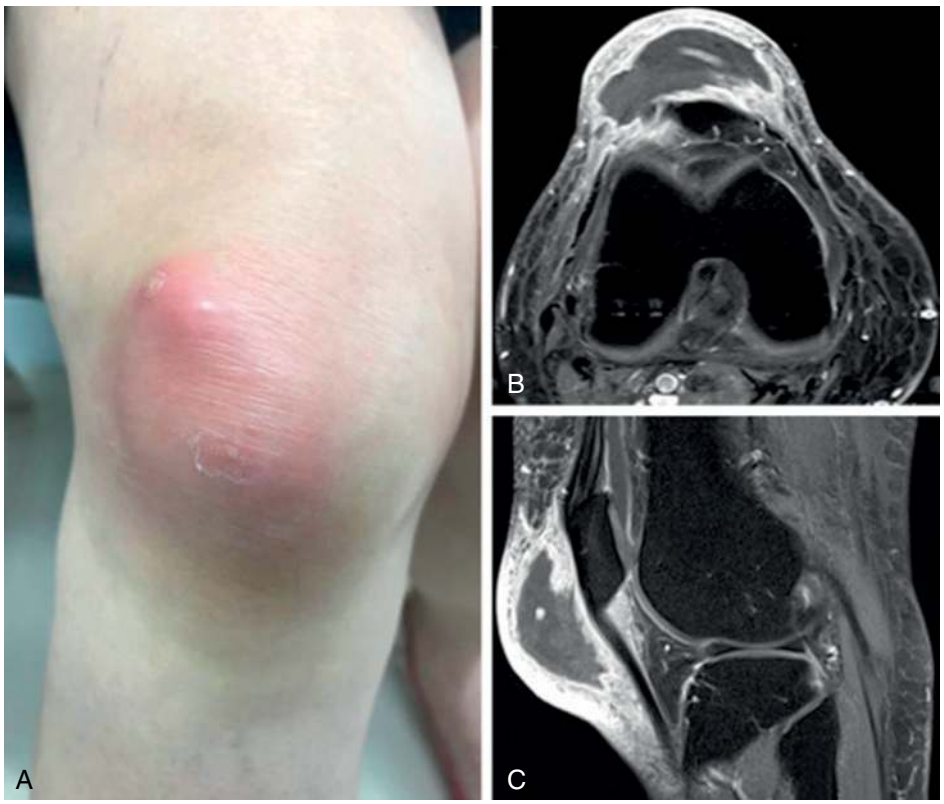

FIG 122.1 Septic prepatellar bursitis. **(A)** Physical examination of right knee revealed a well-defined erythematous mass (cellulitis-like) with draining sinus at the prepatellar area; **(B)** magnetic resonance imaging of the right knee demonstrated a well-defined rim enhancing fluid collection in the prepatellar area with no evidence of intrasynovial extension; **(C)** the sagittal view demonstrated the collection was locally confined to the prepatellar area. (Reproduced from Katwilat P, Chongtrakool P, Muangsomboon S, Jitmuang A. *Prototheca wickerhamii* prepatellar bursitis in an immunocompetent woman: a case report. *J Mycol Méd*. 2019;29(4):361–364. Copyright © 2019. Elsevier Masson SAS. All rights reserved.)

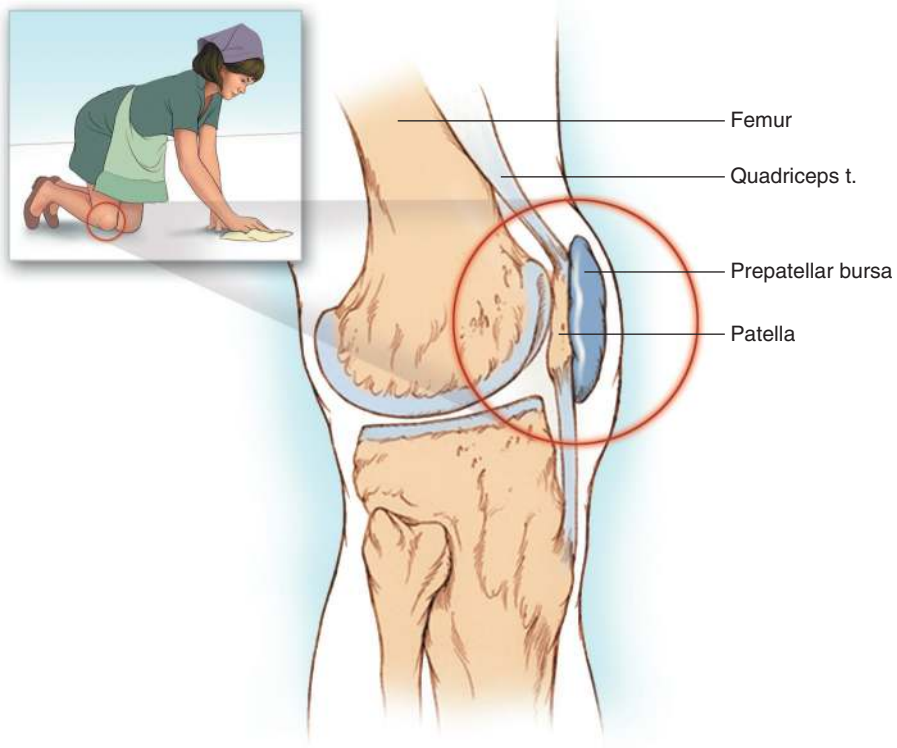

FIG 122.2 Prepatellar bursitis is also known as housemaid's knee because of its prevalence among people whose work requires prolonged crawling or kneeling.

SIGNS AND SYMPTOMS

Patients suffering from prepatellar bursitis complain of pain and swelling in the anterior knee over the patella that can radiate superiorly and inferiorly into the surrounding area (Fig. 122.3). Often, patients are unable to kneel or walk downstairs. A positive ballottement test may also be present (Fig. 122.4). Patients may also complain of a sharp "catching" sensation with range of motion of the knee, especially on first arising. Prepatellar bursitis often coexists with arthritis and tendinitis of the knee, which can confuse the clinical picture.

TESTING

Plain radiographs and magnetic resonance imaging (MRI) of the knee may reveal calcification of the bursa and associated structures, including the quadriceps tendon, consistent with chronic inflammation (Fig. 122.5). MRI is indicated if internal derangement, an occult mass, infection, or a tumor of the knee is suspected (Figs. 122.6 and 122.7). Ultrasound imaging can also help clarify the diagnosis (Figs. 122.8 and 122.9). Electromyography can distinguish prepatellar bursitis from femoral neuropathy, lumbar radiculopathy, and plexopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver. Antinuclear antibody testing is indicated if collagen vascular disease is suspected. If infection is a possibility, aspiration, Gram stain, and culture of bursal fluid should be performed on an emergency basis (Fig. 122.10).

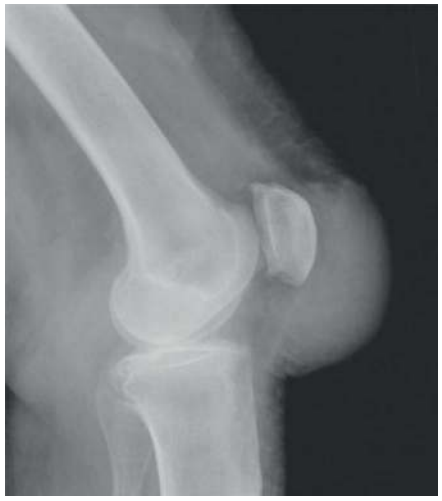

FIG 122.3 Lateral radiograph of the knee in a patient with significant anterior swelling of the knee with associated pain. (From Waldman SD, Campbell RSD. *Imaging of Pain*. Philadelphia: Elsevier; 2011.)

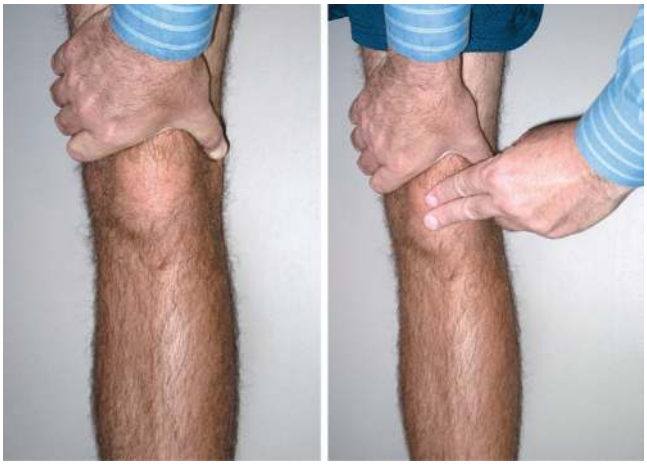

FIG 122.4 The ballottement test for large joint effusions. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. St Louis: Elsevier; 2016, Figure 203–1.)

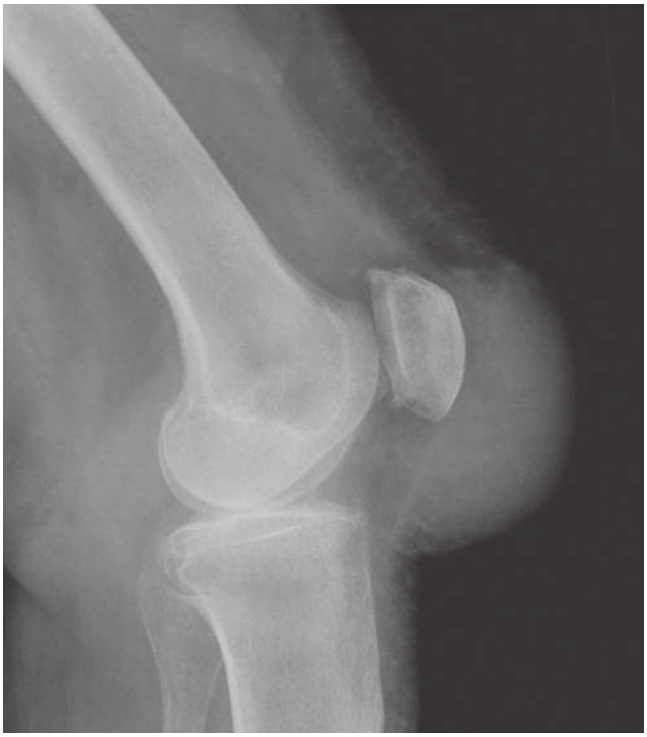

FIG 122.5 Lateral radiograph of the knee of a patient with an acute attack of gout. There is prominent prepatellar soft tissue swelling owing to gouty bursitis. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

DIFFERENTIAL DIAGNOSIS

Because of the anatomy of the region, the associated tendons and other bursae of the knee can become inflamed along with the prepatellar bursa, thus confusing the diagnosis. Both the quadriceps tendon and the prepatellar bursa are subject to inflammation from overuse, misuse, or direct trauma. The tendon fibers, called expansions, are vulnerable to strain, and the tendon proper is subject to the development of tendinitis. The suprapatellar, infrapatellar, and prepatellar bursae may also become inflamed with dysfunction of the quadriceps tendon. Anything that alters the normal biomechanics of the knee can result in inflammation of the prepatellar bursa.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs), or cyclooxygenase-2 inhibitors and a knee brace to prevent further trauma is the first step in the treatment of prepatellar bursitis. If patients do not experience rapid improvement, injection is a reasonable next step.

To perform the injection, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the patella is prepared with antiseptic solution. A sterile syringe containing 2-mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The center of the medial patella is identified. Just above this point, the needle is inserted horizontally to slide subcutaneously into the prepatellar bursa (Fig. 122.11). If the needle strikes the patella, it is withdrawn slightly and is redirected with a more anterior trajectory. When the needle is positioned in proximity to the prepatellar bursa, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

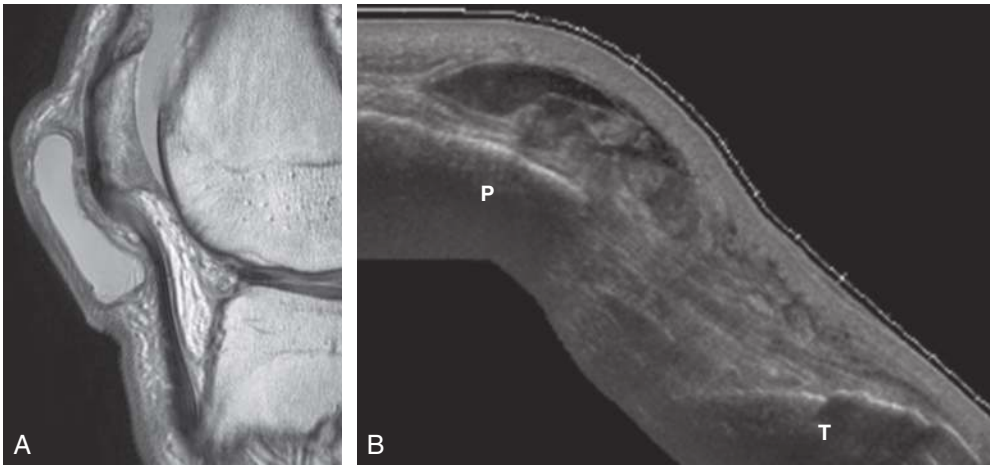

FIG 122.6 A, Sagittal T2-weighted magnetic resonance image showing prominent high–signal intensity (SI) fluid within the prepatellar bursa. There is also an advance osteoarthritic change in the patellofemoral joint. B, The corresponding longitudinal ultrasound image shows the extensive low-echo fluid collection. *P*, Patella; *T*, tibia. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

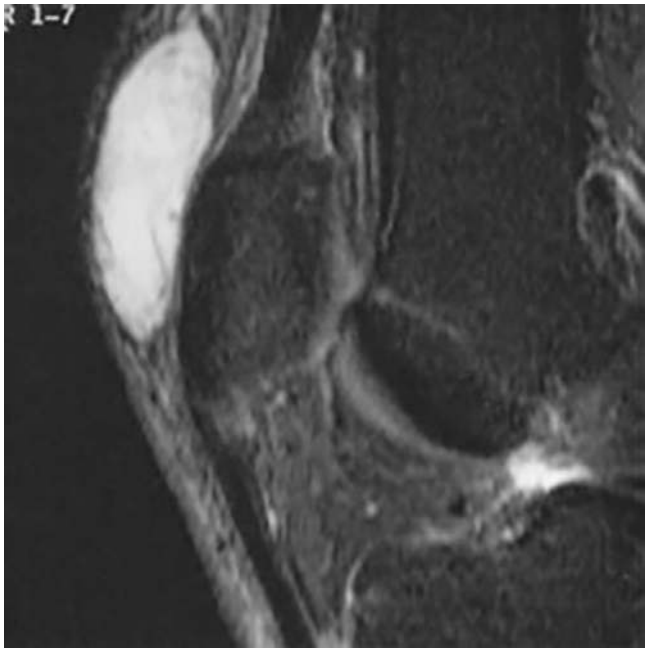

FIG 122.7 Prepatellar bursitis. A sagittal short tau inversion recovery (Repetition time (TR)/Time to echo (TE), 5300/30; inversion time, 150 ms) magnetic resonance image shows fluid and synovial tissue in the prepatellar bursa. (From Resnick D. Diagnosis of Bone and Joint Disorders. ed 4. Philadelphia, PA: Saunders; 2002.)

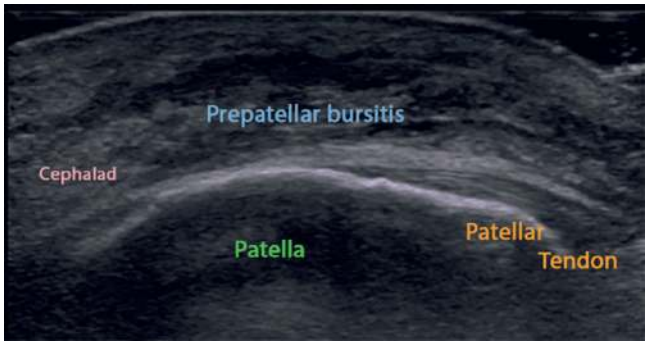

FIG 122.8 Longitudinal ultrasound image demonstrating a large prepatellar bursitis.

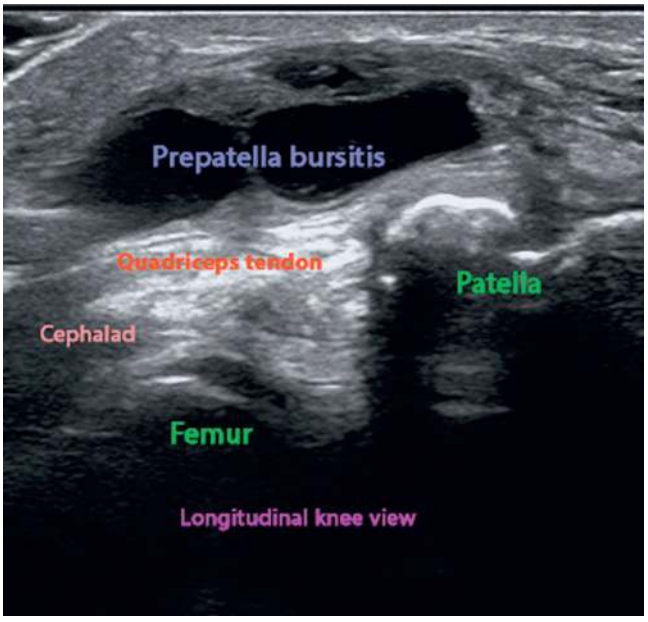

FIG 122.9 Longitudinal ultrasound image demonstrating prepatellar bursitis.

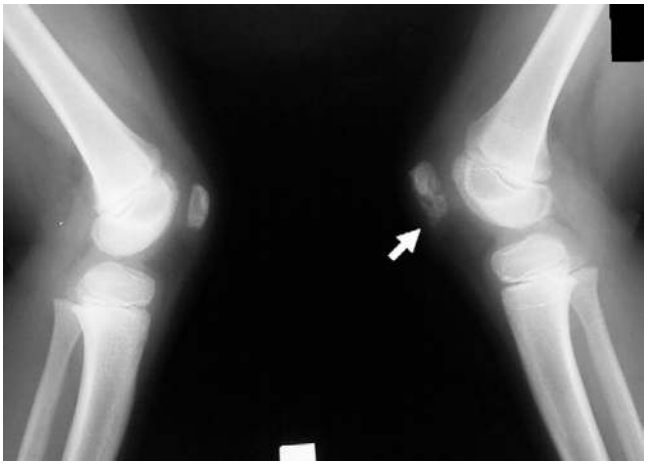

FIG 122.10 Radiograph of the knee in a patient suspected of having septic prepatellar bursitis depicts fragmentation and elongation of the patella consistent with osteomyelitis of the patella (*arrow*). (From Choi H-R. Patellar osteomyelitis presenting as prepatellar bursitis. *Knee*. 2007;14(4):333–335, Figure 5.)

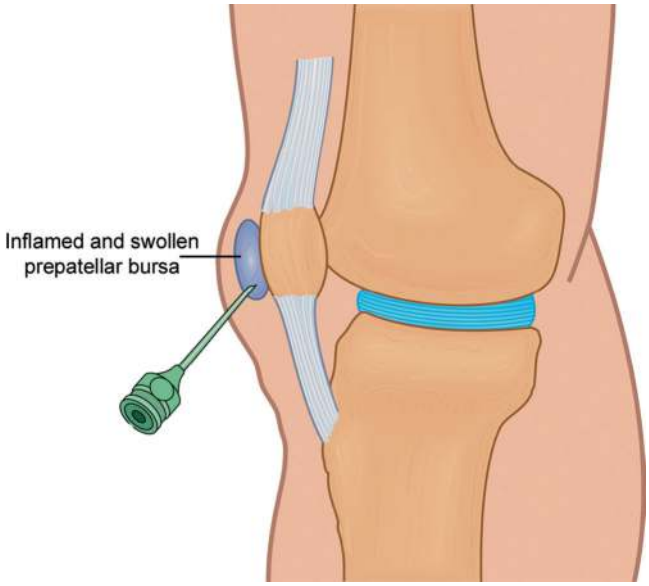

FIG 122.11 Correct needle placement for injection of the prepatellar bursa. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of prepatellar bursitis.

SUGGESTED READINGS

Choi H-R. Patellar osteomyelitis presenting as prepatellar bursitis. *Knee*. 2007;14(4):333–335.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin North Am*. 2013;51(3):433–454.

Waldman SD. Suprapatellar bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:786–791.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308.

Waldman SD. Prepatellar bursa injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:527–529.

Waldman SD, Campbell RSD. Prepatellar bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:405–406.

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Superficial Infrapatellar Bursitis

ICD-10 CODE M76.899

THE CLINICAL SYNDROME

The superficial infrapatellar bursa lies between the subcutaneous tissues and the upper part of the patellar ligament. This bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The superficial infrapatellar bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are caused by direct trauma to the bursa during falls onto the knee or patellar fracture. Overuse injuries are caused by running on soft or uneven surfaces or doing jobs that require crawling or kneeling, such as carpet laying or scrubbing floors (Fig. 123.1). If inflammation of the superficial infrapatellar bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Patients with superficial infrapatellar bursitis complain of pain and swelling in the anterior knee over the patella that can radiate superiorly and inferiorly into the surrounding area. Often, patients are unable to kneel or walk down stairs. Patients may also complain of a sharp "catching" sensation with range of motion of the knee, especially on first arising. Superficial infrapatellar bursitis often coexists with arthritis and tendinitis of the knee, which can confuse the clinical picture.

TESTING

Plain radiographs, ultrasound imaging, and magnetic resonance imaging (MRI) of the knee may reveal calcification of the bursa and associated structures, including the quadriceps and patellar tendons, findings consistent with chronic inflammation (Figs. 123.2 and 123.3). Other occult abnormalities may also be identified that may mimic the pain of infrapatellar bursitis (Fig. 123.4). MRI is indicated if internal derangement, an occult mass, or a tumor of the knee is suspected. Electromyography can distinguish superficial infrapatellar bursitis from femoral neuropathy, lumbar radiculopathy, and plexopathy. The injection technique

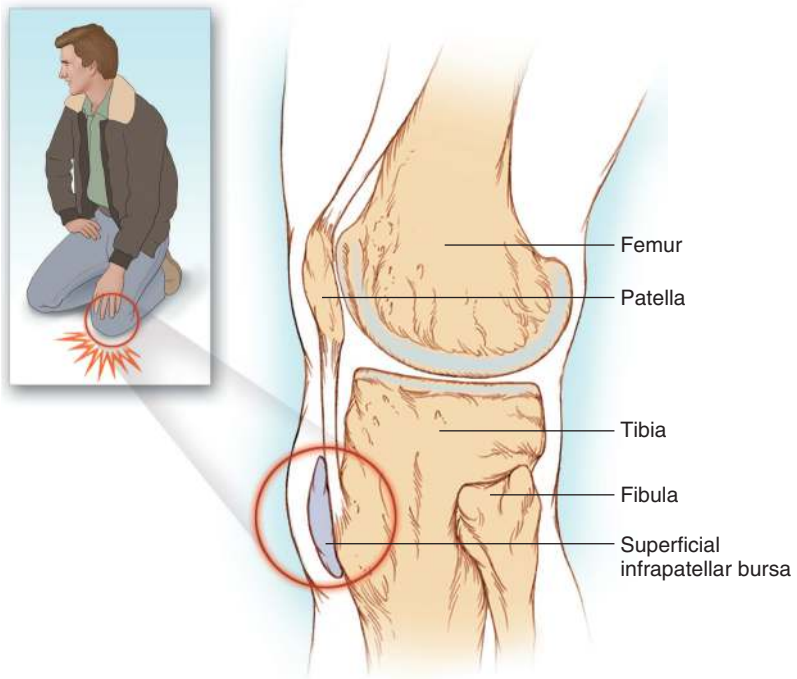

FIG 123.1 Infrapatellar bursitis is a common cause of inferior knee pain.

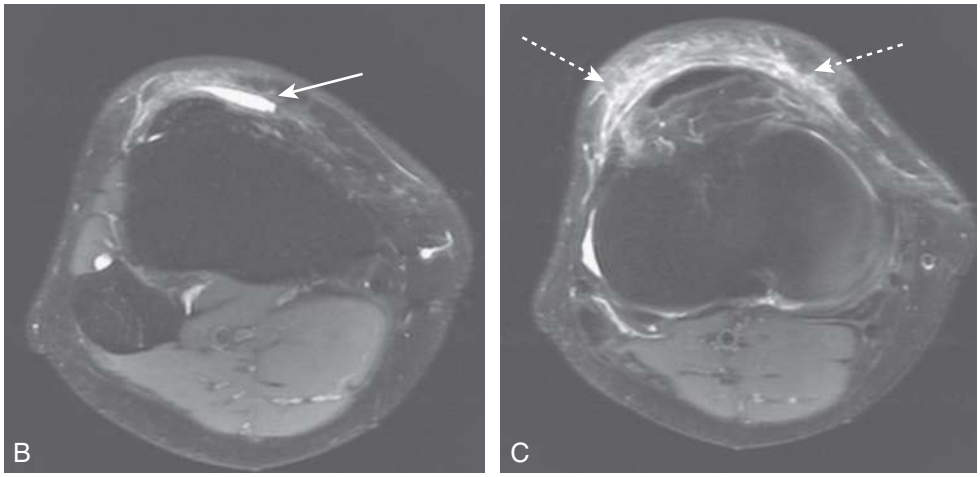

FIG 123.2 **A,** Sagittal T2-weighted with fat suppression (FST2W) magnetic resonance (MR) image demonstrating a small area of high–signal intensity (SI) fluid superficial to the distal patellar tendon and tibial tuberosity (*white arrow*). **B,** This area of fluid is also evident on the axial FST2W MR image. A small amount of fluid may be a normal finding. **C,** In this case, however, more extensive diffuse high-SI edema (*broken white arrows*) is demonstrated in the adjacent soft tissues on the proximal axial FST2W MR image, representing a diffuse adventitial bursitis. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

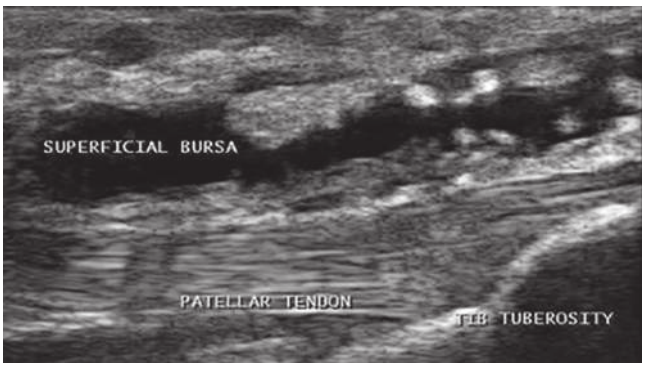

FIG 123.3 Ultrasound image of the knee joint demonstrating the superficial infrapatellar bursa.

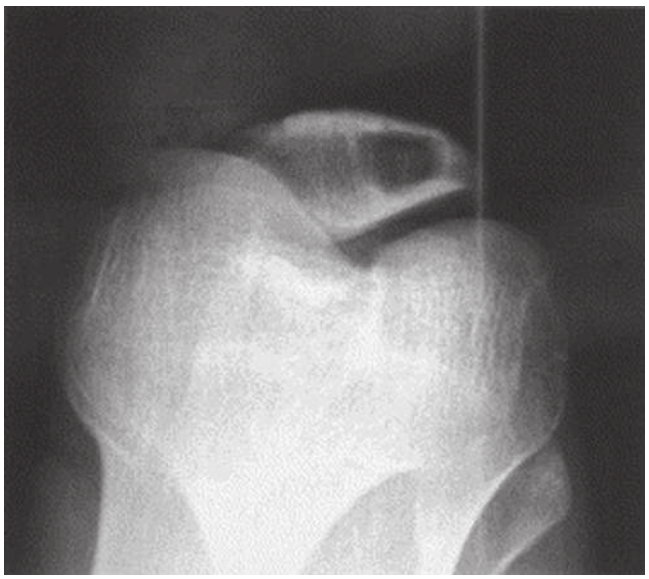

FIG 123.4 Skyline view of the knee showing a well-defined contained osteolytic lesion in the patella with a sequestrum consistent with a diagnosis of tuberculosis of the patella. (From Mittal R, Trikha V, Rastogi S. Tuberculosis of patella. *Knee*. 2006;13(1):54–56.)

described later serves as both a diagnostic and a therapeutic maneuver. Antinuclear antibody testing is indicated if collagen vascular disease is suspected. If infection is a possibility, aspiration, Gram stain, and culture of bursal fluid should be performed on an emergency basis.

DIFFERENTIAL DIAGNOSIS

Because of the anatomy of the region, the associated tendons and other bursae of the knee can become inflamed along with the superficial infrapatellar bursa, thus confusing the diagnosis. Both the quadriceps tendon and the superficial infrapatellar bursa are subject to inflammation from overuse, misuse, or direct trauma. The tendon fibers, called expansions, are vulnerable to strain, and the tendon proper is subject to the development of tendinitis. The infrapatellar fat pad is also subject to various abnormalities including Hoffa's disease, which can mimic the pain of superficial infrapatellar bursitis. The suprapatellar, infrapatellar, and prepatellar bursae may also become inflamed with dysfunction of the quadriceps tendon. Anything that alters the normal biomechanics of the knee can result in inflammation of the superficial infrapatellar bursa.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs)

or cyclooxygenase-2 inhibitors, and a knee brace to prevent further trauma is the first step in the treatment of superficial infrapatellar bursitis. If patients do not experience rapid improvement, injection is a reasonable next step.

To inject the superficial infrapatellar bursa, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the patella is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The center of the lower pole of the patella is identified. Just below this point, the needle is inserted at a 45-degree angle to slide subcutaneously into the superficial infrapatellar bursa. If the needle strikes the patella, it is withdrawn slightly and is redirected with a more inferior trajectory. When the needle is positioned in proximity to the superficial infrapatellar bursa, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of superficial infrapatellar bursitis.

SUGGESTED READINGS

Choi H-R. Patellar osteomyelitis presenting as prepatellar bursitis. *Knee*. 2007;14(4):333–335.

Hong E, Kraft MC. Evaluating anterior knee pain. *Med Clin North Am*. 2014;98(4):697–717.

Lormeau C, Cormier G, Sigaux J, et al. Management of septic bursitis. *Joint Bone Spine*. 2019;86(5):583–588. https://doi.org/10.1016/j.jbspin.2018.10.006.

Naredo E, Canoso JJ, Yinh J, et al. Dynamic changes in the infrapatellar knee structures with quadriceps muscle contraction. An in vivo study. *Ann Anat*. 2021;235:151663. https://doi.org/10.1016/j.aanat.2020.151663.

O'Keeffe SA, Hogan BA, Eustace SJ, et al. Overuse injuries of the knee. *Magn Reson Imaging Clin N Am*. 2009;17(4):725–739.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin North Am*. 2013;51(3):433–454.

Vicentini JRT, Chang CY. MR imaging of the knee bursae and bursal pathology. *Magn Reson Imaging Clin N Am*. 2022;30(2):241–260. https://doi.org/10.1016/j.mric.2021.11.005.

Waldman SD. Superficial infrapatellar bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:792–798.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308.

Waldman SD. Superficial infrapatellar bursa injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:713–717.

Waldman SD, Campbell RSD. Superficial infrapatellar bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:407–408.

Deep Infrapatellar Bursitis

ICD-10 CODE M76.899

THE CLINICAL SYNDROME

The deep infrapatellar bursa lies between the patellar ligament and the tibia. This bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The deep infrapatellar bursa is vulnerable to injury from both acute trauma and repeated microtrauma. Acute injuries are caused by direct trauma to the bursa during falls onto the knee (Fig. 124.1) or patellar fracture. Overuse injuries are caused by running on soft or uneven surfaces or jobs that require crawling and kneeling, such as carpet laying or scrubbing floors. If inflammation of the deep infrapatellar bursa becomes chronic, calcification may occur.

SIGNS AND SYMPTOMS

Patients with deep infrapatellar bursitis complain of pain and swelling in the anterior knee below the patella that can radiate inferiorly into the surrounding area. Often, patients are unable to kneel or walk downstairs. They may also complain of a sharp "catching" sensation with range of motion of the knee, especially on first arising. Infrapatellar bursitis often coexists with arthritis and tendinitis of the knee, which can confuse the clinical picture.

Physical examination may reveal point tenderness in the anterior knee just below the patella. Swelling and fluid accumulation surrounding the lower patella are often present (Fig. 124.2). Passive flexion and actively resisted extension of the knee reproduce the pain. Sudden release of resistance during this maneuver causes a marked increase in pain. The deep infrapatellar bursa is not as susceptible to infection as the superficial infrapatellar bursa.

TESTING

Plain radiographs, ultrasound, and magnetic resonance imaging (MRI) of the knee may reveal calcification of the bursa

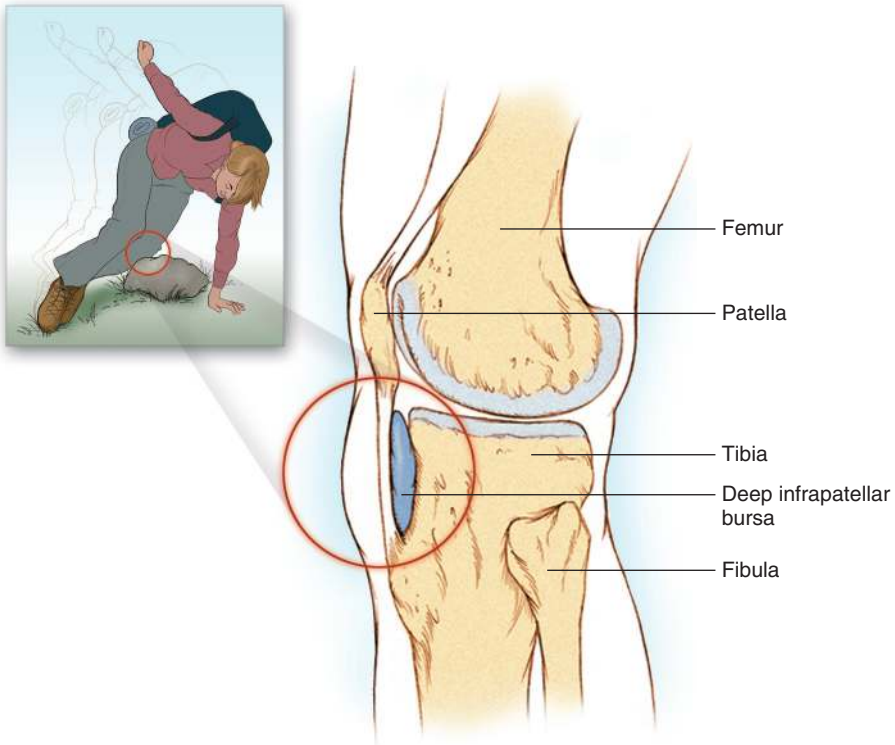

FIG 124.1 Deep infrapatellar bursitis may result from direct trauma, such as falling on the knee.

and associated structures, including the quadriceps tendon, findings consistent with chronic inflammation (Figs. 124.3 and 124.4). MRI and ultrasound imaging are indicated if internal derangement, an occult mass, or a tumor of the knee is suspected as well as to identify other causes of the patient's knee pain (Fig. 124.5). Electromyography can distinguish deep infrapatellar bursitis from femoral neuropathy, lumbar radiculopathy, and plexopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver. Antinuclear antibody testing is indicated if collagen vascular disease is suspected. If infection is a possibility, aspiration, Gram stain, and culture of bursal fluid should be performed on an emergency basis.

DIFFERENTIAL DIAGNOSIS

Because of the anatomy of the region, the associated tendons and other bursae of the knee can become inflamed along with the deep infrapatellar bursa, thus confusing the diagnosis. Both the quadriceps tendon and the deep infrapatellar bursa are subject to inflammation from overuse, misuse, or direct trauma. The tendon fibers, called expansions, are vulnerable to strain, and the tendon proper is subject to the development of tendinitis. The suprapatellar, infrapatellar, and prepatellar bursae may also become inflamed with dysfunction of the quadriceps tendon. Anything that alters the normal biomechanics of the knee can result in inflammation of the deep infrapatellar bursa.

FIG 124.2 Infrapatellar bursitis is caused by direct trauma to the knee as well as overuse injuries caused by running on soft or uneven surfaces or by crawling or kneeling. (From Steven D. Waldman, Chapter 9 - Arif Abad: A 29-Year-Old Electrical Engineer With Severe Left Knee and Upper Leg Pain and Swelling, ed. Steven D. Waldman, The Knee. Elsevier, 2022:116–127.)

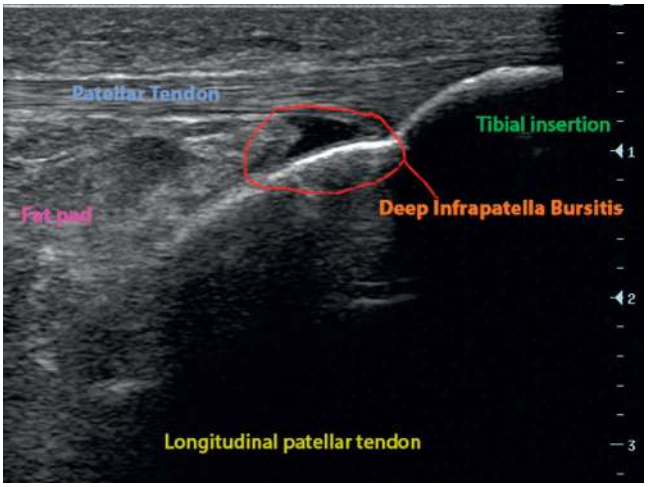

FIG 124.4 Longitudinal ultrasound image of the knee joint demonstrating the deep infrapatellar bursitis.

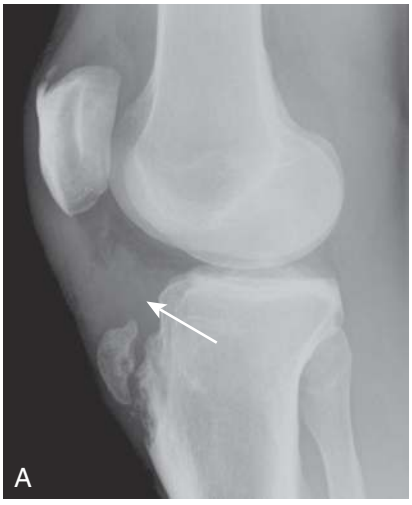

FIG 124.3 A, Lateral radiograph of a patient with chronic insertional tendinopathy of the patellar tendon with nonfused tibial apophysis. There is soft tissue shadowing deep to the distal patellar tendon (*white arrow*) that partly obliterates the Hoffa fat pad. The sagittal (B) and axial (C) T2-weighted with fat suppression magnetic resonance images clearly show the high–signal intensity (SI) fluid within the deep infrapatellar bursa (*white arrows*). There is also high-SI marrow edema within the unfused apophysis and adjacent tibia (*broken white arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier.)

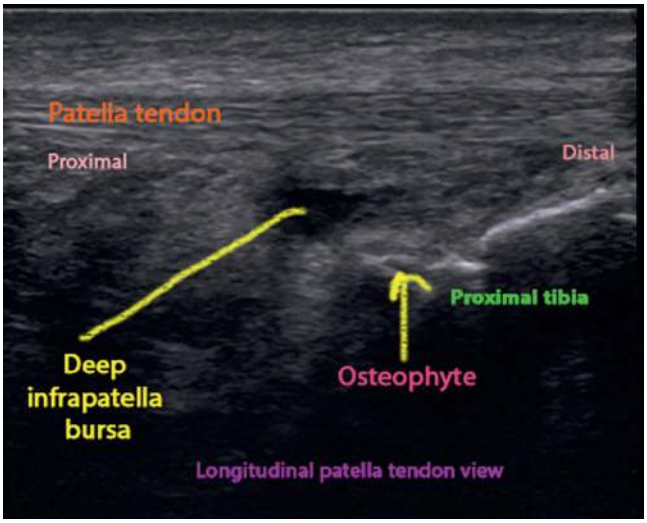

FIG 124.5 Longitudinal ultrasound image demonstrating bursitis of the deep infrapatellar bursa. *Note*: The osteophyte is just distal to the bursa that may be serving as the nidus for the inflammatory process.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors, and a knee brace to prevent further trauma is the first step in the treatment of deep infrapatellar bursitis. If patients do not experience rapid improvement, injection is a reasonable next step.

To inject the deep infrapatellar bursa, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin overlying the medial portion of the lower margin of the patella is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle by using strict aseptic technique. The medial lower margin of the patella is identified. Just below this point, the needle is inserted at a right angle to the patella to slide beneath the patellar ligament into the deep infrapatellar bursa. If the needle strikes the patella, it is withdrawn slightly and is redirected with a more inferior trajectory. When the needle is positioned in proximity to the deep infrapatellar bursa, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of deep infrapatellar bursitis.

SUGGESTED READINGS

Blankenbaker DG, Davis KW, Soninn A, et al. Deep infrapatellar bursitis. In: *Diagnostic imaging: musculoskeletal trauma*. 2nd ed. Philadelphia: Elsevier; 2016:840–841. https://doi.org/10.1016/B978-0-323-39253-2.50198-1.

Lormeau C, Cormier G, Sigaux J, et al. Management of septic bursitis. *Joint Bone Spine*. 2019;86(5):583–588. https://doi.org/10.1016/j.jbspin.2018.10.006.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin N Am*. 2013;51(3):433–454.

Waldman SD. Deep infrapatellar bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:799–804.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308.

Waldman SD. Deep infrapatellar bursa injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:533–535.

Waldman SD, Campbell RSD. Deep infrapatellar bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:408–410.

Osgood-Schlatter Disease

ICD-10 CODE M92.50

THE CLINICAL SYNDROME

Osgood-Schlatter disease is characterized by anterior knee pain that is exacerbated by stress on the quadriceps mechanism and by direct pressure on the tibial tuberosity. Although the disease can affect all ages, most cases occur in adolescents, with a peak incidence at approximately 13 years of age. Boys and men are affected two to three times more often than girls and women, although some investigators believe that the number of female cases is on the rise as a result of increased female participation in competitive sports. The pain and functional disability associated with Osgood-Schlatter disease is bilateral in 25%–30% of patients, and one side often has more severe symptoms. Osgood-Schlatter disease is usually the result of overuse or misuse of the knee joint caused by running, jumping, or overtraining on hard surfaces, as well as any other activities that require repetitive quadriceps contraction (Fig. 125.1). Competitive sports are most often implicated in the development of Osgood-Schlatter disease include soccer, gymnastics, basketball, ballet, track, hockey, baseball, and Irish and Scottish Highland–style dancing (Box 125.1).

The quadriceps tendon is made up of fibers from the four muscles that constitute the quadriceps muscle: vastus lateralis, vastus intermedius, vastus medialis, and rectus femoris. These muscles are the primary extensors of the lower extremity at the knee. The tendons of these muscles converge and unite to form a single, exceedingly strong tendon. The patella functions as a sesamoid bone within the quadriceps tendon, with fibers of the tendon expanding around the patella and forming the medial and lateral patella retinacula, which strengthen the knee joint. The patellar tendon extends from the patella to the tibial tuberosity. The tibial tuberosity is the nidus of the pain and functional disability associated with Osgood-Schlatter disease because the repetitive stresses applied to the tibial tuberosity by contraction of the quadriceps mechanism result in apophysitis and heterotopic bone growth. These responses to the damage induced by repetitive stress are most often seen during the period of rapid skeletal growth associated with adolescence, although as mentioned earlier, this disease has been reported in all age groups.

SIGNS AND SYMPTOMS

Patients with Osgood-Schlatter disease present with pain over the anterior knee and with pressure on the tibial tuberosity. Patients note increased pain on walking down slopes or up and downstairs, as well as during any activity that involves contraction of the quadriceps mechanism. Activity using the knee makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. On physical examination, the patient notes significant pain on palpation of the tibial tuberosity, as well as tenderness to palpation of the patellar tendon. Enlargement of the tibial tuberosity is often readily apparent, and this cosmetic defect can cause significant anxiety in the patient and parents. Generalized swelling of the joint may be present, and thickening of the patellar tendon may be appreciated on careful physical examination. Actively resisted extension of the knee reproduces the pain, as does pressure on the tibial tuberosity. Coexistent suprapatellar and infrapatellar bursitis, tendinitis, arthritis, or internal derangement of the knee may confuse the clinical picture after trauma to the knee.

BOX 125.1 Sports Commonly Associated With Osgood-Schlatter Disease

- Soccer
- Gymnastics
- Basketball
- Baseball
- Track
- Hockey
- Ballet
- Irish-style line dancing
- Scottish Highland–style dancing

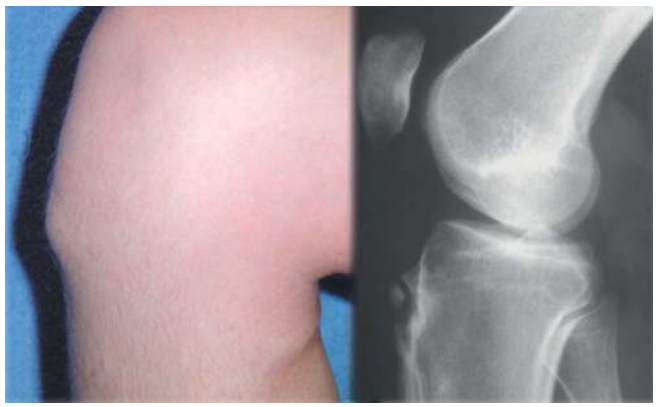

FIG 125.1 Osgood-Schlatter disease. (From Macnicol MF. Paediatric knee problems. *Orthop Trauma*. 2010;24(5):369–380.)

TESTING

Plain radiographs are indicated in all patients who present with knee pain and who are suspected of suffering from Osgood-Schlatter disease (Fig. 125.2). Magnetic resonance imaging of the knee is indicated if Osgood-Schlatter disease is suspected, because it readily detects any disorder of the patellar tendon, as well as the condition of the tibial tuberosity (Fig. 125.3). Ultrasound imaging may also provide beneficial information regarding the vascularity and integrity of the patellar tendons and the presence of tibial tuberosity abnormalities (Figs. 125.4–125.6). Bone scan may be useful to identify occult stress fractures involving the joint, especially if trauma has occurred. Based on the patient's clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Osgood-Schlatter disease is a repetitive stress disorder that is a distinct clinical entity from tendinitis of the patellar tendons or quadriceps expansion syndrome. However, such tendinitis, bursitis, and other painful conditions that affect the anterior knee may coexist with Osgood-Schlatter disease and may confuse the clinical picture. Diseases that may mimic Osgood-Schlatter diseases are listed in Box 125.2. Quadriceps expansion syndrome has a predilection for the medial side of the superior pole of the patella. The quadriceps tendon is also subject to acute calcific tendinitis, which may coexist with acute strain injuries and the more chronic changes of Osgood-Schlatter disease. Calcific tendinitis of the quadriceps has a characteristic radiographic appearance of whiskers on the anterosuperior patella. The suprapatellar, infrapatellar, and prepatellar bursae also may become inflamed with dysfunction of the quadriceps tendon. Hoffa's syndrome, which is a disease affecting the infrapatellar fat pad, may also coexist with Osgood-Schlatter disease.

TREATMENT

Initial treatment of the pain and functional disability associated with Osgood-Schlatter disease includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and rest, with the patient avoiding activities that involve contraction of the quadriceps mechanism. The use of therapeutic cold may also provide symptom relief. Gentle physical therapy consisting of stretching of the quadriceps mechanism and the opposing hamstring muscles should be implemented as the patient's symptoms allow. A nighttime splint and knee pads to protect the knee, as well as the use of an infrapatellar strap during activity, may also help relieve symptoms. For patients who do not respond to these treatment modalities, injection of the tibial tuberosity with local anesthetic and steroid is a reasonable next step.

To perform the injection, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The patellar tendon is identified, and the skin overlying the medial portion of the lower margin of the patella is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The medial lower margin of the patella is identified. Just below this point, the needle is inserted at a right angle to the patella to slide beneath the patellar ligament into the region of the deep infrapatellar bursa. If the needle strikes the patella, it is withdrawn slightly and is redirected with a more inferior trajectory. When the needle is in position in proximity to the region beneath the patellar tendon, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

If the foregoing modalities fail to relieve the patient's symptoms, some experts recommend complete rest of the affected knee by application of a cast for a 4- to 6-week period. In recalcitrant cases, excision of the tibial tuberosity and associated ossicles may be required.

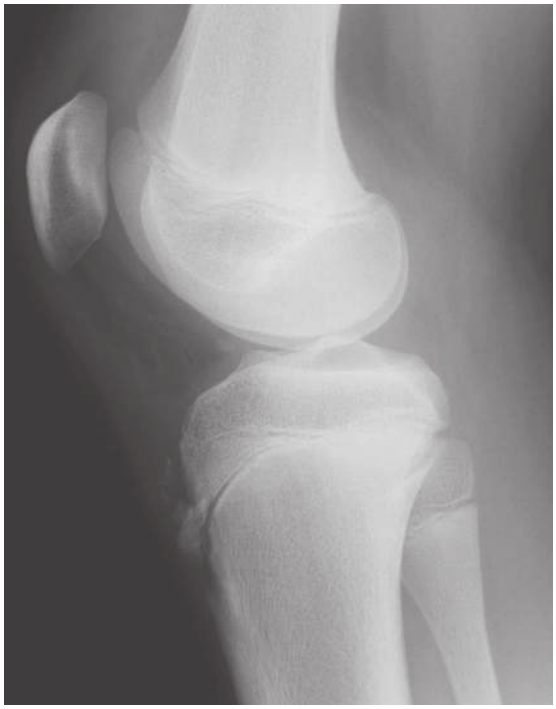

FIG 125.2 Lateral radiograph of an adolescent with anterior knee pain, demonstrating fragmentation of the tibial tuberosity caused by Osgood-Schlatter disease. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier.)

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if

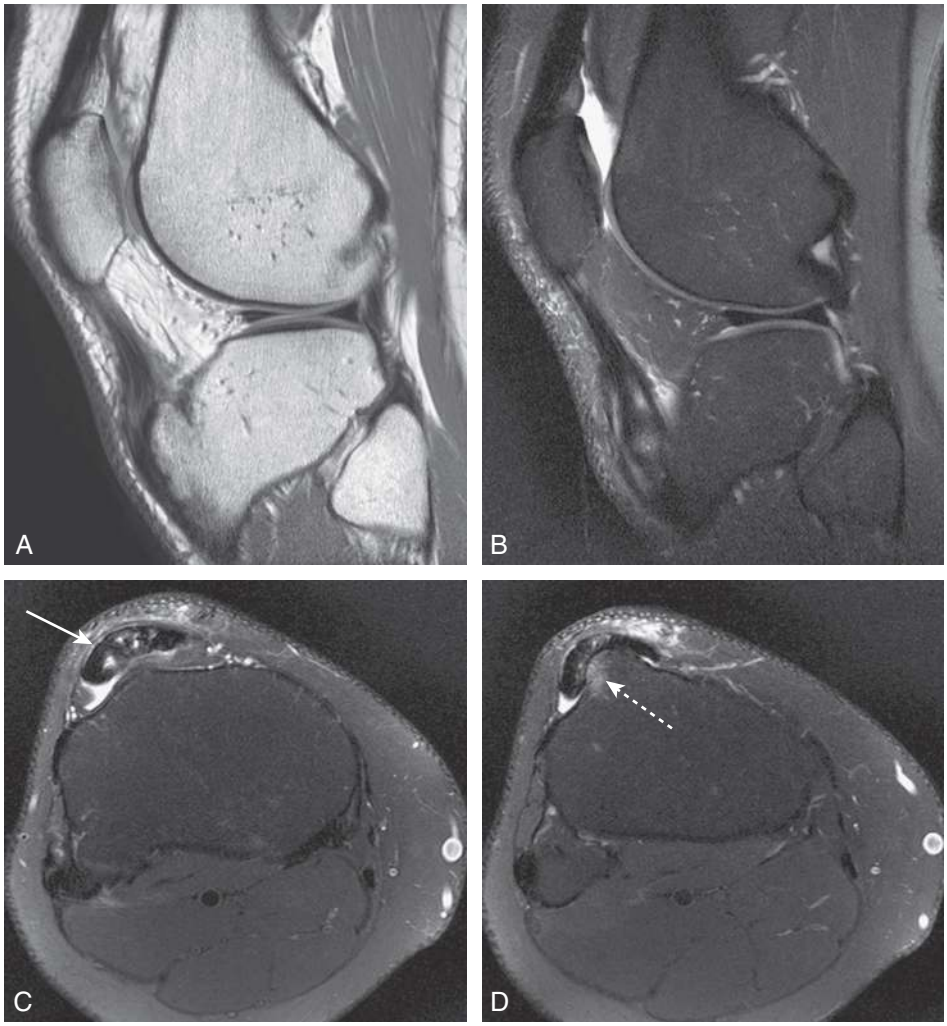

FIG 125.3 **A,** Sagittal proton density magnetic resonance (MR) image of a young adult with anterior knee pain. The tibial tuberosity is prominent as a result of adolescent Osgood-Schlatter disease. **B,** The sagittal T2-weighted with fat suppression (FST2W) MR image shows associated increased Signal Intensity (SI) within a thickened distal patellar tendon. The axial FST2W MR images **(C** and **D)** also show the tendon changes (*white arrow*) and the marrow edema in the tibial tuberosity (*broken white arrow*) owing to chronic insertional tendinopathy. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier.)

BOX 125.2 Differential Diagnosis for Osgood-Schlatter Disease

- Sinding-Larsen-Johansson syndrome
- Legg-Perthes disease
- Jumper's knee
- Superficial infrapatellar bursitis
- Deep infrapatellar bursitis
- Prepatellar bursitis
- Septic arthritis
- Cellulitis
- Quadriceps tendinopathy
- Chondromalacia patellae
- Patellar tendonitis
- Osteogenic sarcoma
- Soft-tissue malignancy
- Accessory ossicles
- Synovitis
- Tibial plateau fractures
- Anterior cruciate ligament injuries
- Osteomyelitis of the patella
- Osteomyelitis of the tibia
- Hoffa's syndrome
- Foreign body
- Synovial plica injury
- Tibial tubercle fracture
- Proximal tibiofibular joint disorders
- Patellofemoral joint disorders

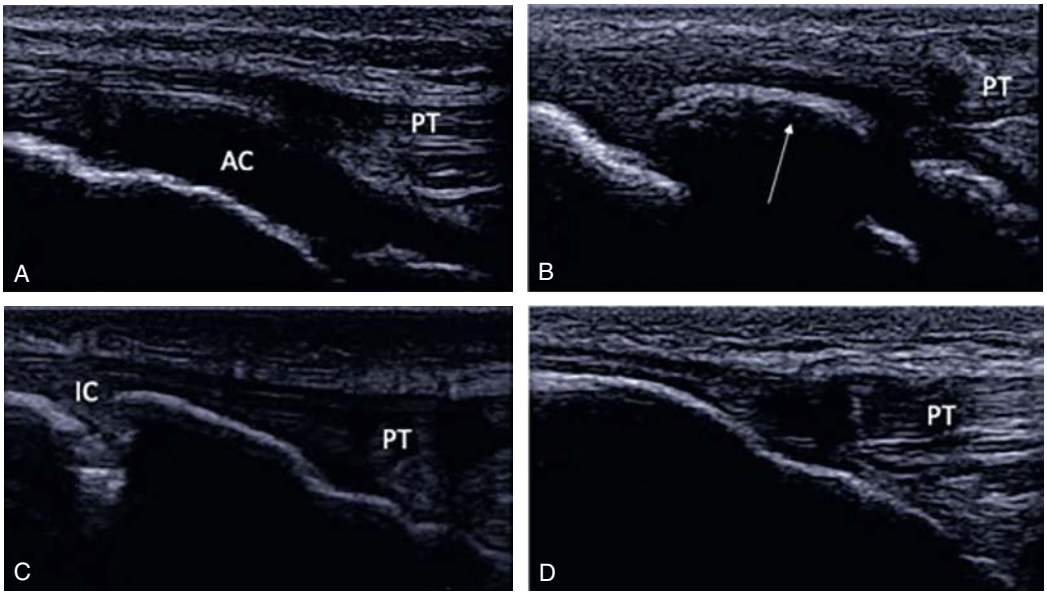

FIG 125.4 Sagittal view of ultrasonographic characteristics of the tibial tuberosity. **A,** Cartilaginous stage: characterized by a large amount of apophyseal cartilage (AC) without a secondary ossification center. **B,** Apophyseal stage: characterized by secondary ossification centers (*arrow*) in the apophysis. **C,** Epiphyseal stage: characterized by the patellar tendon (PT) attaching to the bone surface and a thin layer of insertional cartilage (IC) is still present. **D,** Bony stage: characterized by the patellar tendon attaching to the tubercle and the absence of any apophyseal cartilage. (From Schultz M, Tol JL, Veltman L, et al. Osgood-Schlatter Disease in youth elite football: minimal time-loss and no association with clinical and ultrasonographic factors. *Phys Ther Sport*. 2022;55:98–105.)

FIG 125.5 Osgood-Schlatter disease. **A,** Comparative sagittal ultrasound scans: cartilage swelling can be seen in the right tibial apophysis (*arrows*). **B,** Comparative sagittal ultrasound scans: fragmentation of the right tibial tubercle ossification center. **C,** The sagittal ultrasound scan shows reactive bursitis of the deep tibial patellar bursa (*arrows*), besides fragmentation of the tibial tubercle ossification center. (From Draghi F, Danesino GM, Coscia D, et al. Overload syndromes of the knee in adolescents: sonographic findings. *J Ultrasound*. 2008;11(4):151–157.)

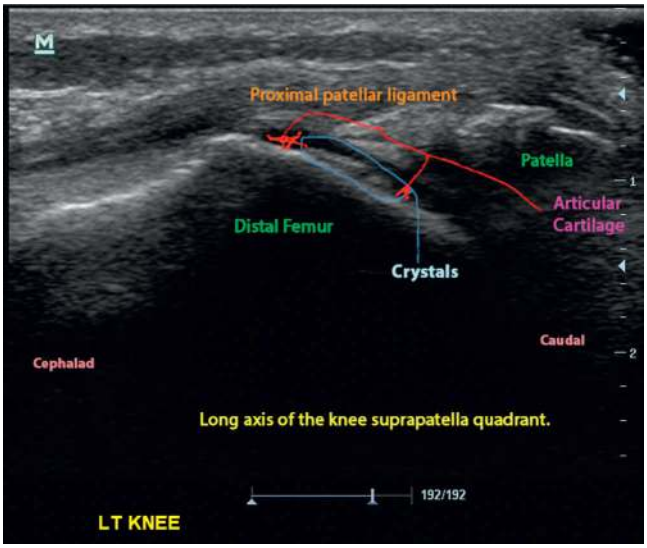

FIG 125.6 Longitudinal ultrasound image demonstrating crystal deposition disease affecting the patellar tendon.

careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although it should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of Osgood-Schlatter disease. Patients and their families often require repeated reassurance regarding the enlargement of the tibial tuberosity and clear statements that such enlargement is definitely not cancer. Such reassurance is the only thing that will lessen their anxiety regarding the common cause of anterior knee pain.

SUGGESTED READINGS

Belhaj K, Meftah S, Lahrabli S, et al. Osgood-Schlatter and patellar instability: fortuitous association or complication? *Ann Phys Rehabil Med*. 2014;57(suppl 1):e275.

Elangovan SM, Meyers AB. Pearls and Pitfalls of imaging of the developing pediatric skeleton: differentiating normal and pathology with MRI. *Semin Ultrasound CT MRI*. 2022;43(1):73–87. https://doi.org/10.1053/j.sult.2021.05.008.

Hong E, Kraft MC. Evaluating anterior knee pain. *Med Clin N Am*. 2014;98(4):697–717.

Neuhaus C, Appenzeller-Herzog C, Faude O. A systematic review on conservative treatment options for OSGOOD-Schlatter disease. *Phys Ther Sport*. 2021;49:178–187. https://doi.org/10.1016/j.ptsp.2021.03.002.

Schultz M, Tol JL, Veltman L, et al. Osgood-Schlatter disease in youth elite football: minimal time-loss and no association with clinical and ultrasonographic factors. *Phys Ther Sport*. 2022;55:98–105. https://doi.org/10.1016/j.ptsp.2022.02.024.

Talawadekar GD, Mostofi B, Housden P. Unusually large sized bony ossicle in Osgood-Schlatter disease. *Eur J Radiol Extra*. 2009;69(1):e37–e39.

Waldman SD. Osgood-Schlatter disease. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:810–819.

Waldman SD, Campbell RSD. Osgood-Schlatter disease. In: *Imaging of pain*. Philadelphia: Saunders; 2011:408–410.

Baker's Cyst of the Knee

ICD-10 CODE M70.20

THE CLINICAL SYNDROME

When bursae become inflamed, they may overproduce synovial fluid, which can become trapped in a saclike cyst. Because of a one-way valve effect, this cyst gradually expands. Baker's cyst is the result of an abnormal accumulation of synovial fluid in the medial aspect of the popliteal fossa. Often, a tear of the medial meniscus or tendinitis of the medial hamstring is responsible for the development of Baker's cyst. Patients suffering from rheumatoid arthritis are especially susceptible to the development of Baker's cysts.

SIGNS AND SYMPTOMS

Patients with Baker's cysts complain of a feeling of fullness behind the knee (Fig. 126.1). They may notice a lump behind the knee that becomes more apparent when they flex the knee. The cyst may continue to enlarge and dissect inferiorly into the calf (Fig. 126.2). Patients suffering from rheumatoid

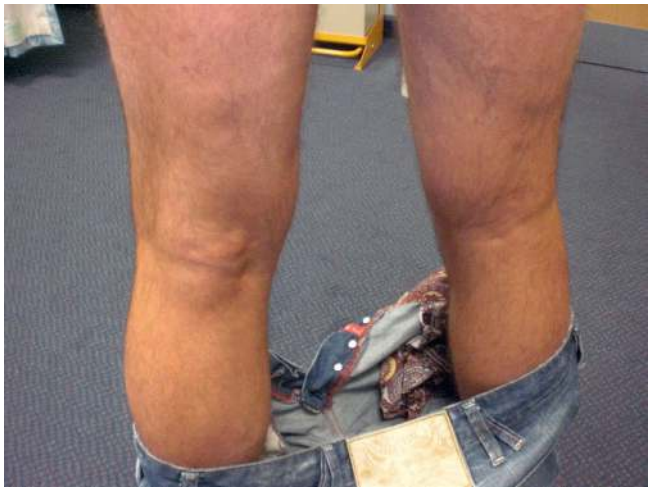

FIG 126.1 Classic appearance of a Baker's cyst. (From Ali F. Clinical examination of the knee. *Orthop Trauma*. 2013;27(1): 50–55.)

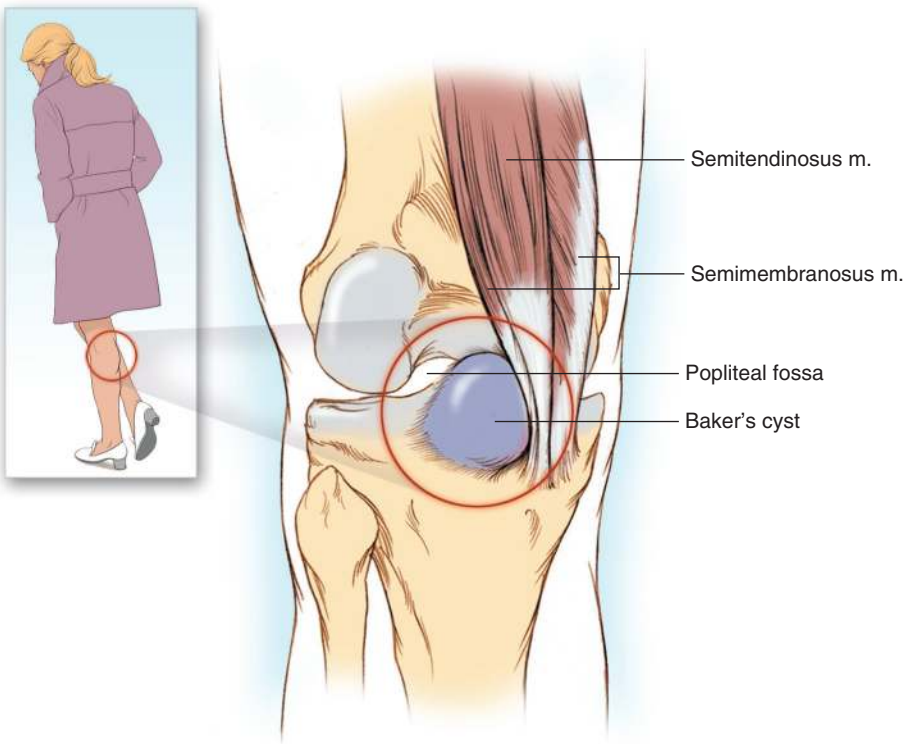

FIG 126.2 Patients with Baker's cyst often complain of a sensation of fullness or a lump behind the knee.

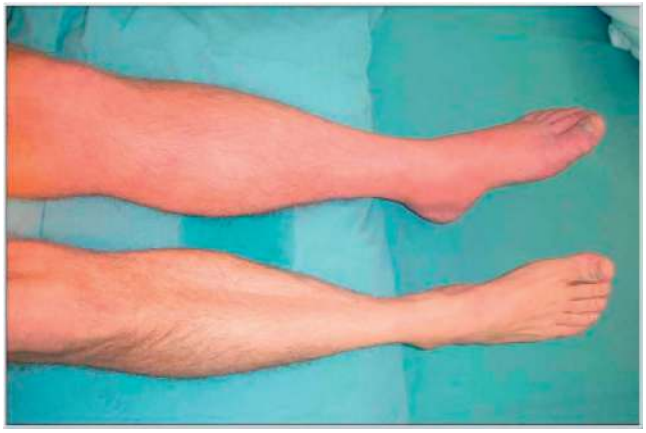

FIG 126.3 Ruptured Baker cyst can mimic thrombophlebitis. (From WEI ZHOU, PETER H. LIN, RUTH L. BUSH, ERIC K. PEDEN, ALAN B. LUMSDEN, CHAPTER 51 – Mechanical Thrombectomy and Thrombolysis for Acute Deep Venous Thrombosis, ed. John J. Bergan, The Vein Book, Academic Press, 2007:477–483.)

arthritis are susceptible to this phenomenon, and the pain associated with dissection into the calf may be confused with thrombophlebitis, thus leading to inappropriate treatment with anticoagulants. Occasionally, Baker's cyst spontaneously ruptures, usually after frequent squatting. In this case, rubor and calor may be evident in the calf, again mimicking thrombophlebitis; however, Homan's sign is negative, and no cords are palpable (Fig. 126.3).

On physical examination, patients with Baker's cysts have a cystic swelling in the medial aspect of the popliteal fossa that may be quite large. Activity that includes squatting or walking makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep.

TESTING

Plain radiographs, magnetic resonance imaging (MRI), and ultrasound imaging of the knee are indicated for all patients who present with Baker's cyst (Figs. 126.4 and 126.5). MRI and ultrasound imaging can also detect internal derangement, an occult mass, or a tumor (Fig. 126.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

As mentioned, Baker's cysts may rupture spontaneously, thus leading to a misdiagnosis of thrombophlebitis (Box 126.1). Occasionally, tendinitis of the medial hamstring or injury to the medial meniscus is confused with Baker's cyst. Primary or metastatic tumors in the region, although rare, must also be considered in the differential diagnosis as should infected Baker's cyst. Care must be taken not to mistake a popliteal artery aneurysm for Baker's cyst (Fig. 126.7).

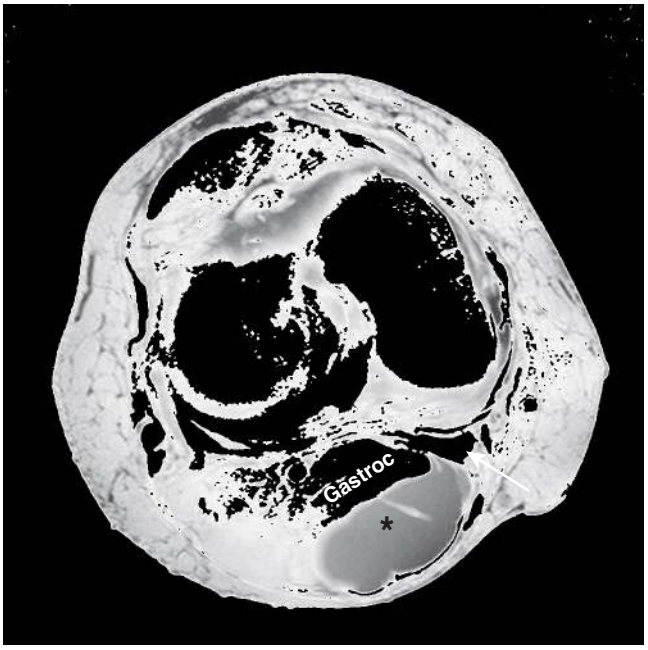

FIG 126.4 A T2-weighted with fat suppression magnetic resonance image of a Baker's cyst in a different patient. The features are identical, with the high-signal intensity, fluid-filled cyst (*asterisk*) arising between the gastrocnemius muscle (Gastroc) and the semimembranosus tendon (*white arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

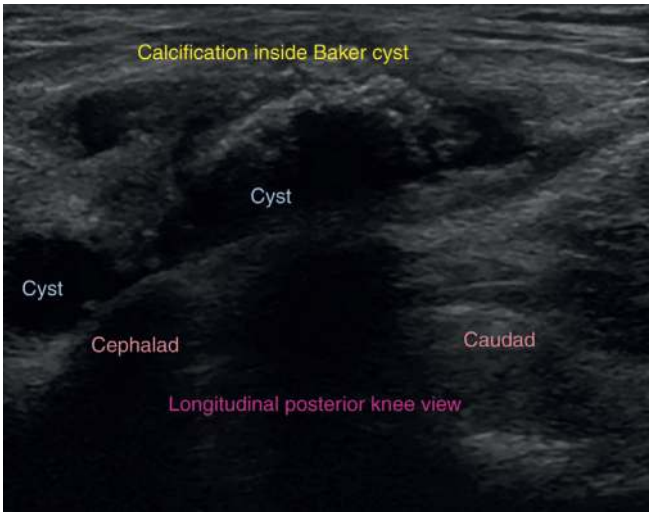

FIG 126.5 Transverse ultrasound image demonstrating a hypoechoic Baker's cyst

Careful palpation of the popliteal fossa should reveal a pulsatile mass if the artery is involved.

TREATMENT

Although surgery is often required to treat Baker's cyst, a short trial of conservative therapy consisting of an elastic bandage and nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors is warranted. If these conservative measures fail, injection is a reasonable next step.

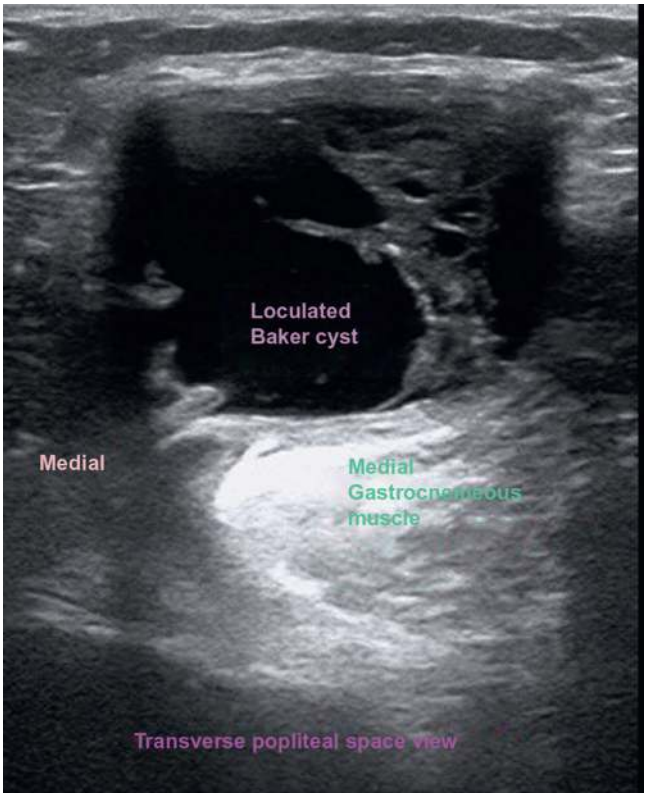

FIG 126.6 Ultrasound image demonstrating a large loculated Baker's cyst.

BOX 126.1 Differential Diagnosis of Posterior Knee Swelling and Pain

Baker cyst	Abscess
Popliteal aneurysm	Lymphadenopathy
Sarcoma	Varicosities
Lipoma	Arteriovenous fistula
Metastatic disease	Hematoma
Pigmented villonodular synovitis	Gastrocnemius tear
Ganglion cyst	Planteris muscle tear
Glomus tumor	Synovitis

To inject Baker's cyst, the patient is placed in the prone position with the anterior ankle resting on a folded towel to flex the knee slightly. The middle of the popliteal fossa is identified, and at a point two finger breadths medial to and two finger breadths below the popliteal crease, the skin is prepared with antiseptic solution. A syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 2-inch, 22-gauge needle. The needle is carefully advanced through the previously identified point at a 45-degree angle from the medial border of the popliteal fossa directly toward Baker's cyst. While continuously aspirating, the clinician advances the needle very slowly, to avoid trauma to the tibial nerve or popliteal artery or vein. When the cyst is entered, synovial fluid suddenly appears in the syringe. At this point, if no paresthesia is noted in the distribution of the common peroneal or

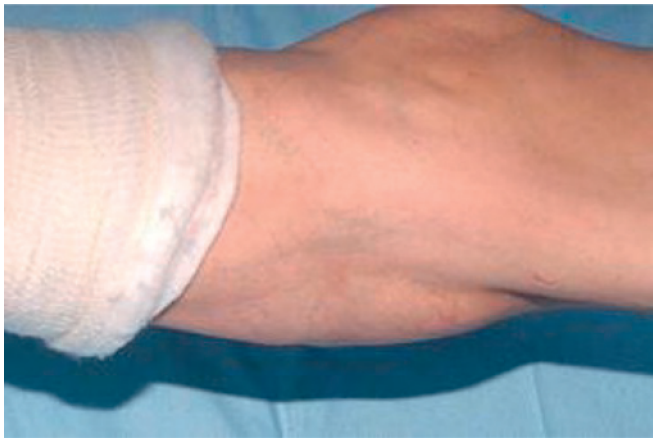

FIG 126.7 View of the medial aspect of the right knee demonstrates the large swelling in the popliteal fossa, which was subsequently diagnosed on clinical and radiological testing to be a large right popliteal artery aneurysm. (From Harkin DW, Mohammed O, Khadim M, et al. Rapid expansion of popliteal artery aneurysm after lower limb graduated compression bandaging for varicose ulcer. *EJVES Extra*. 2006;12(1):6–8.)

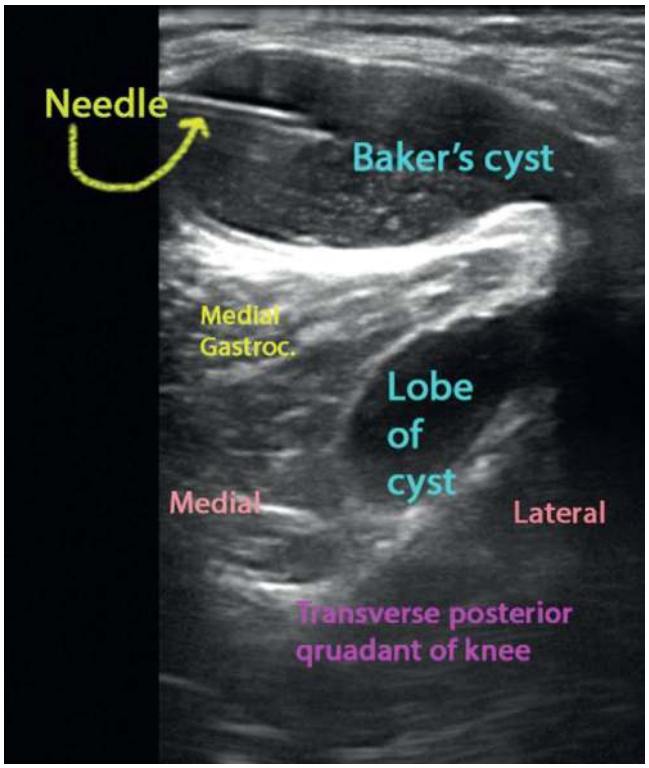

FIG 126.8 Ultrasound-guided aspiration of a large Baker's cyst.

tibial nerve, the contents of the syringe are gently injected. Resistance to injection should be minimal. A pressure dressing is placed over the cyst to prevent fluid reaccumulation. Ultrasound guidance may aid the clinician in needle placement in patients whose anatomic landmarks are difficult to identify as well as to aid in the drainage of large Baker's cysts (Fig. 126.8). Surgical treatment may be required for recurrent Baker's cyst (Fig. 126.9).

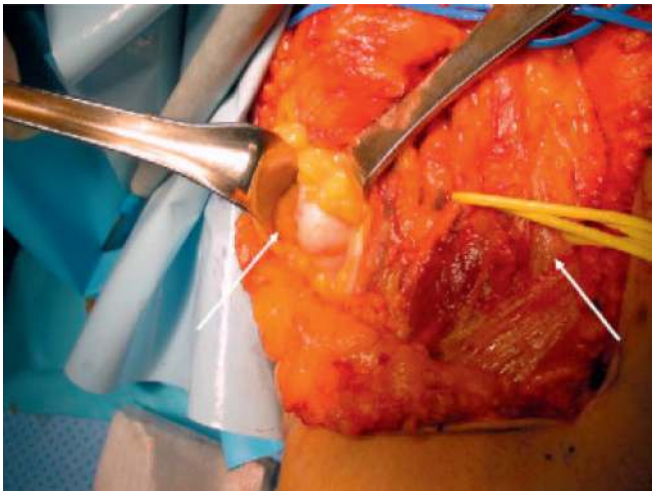

FIG 126.9 Intraoperative photograph shows thick and whitish Baker's cyst was found between medial head of gastrocnemius muscle and semimembranosus tendon which compresses the tibial nerve medially (*arrow*) and displaced the peroneal nerve laterally (*arrow*). (From Ji J-H, Shafi M, Kim W-Y, Park SH, Cheon JO. Compressive neuropathy of the tibial nerve and peroneal nerve by a Baker's cyst: case report. *Knee*. 2007;14(3):249–252.)

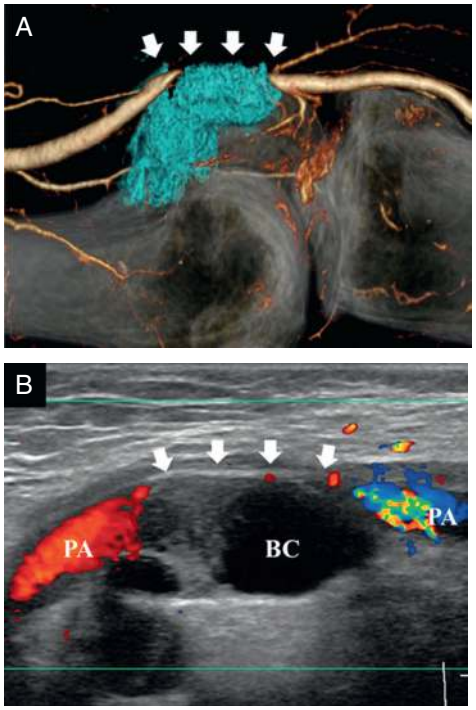

FIG 126.11 Computed tomography angiography image showing focal narrowing of the popliteal artery (*arrows*) at the right knee. **A,** The Baker's cyst appears polycystic (*blue masses*) and attached to the popliteal artery. **B,** Color Doppler ultrasound image demonstrating flow disturbance in the popliteal artery (*PA*) by the Baker cyst (*BC*). (From Fujiyoshi K, Minami Y, Tojo T, Iwase D, Hirata M, Ako J. Lower limb ischemia due to popliteal artery compression by Baker cyst. *J Vascu Surg Cases Innov Tech*. 2018;4(2): 99–101.)

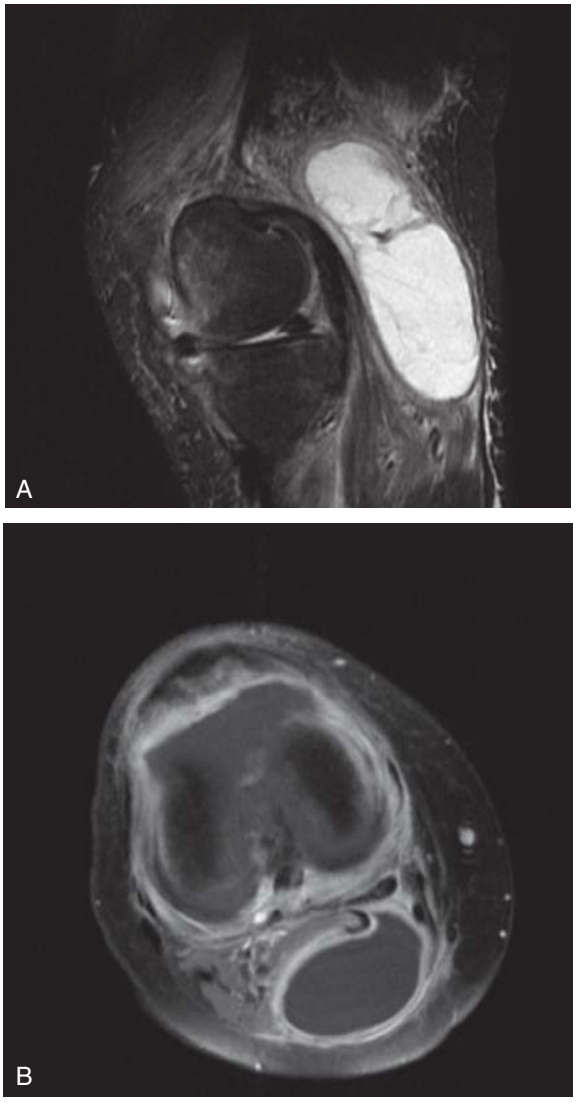

FIG 126.10 Infected Baker cyst. (**A**) Mid-sagittal STIR and (**B**) mid-axial FAT-SAT magnetic resonance images with gadolinium contrast illustrate a 9 × 3 × 5–cm popliteal cyst of the knee. (STIR, short tau inversion recovery; FAT-SAT, fat-saturated.) (From Josef K. Eichinger, Eric M. Bluman, Steven D. Sides, Edward D. Arrington, Surgical Management of Septic Arthritis of the Knee With a Coexistent Popliteal Cyst. *Arthroscopy: The Journal of Arthroscopic & Related Surgery*. 2009;25(6): 696-700.)

COMPLICATIONS AND PITFALLS

Failure to diagnose primary knee disorders, such as tears of the medial meniscus, may lead to further pain and disability. Because of the proximity to the common peroneal and tibial nerves, as well as to the popliteal artery and vein, it is imperative that injection of Baker's cysts be performed only by those familiar with the regional anatomy and experienced in the technique. Many patients complain of a transient increase in pain after injection, and infection, although rare, may occur (Fig. 126.10). In rare instances, the Baker's cyst may compress the popliteal vessels leading to lower extremity ischemia (Fig. 126.11).

CLINICAL PEARLS

The injection technique described is extremely effective in treating the pain and swelling of Baker's cysts. Coexistent semimembranosus bursitis, medial hamstring tendinitis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Alonso-Gómez N, Pérez-Piqueras A, Martínez-Izquierdo A, et al. Giant Baker' cyst. Differential diagnosis of deep vein thrombosis. *Reumatol Clín*. 2015;11(3):179–181.

Drescher MJ, Smally AJ. Thrombophlebitis and pseudothrombophlebitis in the (ED.). *Am J Emerg Med*. 1997;15(7):683–685.

Marra MD, Crema MD, Chung M, et al. MRI features of cystic lesions around the knee. *Knee*. 2008;15(6):423–438.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin North Am*. 2013;51(3):433–454.

Torreggiani WC, Al-Ismail K, Munk PL, et al. The imaging spectrum of Baker's (popliteal) cysts. *Clin Radiol*. 2002;57(8):681–691.

Waldman SD. Baker cyst. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:853–860.

Waldman SD. Injection cyst for Baker cyst. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:745–750.

Waldman SD, Campbell RSD. Baker cyst. In: *Imaging of pain*. Philadelphia: Saunders; 2011:413–415.

127

Pes Anserine Bursitis

ICD-10 CODE M76.899

THE CLINICAL SYNDROME

The pes anserine bursa lies beneath the pes anserine tendon, which is the insertional tendon of the sartorius, gracilis, and semitendinous muscles on the medial side of the tibia (Fig. 127.1). This bursa may exist as a single bursal sac or, in some patients, as a multisegmented series of loculated sacs. The pes anserine bursa is susceptible to the development of inflammation from overuse, misuse, or direct trauma. If inflammation of the pes anserine bursa becomes chronic, calcification may occur. Rarely, the pes anserine bursa becomes infected.

With trauma to the medial knee, the medial collateral ligament is often involved, along with the pes anserine bursa. This broad, flat, bandlike ligament runs from the medial condyle of the femur to the medial aspect of the shaft of the tibia, where it attaches just above the groove of the semimembranosus muscle; it also attaches to the edge of the medial semilunar cartilage. The medial collateral ligament is crossed at its lower part by the tendons of the sartorius, gracilis, and semitendinosus muscles.

SIGNS AND SYMPTOMS

Patients with pes anserine bursitis present with pain over the medial knee joint and increased pain on passive valgus and external rotation of the knee. Activity, especially that involving flexion and external rotation of the knee, makes the pain worse, whereas rest and heat provide some relief. Often, patients are unable to kneel or walk downstairs (Fig. 127.2). The pain of pes anserine bursitis is constant and is characterized as aching; it may interfere with sleep. Coexistent bursitis, tendinitis, arthritis, or internal derangement of the knee may confuse the clinical picture after trauma to the knee.

Physical examination may reveal point tenderness in the anterior knee just below the medial knee joint at the tendinous insertion of the pes anserine. Swelling and fluid accumulation surrounding the bursa are often present (Fig. 127.3). Actively resisted flexion of the knee reproduces the pain. Sudden release of resistance during this maneuver causes a marked increase in pain.

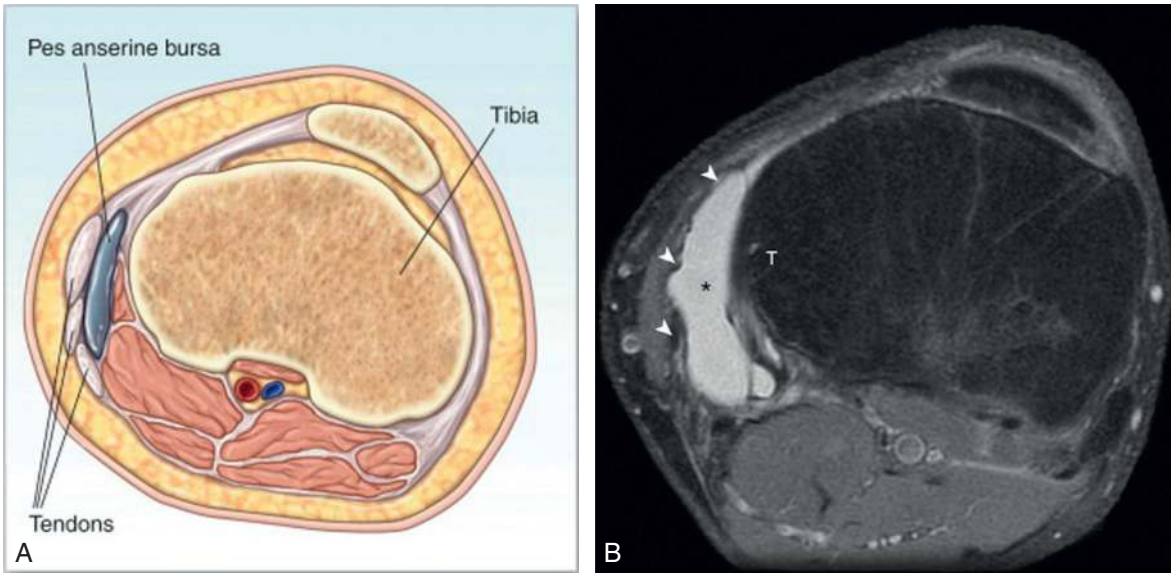

FIG 127.1 **A,** Drawing illustrating pes anserine bursitis. This axial view shows the anserine bursa (*blue*) located between the medial aspect of the tibia and the tendons forming the pes anserinus (from anterior to posterior: sartorius, gracilis, and semitendinosus). **B,** Axial proton density (PD)-weighted image with fat suppression shows a fluid collection *(asterisk)* located between the pes anserinus (*arrowheads*) and the surface of the medial tibial condyle (*T*), consistent with anserine bursitis. (From Marra MD, Crema MD, Chung M, et al. MRI features of cystic lesions around the knee. *Knee*. 2008;15:423–438.)

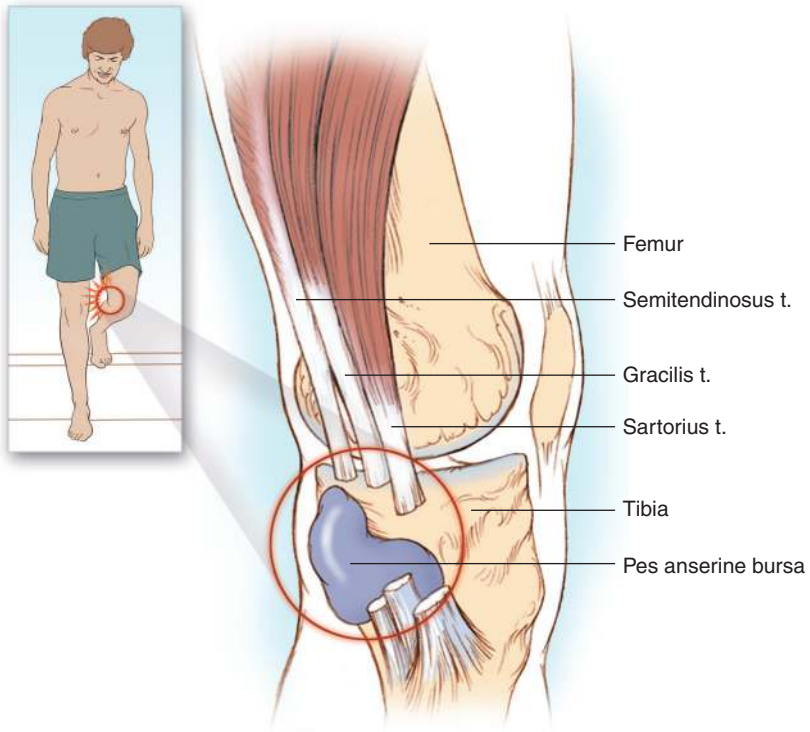

FIG 127.2 Patients with pes anserine bursitis complain of medial knee pain that is made worse with kneeling or walking downstairs.

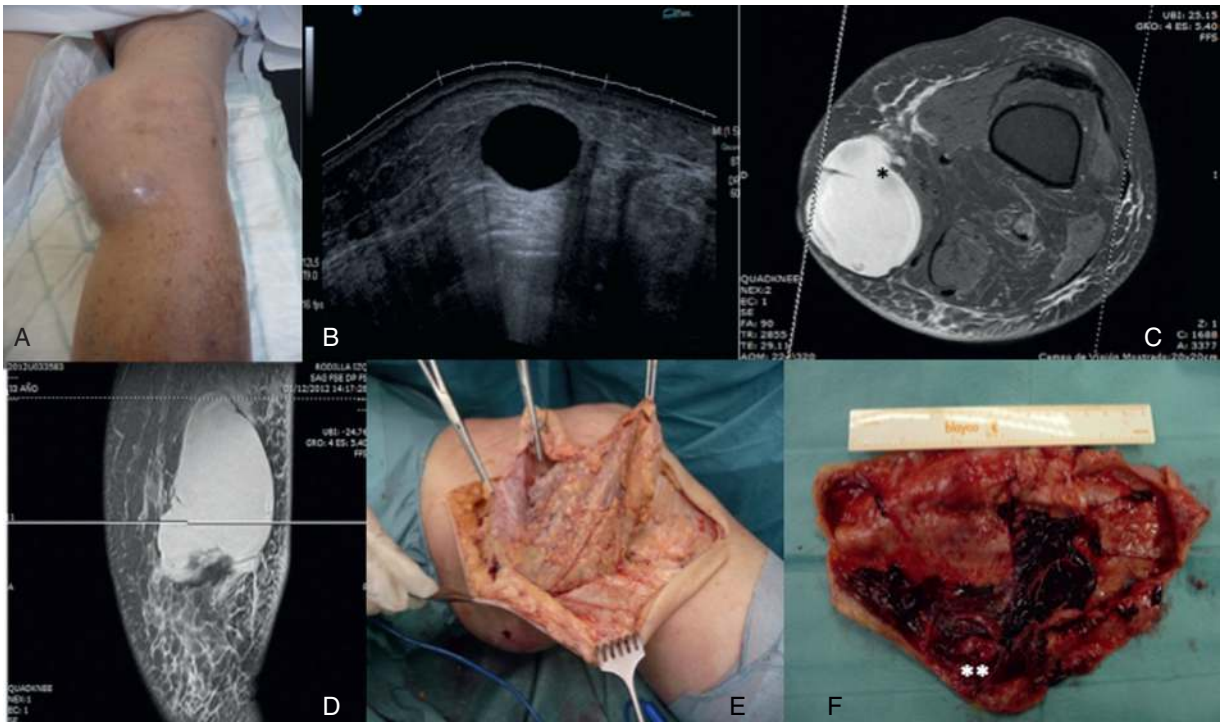

FIG 127.3 Pes anserine bursitis. **A,** Clinical aspect of the soft tissue mass in the medial knee, **B,** gray scale ultrasound showing a 16 cm long well defined anechoic mass (area: >15.6 cm^2) without synovial hypertrophy in the pes anserinus bursa, **C,** MRI with axial T1 view showed a homogeneous soft tissue mass at the enthesis of pes anserinus conjoint tendon with peripheral enhancement (*). Absence of hemosiderin deposits and no evidence of bone or joint involvement, **D,** T1 coronal view of the bursa, **E,** Intraoperative image of the lesion showed multiple yellow to brown nodules inside the pes anserinus bursa, **F,** 15 cm large bursa excision pathology confirmed the pathology (**). (From Hèctor Corominas, Ramon Balius, Paula Estrada-Alarcón, Dèlia Reina, Patricia Moya, Miquel Videla, Giant pes anserinus bursitis: A rare soft tissue mass of the medial knee. *Reumatología Clínica* (English Edition). 2021;17(7):420–421.)

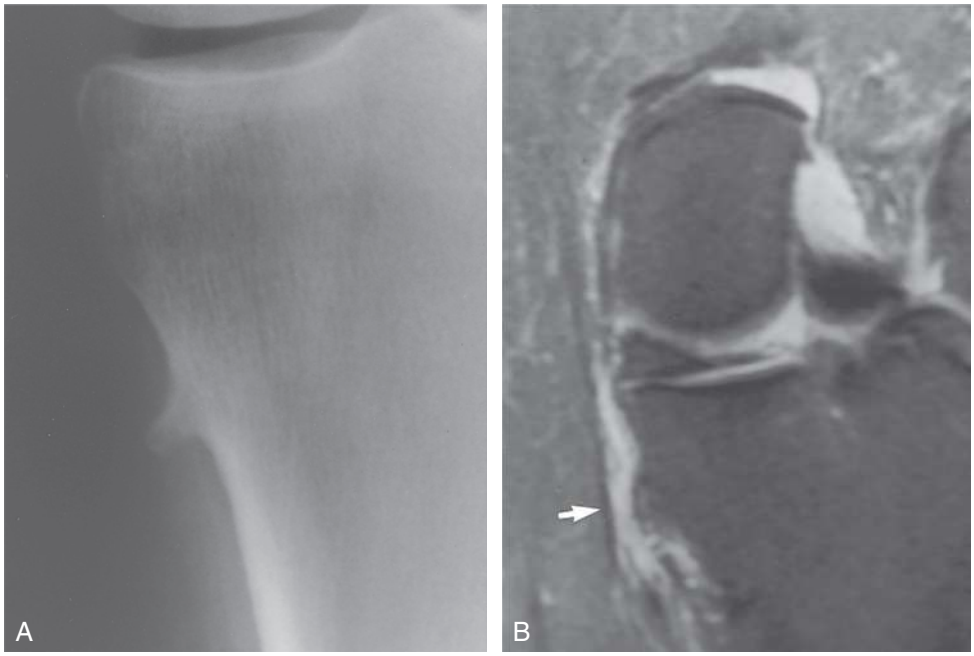

FIG 127.4 Pes anserine spurs. In this 65-year-old woman with a history of pes anserine bursitis, a conventional radiograph **(A)** reveals a small excrescence in the medial portion of the tibia. On a coronal, fat-suppressed, fast spin-echo magnetic resonance image **(B)**, fluid of high signal intensity (*arrow*) is seen about the bone outgrowth. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:3898)

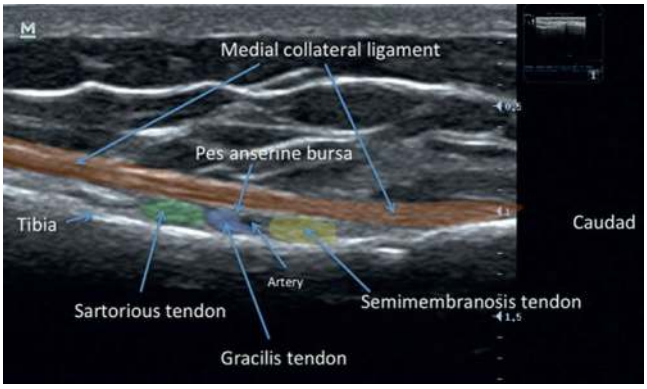

FIG 127.5 Ultrasound image of the knee joint demonstrating the pes anserine bursa lying beneath the pes anserine tendon.

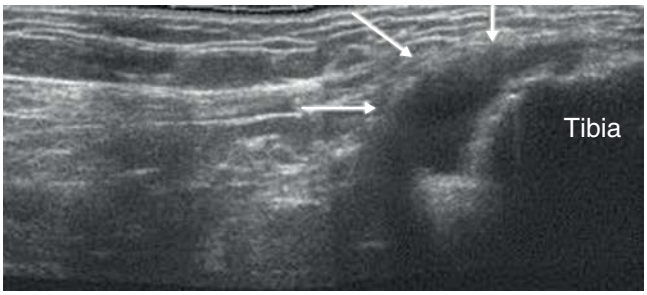

FIG 127.6 Ultrasound image demonstrating pes anserine bursitis.

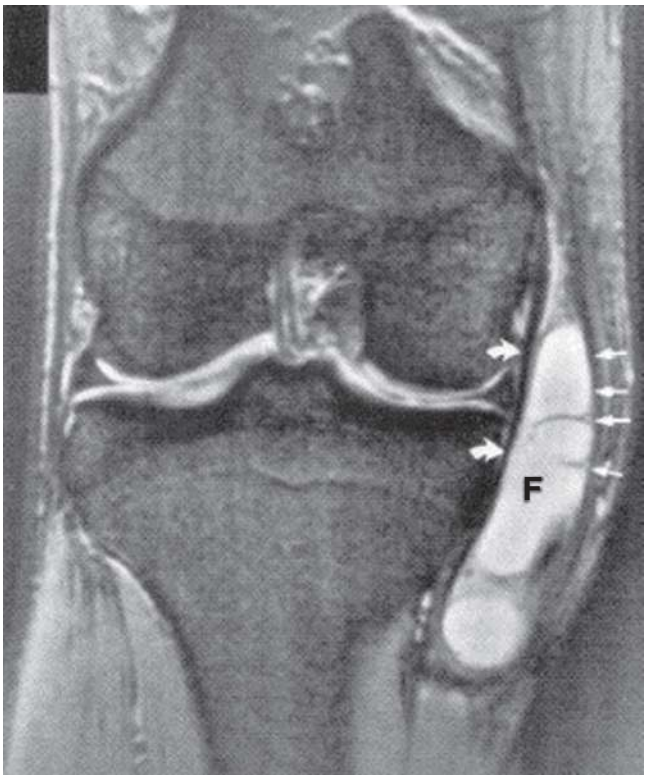

FIG 127.7 Pes anserine bursitis. A large lobulated mass of fluid signal (*F*) represents the inflamed fluid-filled pes anserine bursa seen between the medial collateral ligament (MCL) (*curved arrows*) and the pes anserine tendon (*small arrows*). (From Gray SD, Kaplan PA, Dussault RG. Imaging of the knee: current status. *Orthop Clin North Am*. 1997;28(4):643–658.)

TESTING

Plain radiographs and ultrasound imaging of the knee may reveal calcification of the bursa and associated structures, including the pes anserine tendon, findings consistent

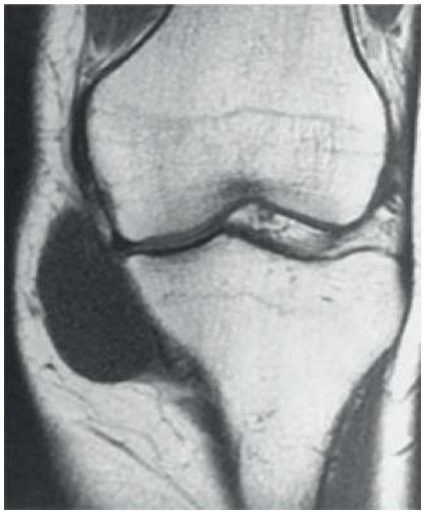

FIG 127.8 Pes anserinus ganglion cyst (bursitis). Magnetic resonance image shows a large, fluid-filled mass adjacent to the anteromedial portion of the tibia. (From Resnick D: Diagnosis of bone and joint disorders, 4th ed., Philadelphia, 2002, Saunders.)

with chronic inflammation. Magnetic resonance imaging (MRI) and ultrasound imaging are indicated if internal derangement, an occult mass, or a tumor of the knee is suspected (Figs. 127.4–127.8). Electromyography can distinguish pes anserine bursitis from neuropathy, lumbar radiculopathy, and plexopathy. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Pes anserine spurs may coexist with pes anserine bursitis, thus confusing the clinical picture. Because of the unique anatomic relationships present in the medial knee, it is often difficult to make an accurate clinical diagnosis that identifies the structure responsible for the patien's pain (Fig. 127.9). MRI can rule out lesions that may require surgical intervention, such as tears of the medial meniscus. Anything that alters the normal biomechanics of the knee can result in inflammation of the pes anserine bursa.

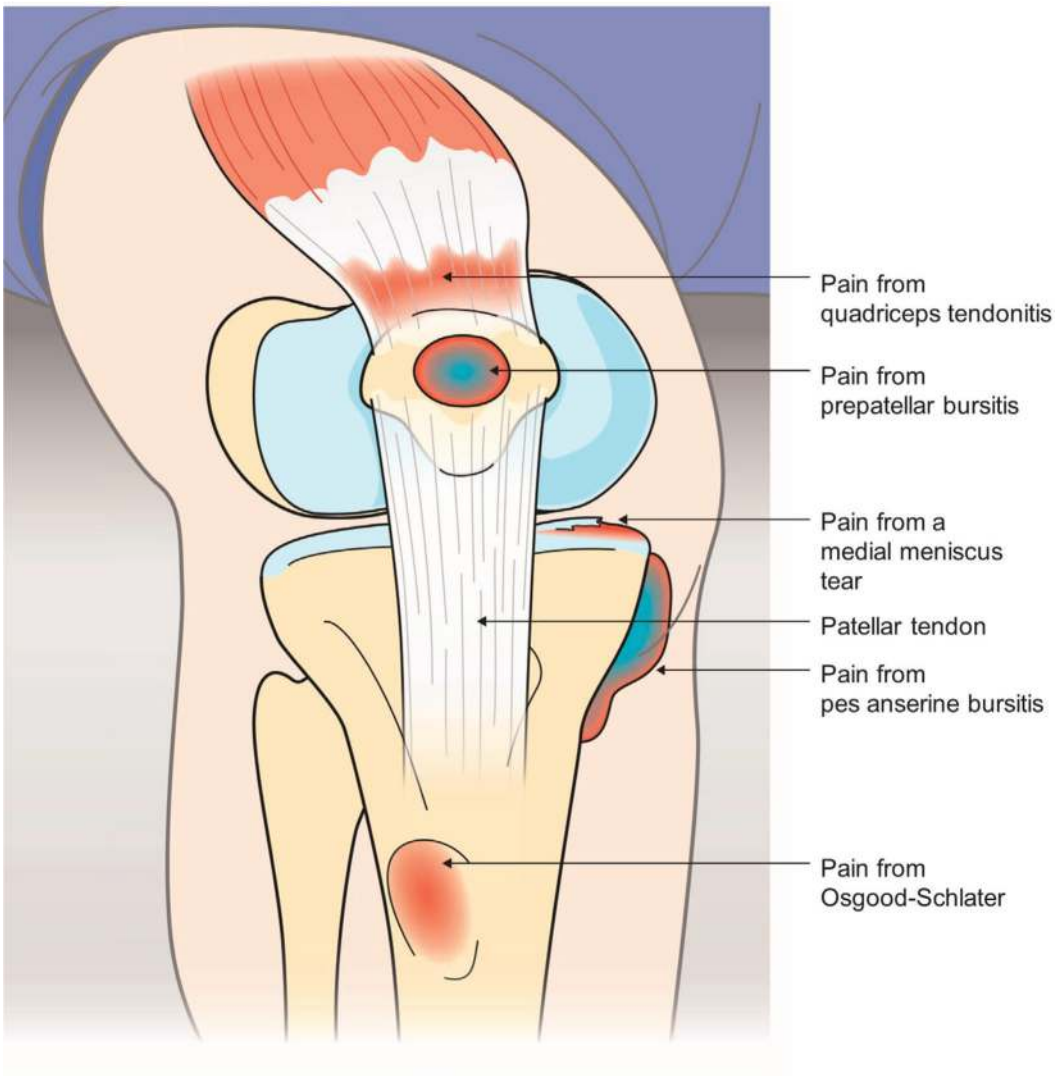

FIG 127.9 The pain of pes anserine bursitis is localized to the anterior medial knee. (From Waldman SD. *Knee: pain medicine*. Philadelphia: Elsevier; 2022:128.)

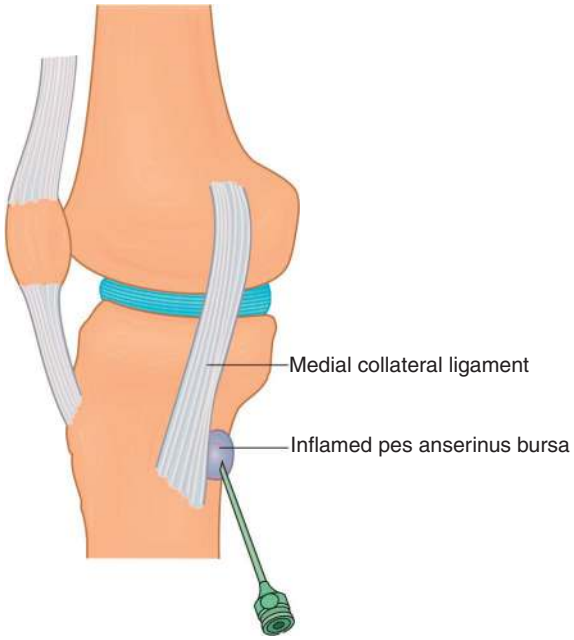

FIG 127.10 Injection of the pes anserine bursa with local anesthetic and steroid may provide symptomatic relief of the pain and swelling associated with pes anserine bursitis. (From Waldman SD. *Atlas of pain management injection techniques*. 4th ed. St Louis: Elsevier; 2017: Fig. 144-3.)

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors, and a knee brace to prevent further trauma is the first step in the treatment of pes anserine bursitis. If patients do not experience rapid improvement, injection is a reasonable next step.

To inject the pes anserine bursa, the patient is placed in the supine position with a rolled blanket underneath the knee to flex the joint gently. The skin just below the medial knee joint is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The pes anserine tendon is identified by having the patient strongly flex his or her leg against resistance. The pes anserine bursa is located at a point distal to the medial joint space where the pes anserine tendon attaches to the tibia. The bursa can usually be identified by point tenderness. At that point, the needle is inserted at a 45-degree angle to the tibia and passes through the skin and subcutaneous tissues into the pes anserine bursa. If the needle strikes the tibia, the needle is withdrawn slightly into the substance of the bursa (Fig. 127.10). When the needle is positioned in proximity to the pes anserine bursa, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the distal femur or knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of pes anserine bursitis.

SUGGESTED READINGS

Maheshwari AV, Muro-Cacho CA, Pitcher JD Jr. Pigmented villonodular bursitis/diffuse giant cell tumor of the pes anserine bursa: a report of two cases and review of literature. *Knee*. 2007;14(5):402–407.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin N Am*. 2013;51(3):433–454.

Waldman SD. Pes anserine bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:805–809.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308.

Waldman SD. Pes anserine bursa injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:722–727.

Sciatic Nerve Entrapment at the Knee

ICD-10 CODE G57.00

THE CLINICAL SYNDROME

The sciatic nerve provides innervation to the distal lower extremity and foot with the exception of the medial aspect of the calf and foot, which are subserved by the saphenous nerve. The largest nerve in the body, the sciatic nerve, is derived from the L4, L5, and S1–S3 nerve roots. The roots fuse in front of the anterior surface of the lateral sacrum on the anterior surface of the piriformis muscle. The nerve travels inferiorly and leaves the pelvis just below or through the piriformis muscle via the sciatic notch. Just beneath the nerve at this point is the obturator internus muscle. The sciatic nerve lies anterior to the gluteus maximus muscle; at this muscle's lower border, the sciatic nerve lies halfway between the greater trochanter and the ischial tuberosity. The sciatic nerve courses downward past the lesser trochanter to lie posterior and medial to the femur. In the mid-thigh, the nerve gives off branches to the hamstring muscles and the adductor magnus muscle. In most patients, the nerve divides to form the tibial and common peroneal nerves in the upper portion of the popliteal fossa, although in some patients these nerves can remain separate through their entire course (Fig. 128.1). The tibial nerve continues downward to provide innervation to the distal lower extremity, whereas the common peroneal nerve travels laterally to innervate a portion of the knee joint and, via its lateral cutaneous branch, provides sensory innervation to the back and lateral side of the upper calf.

The most common pain syndrome mediated via the sciatic nerve is piriformis syndrome which is caused by compromise of the sciatic nerve by the piriformis muscle (Fig. 128.2). The sciatic nerve can also be compromised at the popliteal fossa by popliteal artery aneurysms as well as Baker's synovial cysts (Figs. 128.3 and 128.4). Distal hamstring avulsions can also cause compromise of the sciatic nerve. The symptoms associated with sciatic neuralgia depend on the point at which the nerve is compromised.

SIGNS AND SYMPTOMS

Initial symptoms of sciatic nerve entrapment at the knee include pain in the posterior distal thigh and knee that may radiate into the lower extremity and foot. Patients suffering from sciatic nerve entrapment will complain of both motor and sensory symptoms. Burning, tingling, numbness, and dysesthesias in the sensory distribution of the sciatic nerve, which may worsen at night, are frequent complaints, as is allodynia (Fig. 128.5). Weakness of the muscles innervated by the sciatic nerve will cause the patient to develop an altered gait, leading to coexistent sacroiliac, back, hip, and knee pain that confuses the clinical picture. Physical findings include tenderness over the sciatic nerve at the point of entrapment. A positive Tinel's sign at the point of entrapment is often present. A positive straight leg raising test may be present, especially if the nerve is tethered down by a large Baker's cyst, hematoma, tumor, or aneurysm.

TESTING

Electromyography can distinguish lumbar radiculopathy from piriformis syndrome. Plain radiographs of the back,

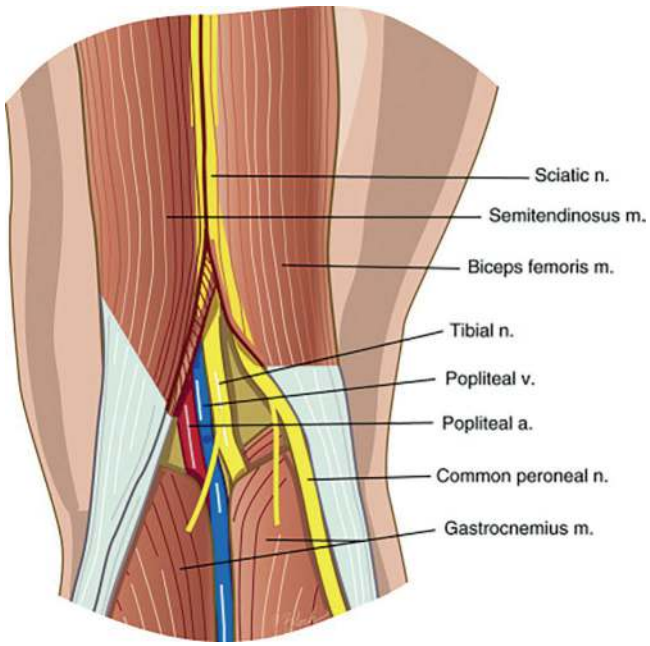

FIG 128.1 The sciatic nerve courses downward past the lesser trochanter to lie posterior and medial to the femur. In the middle thigh, the nerve gives off branches to the hamstring muscles and the adductor magnus muscle. In most patients, the nerve divides to form the tibial and common peroneal nerves in the upper portion of the popliteal fossa, although these nerves sometimes remain separate through their entire course. *a.*, Artery; *m.*, muscle; *n.*, nerve; *v.*, vein. (From Waldman SD. *Atlas of interventional pain management,* 4th ed. Philadelphia: Saunders; 2015:728.)

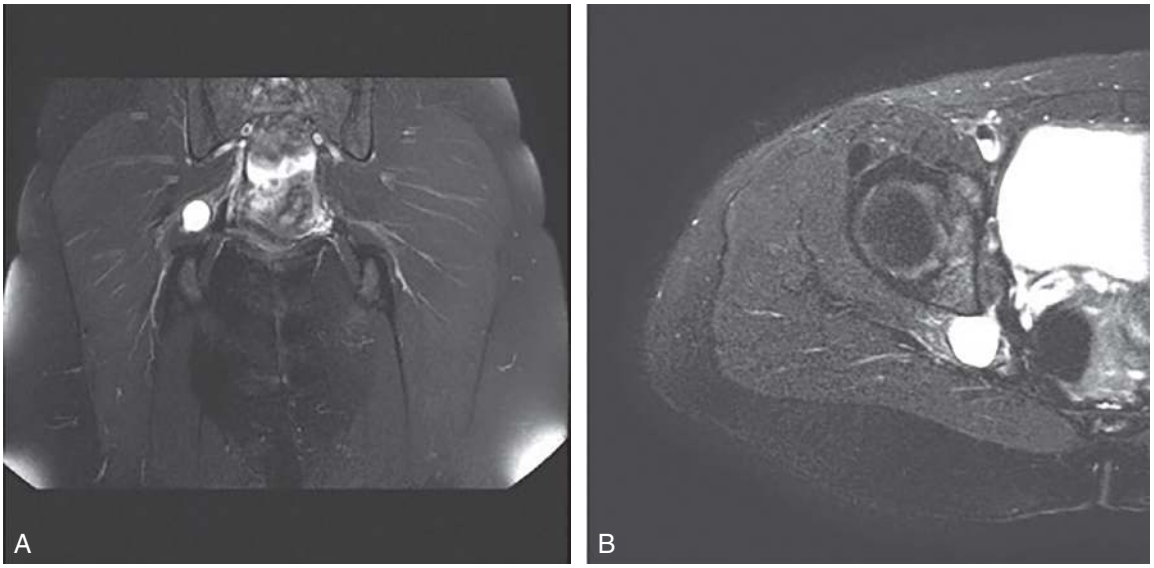

FIG 128.2 Magnetic resonance imaging (MRI) findings of a 32-year-old female patient. Coronal **(A)** and axial **(B)** MR images demonstrating about a 2 cm sized multilobulated cystic mass located in the piriformis muscle that showed high intensity on T2-weighted and low intensity on T1-weighted image. (From Park JH, Jeong HJ, Shin HK, et al. Piriformis ganglion: an uncommon cause of sciatica. *Orthop Traumatol Surg Res*. 2016;102(2):257–260.)

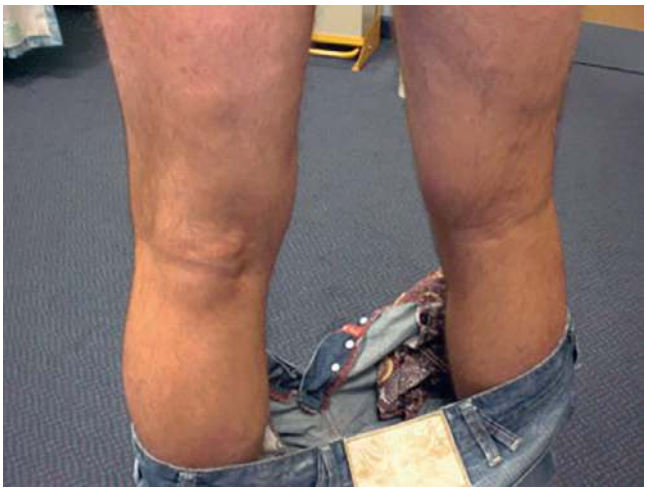

FIG 128.3 Classic appearance of a Baker's cyst. (From Ali F. Clinical examination of the knee. *Orthop Trauma*. 2013; 27(1):50–55.)

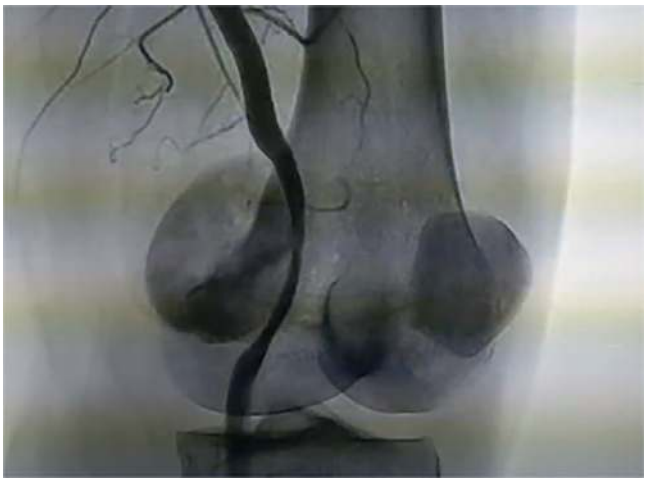

FIG 128.4 Angiogram showing a large popliteal aneurysm compressing the popliteal artery. (From Jaber C, Moulahi N, Mrad IB, et al. Endovascular management of a ruptured popliteal artery aneurysm in a patient with Behçet's disease. *Ann Vasc Surg Brief Rep Innov*. 2022: 100097.)

hip, and pelvis are indicated in all patients who present with piriformis syndrome to rule out occult bony disorders. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging (MRI) of the back is indicated if herniated disk, spinal stenosis, or space-occupying lesion of the popliteal fossa are suspected. MRI and ultrasound imaging of the hip, piriformis muscle, and knee may elucidate the cause of compression of the sciatic nerve (Figs. 128.6 and 128.7). Comprehensive metabolic profile and thyroid function testing should be obtained to rule out systemic and endocrine diseases that may cause vulnerable nerve syndrome (e.g., diabetes). Antinuclear antibody testing is indicated if collagen vascular disease is suspected. Injection in the region of the sciatic nerve at this level serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of sciatic nerve entrapment is a two-step process. The first step is to ascertain the exact anatomic level of sciatic nerve compromise. The most common site of sciatic nerve compromise is as the nerve travels under

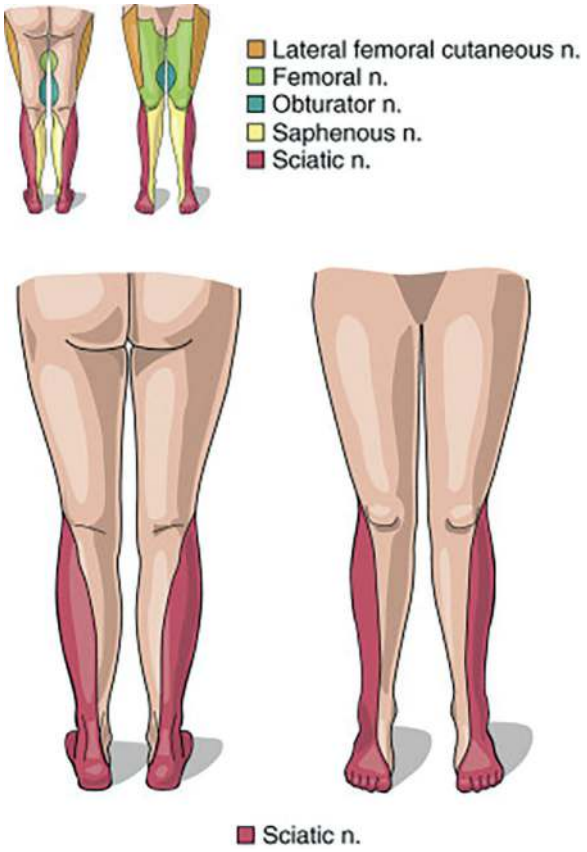

FIG 128.5 Sensory distribution of the sciatic nerve. (From Waldman SD. *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021.)

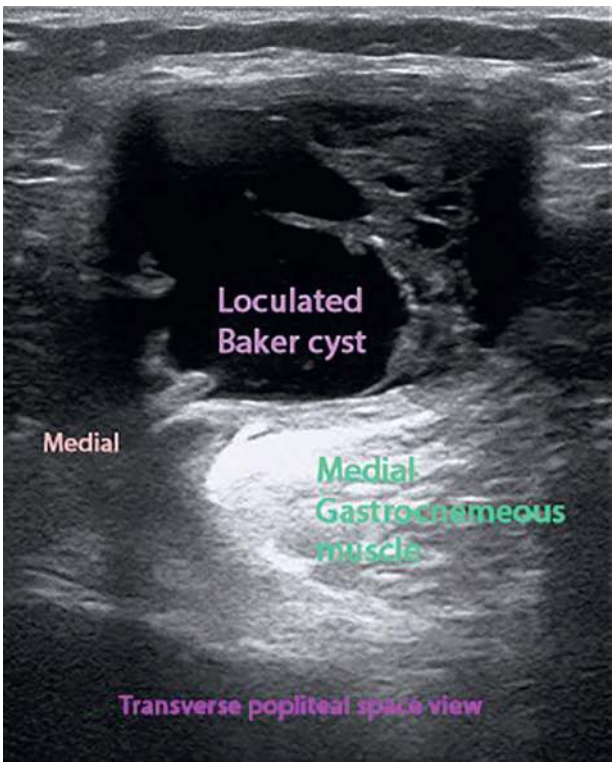

FIG 128. 6 Ultrasound image demonstrating a large loculated Baker cyst.

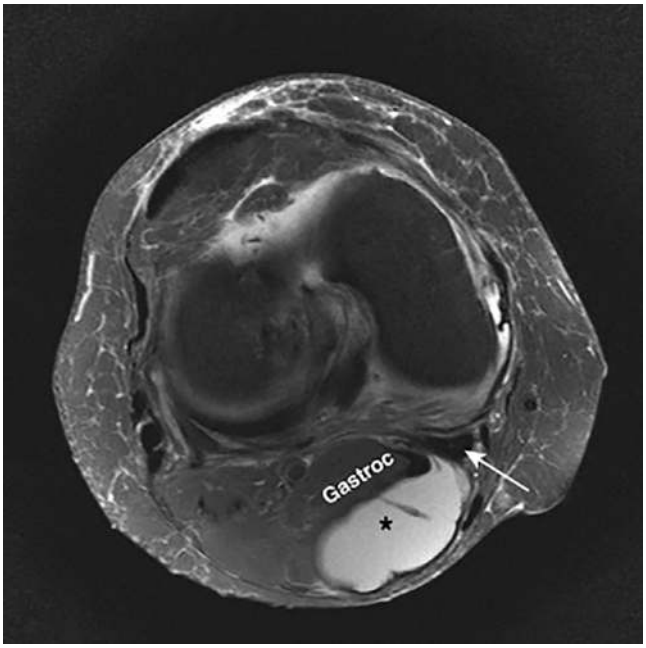

FIG 128.7 A T2-weighted with fat suppression magnetic resonance image of a Baker's cyst in a different patient. The features are identical, with the high–signal intensity, fluid-filled cyst (*asterisk*) arising between the gastrocnemius muscle (Gastroc) and the semimembranosus tendon (*white arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain.* Philadelphia: Elsevier; 2011.)

BOX 128.1 Differential Diagnosis of Sciatic Nerve Entrapment at the Knee

Baker's cyst	Abscess
Popliteal aneurysm	Lymphadenopathy
Sarcoma	Varicosities
Lipoma	Arteriovenous fistula
Metastatic disease	Hematoma
Pigmented villonodular synovits	Gastrocnemius tear
Ganglion cyst	Planteris muscle tear
Glomus tumor	Synovitis

the piriformis muscle rather than the knee. This first step is best accomplished with the use of careful physical examination combined with electrodiagnostic testing and medical imaging to confirm the clinical diagnosis. Step two is to rule out other pathologic processes that may be responsible for compression of the sciatic nerve at the knee. Common causes of sciatic nerve compression at the knee include Baker's cysts, popliteal artery aneurysms, tumors, and hematoma (Box 128.1). Primary tumors of the sciatic nerve at this level can also present as entrapment neuropathy.

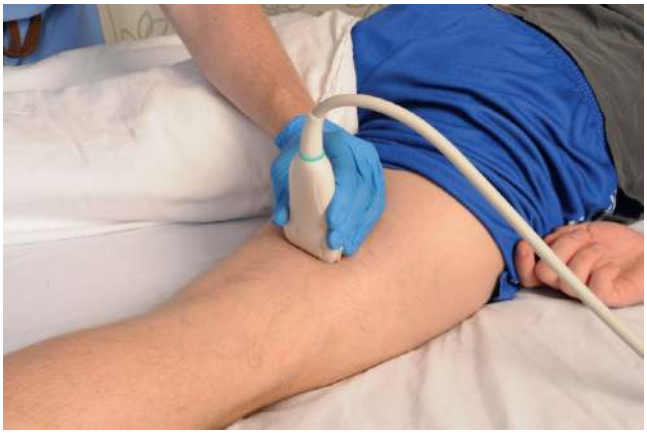

FIG 128.8 Proper transverse position of the ultrasound transducer approximately 8 cm above the popliteal crease for ultrasound-guided sciatic nerve block at the popliteal fossa.

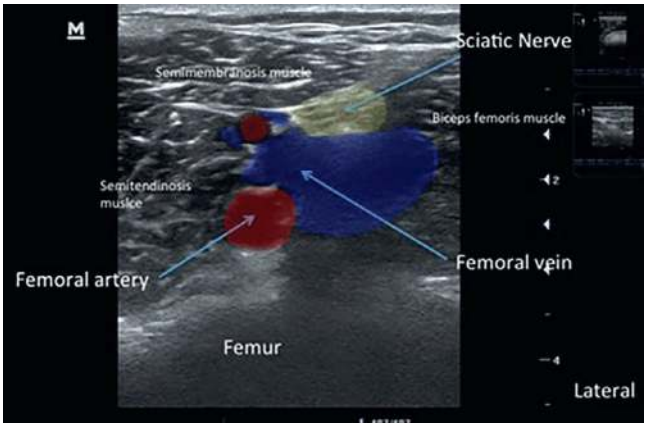

FIG 128.9 Transverse ultrasound image demonstrating the popliteal artery and vein and the sciatic nerve just above and lateral to the sciatic nerve.

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors, and the use of an ankle-foot orthosis will help prevent further trauma to the affected musculotendinous units and to decrease the risk of falling is the first step in the treatment of common peroneal nerve entrapment. Removal of the source of nerve entrapment or compression is crucial to prevent permanent nerve damage. Tricyclic antidepressants may help with sleep disturbance. If patients do not experience rapid improvement, injection is a reasonable next step.

To perform sciatic nerve block at the knee under ultrasound guidance using the posterior approach, the patient is placed in the prone position An ultrasound transducer is placed in a transverse orientation at the popliteal fossa and the tibial artery, popliteal vein, and the peroneal and tibial nerves are identified (Fig. 128.8). After the tibial artery, popliteal vein, and the tibial nerve are identified, the transversely placed ultrasound transducer is slowly moved superiorly toward the hip while tracing the path of the tibial nerve until its origin at the bifurcation of the sciatic nerve is visualized. The sciatic nerve is then traced superiorly to a point just above the bifurcation of the tibial and peroneal nerves (Fig. 128.9).

After the sciatic nerve just proximal to its bifurcation is identified, a 25-gauge, 3½-inch needle is then slowly advanced perpendicular to the skin using an in-plane approach toward the sciatic nerve under continuous ultrasound guidance (see Fig.128.9). The needle is slowly advanced toward the sciatic nerve until the needle tip is in proximity to the sciatic nerve. After careful aspiration, a small amount of solution is injected to confirm location of the needle tip. After satisfactory confirmation of the needle tip is confirmed, after careful aspiration, 18 mL of 1.0% preservative-free lidocaine is slowly injected in incremental doses. Care must be taken not to advance the needle into the substance of the nerve during the injection and inject solution intraneurally. Given the proximity to the superficial femoral artery and vein, the possibility of inadvertent intravascular injection remains an ever-present possibility.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and injected in incremental doses. Subsequent daily nerve blocks are performed in a similar manner, substituting 40 mg of methylprednisolone for the initial 80 mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation. The use of a nerve stimulator may aid in more accurate needle placement. Proper needle placement in proximity to the sciatic nerve is evidenced by twitching in the toes and feet when stimulated at a level of 0.2–0.5 mA.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

It should be remembered that the most common causes of sciatica are herniated lumbar disk and degenerative arthritis of the lumbar spine, not disorders involving the sciatic nerve per se. Electromyography and magnetic resonance imaging of the lumbar spine, combined with the information gleaned from clinical history and physical examination, will help to sort out the etiology of sciatic pain.

SUGGESTED READINGS

Hunter OO, Kim TE, Mariano ER, Harrison TK. Care of the patient with a peripheral nerve block. *J PeriAnesth Nurs*. 2018:87–93.

Jaber C, Moulahi N, Ben Mrad I, et al. Endovascular management of a ruptured popliteal artery aneurysm in a patient with Behçet's disease. *Ann Vasc Surg Brief Rep Innov*. 2022:100097.

Pichamuthu JE, Maiti S, Gan MG, et al. Mechanics of anesthetic needle penetration into human sciatic nerve. *J Biomech*. 2018;74:92–97.

Waldman SD. Piriformis syndrome and other abnormalities of the sciatic nerve. In: *Waldman's comprehensive atlas of ultrasound diagnosis of painful conditions*. Philadelphia: Wolters Kluwer; 2016:695–702.

Waldman SD. Sciatic nerve block. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:531–534.

Waldman SD. Sciatic nerve. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:121–122.

129

Common Peroneal Nerve Entrapment

ICD-10 CODE G57.30

THE CLINICAL SYNDROME

The common peroneal nerve, also known as the common fibular nerve, is commonly entrapped or compressed as it crosses the head of the fibula; it is known as cross leg or yoga palsy. Symptoms of entrapment of the common peroneal nerve at this anatomic location are numbness and foot drop. The common peroneal nerve is also subject to compromise from a number of pathologic conditions including neuropathy, leprosy, and vasculitis. Tumors of the common peroneal nerve as well extrinsic masses including ganglion cysts may also entrap the nerve. Plaster casts and orthotic braces must be carefully fitted to avoid compression of the nerve (Box 129.1). The common yoga position vajrasana has also been implicated in the evolution of this lower extremity nerve entrapment (Fig. 129.1).

SIGNS AND SYMPTOMS

Patients suffering from common peroneal nerve entrapment will complain of both motor and sensory symptoms. Burning, tingling, numbness, and dysesthesia in the sensory distribution of the common peroneal nerve, which may worsen at night, are frequent complaints, as is allodynia (Fig. 129.2). Weakness of the dorsiflexors and evertors of the foot and ankle are often present, and the patient may adopt a steppage gait to compensate for the drop foot (Fig. 129.3).

BOX 129.1 Causes of Common Peroneal Nerve Entrapment

- External compression
  - During anesthesia, coma, sleep, bed rest
  - Plaster casts, braces
  - Habitual leg crossing
  - Sitting cross-legged
  - Prolonged squatting, kneeling
- Direct trauma
  - Blunt injuries, lacerations
  - Fractures of the fibula
  - Adduction injuries and dislocations of the knee
  - Surgery and arthroscopy in popliteal fossa and knee
- Traction injuries
  - Acute ankle injuries
- Masses
  - Ganglia, Baker's cysts, callus, fibular tumors, osteomas, hematomas
- Tumors
  - Nerve sheath tumors
  - Nerve sheath ganglia
  - Lipomas
- Entrapment
  - In the fibular tunnel
  - Anterior (tibial) compartment syndrome
- Vascular
  - Vasculitis, local vascular disease
- Diabetes mellitus: susceptibility to compression, ischemic damage
- Leprosy
- Idiopathic

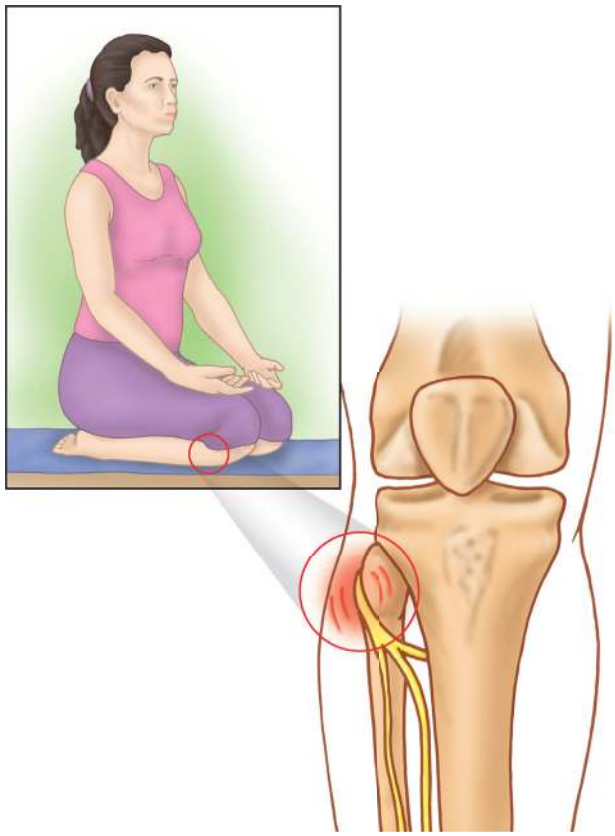

FIG 129.1 Common peroneal nerve entrapment, also known as yoga palsy, is caused by compression of the common peroneal nerve as it passes over the head of the fibula.

TESTING

Plain radiographs, ultrasound, and magnetic resonance imaging (MRI) of the knee may reveal calcification of the bursa and associated structures as well as other masses including Baker's cyst or ganglion cysts that may be compressing the common peroneal nerve (Figs. 129.4–129.6). Electrodiagnostic testing should be considered in all patients who suffer from common peroneal nerve dysfunction to provide both neuroanatomic and neurophysiologic information regarding nerve function. Comprehensive metabolic profile and thyroid function testing should be obtained to rule out systemic and endocrine diseases that may cause vulnerable nerve syndrome (e.g., diabetes). Antinuclear antibody testing is indicated if collagen vascular disease is suspected.

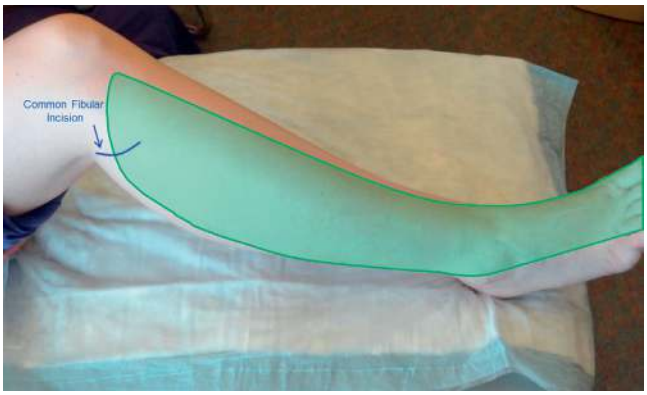

FIG 129.2 Common peroneal (fibular nerve) sensory distribution. (From Anderson JC. Common fibular nerve compression: anatomy, symptoms, clinical evaluation, and surgical decompression. *Clin Podiatr Med Surg*. 2016;33(2):283–291.)

DIFFERENTIAL DIAGNOSIS

Because of the anatomy of the region, the associated tendons and structures of the knee can become inflamed, thus confusing the diagnosis. Anything that compresses, entraps, or damages the common peroneal nerve can contribute to the patient's pain and functional disability (Figs. 129.7 and 129.8; see Box 129.1).

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors, and an ankle-foot orthosis will aid in prevention of further trauma to the affected musculotendinous units and will also decrease the risk of falling. Removal of the source of nerve entrapment or compression is crucial to prevent permanent nerve damage. Tricyclic antidepressants may help with sleep disturbance. If patients do not experience rapid improvement, injection is a reasonable next step.

To inject the common peroneal nerve, the patient is placed in the supine position with the leg slightly flexed. The superior margin of the patella is identified and an imaginary line is envisioned extending laterally to a point overlying the groove between the margins of the vastus lateralis and biceps femoris muscles. The margins of these muscles can be more easily identified by having the patient flex his or her leg under resistance. At this point, the skin is prepared with antiseptic solution. A 25-gauge, 3½-inch needle is then slowly advanced perpendicular to the skin through this point toward the common peroneal nerve which lies on the lateral aspect of the lower extremity (Fig. 129.9). The needle is slowly advanced toward

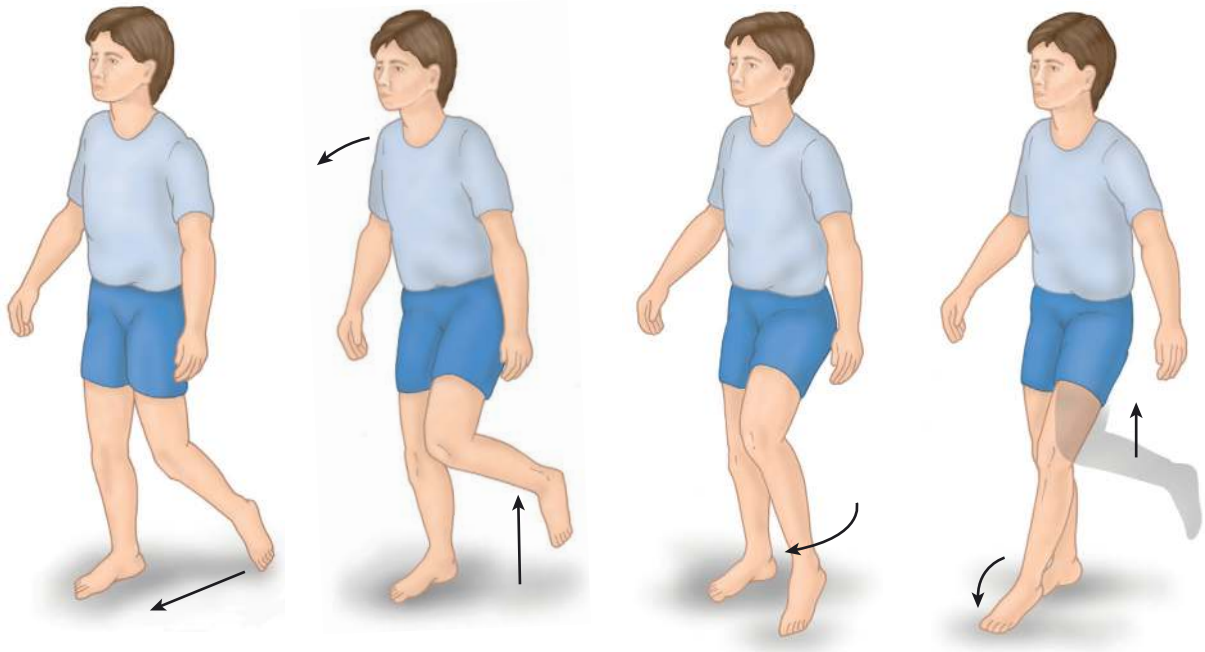

FIG 129.3 Compensating gaits seen in patients with foot drop.

FIG 129.4 **A,** Coronal left knee proton density fast spin echo fat saturation (TR: 2280; TE: 12; ST: 3; Spacing: 3.3; Siemens; Matrix: 320/0/0/240). Coronal image at the level of the fibular head including the posterior lateral femoral condyle. Marked soft tissue swelling and edema in the lateral aspect of the left knee. A redundant portion of the ruptured common peroneal nerve (distal portion) is seen in the soft tissues superior to the fibular head (*arrows*). **B,** Sagittal T2 turbo spin echo fat saturation (Siemens; TR: 5691.7; TE: 79; ST: 4; Spacing: 4.4; Matrix: 320/0/0/224; pixel: 0.25/0.25 mm) Sagittal slice of the left knee just medial to the intercondylar notch. Avulsed proximal stump of the common peroneal nerve (*long arrow*) just distal to the branch point from the sciatic nerve (*short arrow*). Soft tissue edema surrounding the avulsed nerve. **C,** Sagittal T2 turbo spin echo fat saturation (Siemens; TR: 5691.7; TE: 79; ST: 4; Spacing: 4.4; Matrix: 320/0/0/224; pixel: 0.25/0.25 mm). Sagittal slice of the left knee lateral to the fibular head. Ruptured and retracted common peroneal nerve is seen within the soft tissues of the lateral knee. The proximal segment (*long arrow*) of the nerve is looped back to the intact distal segment (*short arrow*), resulting in a "lariat" shape. TE, echo time (milliseconds); TR, repitition time (milliseconds). (From Brandon L. Morris, Anders S. Grinde, Hannah Olson, Jacob W. Brubacher, J. Paul Schroeppel, B. MacNeille Everist, Lariat sign: An MRI finding associated with common peroneal nerve rupture, Radiology Case Reports. 2018;13(3):743–746.)

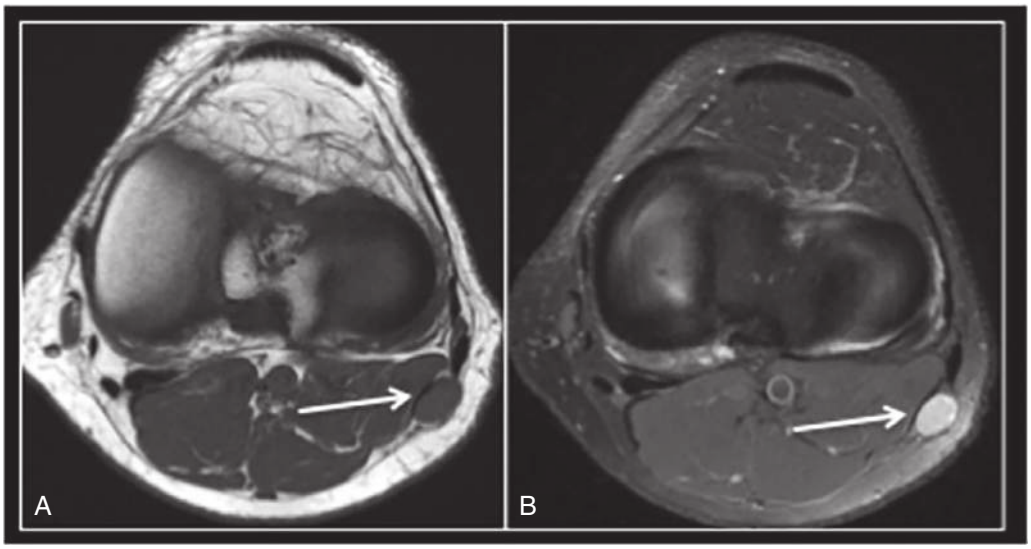

FIG 129.5 Axial T1 **(A)** and Short Tau Inversion Recovery (STIR) magnetic resonance imaging **(B)** showing nerve sheath tumor of common peroneal nerve (*arrow*) with typical split fat sign. (From Iyengar KP, Botchu R. Typical and atypical lesions of common peroneal nerve (CPN). *J Orthop Rep*. 2022;1(1):67–69.)

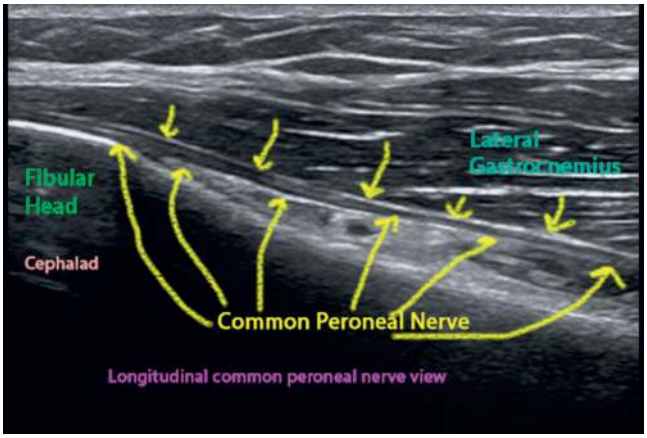

FIG 129.6 Longitudinal ultrasound of the upper lateral leg showing the common peroneal nerve entering the upper calf after coursing around the fibular head.

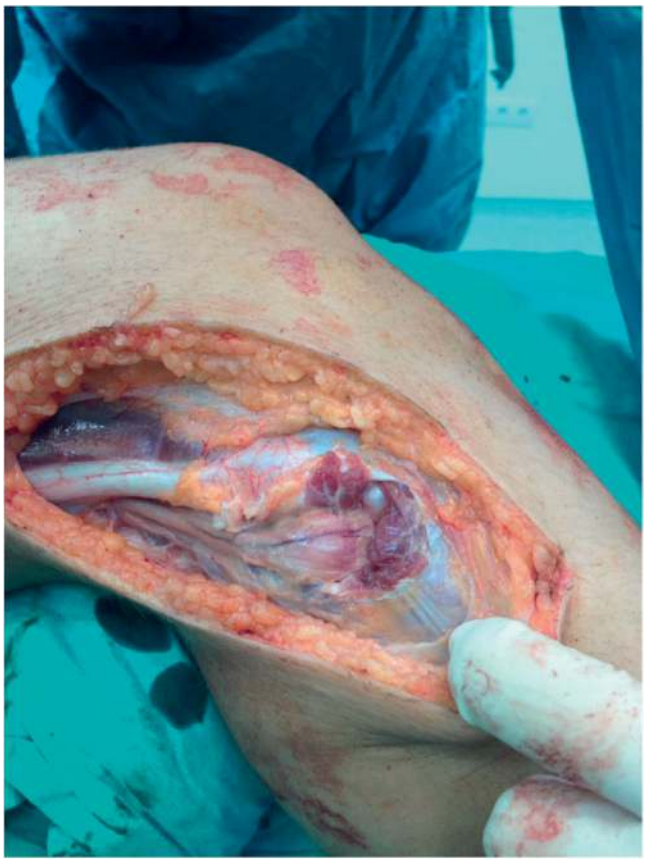

FIG 129.7 Common peroneal nerve entrapment is secondary to osteochondroma. Peroneal nerve looks edematous and inflamed with tibial osteochondroma at the level of fibular neck. (From Demiroğlu M, Özkan K, Kılıç B, Akçal A, Akkaya M, Özkan FÜ. Deep peroneal nerve palsy due to osteochondroma arising from fibular head and proximal lateral tibia. *Int J Surg Case Rep*. 2017;31:200–202.)

the common peroneal nerve until a paresthesia is elicited in the distribution of the common peroneal nerve. The patient should be warned to expect a paresthesia and should be told to say "There!" immediately on perceiving the paresthesia.

Once a paresthesia is elicited in the distribution of the common peroneal nerve, the needle is withdrawn 1 mm and then determine whether any persistent paresthesia exists. If no persistent paresthesia is present after careful aspiration 1.0 mL of 1.0% preservative-free lidocaine is slowly injected. Care must be taken not to advance the needle into the substance of the nerve during the injection and to inject the solution intraneurally. Given the proximity to the superficial femoral artery and vein, the possibility of inadvertent intravascular injection remains an ever-present possibility.

If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are carried out in a similar manner, substituting 40 mg of methylprednisolone for the initial 80 mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation. Use of a nerve stimulator may aid in more accurate needle placement. Proper needle placement in proximity to the sciatic nerve is evidenced by twitching in the toes when stimulated at a level of 0.2–0.5 mA. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of common peroneal nerve entrapment.

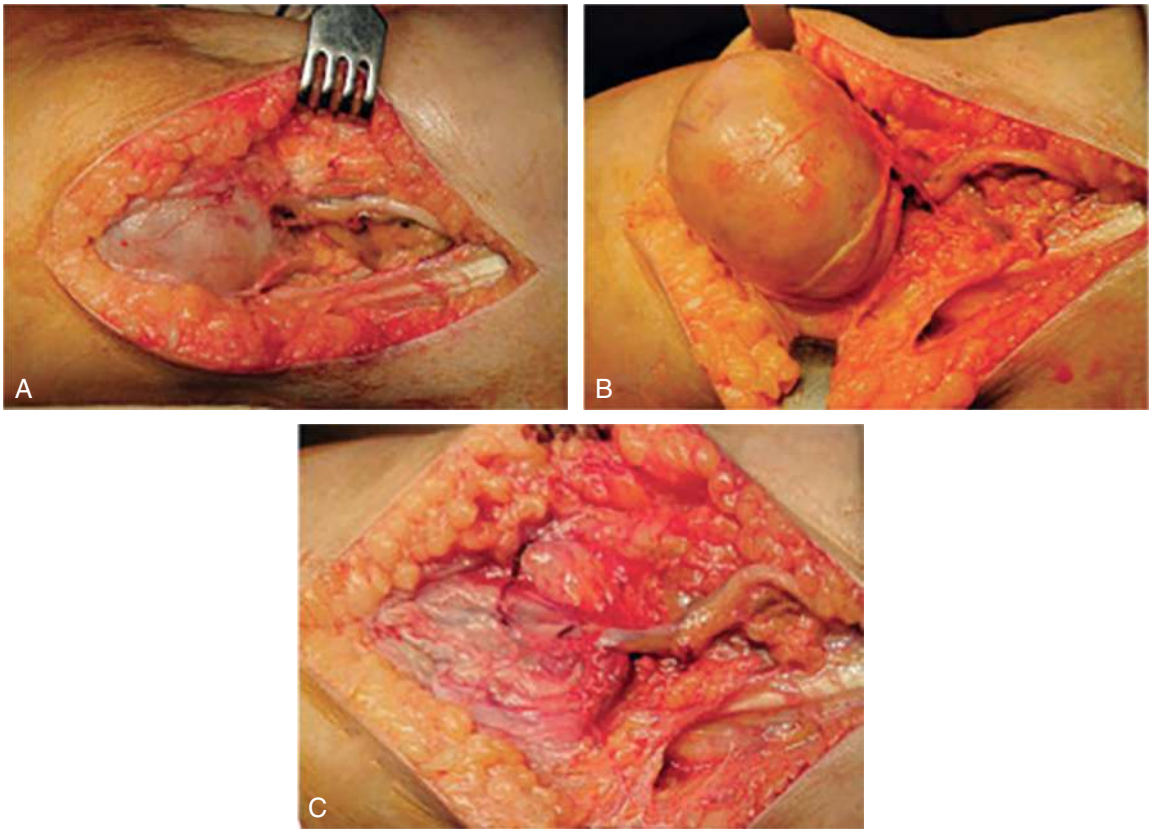

FIG 129.8 Intraoperative partial **(A)** and complete **(B)** dissection of the common peroneal nerve enveloping the giant tumor with branch-like fascicles. The "deflated" common peroneal nerve after enucleation of the tumor **(C)**. (From Georgiev GP, Ananiev J, Slavchev SA. An unusual case of a giant schwannoma of the common peroneal nerve with duration of twenty years. *Curr Probl Cancer Case Rep*. 2021;3:100061.)

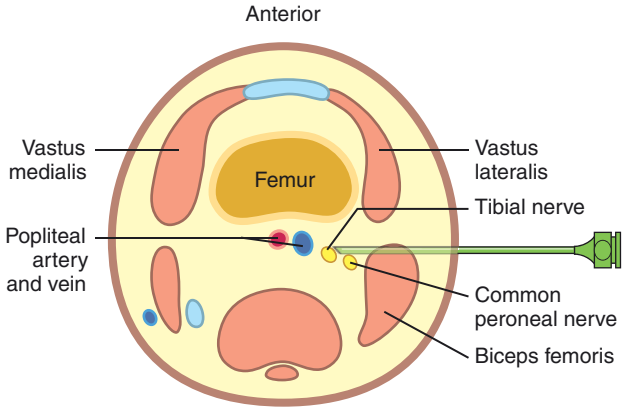

FIG 129.9 Cross-sectional view of the needle placed within the previously identified groove between the vastus lateralis and biceps femoris muscles. The needle is slowly advanced past the common peroneal nerve until it is in proximity to the tibial nerve. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015:740.)

SUGGESTED READINGS

Anderson JC. Common fibular nerve compression: anatomy, symptoms, clinical evaluation, and surgical decompression. *Clin Podiatr Med Surg*. 2016;33(2):283–291.

Broekx S, Van Der Straeten R, D'Haen B, et al. Intraneural ganglion cyst of the common peroneal nerve causing foot drop in a 12-year old child. *Clin Neurol Neurosurg*. 2021;209:106915. https://doi.org/10.1016/j.clineuro.2021.106915.

Iyengar KP, Botchu R. Typical and atypical lesions of common peroneal nerve (CPN). *J Orthop Rep*. 2022;1(1):67–69. https://doi.org/10.1016/j.jorep.2022.03.009.

Lisovski V, Minderis M. Intraneural ganglion cyst: a case report and a review of the literature. *Acta Med Litu*. 2019;26(2):147–151. https://doi.org/10.6001/actamedica.v26i2.4036.

Milenković SS, Mitković MM. Common peroneal nerve schwannoma. *Hippokratia*. 2018;22:91.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin North Am*. 2013;51(3):433–454.

Waldman SD. Deep infrapatellar bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:799–804.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308.

Waldman SD, Campbell RSD. Deep infrapatellar bursitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:408–410.

Tibial Nerve Entrapment at the Knee

ICD-10 CODE G57.30

THE CLINICAL SYNDROME

The tibial nerve is one of the two major continuations of the sciatic nerve, and the other is the common peroneal nerve. It provides sensory innervation to the posterior portion of the calf, the heel, and the medial plantar surface. The nerve splits from the sciatic nerve at the superior margin of the popliteal fossa and descends in a slightly medial course through the popliteal fossa (Fig. 130.1). The tibial nerve block at the knee lies just beneath the popliteal fascia and is readily accessible for neural blockade. The tibial nerve continues its downward course, running between the two heads of the gastrocnemius muscle, passing deep to the soleus muscle. The nerve courses medially between the Achilles tendon and the medial malleolus, where it divides into the medial and lateral plantar nerves, providing sensory innervation to the heel and medial plantar surface (Figs. 130.2 and 130.3). The tibial nerve is occasionally subject to compression at this point, which is known as posterior tarsal tunnel syndrome.

The tibial nerve may be entrapped or compressed within the popliteal fossa. The tibial nerve can be compromised at the popliteal fossa by popliteal artery aneurysms and cysts as well as Baker's synovial cysts (Figs. 130.4 and 130.5). The symptoms associated with tibial nerve compromise depend on the point at which the nerve is compromised with entrapment at the ankle a common clinical presentation (Fig. 130.6).

SIGNS AND SYMPTOMS

Patients suffering from tibial nerve entrapment will experience burning, tingling, numbness, and dysesthesias in the sensory distribution of the tibial l nerve (see Fig. 130.3). These symptoms may worsen at night. Physical findings include tenderness over the tibial nerve at the point of entrapment. A positive Tinel's sign at the point of entrapment is often present. If the nerve is tethered down by a large Baker's cyst, hematoma, tumor, or aneurysm, extension of the affected knee may increase the patient's pain.

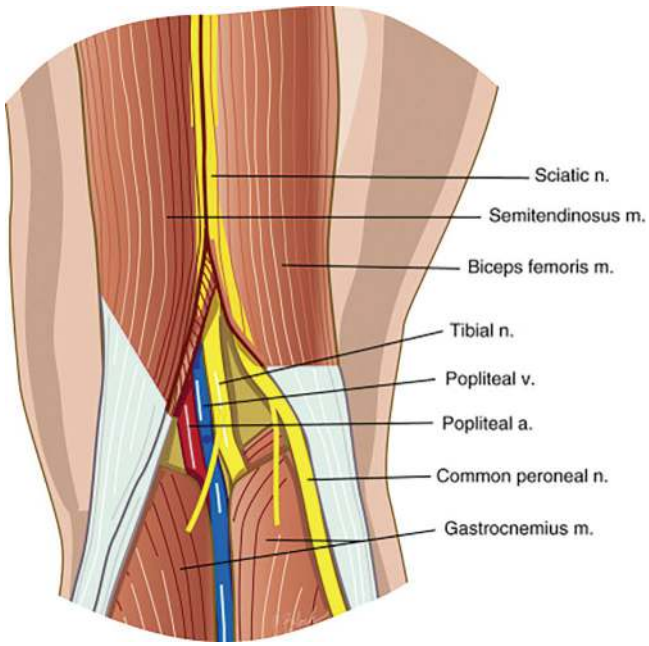

FIG 130.1 Anatomy of the tibial nerve at the popliteal fossa. *a*, Artery; *m*, muscle; *n*, nerve; *v*, vein. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015:733.)

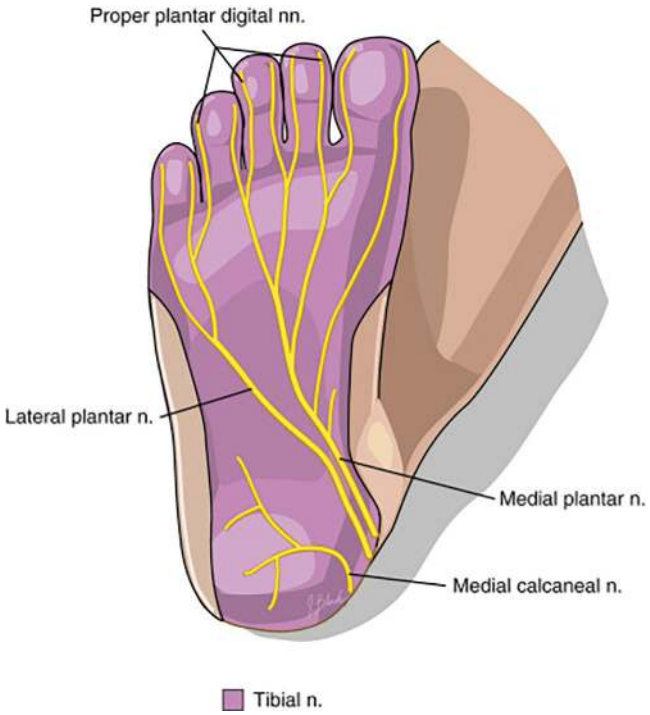

FIG 130.2 Distribution of the branches of the tibial nerve across the plantar surface. *n/nn*, Nerve/nerves. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Saunders; 2015:733.)

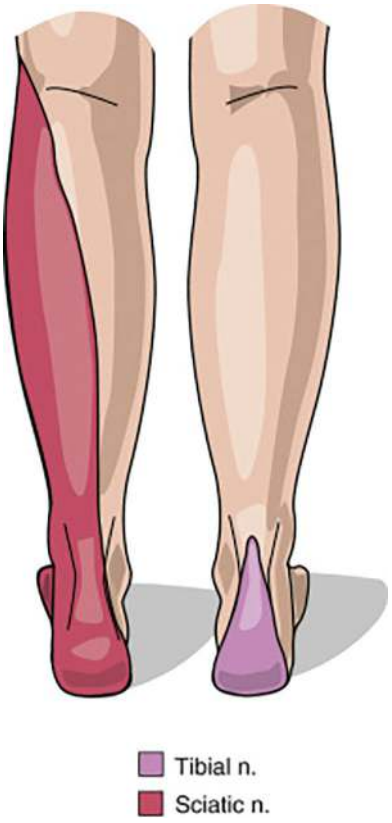

FIG 130.3 Sensory distribution of the tibial nerve. *n.*, Nerve. (From Waldman SD. *Atlas of interventional pain management.* 4th ed. Philadelphia: Saunders; 2015:733.)

TESTING

Plain radiographs, ultrasound, arteriography, and magnetic resonance imaging of the knee may reveal calcification of the bursa and associated structures as well as other masses including Baker's cyst, popliteal artery cysts, tumor, or popliteal artery aneurysm (Fig. 130.7). Electrodiagnostic testing should be considered in all patients who suffer from tibial nerve dysfunction to provide both neuroanatomic and neurophysiologic information regarding nerve function. Comprehensive metabolic profile and thyroid function testing should be obtained to rule out systemic and endocrine diseases that may cause vulnerable nerve syndrome (e.g., diabetes). Antinuclear antibody testing is indicated if collagen vascular disease is suspected.

DIFFERENTIAL DIAGNOSIS

Because of the anatomy of the region, the associated tendons and structures of the knee can become inflamed, thus confusing the diagnosis. Anything that compresses, entraps, or damages the tibial nerve can contribute to the patient's pain and functional disability (Figs. 130.8 and 130.9; Box 130.1).

TREATMENT

A short course of conservative therapy consisting of simple analgesics, nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors, and gait training to decrease the risk of falling is the first step in the treatment of tibial nerve entrapment. Removal of the source of nerve entrapment or compression is crucial to prevent permanent nerve damage. Tricyclic antidepressants may help with sleep disturbance. If patients do not experience rapid improvement, injection is a reasonable next step.

To inject the tibial nerve at the knee, the patient is placed in the prone position with the leg slightly flexed. The skin crease of the knee and margins of the semitendinosus and biceps femoris muscles in the upper popliteal fossa are palpated. The margins of these muscles can be more easily identified by having the s. An imaginary triangle is envisioned, with the apex at the convergence of these two muscles and the base at the skin crease of the knee. At a point in the center of this imaginary apex, the skin is prepared with antiseptic solution. A 25-gauge, 1½-inch needle is then slowly advanced perpendicular to the skin through this point toward the tibial nerve until paresthesia is elicited in the distribution of the tibial nerve. The patient should be warned to expect paresthesia and should be told to say "there!" as soon as the paresthesia is felt. Paresthesia usually is elicited at a depth of ½ to ¾ inch. If it is not elicited, the needle is withdrawn and redirected slightly more medially until paresthesia is obtained. Once paresthesia is elicited in the distribution of the tibial nerve, the needle is withdrawn 1 mm, and the patient is observed to rule out any persistent paresthesia. If no persistent paresthesia is present, and after careful aspiration, 8 mL of 1.0% preservative-free lidocaine is slowly injected. Care must be taken not to advance the needle into the substance of the nerve during the injection and inject solution intraneurally. Given the proximity to the common peroneal nerve, this nerve may also be blocked when performing tibial nerve block at the knee. If the pain has an inflammatory component, the local anesthetic is combined with 80 mg of methylprednisolone and is injected in incremental doses. Subsequent daily nerve blocks are performed in a similar manner, substituting 40 mg of methylprednisolone for the initial 80 mg dose. After injection of the solution, pressure is applied to the injection site to decrease the incidence of postblock ecchymosis and hematoma formation. The use of a nerve stimulator may aid in more accurate needle placement. Proper needle placement in proximity to the sciatic nerve is evidenced by twitching in the toes and feet when stimulated at a level of 0.2–0.5 mA. Ultrasound guidance may be useful in assisting in needle placement if difficulty is encountered (Fig. 130.10). Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

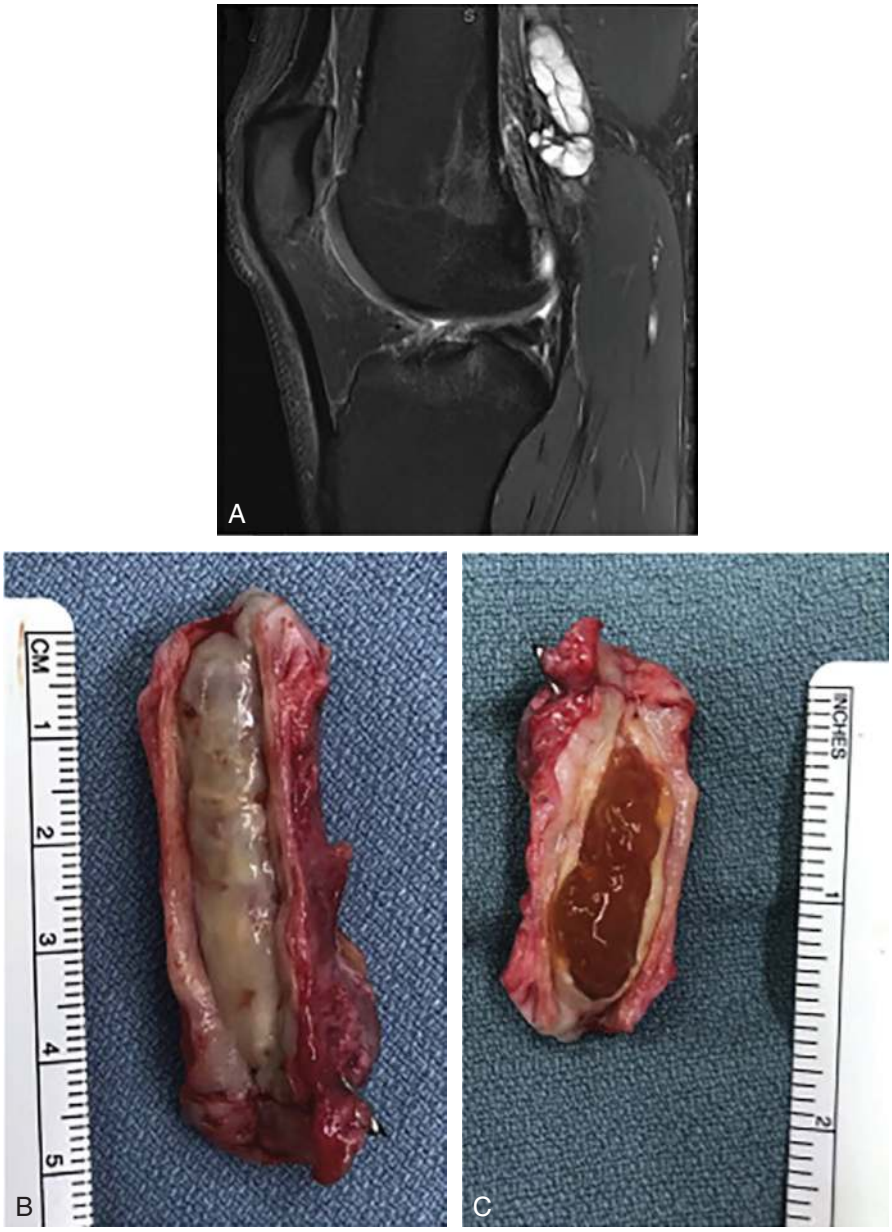

FIG 130.4 Popliteal artery cyst compressing the contents of the popliteal fossa. **A,** Magnetic resonance angiography (MRA) showing popliteal cyst. **B,** Surgical specimen of popliteal cyst demonstrating intraluminal mucinous material. **C,** Cyst cavity after mucinous removal. (From Konda S, Reed AB. Popliteal artery adventitial cystic disease in an athlete. *J Vasc Surg Cases Innov Tech.* 2022;8(2):140–141.)

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the knee joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, tendinitis, arthritis, and internal derangement of the knee may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of common peroneal nerve entrapment.

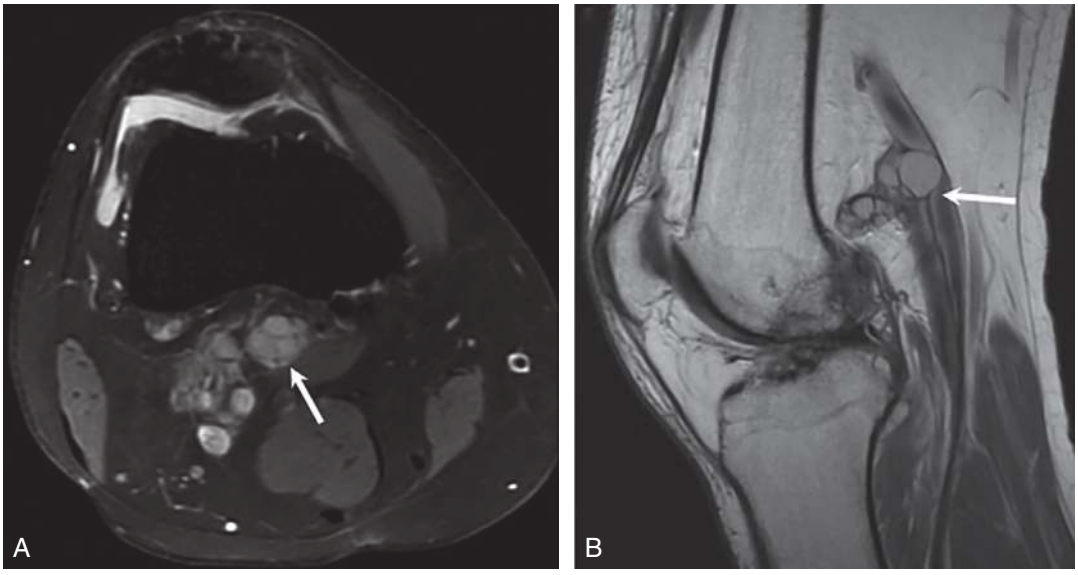

FIG 130.5 Magnetic resonance images showing cystic mass (*white arrow*) adjacent to the popliteal artery compressing the tibial nerve and popliteal artery. **A,** T1-weighted image. **B,** T2-weighted image. (From Chan MC-Y, Cornwall J, Ilonzo N, McKinsey J. Cystic adventitial disease of the popliteal vein and artery in siblings. *J Vasc Surg Cases Innov Tech*. 2021;7(3):545–548.)

FIG 130.6 Compression of the tibial nerve in the popliteal fossa may result in pain in the posterior knee and numbness across the plantar surface.

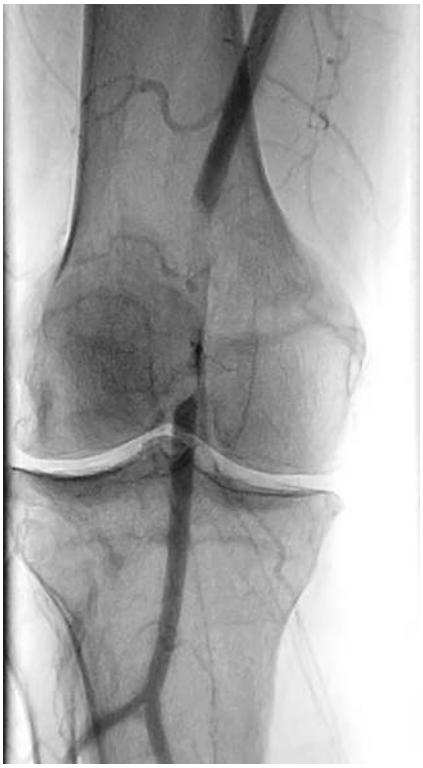

FIG 130.7 Conventional angiogram demonstrating hourglass-shaped subtotal occlusion from extrinsic compression in the popliteal artery (scimitar sign). At surgery, this compression was found to be secondary to a popliteal artery cyst. (From Chan MC-Y, Cornwall J, Ilonzo N, McKinsey J. Cystic adventitial disease of the popliteal vein and artery in siblings. *J Vasc Surg Cases Innov Tech*. 2021;7(3):545–548.)

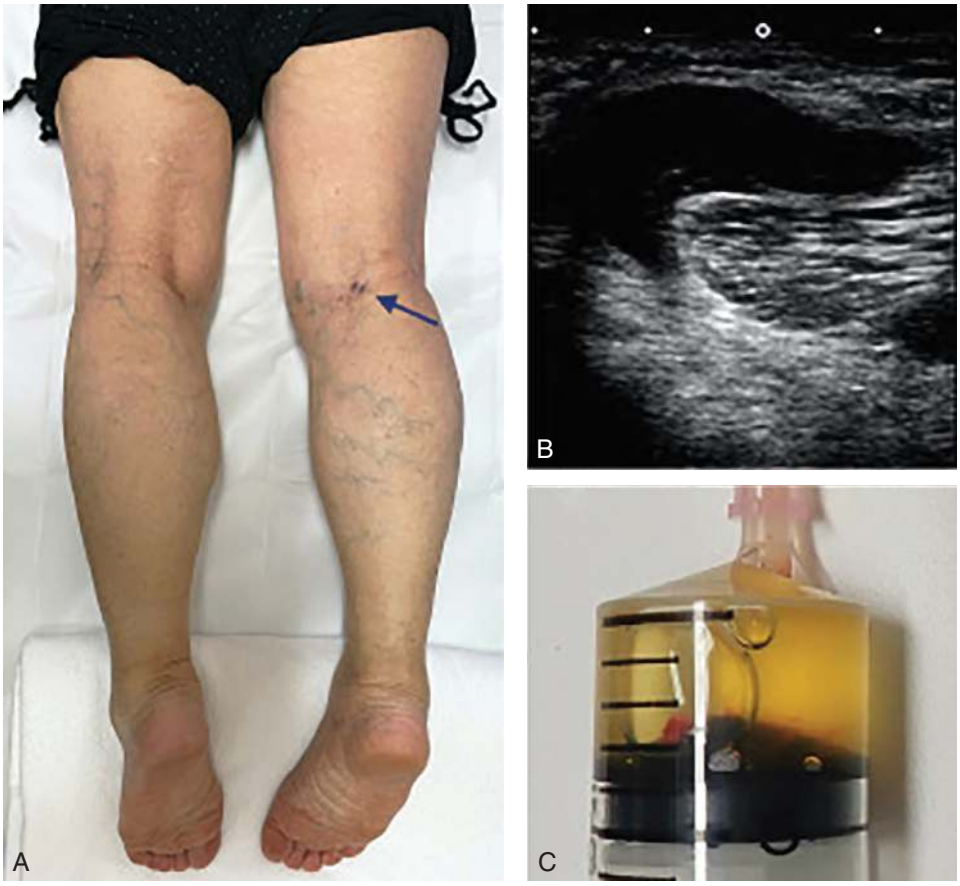

FIG 130.8 **A,** Swelling of the popliteal region with ecchymosis (*arrow*) in the popliteal fossa. **B,** Ultrasonography visualized a cyst below the ecchymosis. **C,** The fluid drained by aspiration of the popliteal cyst was clear, viscous, and yellow and showed an admixture of blood resulting from the trauma tap. (From Kano Y, Harada Y. Popliteal ecchymosis in ruptured Baker's cyst. *Am J Med.* 2021;134(4):e277.)

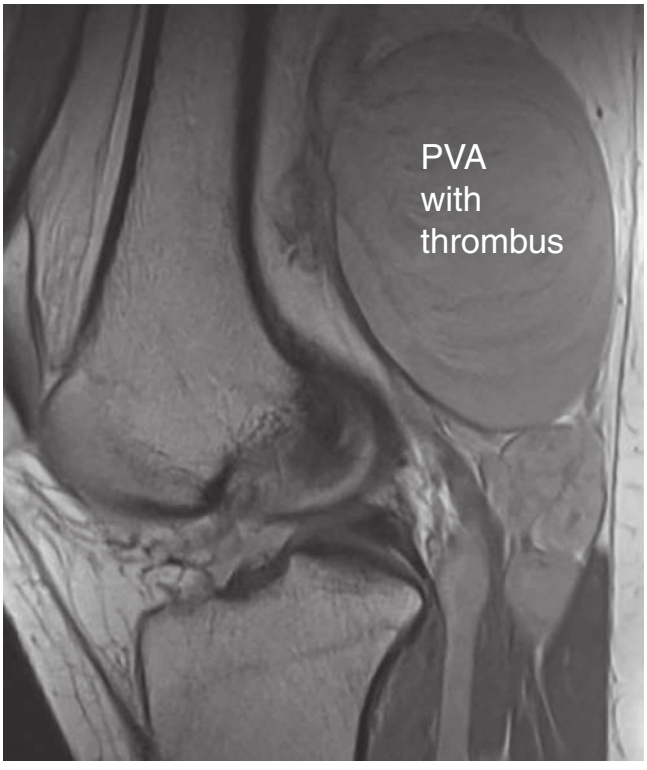

FIG 130.9 Popliteal vein aneurysm compressing contents of the popliteal fossa. Magnetic resonance imaging of the right knee. *PVA*, popliteal vein aneurysm. (From Krishan A, Droste JC, Molloy K, et al. Popliteal vein aneurysm masquerading as a Baker's cyst leading to pulmonary embolism. *Am J Med.* 2021;134(12):1495–1498.)

BOX 130.1 Causes of Tibial Nerve Entrapment at the Knee

- Direct trauma
  - Blunt injuries, lacerations
  - Fractures of the tibia and fibula
  - Adduction injuries and dislocations of the knee
  - Surgery and arthroscopy in popliteal fossa and knee
- Traction injuries
  - Acute ankle injuries
- Masses
  - Ganglia
  - Baker's cysts
  - Popliteal cysts
  - Bony callus formation
  - Fibular tumors
  - Osteomas
  - Hematomas
- Tumors
  - Nerve sheath tumors
  - Nerve sheath ganglia
  - Lipomas
- Entrapment
  - In the popliteal fossa
  - Anterior (tibial) compartment syndrome
- Vascular
  - Vasculitis, local vascular disease
- Diabetes mellitus: susceptibility to compression, ischemic damage
- Leprosy
- Idiopathic

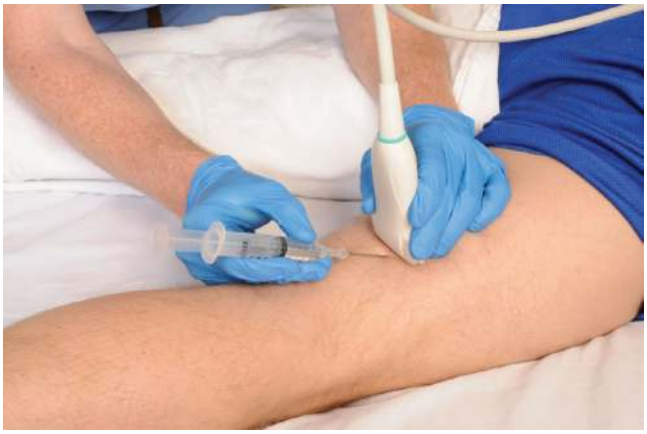

FIG 130.10 Proper out-of-plane needle position for performing tibial nerve block at the popliteal fossa.

SUGGESTED READINGS

Anderson JC. Common fibular nerve compression: anatomy, symptoms, clinical evaluation, and surgical decompression. *Clin Podiatr Med Surg*. 2016;33(2):283–291.

Chan MC-Y, Cornwall J, Ilonzo N, McKinsey J. Cystic adventitial disease of the popliteal vein and artery in siblings. *J Vasc Surg Cases Innov Tech*. 2021;7(3):545–548. https://doi.org/10.1016/j.jvscit.2021.04.011.

Kano Y, Harada Y. Popliteal ecchymosis in ruptured Baker's cyst. *Am J Med*. 2021;134(4):e277. https://doi.org/10.1016/j.amjmed.2020.09.039.

Konda S, Reed AB. Popliteal artery adventitial cystic disease in an athlete. *J Vasc Surg Cases Innov Tech*. 2022;8(2):140–141. https://doi.org/10.1016/j.jvscit.2022.01.004.

Krishan A, Droste JC, Molloy K, et al. Popliteal vein aneurysm masquerading as a Baker's cyst leading to pulmonary embolism. *Am J Med*. 2021;134(12):1495–1498. https://doi.org/10.1016/j.amjmed.2021.06.026.

Lisovski V, Minderis M. Intraneural ganglion cyst: a case report and a review of the literature. *Acta Medica Litu*. 2019;26(2):147–151. https://doi.org/10.6001/actamedica.v26i2.4036.

Steinbach LS, Stevens KJ. Imaging of cysts and bursae about the knee. *Radiol Clin North Am*. 2013;51(3):433–454.

Waldman SD. Bursitis syndromes of the knee. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:306–308.

Saphenous Neuralgia

ICD-10 G58.8

THE CLINICAL SYNDROME

Saphenous neuralgia, which is also known as gonalgia paresthetica, is caused by compression of the saphenous nerve by the sartorius muscle and the adductor longus and magnus muscles as the nerve passes through the Hunter canal or, more commonly, as the nerve passes over the medial condyle of the femur (Fig. 131.1). This entrapment neuropathy manifests as pain, numbness, and dysesthesia in the distribution of the saphenous nerve. These symptoms often begin as a burning pain over the medial knee. Patients with saphenous neuralgia note that sitting or squatting often causes the symptoms of saphenous neuralgia to worsen (Fig. 131.2). Although traumatic lesions to the saphenous nerve after vein stripping surgery and vein harvest surgery for coronary artery bypass surgery have been implicated in the onset of saphenous neuralgia, in most patients no obvious antecedent trauma can be identified (Fig. 131.3).

SIGNS AND SYMPTOMS

Physical findings include tenderness over the saphenous nerve just medial to the midline of the midthigh. A positive Tinel sign over the saphenous nerve as it passes over the medial femoral condyle may be present. Careful sensory examination of the medial thigh reveals a sensory deficit in the distribution of the saphenous nerve (Fig. 131.4). No motor deficit should be present. Sitting or squatting, which compress the saphenous nerve, may exacerbate the symptoms of saphenous neuralgia.

TESTING

Saphenous neuralgia often is misdiagnosed as lumbar radiculopathy or is attributed to primary knee disease. Radiographs, ultrasonography, and magnetic resonance imaging (MRI) of the knee and electromyography help distinguish saphenous neuralgia from radiculopathy or pain emanating from the

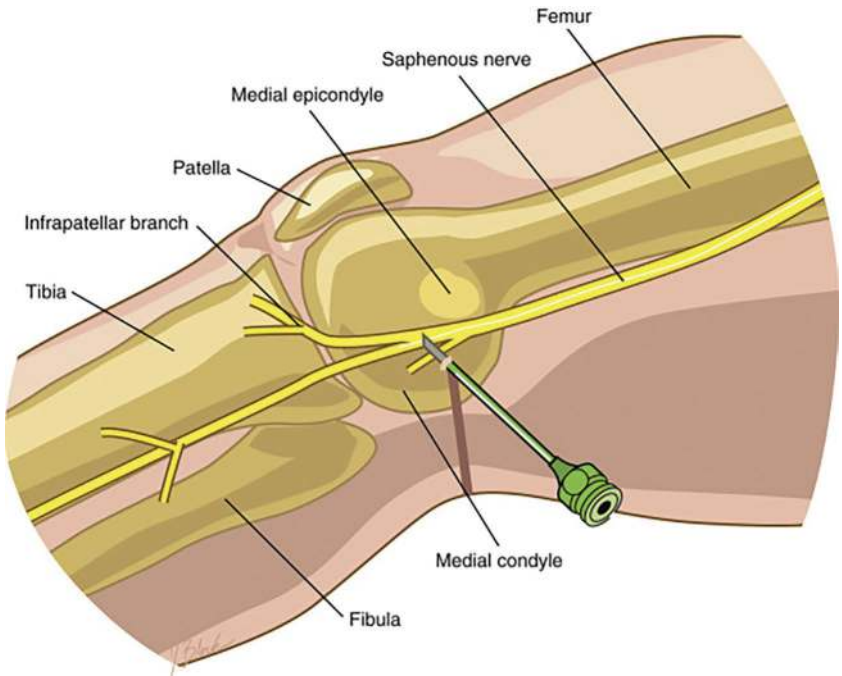

FIG 131.1 Anatomy of the saphenous nerve at the knee. (From Waldman SD. Saphenous nerve block at the knee. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:897–902.)

knee (Fig. 131.5). Most patients with a lumbar radiculopathy have back pain associated with reflex, motor, and sensory changes and associated with neck pain, whereas patients with saphenous neuralgia have no back pain and no motor or reflex changes. The sensory changes of saphenous neuralgia are limited to the distribution of the saphenous nerve. Lumbar radiculopathy and saphenous nerve entrapment may coexist as the so-called double crush syndrome. Occasionally, diabetic femoral neuropathy may produce anterior thigh pain, which may confuse the diagnosis.

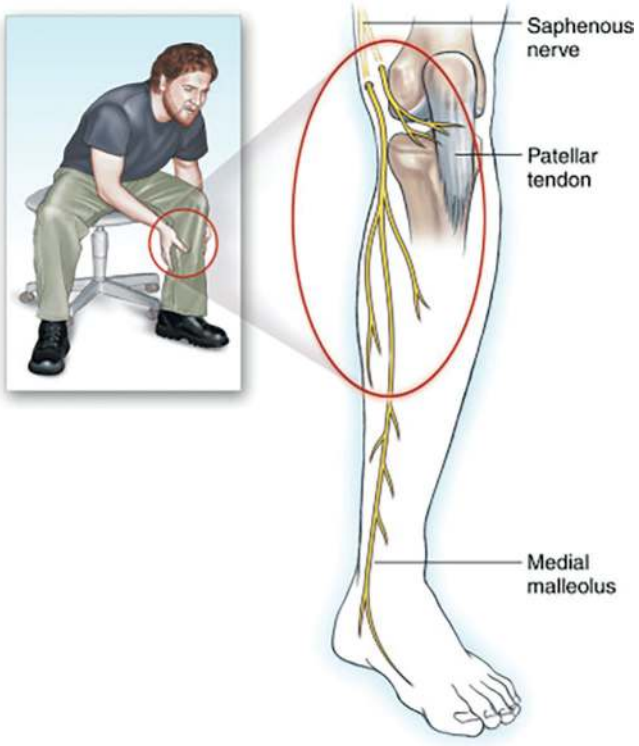

FIG 131.2 Patients with saphenous neuralgia present with burning pain that radiates into the medial calf to the medial malleolus that is made worse with sitting or squatting. (From Waldman SD. Saphenous nerve block at the knee. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:897–902.)

Electromyography helps distinguish lumbar radiculopathy and diabetic femoral neuropathy from saphenous neuralgia. Plain radiographs of the back, hip, and pelvis are indicated for all patients with saphenous neuralgia to rule out occult bony disease. On the basis of the patient's clinical presentation, additional testing may be indicated, including complete

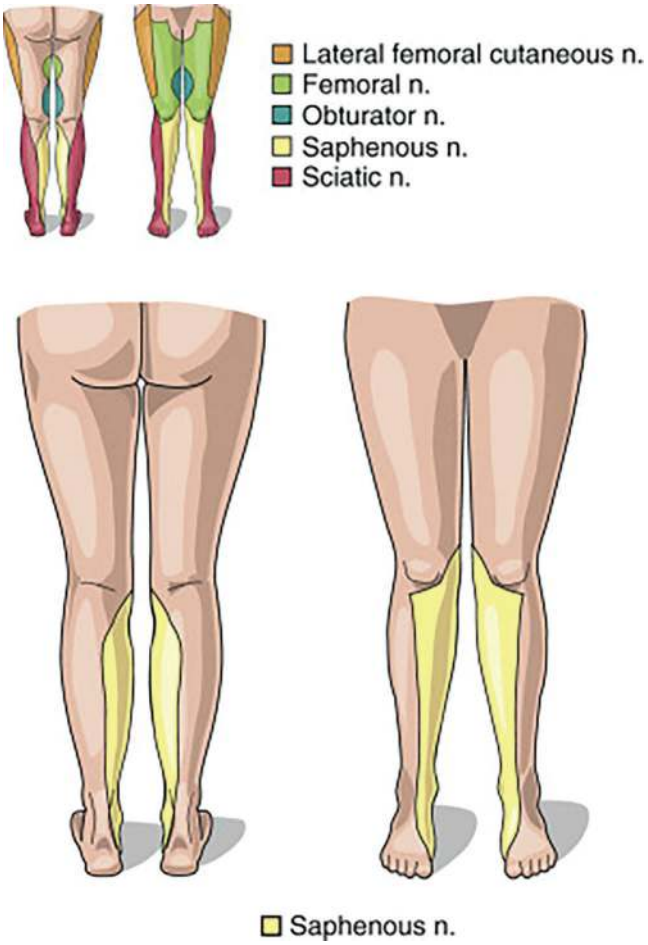

FIG 131.4 The sensory distribution of the saphenous nerve. (From Waldman SD. Saphenous nerve block at the knee. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:897–902.)

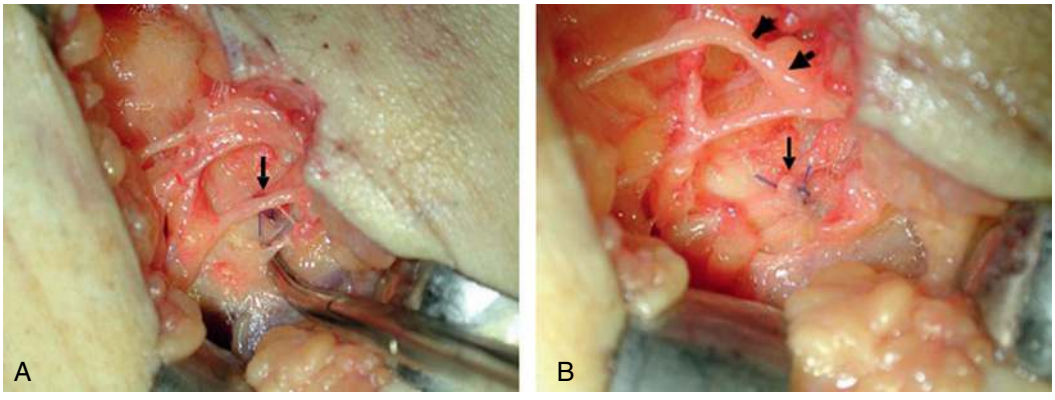

FIG 131.3 **A,** Open saphenous nerve dissection showing suture from meniscus repair wrapped around a posteromedial branch off the sartorial branch of saphenous nerve. **B,** Higher magnification view; *arrows*, posteromedial branches of sartorial nerve; *arrowheads*, sartorial branch of saphenous nerve. (From Diana C. Patterson, Carl M. Cirino, James N. Gladstone, No safe zone: The anatomy of the saphenous nerve and its posteromedial branches. *The Knee*. 2019;26(3):660–665.)

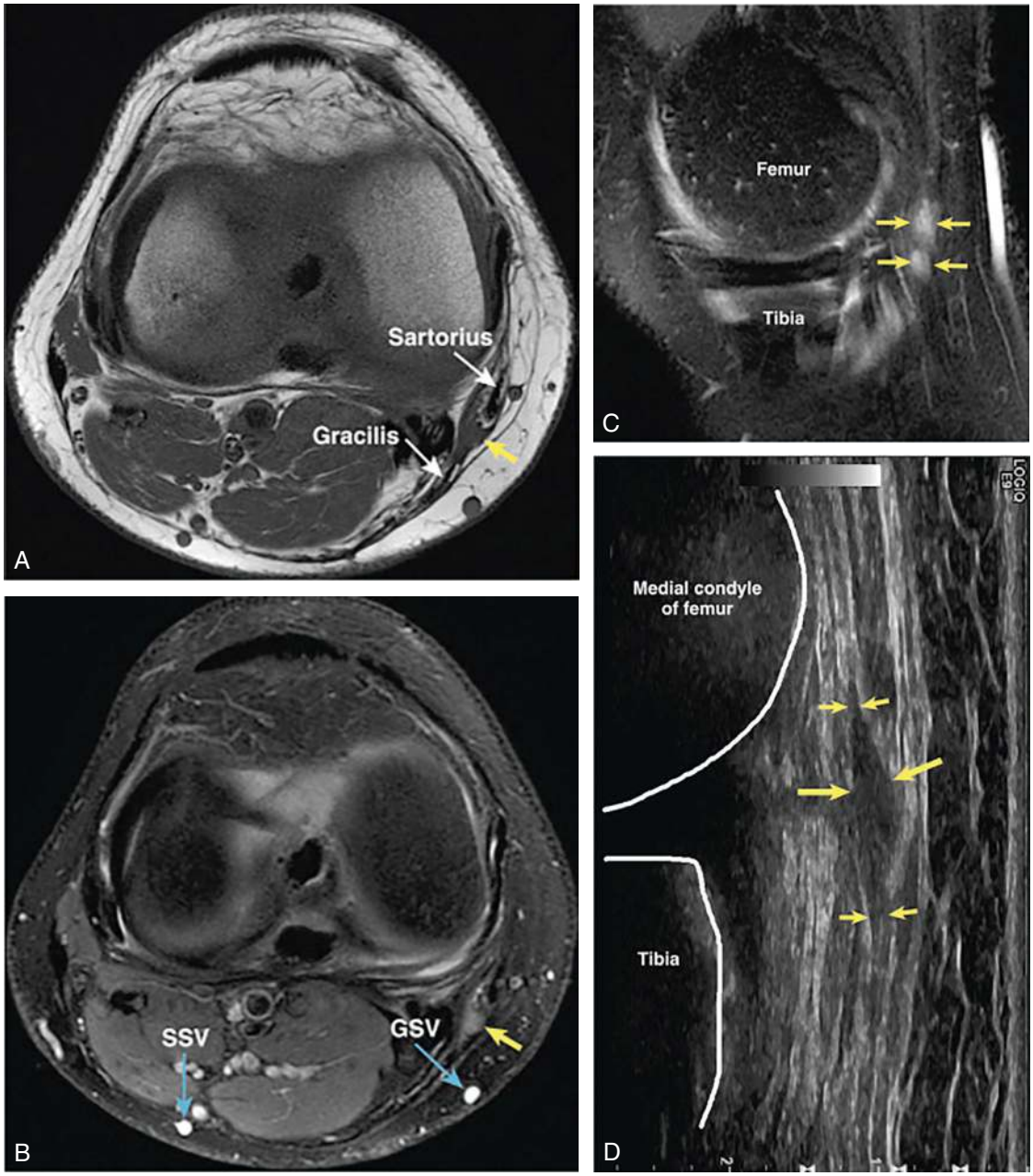

FIG 131.5 Axial T1-weighted **(A)** and PD-weighted fat-suppressed **(B)** images, sagittal PD-weighted fat-suppressed **(C)** and ultrasound (*arrows* delineate postsurgical neuroma) **(D)** images of a right knee. Postsurgery neuroma of the sartorial branch of the saphenous nerve at the point where it becomes superficial between the sartorius (*S*) and gracilis (*G*) tendons. *GSV*, Great saphenous vein; *SSV*, small saphenous vein. (From Damarey B, Demondion X, Wavreille G, et al. Imaging of the nerves of the knee region. *Eur J Radiol*. 2013;82:27–37 (Fig. 9).)

blood cell count, uric acid, sedimentation rate, and antinuclear antibody testing. MRI of the back is indicated if herniated disk, spinal stenosis, or a space-occupying lesion is suggested. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Saphenous neuralgia often is misdiagnosed as lumbar radiculopathy or is attributed to primary knee disease. Radiographs of the knee and electromyography help distinguish saphenous neuralgia from radiculopathy or pain emanating from the knee. Most patients with a lumbar radiculopathy have back pain associated with reflex, motor, and sensory changes and associated with neck pain, whereas patients with saphenous neuralgia have no back pain and no motor or reflex changes. The sensory changes of saphenous neuralgia are limited to the distribution of the saphenous nerve. Lumbar radiculopathy and saphenous nerve entrapment may coexist as the so-called double crush syndrome. Occasionally,

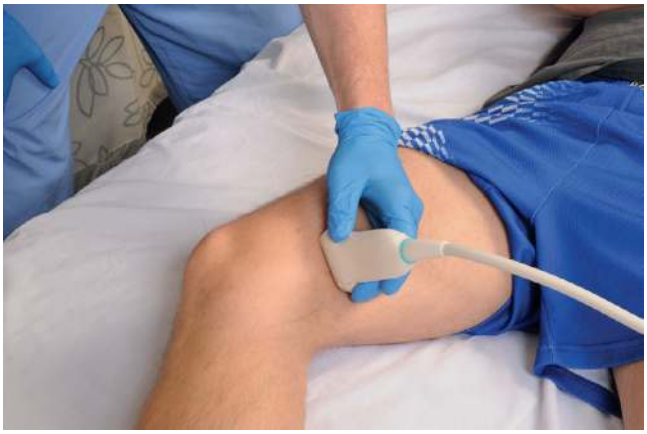

FIG 131.6 The transversely placed ultrasound transducer is moved medially to identify the sartorius muscle and the saphenous nerve beneath it.

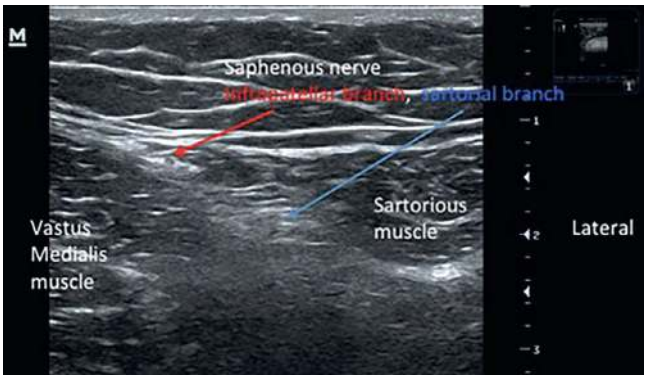

FIG 131.7 Ultrasound image demonstrating relationship of the saphenous nerve and sartorius muscle.

diabetic femoral neuropathy may produce anterior thigh pain, which may confuse the diagnosis. Electromyography helps distinguish lumbar radiculopathy and diabetic femoral neuropathy from saphenous neuralgia.

TREATMENT

To perform ultrasound-guided saphenous nerve block at the knee, the patient is placed in the supine position with the arms resting comfortably across the chest and the affected lower extremity externally rotated. A total of 8 mL of local anesthetic is drawn up in a 12-mL sterile syringe. If the painful condition being treated is thought to have an inflammatory component, 40–80 mg of depot steroid is added to the local anesthetic. A point approximately 5 cm above the patella on the anteromedial femur is then identified by palpation. A high-frequency linear ultrasound transducer is placed in a transverse plane over the previously identified point on the anteromedial femur and a sonogram is obtained (Fig. 131.6). The hyperechoic anteromedial border of the femur will be visualized as well as the vastus medialis muscle just anteromedial to it. The ultrasound transducer is then slowly moved in a more medial direction until the sartorius muscle, which lies posteromedial to the vastus medialis muscle, is visualized. The saphenous nerve lies just in the fascial plane just below the sartorius muscle (Fig. 131.7). When the fascial plane below the sartorius muscle is identified on ultrasound imaging, the skin is prepped with anesthetic solution, and a 1½-inch, 22-gauge needle is advanced from the lateral border of the ultrasound transducer and advanced using an in-plane approach with the trajectory adjusted under real-time ultrasound guidance until the needle tip is resting within the fascial plane beneath the sartorius muscle in proximity to the saphenous nerve (Fig. 131.8). When the tip of needle is thought to be in satisfactory position, a small amount of local anesthetic and steroid is injected under real-time ultrasound guidance to confirm that the needle tip is correctly beneath the sartorius muscle in proximity to the saphenous nerve. There should be minimal resistance to injection. After needle tip placement is confirmed, the remainder of the contents of the syringe is slowly injected. The needle is then removed, and a sterile pressure dressing and ice pack are placed at the injection site.

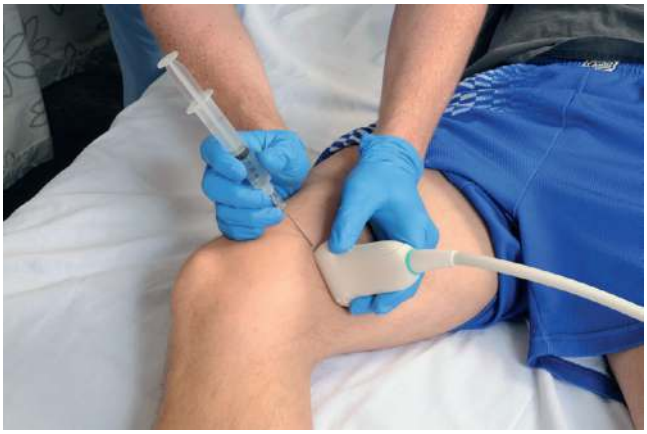

FIG 131.8 Proper in-plane needle position for performing saphenous nerve block.

COMPLICATIONS AND PITFALLS

The main side effect of saphenous nerve block at the knee is postblock ecchymosis and hematoma formation because the nerve is in close proximity to the greater saphenous artery. As mentioned earlier, pressure should be maintained on the injection site after block to avoid ecchymosis and hematoma formation. Because a paresthesia is elicited with this technique, needle-induced trauma to the saphenous nerve remains a possibility. By advancing the needle slowly and then withdrawing the needle slightly away from the nerve, needle-induced trauma to the saphenous nerve can be avoided.

CLINICAL PEARLS

Saphenous nerve block at the knee is a simple technique that can produce dramatic relief for patients with saphenous neuralgia. Careful preblock neurologic assessment is important to avoid preexisting neurologic deficits later attributed to the saphenous nerve block at the knee. These assessments are especially important in patients who have sustained trauma to the distal femur, have undergone vascular procedures on the lower extremity, or have diabetic neuropathy in which saphenous nerve block at the knees is used for acute pain

control. Compressive neuropathy of the saphenous nerve at the knee is sometimes seen in musicians who play the cello; this painful syndrome is called *viol paresthesia*.

The most common cause of pain radiating into the lower extremity is herniated lumbar disk or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the saphenous nerve per se. Other pain syndromes that may be confused with saphenous nerve entrapment include lesions above the origin of the saphenous nerve, such as lesions of the femoral nerve, and lesions of the saphenous nerve at the ankle. Electromyography and MRI of the lumbar spine combined with the clinical history and physical examination can help sort out the cause of distal lower pain.

SUGGESTED READINGS

Cançado de Morais Ribeiro G, Damaceno Emiliano J, Campolina Fonseca Á, et al. Anatomical variation of the saphenous nerve path: a case report. *Morphologie*. 2022;106(354):199–202. https://doi.org/10.1016/j.morpho.2021.05.003.

Garwood ER, Duarte A, Bencardino JT. MR imaging of entrapment neuropathies of the lower extremity. *Radiol Clin North Am*. 2018;56(6):997–1012. https://doi.org/10.1016/j.rcl.2018.06.012.

Horteur C, Cavalié G, Gaulin B, et al. Saphenous nerve injury after anterior cruciate ligament reconstruction: reduced numbness area after ligamentoplasty using quadriceps tendon compared with hamstring tendon. *Knee*. 2020;27(4):1151–1157. https://doi.org/10.1016/j.knee.2020.05.020.

Waldman SD. Abnormalities of the saphenous nerve at the knee. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:820–824.

Waldman SD. Saphenous nerve block technique. In: *Pain review*. 2nd ed. Philadelphia: W.B. Saunders; 2017:537–539.

Waldman SD. Ultrasound guided saphenous nerve block at the knee. In: *Waldman's comprehensive atlas of ultrasound guided pain management injection techniques*. 2nd ed. Philadelphia: Wolters Kluwer; 2020:1092–1099.

Waldman SD. Saphenous nerve block at the knee. In: *Atlas of interventional pain management*. 5th ed. Philadelphia: Elsevier; 2021:897–902.

Waldman SD. Saphenous neuralgia. In: *Atlas of uncommon pain syndromes*. 4th ed. Philadelphia: Elsevier; 2021:365–367.

132

Tennis Leg

ICD-10 CODE S86.919A

THE CLINICAL SYNDROME

Tennis leg is the term applied to acute injury of the musculotendinous unit of the gastrocnemius muscle. This injury occurs most commonly following an acute, forceful push-off with the foot of the affected leg. Although this injury has been given the name tennis leg because of its common occurrence in tennis players, tennis leg can also be seen in divers, jumpers, hill runners, and basketball players. Occurring most commonly in men in the fourth to sixth decade, tennis leg is usually the result of an acute traumatic event secondary to a sudden push-off or lunge with the back leg while the knee is extended and the foot dorsiflexed, thus placing maximal eccentric tension on the lengthened gastrocnemius muscle (Fig. 132.1). Tennis leg has also been reported during namaz praying owing to simultaneously forced dorsiflexion of ankle and extension of the knee.

The main functions of the gastrocnemius muscle are to plantar flex the ankle and to provide stability to the posterior knee. The medial head of the muscle finds its origin at the posterior aspect of the medial femoral condyle, and, coursing inferiorly, it merges with the musculotendinous unit of the soleus muscle to form the Achilles tendon. Several tendinous insertions are spread throughout the belly of the gastrocnemius muscle, and strain or complete rupture is most likely to occur at these points (Fig. 132.2).

SIGNS AND SYMPTOMS

In most patients, the pain of tennis leg occurs acutely; it is often quite severe and is accompanied by an audible pop or snapping sound. The pain is constant and severe and is localized to the medial calf. The patient often complains that it felt like a knife was suddenly stuck into the medial calf. Patients with complete rupture of the gastrocnemius musculotendinous unit experience significant swelling, ecchymosis, and hematoma formation that may extend from the medial thigh to the ankle (Fig. 132.3). If this swelling is not too severe, the clinician may identify a palpable defect in the medial calf, as well as obvious asymmetry when compared with the uninjured side. The clinician can elicit pain by passively dorsiflexing the ankle of the patient's affected lower extremity and by having the patient plantar flex the ankle against active resistance. The Thompson Squeeze Test will help identify Achilles tendon rupture (Fig. 132.4).

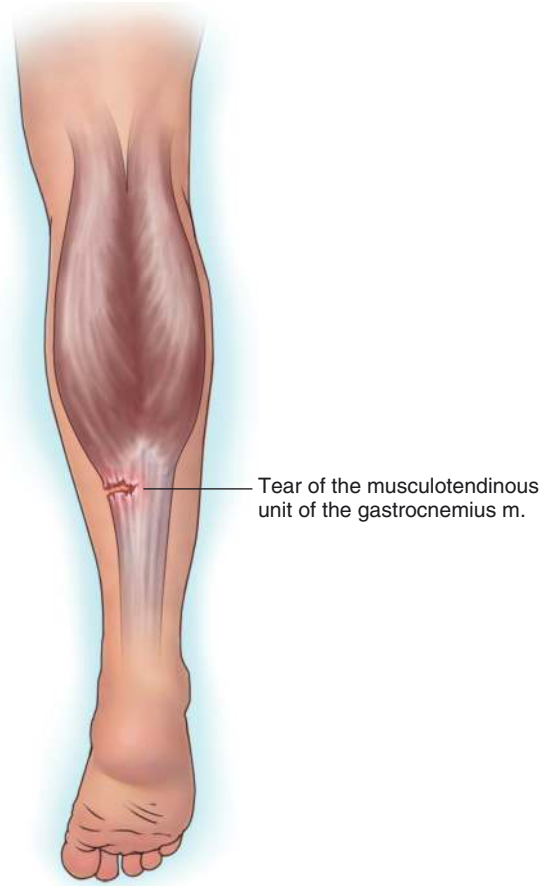

FIG 132.1 The pain of tennis leg occurs acutely and is accompanied by an audible snap or pop that emanates from the tearing of the musculotendinous unit of the gastrocnemius muscle.

TESTING

Magnetic resonance imaging of the calf is indicated if tennis leg is suspected and to rule out other disorders that may mimic this condition (Figs. 132.5 and 132.6). Ultrasound imaging may also aid in diagnosis; the common finding of fluid between the gastrocnemius and soleus muscles is highly suggestive of the diagnosis of tennis leg (Figs. 132.7 and 132.8). Ultrasound imaging can also identify defects in the musculotendinous unit (Fig. 132.9). Based on the patient's

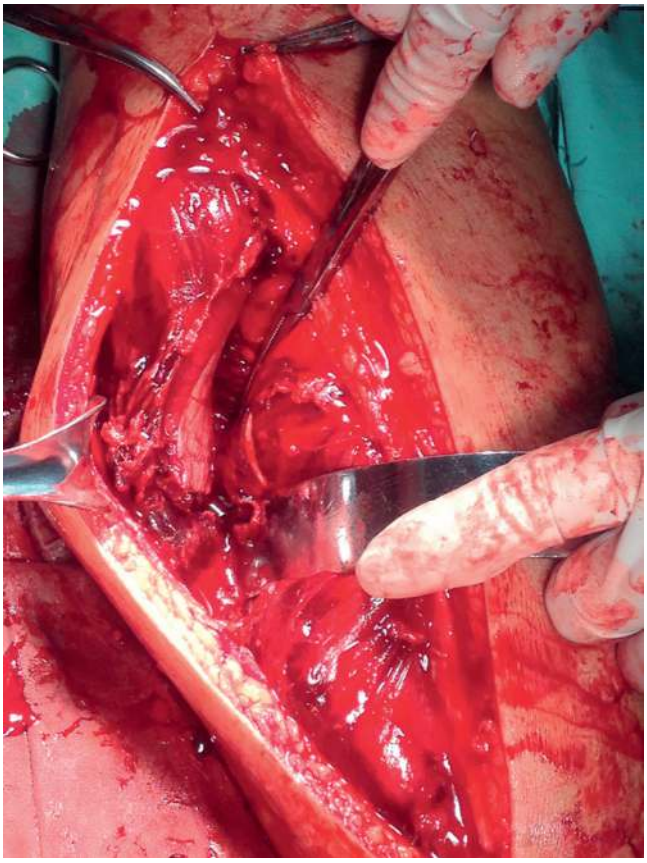

FIG 132.2 Complete rupture of the medial head of gastrocnemius muscle at the myotendinous junction after evacuation of the hematoma. (From Li T, Huang J, Ding M, et al. Acute compartment syndrome after gastrocnemius rupture (tennis leg) in a nonathlete without trauma. *J Foot Ankle Surg*. 2016;55(2):303–305.)

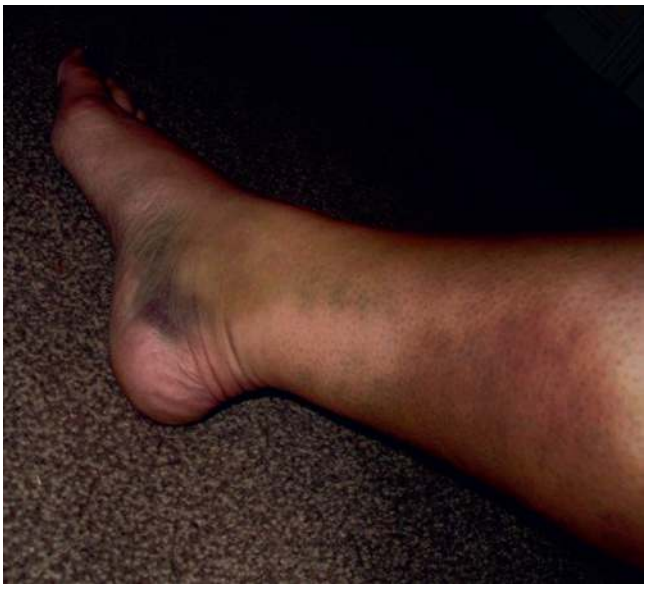

FIG 132.3 Patients with significant tearing or complete rupture of the gastrocnemius musculotendinous unit will experience significant swelling and ecchymosis and hematoma formation that may extend from the medial calf to the ankle.

FIG 132.4 The Thompson Squeeze Test for Achilles tendon rupture. (From Waldman, S. *Physical diagnosis of pain: an atlas of signs and symptoms*. 3rd ed. Philadelphia: Elsevier; 2016.)

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FIG 132.5 A 53-year-old woman reported a sudden pop and sharp pain in the back of her leg during arising from sajdah (the placing of the forehead on the prayer mat-covered floor during prayer). A T2-weighted coronal magnetic resonance image reveals fluid (*arrows*) between the gastrocnemius and soleus muscles. Also noted is an increased signal (*star*) within the medial gastrocnemius head near the midtarsal joint that indicates muscle injury. (From Yilmaz C, Orgenc Y, Ergenc R, Erkan N. Rupture of the medial gastrocnemius muscle during namaz praying: an unusual cause of tennis leg. *Comput Med Imaging Graph*. 2008;32(8):728–731.)

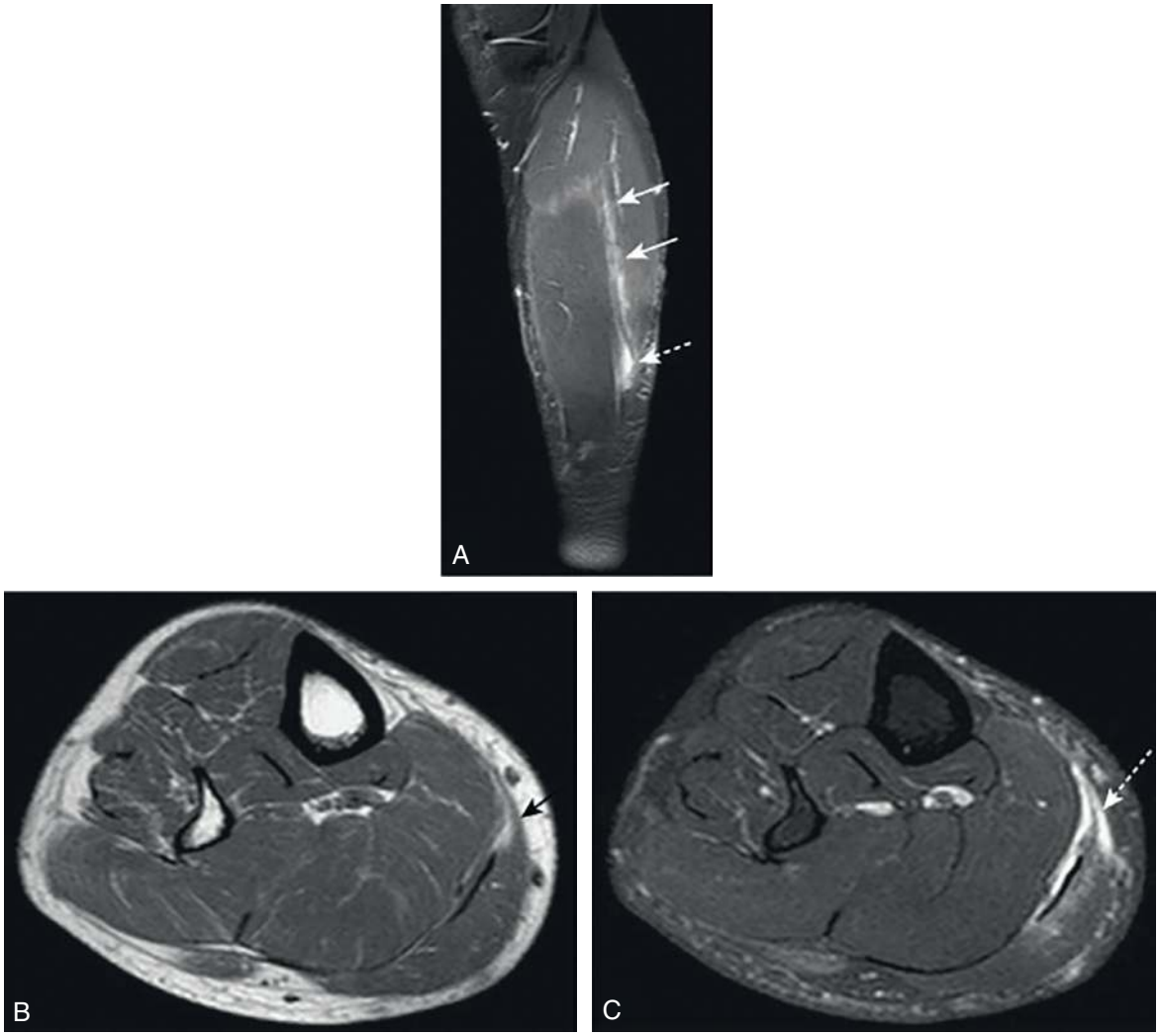

FIG 132.6 Tennis leg. **A,** Sagittal STIR magnetic resonance (MR) image of a patient with an acute medial gastrocnemius muscle tear. There are high-SI diffuse edema within the muscle belly (*white arrows*) and a focal area of high-signal intensity (SI) hematoma (*broken white arrow*). **B,** The axial T1W MR image shows the partly retracted muscle belly (*black arrow*). **C,** The high-SI hematoma (*white arrow*) can be seen on the axial T2W with fat suppression MR image. (From Waldman S, Campbell R. *Imaging of pain*. Philadelphia: Saunders; 2011.)

clinical presentation, additional testing may be indicated, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Tennis leg is usually a straightforward clinical diagnosis that can be made on the basis of history and clinical findings. Occasionally, thrombophlebitis may mimic tennis leg. However, coexisting bursitis or tendinitis of the knee and distal lower extremity from overuse or misuse may confuse the diagnosis. In some clinical situations, consideration should be given to primary or secondary tumors involving the affected region. Nerve entrapments of the lower extremity secondary to compression by massive hematoma formation (especially in anticoagulated patients) can also confuse the diagnosis.

TREATMENT

Initial treatment of the pain and functional disability associated with tennis leg includes rest, elevation, use of elastic compressive wraps, and application of ice to the affected extremity to reduce swelling and pain. The use of acetaminophen or cyclooxygenase-2 inhibitors, with or without the addition of a short-acting opioid such as hydrocodone, is indicated for pain. Aspirin should be avoided because of its effects on platelets, given the sometimes-significant bleeding associated with the injury of tennis leg. Gentle physical therapy to normalize gait and to maintain range of motion should be implemented in a few days, as the swelling subsides.

Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Occasionally, surgical repair of the tendon is undertaken if the patient is experiencing significant functional disability or is unhappy with

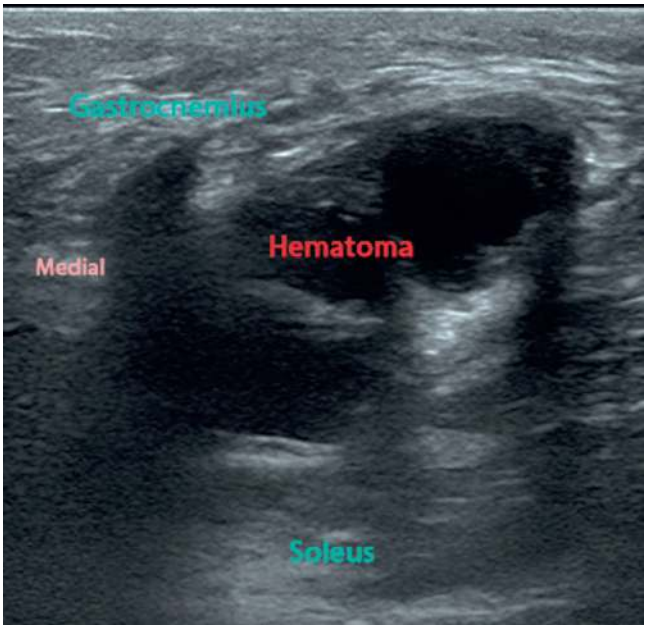

FIG 132.7 Transverse ultrasound image demonstrating a large hematoma within the torn gastrocnemius muscle.

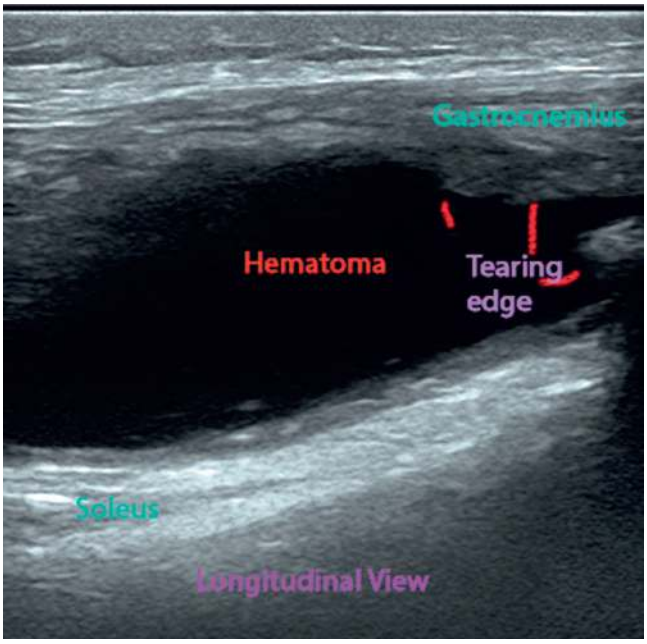

FIG 132.8 Transverse ultrasound image demonstrating a large hematoma within the torn gastrocnemius muscle. Note the tearing edge of the muscle injury.

the cosmetic defect resulting from the retracted tendon and muscle.

COMPLICATIONS AND PITFALLS

Careful observation for the development of lower extremity compartment syndrome during the early phase of this condition is important if bleeding is significant, especially in anticoagulated patients.

Given the overlap of symptoms of tennis leg with deep venous thrombosis, the clinician must have a high index of suspicion for the development of deep venous thrombosis, especially during the rest phase of recovery or if anticoagulants have been discontinued. As the damage to the musculotendinous unit heals, scar formation can occur and can lead to chronic pain and functional disability. If this occurs, surgical excision and reconstruction of the musculotendinous unit may be required.

FIG 132.9 Ultrasound image of a patient with complete rupture (*asterisks*) of the medial head of the gastrocnemius; the soleus muscle is also visible. *MHG*, medial head of the gastrocnemius muscle. (From Flecca D, Tomei A, Ravazzolo N, et al. US evaluation and diagnosis of rupture of the medial head of the gastrocnemius (tennis leg). *J Ultrasound*. 2007;10(4):194–198.)

CLINICAL PEARLS

The amount of swelling and bruising associated with the acute injury causing tennis leg may be impressive and may cause extreme anxiety for the injured patient and his or her family. Reassurance should be given early and often. A high index of suspicion for the insidious onset of lower extremity compartment syndrome or deep venous thrombosis is important, to avoid disaster.

SUGGESTED READINGS

Armfield DR, Kim DH, Towers JD, et al. Sports-related muscle injury in the lower extremity. *Clin Sports Med*. 2006;25(4):803–842.

Flecca D, Tomei A, Ravazzolo N, et al. US evaluation and diagnosis of rupture of the medial head of the gastrocnemius (tennis leg). *J Ultrasound*. 2007;10(4):194–198.

Harwin JR, Richardson ML. "Tennis leg": gastrocnemius injury is a far more common cause than plantaris rupture. *Radiol Case Rep*. 2017;12(1):129–123.

Kwak H-S, Lee K-B, Han Y-M. Ruptures of the medial head of the gastrocnemius ("tennis leg"): clinical outcome and compression effect. *Clin Imaging*. 2006;30(1):48–53.

Li T, Huang J, Ding M, et al. Acute compartment syndrome after gastrocnemius rupture (tennis leg) in a nonathlete without trauma. *J Foot Ankle Surg*. 2016;55(2):303–305.

Pai V, Pai V. Acute compartment syndrome after rupture of the medial head of gastrocnemius in a child. *J Foot Ankle Surg*. 2007;46(4):288–290.

Waldman SD. Tennis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:866–874.

133

Arthritis Pain of the Ankle

ICD-10 CODE M19.90

THE CLINICAL SYNDROME

Arthritis of the ankle is a common condition. The ankle joint is susceptible to the development of arthritis from various conditions that can damage the joint cartilage. Osteoarthritis is the most common form of arthritis that results in ankle pain; rheumatoid arthritis and posttraumatic arthritis are also frequent causes of ankle pain. Less common causes include the collagen vascular diseases, infection, villonodular synovitis, and Lyme disease (Fig. 133.1). Acute infectious arthritis

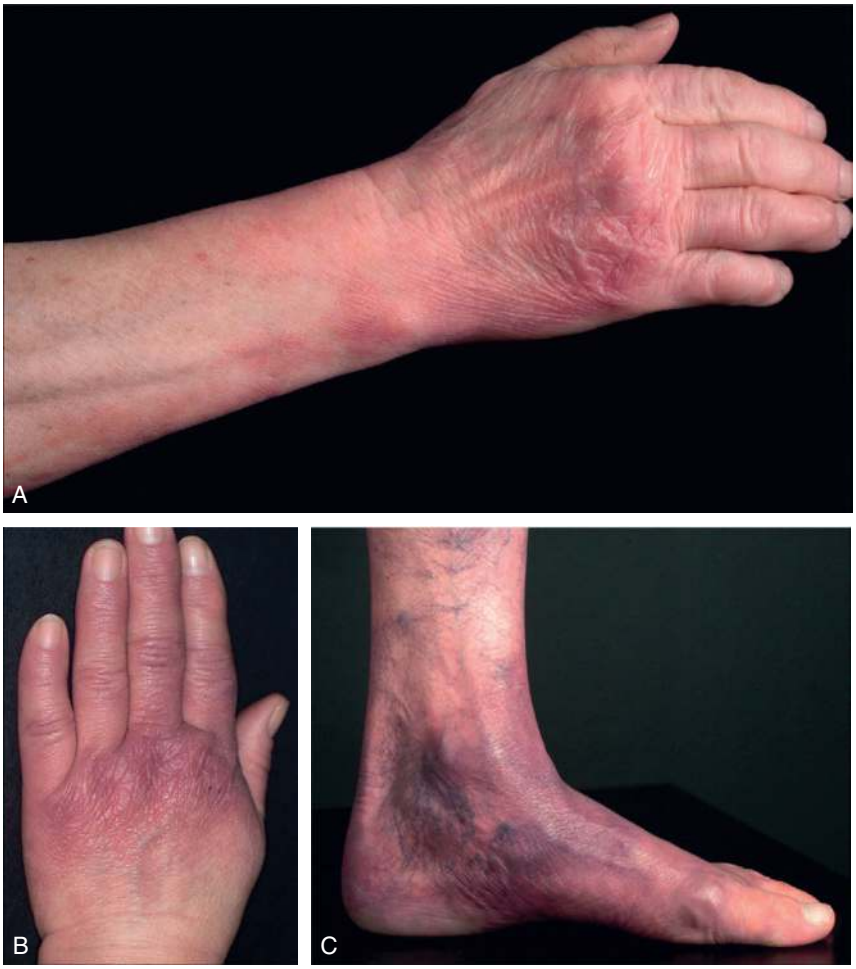

FIG 133.1 Lyme disease is an uncommon cause of ankle pain that must be included in the differential diagnosis. Acrodermatitis chronic atrophicans is an uncommon cutaneous manifestation of European Lyme disease and is typically located on the extensor sites of extremities: **(A)** ulnar and hand lesions, **(B)** bluish-red lesion on the back of a patient's hand and waxy appearance of the skin of fingers, and **(C)** lesions on a patient's left foot and lower leg. (Reprinted with permission from Elsevier., Stanek G, Wormser GP, Gray J, et al. Lyme borreliosis. *Lancet*. 2012;379(9814):461–473.)

is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the ankle joint, although ankle pain secondary to collagen vascular disease responds exceedingly well to the treatment modalities described here.

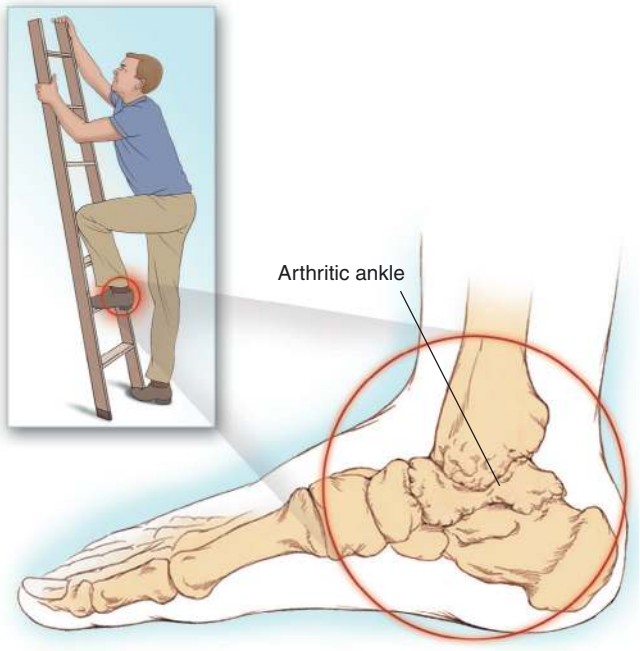

FIG 133.2 Arthritis of the ankle is often made worse with activity.

SIGNS AND SYMPTOMS

Most patients complain of pain localized around the ankle and distal lower extremity. Activity makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical examination.

In addition to pain, patients with arthritis of the ankle often experience a gradual decrease in functional ability because of reduced ankle range of motion that makes simple everyday tasks, such as walking and climbing stairs and ladders, quite difficult (Fig. 133.2). With continued disuse, muscle wasting may occur, and a frozen ankle secondary to adhesive capsulitis may develop.

TESTING

Plain radiographs are indicated in all patients who present with ankle pain (Fig. 133.3). Magnetic resonance imaging,

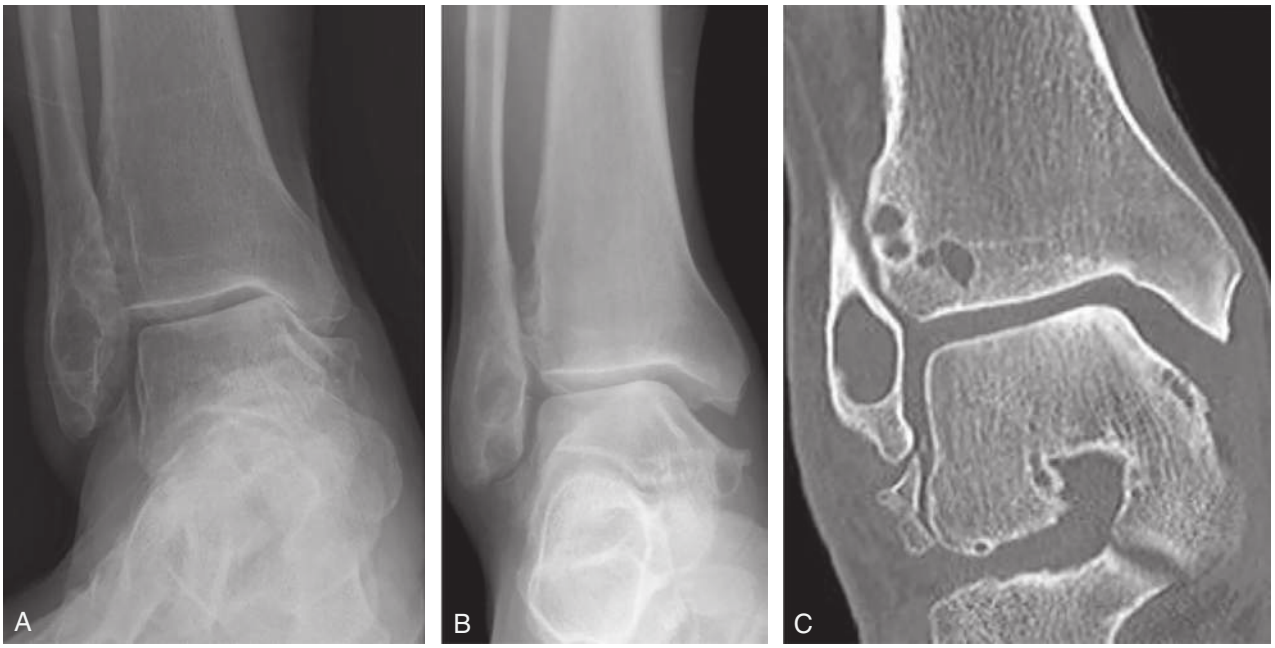

FIG 133.3 A case of varus ankle osteoarthritis with erosion of the medial malleolus. **A,** The anteroposterior weight-bearing radiograph shows medial translation of the talus relative to the tibia and medial gutter obliteration. **B,** A non–weight-bearing anteroposterior radiograph shows widening of the medial gutter and suspicious erosion of the medial malleolus. **C,** A coronal computed tomography image shows complete erosion of the articular facet of the medial malleolus about 2 mm inferior to the tibial plafond. (From Lee WC. Extraarticular supramalleolar osteotomy for managing varus ankle. Osteoarthritis, alternatives for osteotomy: how and why? *Foot Ankle Clin*. 2016;21(1):27–35.)

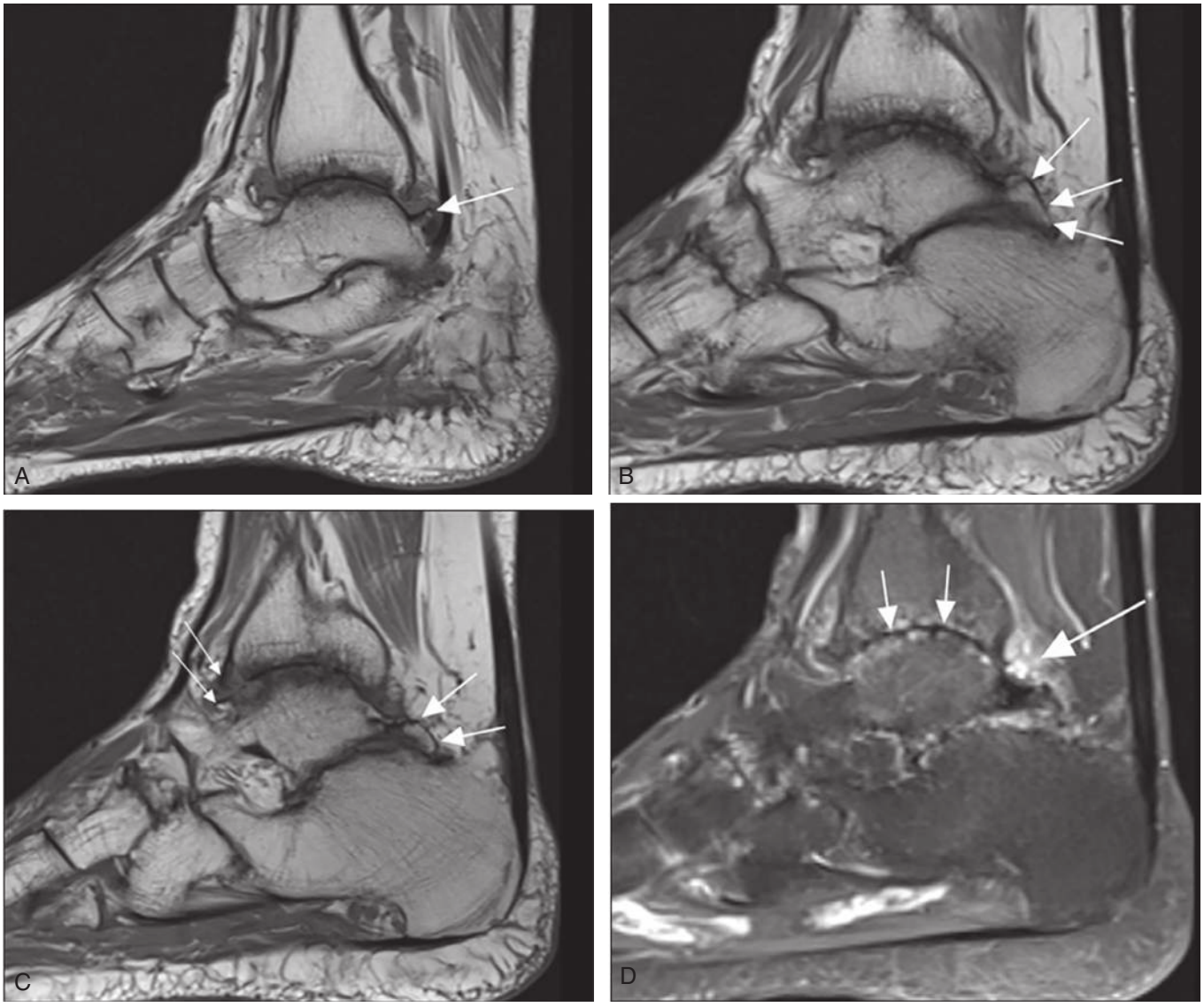

FIG 133.4 A 58-year-old male active runner with advanced posttraumatic osteoarthritis of the ankle joint and posterior pain. **A,** Sagittal T1-weighted magnetic resonance image (MRI) shows advanced osteoarthritis of the tibio-talar joint and a large posterior talar osteophyte that impinges into the posterior joint capsule, especially on plantar flexion (*arrow*). **B,** An additional large osteophyte is seen at the posterior margin of the talus leading to impingement at the calcaneo-talar joint (*arrows*). **C,** The osteophyte reaches far laterally (*large arrows*) and also anterior tibia-talar osteophytes are observed (*small arrows*). **D,** Sagittal contrast enhanced T1-weighted fat-suppressed MRI shows that reactive synovitis is primarily observed posteriorly (*large arrow*), which correlates well with the clinical picture. There is complete obliteration of the tibio-talar joint (*small arrows*). (From Hayashi D, Roemer FW, D'Hooghe P, et al. Posterior ankle impingement in athletes: pathogenesis, imaging features and differential diagnoses. *Eur J Radiol*. 2015;84(11):2231–2241.)

computerized tomography, and ultrasound imaging of the ankle are indicated in the case of trauma, if the diagnosis is in question, or if an occult mass or tumor is suspected (Figs. 133.4–133.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, comprehensive metabolic profile, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Lumbar radiculopathy may mimic the pain and disability of arthritis of the ankle; however, results of the ankle examination are negative. Bursitis of the ankle and entrapment neuropathies, such as tarsal tunnel syndrome, may also confuse the diagnosis; both of these conditions may coexist with arthritis of the ankle. Primary and metastatic tumors of the

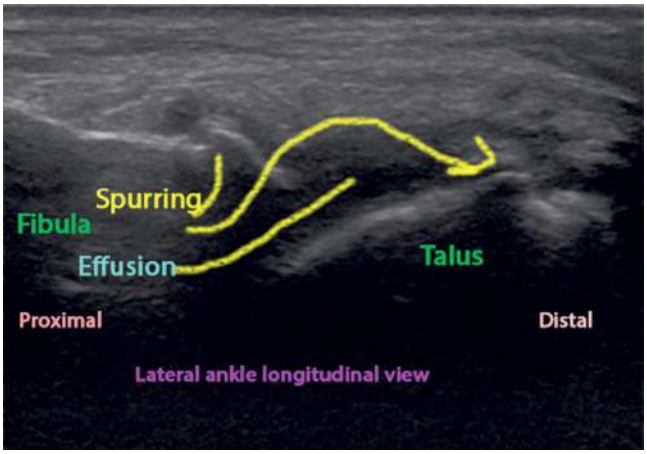

FIG 133.5 Longitudinal ultrasound image of the lateral ankle demonstrating significant osteoarthritis of the ankle joint. Note the osteophytic spurring and effusion.

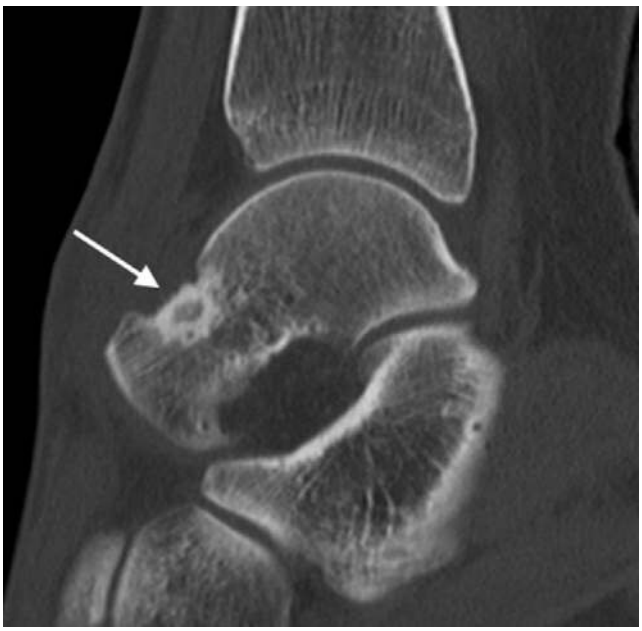

FIG 133.6 Computed tomography of an osteoid osteoma of the talar neck with typical perifocal sclerosis and a central nidus. (From Toepfer A. Tumors of the foot and ankle—a review of the principles of diagnostics and treatment. *Fuß & Sprunggelenk*. 2017;15(2):82–96, Fig. 7.)

distal tibia and fibula and spine, as well as occult fractures, may also manifest in a manner similar to arthritis of the ankle (Box 133.1 and Table 133.1).

TREATMENT

Initial treatment of the pain and functional disability associated with arthritis of the ankle includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, as well as short-term immobilization of the ankle joint, may provide relief.

BOX 133.1 Differential Diagnosis of Ankle Pain

- Osteoarthritis
- Avulsion fracture
- Intraarticular fracture
- Stress fractures
- Achilles tendinitis
- Achilles bursitis
- Bursitis
- Ligamentous sprains
- Ligamentous strains
- Gout
- Other crystal arthropathies
- Psoriatic arthritis
- Reactive arthritis
- Rheumatoid arthritis
- Septic arthritis
- Osteochondritis dissecans
- Anterior tarsal tunnel syndrome
- Posterior tarsal tunnel syndrome
- Lyme disease

From Waldman SD. *Ankle and foot: pain medicine*, Chapters 1 and 2–18.

For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

To perform intraarticular injection of the ankle, the patient is placed in the supine position, and the skin overlying the ankle joint is prepared with an antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. With the patient's foot in the neutral position, the junction of the tibia and fibula just above the talus is identified. At this point, a triangular indentation indicating the joint space is easily palpable (Fig. 133.7). The needle is carefully advanced through the skin, subcutaneous tissues, and joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly and slightly more medially. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. The injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of ankle arthritis.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

TABLE 133.1 Painful Disorders of the Ankle and the Foot

Articular	
Arthritis	RA, OA, PsA, gout
Toe disorders	Hallux valgus, hallux rigidus, hammer toe
Arch disorders	Pes planus, pes cavus
Periarticular	
Cutaneous	Corn, callosity
Subcutaneous	RA nodules, tophi
	Ingrown toenail
Plantar fascia	Plantar fasciitis
	Plantar nodular fibromatosis
Tendons	Achilles tendinitis
	Achilles tendon rupture
	Tibialis posterior tenosynovitis
	Peroneal tenosynovitis
Bursae	Bunion, bunionette
	Retrocalcaneal, retroachilleal, and subcalcaneal bursitis
	Medial and lateral malleolar bursitis
Acute calcific periarthritis	Hydroxyapatite pseudopodagra (first MTP)
Osseous	
Fracture (traumatic, stress)	
Sesamoiditis	
Neoplasm	
Infection	
Epiphysitis (osteochondritis)	Second metatarsal head (Freiberg disease)
	Navicular (Köhler disease)
	Calcaneus (Sever disease)
Painful accessory ossicles	Accessory navicular
	Os trigonum (near talus)
	Os intermetatarseum (first and second)
Neurologic	
Tarsal tunnel syndrome	
Interdigital (Morton) neuroma	
Peripheral neuropathy	
Radiculopathy (lumbar disk)	
Vascular	
Ischemic	Atherosclerosis, Buerger disease
Vasospastic disorder (Raynaud disease)	
Cholesterol emboli with "purple toes"	
Referred	
Lumbosacral spine	
Knee	
• Chronic regional pain syndrome I	
• Chronic regional pain syndrome II	

MTP, metatarsophylangeal; *OA*, osteoarthritis; *PsA*, Psoriatic arthritis; *RA*, rheumatoid arthritis

Modified from Lawry G, Kreder H, Hawker G, et al. *Fam's musculoskeletal examination and joint injection techniques*. 2nd ed. Philadelphia: Mosby; 2010:89–101.

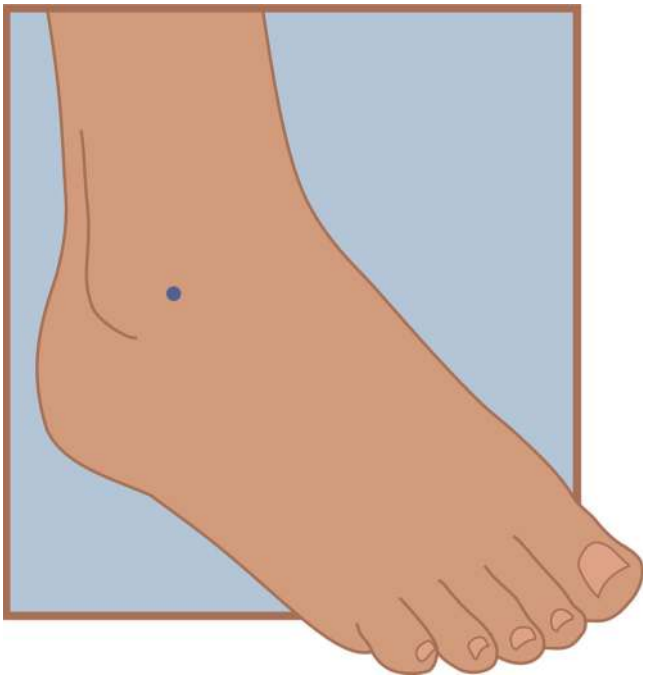

FIG 133.7 With the foot in the neutral position, the junction of the tibia and fibula just above the talus can be identified. At this point, a triangular indentation indicating the joint space is easily palpable. (From Waldman SD. *Atlas of pain management injection techniques*. 5th ed. 2022)

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the ankle that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of intraarticular injection of the ankle is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's ankle pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of arthritis of the ankle joint.

SUGGESTED READINGS

Alcaraz MJ, Megías J, García-Arnandis I, et al. New molecular targets for the treatment of osteoarthritis. *Biochem Pharmacol*. 2010;80(1):13–21.

Repetto I, Biti B, Cerruti P, et al. Conservative treatment of ankle osteoarthritis: can platelet-rich plasma effectively postpone surgery? *J Foot Ankle Surg*. 2017;56(2):362–365.

Robinson AHN, Keith T. Osteoarthritis of the ankle. *Orthop Trauma*. 2016;30(1):59–67.

Waldman SD. Arthritis and other abnormalities of the ankle joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:875–882.

Waldman SD. Functional anatomy of the ankle and foot. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:149–150.

Waldman SD. Intra-articular injection of the ankle joint. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:794–797.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

134

Arthritis of the Midtarsal Joints

ICD-10 CODE M19.90

THE CLINICAL SYNDROME

Arthritis of the midtarsal joints is a common condition. The midtarsal joints are susceptible to the development of arthritis from various conditions that can damage the joint cartilage. Osteoarthritis is the most common form of arthritis that results in midtarsal joint pain; rheumatoid arthritis and posttraumatic arthritis are also frequent causes of midtarsal pain. Less common causes include the collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Charcot midtarsal joints may occur from a variety of peripheral neuropathies (Fig. 134.1). Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the midtarsal joint, although midtarsal pain secondary to collagen vascular disease responds exceedingly well to the treatment modalities described here.

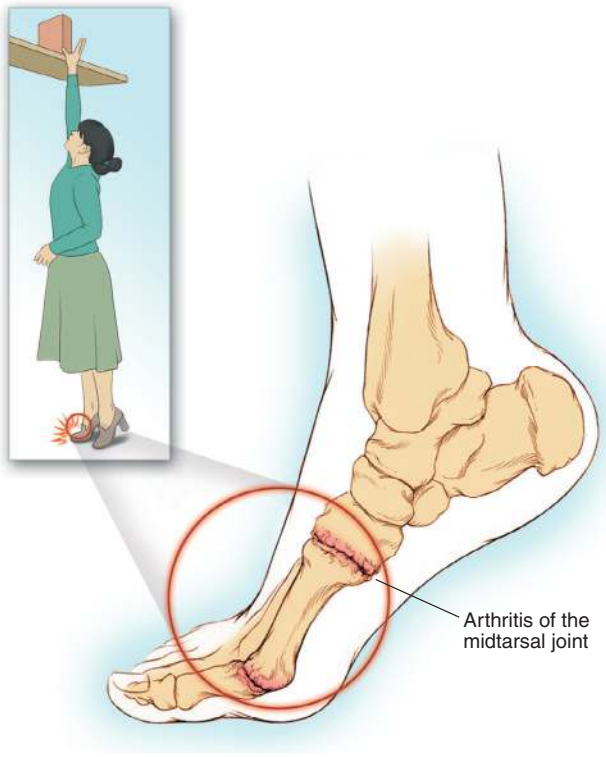

FIG 134.1 The midtarsal joints are susceptible to the development of arthritis from various conditions that can damage the joint cartilage.

SIGNS AND SYMPTOMS

Most patients present with pain localized to the dorsum of the foot. Activity, especially that involving inversion and adduction of the midtarsal joint, worsens the pain (Fig. 134.2), whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with the use of the joints, and crepitus may be present on physical examination. In addition to pain, patients with arthritis of the midtarsal joint often experience a gradual decrease in functional ability because of reduced midtarsal range of motion that makes simple everyday tasks, such as walking and climbing stairs, quite difficult.

TESTING

Plain radiographs and ultrasound imaging are indicated for all patients who present with midtarsal pain (Fig. 134.3). Magnetic resonance imaging of the midtarsal joint is indicated if aseptic necrosis, inflammatory arthritis, an occult

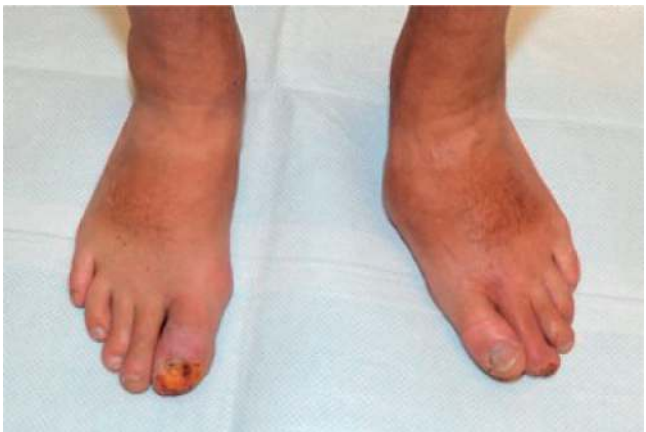

FIG 134.2 Charcot neuroarthropathy of the midtarsal joint manifests as pain in the dorsum of the foot that is made worse with inversion and adduction of the affected joint. (From Young N, Neiderer K, Martin B, et al. HIV neuropathy induced Charcot neuroarthropathy: a case discussion. *Foot (Edinb)*. 2012;22(3):112–116.)

mass, fracture, or a tumor is suspected as well as to confirm the diagnosis (Figs. 134.4 and 134.5). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, comprehensive metabolic profile, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Primary disorders of the foot, including gout and occult fractures, may mimic the pain and disability of arthritis of the midtarsal joint. Bursitis and plantar fasciitis of the foot, as well as entrapment neuropathies such as tarsal tunnel syndrome, may also confuse the diagnosis; these conditions may coexist with arthritis of the midtarsal joint. Primary and metastatic tumors of the foot may also manifest in a manner similar to arthritis of the midtarsal joint.

FIG 134.3 Anteroposterior view of the midfoot demonstrating uniform cartilage loss between all the tarsal bones. (From Brower AC, Flemming DJ, eds. Rheumatoid arthritis. In: *Arthritis in black and white*. 3rd ed. Philadelphia: Saunders; 2012:170–199.)

TREATMENT

Initial treatment of the pain and functional disability associated with arthritis of the midtarsal joint includes a

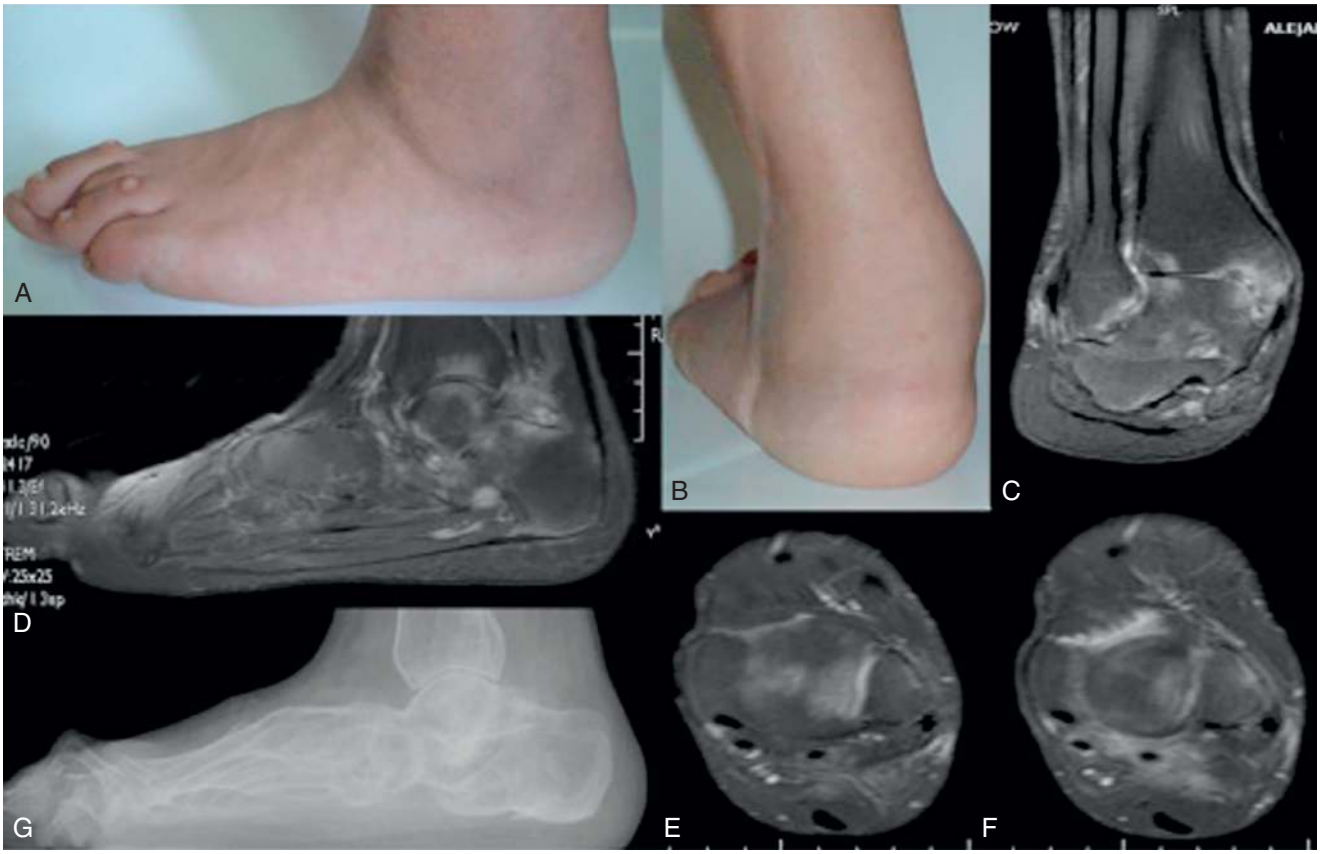

FIG 134.4 Composite images of ankylosing tarsitis in a 16-year-old boy with ankylosing spondylitis of 9 years' duration and complete ankylosis of the tarsal bones and grade 2 bilateral sacroiliitis. **A** and **B,** Flat-foot and swelling around the ankle. **C–F,** Short tau inversion recovery magnetic resonance images showing edema in various tarsal bones, joint spaces **(C, D),** and soft tissues **(E, F)** surrounding the tendons of the posterior aspect of the foot on the coronal view. **G,** Complete ankylosis of the tarsal bones and an enthesophyte at the plantar fascia attachment are seen. (Modified from Burgos-Vargas R. A case of childhood-onset ankylosing spondylitis: diagnosis and treatment. *Nat Clin Pract Rheumatol.* 2009;5:52–57.)

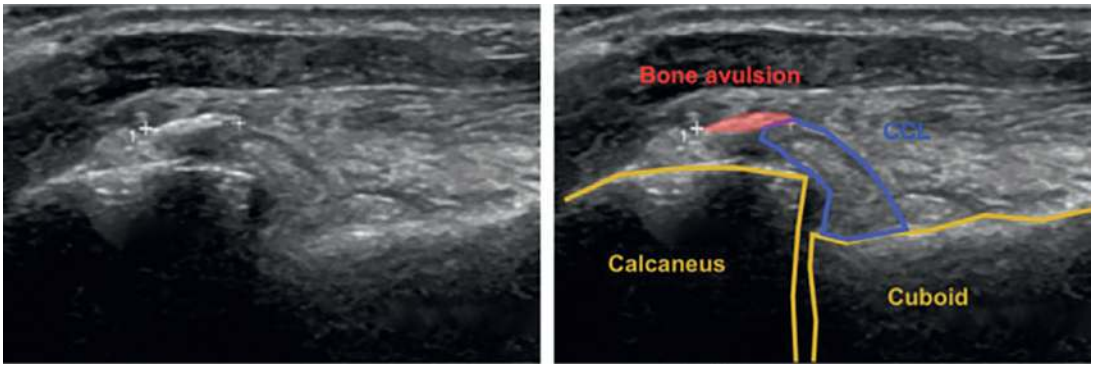

FIG 134.5 Ultrasound image of a grade-3 calcaneocuboid ligament lesion in a patient with midtarsal pain.

combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, as well as short-term immobilization of the midtarsal joint, may provide relief. For patients who do not respond to these treatment modalities, intraarticular injection of local anesthetic and steroid is a reasonable next step.

To perform midtarsal injection, the patient is placed in the supine position, and the skin overlying the tenderest midtarsal joint is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a ⅝-inch, 25-gauge needle using strict aseptic technique. The affected joint space is identified. At this point, the needle is carefully advanced at a right angle to the dorsal aspect of the ankle through the skin, subcutaneous tissues, and joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. After the joint space is entered, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. The use of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of ankle arthritis.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the midtarsal joint that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of intraarticular injection of the midtarsal joint is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of arthritis of the midtarsal joint.

SUGGESTED READINGS

Alcaraz MJ, Megías J, García-Arnandis I, et al. New molecular targets for the treatment of osteoarthritis. *Biochem Pharmacol*. 2010;80(1):13–21.

Repetto I, Biti B, Cerruti P, et al. Conservative treatment of ankle osteoarthritis: can platelet-rich plasma effectively postpone surgery? *J Foot Ankle Surg*. 2017;56(2):362–365.

Robinson AHN, Keith T. Osteoarthritis of the ankle. *Orthop Trauma*. 2016;30(1):59–67.

Waldman SD. Arthritis and other abnormalities of the ankle joint. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:875–882.

Waldman SD. Functional anatomy of the ankle and foot. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:149–150.

Waldman SD. Intra-articular injection of the mid-tarsal. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:803–806.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Deltoid Ligament Strain

ICD-10 CODES S93.429A

THE CLINICAL SYNDROME

The deltoid ligament is exceptionally strong and is not as easily strained as the anterior talofibular ligament. However, the deltoid ligament is susceptible to strain from acute injury resulting from sudden overpronation of the ankle or repetitive microtrauma to the ligament from overuse or misuse, such as long-distance running on soft or uneven surfaces. The deltoid ligament has two layers, both of which attach to the medial malleolus above it (Fig. 135.1). The deep layer attaches below to the medial body of the talus, and the superficial fibers attach to the medial talus, the sustentaculum tali of the calcaneus, and the navicular tuberosity.

SIGNS AND SYMPTOMS

Patients with deltoid ligament strain complain of pain just below the medial malleolus. Plantar flexion and eversion of the ankle joint exacerbate the pain. Often, patients with injury to the deltoid ligament note a pop, followed by significant swelling and the inability to walk (Fig. 135.2).

On physical examination, patients have point tenderness over the medial malleolus. With acute trauma, ecchymosis over the ligament may be noted. Patients with deltoid ligament strain have a positive result of the eversion test, which is performed by passively everting and plantar flexing the affected ankle joint (Fig. 135.3). Coexistent bursitis and arthritis of the ankle and subtalar joint may also be present and may confuse the clinical picture.

TESTING

Plain radiographs are indicated for all patients who present with ankle pain (Fig. 135.4). Magnetic resonance imaging (MRI) and ultrasound imaging of the ankle are indicated if disruption of the deltoid ligament, joint instability, an occult mass, or a tumor is suspected (Figs. 135.5–135.7). Radionuclide bone scanning should be performed if occult fracture is suspected. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Avulsion fracture of the calcaneus, talus, medial malleolus, or base of the fifth metatarsal can mimic deltoid ligament pain.

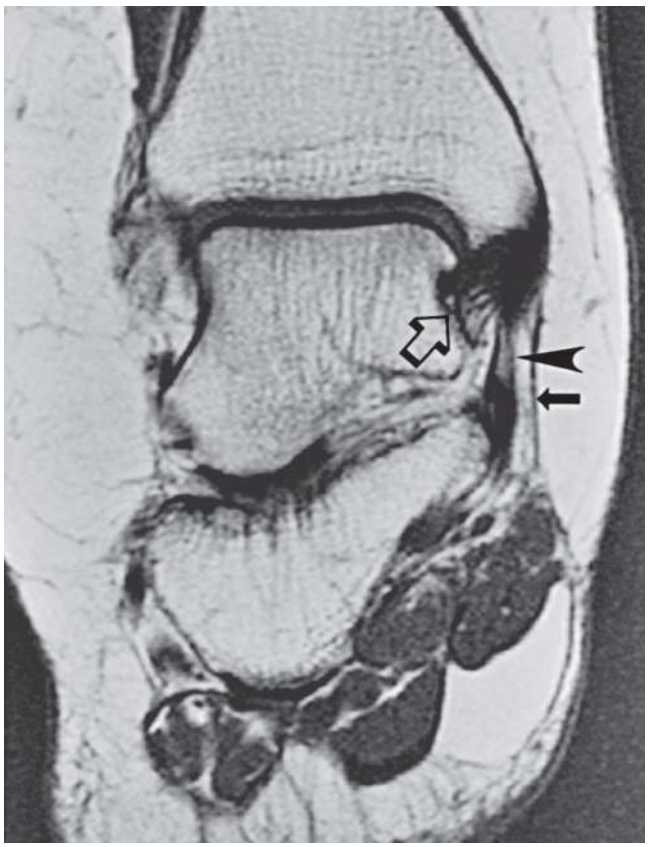

FIG 135.1 Normal medial ankle ligaments on a coronal, T1-weighted magnetic resonance image. The two layers of the deltoid (medial) ligament are seen. The deep tibiotalar ligament is striated (*open arrow*). The more superficial tibiocalcaneal ligament (*arrowhead*) may have vertical striations as well. The thin, vertical, low-signal structure superficial to the tibiocalcaneal ligament is the flexor retinaculum (*solid arrow*). (From Kaplan PA, Helms CA, Dussault R, et al. *Musculoskeletal MRI*. Philadelphia: Saunders; 2001:835.)

Bursitis, tendinitis, and gout of the midtarsal joints may coexist with deltoid ligament strain, thus confusing the diagnosis. Tarsal tunnel syndrome may occur after ankle trauma and further complicate the clinical picture.

TREATMENT

Initial treatment of the pain and functional disability associated with deltoid ligament strain includes a combination of

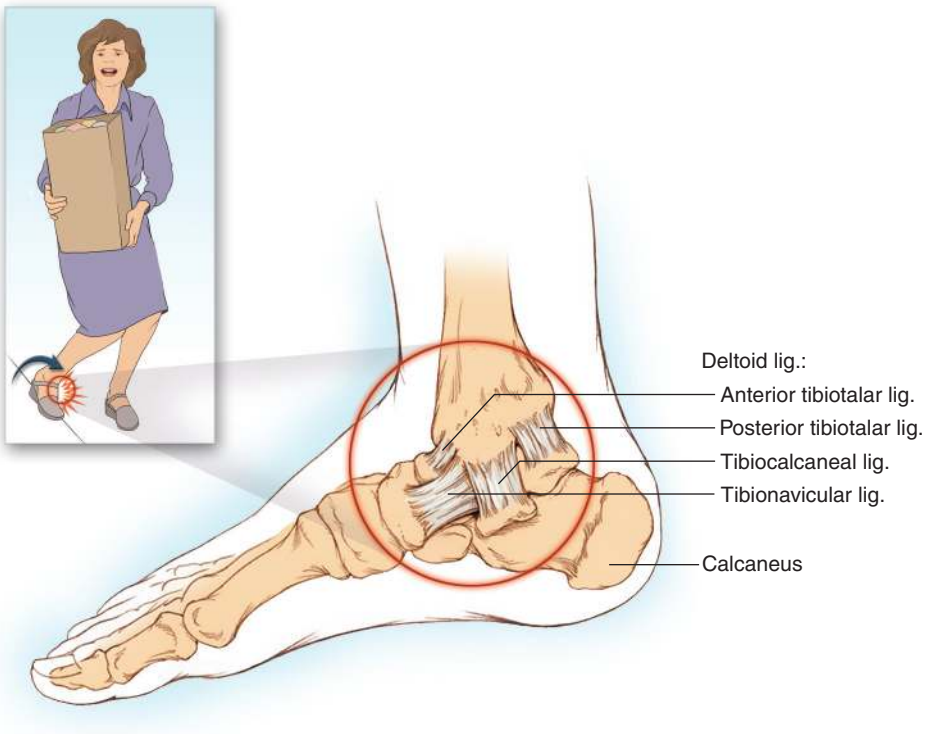

FIG 135.2 With deltoid ligament strain, patients may notice a pop, followed by significant swelling.

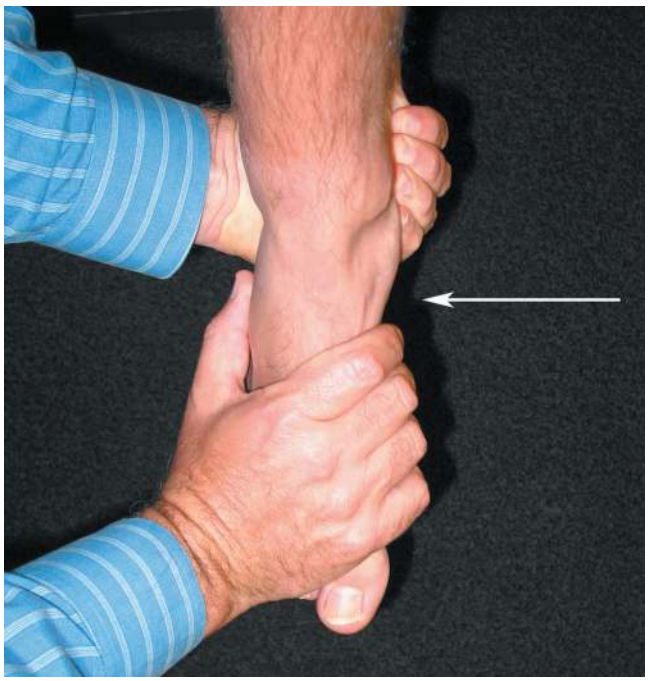

FIG 135.3 Eversion test for deltoid ligament insufficiency. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:369.)

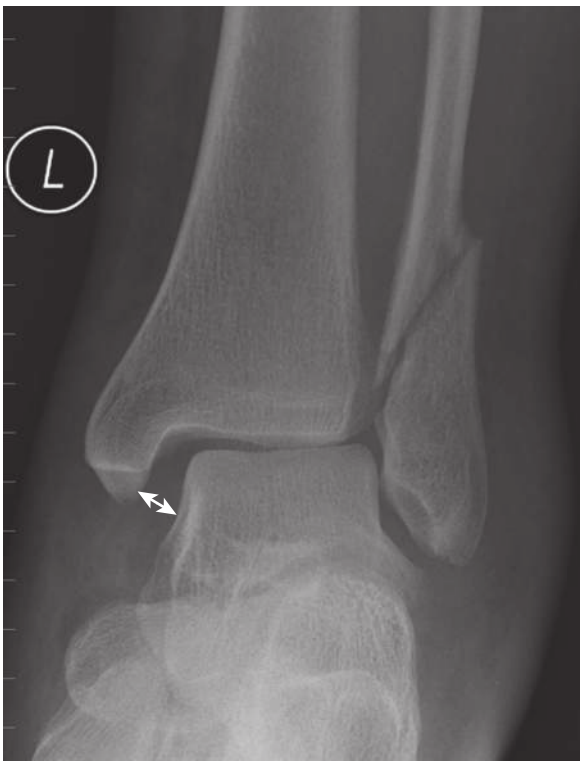

FIG 135.4 Anteroposterior radiograph of a severe acute eversion ankle injury. There is an oblique fracture of the distal fibula. Disruption of the ankle mortise with widening of the medial joint line (*double-headed arrow*) indicates a tear of the deltoid ligament. This pattern of injury is less common than an avulsion fracture of the entire medial malleolus with an intact ligament. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, as well as short-term immobilization of the ankle joint, may

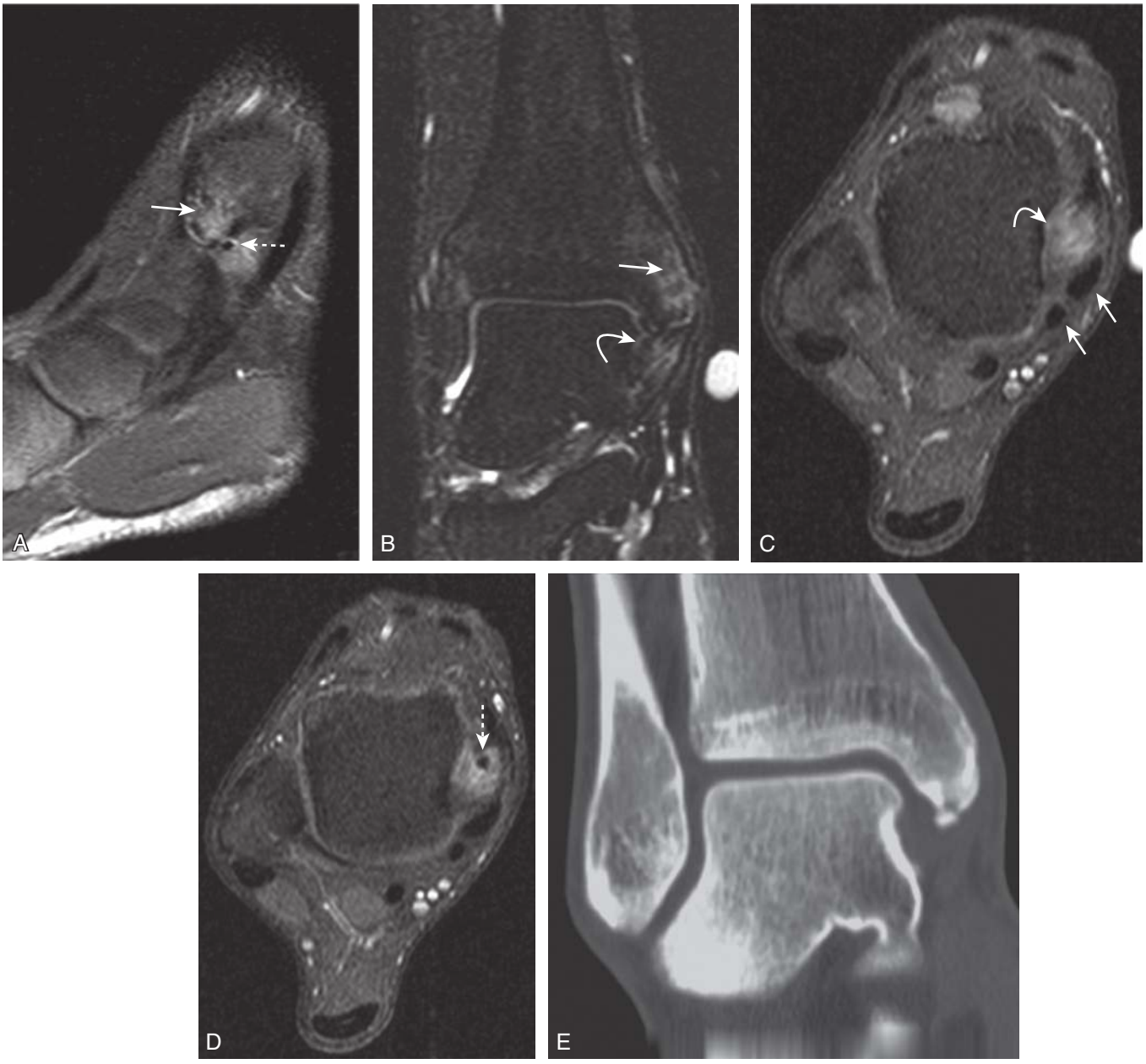

FIG 135.5 **A,** Sagittal fat-suppressed T2-weighted (FST2W) magnetic resonance (MR) image of an athlete with a subacute eversion ankle sprain. There is marrow edema in the tip of the medial malleolus (*white arrow*) and a possible small bony avulsion injury (*broken white arrow*). **B,** The coronal FST2W MR image also shows the marrow edema (*white arrow*), and there is high signal intensity (SI) within the deltoid ligament (*curved white arrow*) as a result of partial tearing. **C,** Consecutive axial FST2W MR images more clearly demonstrate the deltoid ligament edema (*curved white arrow*) anterior to the flexor tendons (*white arrows*). **D,** The bony avulsion fragment is demonstrated as a small round area of low SI (*broken white arrow*). **E,** The coronal computed tomography scan confirms the presence of an avulsion fracture of the tip of the medial malleolus. (From Waldman SD, Campbell RSD. *Imaging of pain.* Philadelphia: Elsevier; 2011.)

provide relief. For patients who do not respond to these treatment modalities, injection is a reasonable next step.

To perform deltoid ligament injection, the patient is placed in the supine position, and the skin overlying the area of the medial malleolus is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. With the lower extremity slightly abducted, the lower margin of the medial malleolus is identified. At this point, the needle is carefully advanced at a 30-degree angle to the ankle through the skin and subcutaneous tissues to impinge on the lower margin of the medial malleolus. The needle is then withdrawn slightly, and the contents of the syringe are gently

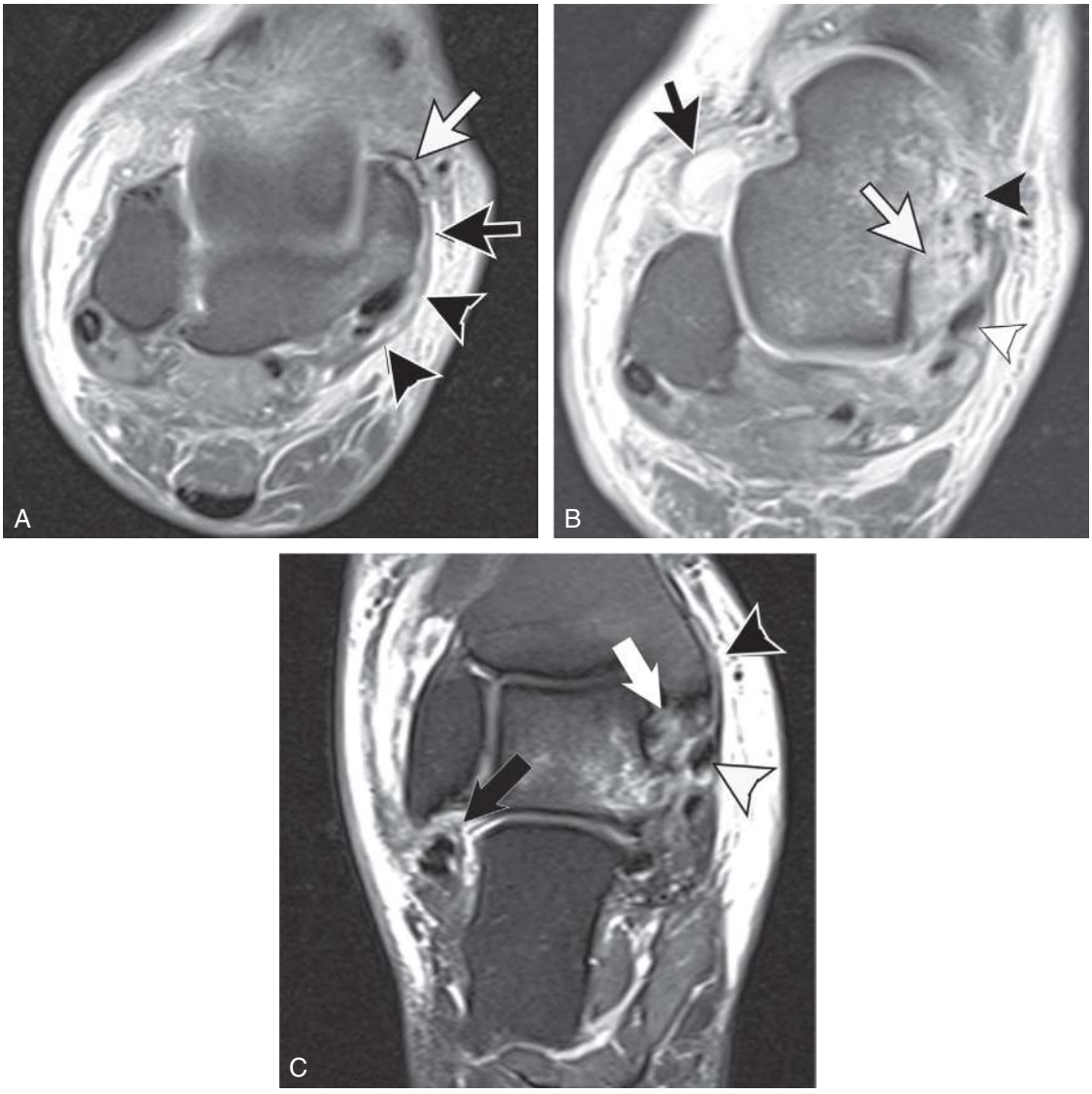

FIG 135.6 Traumatic supination–external rotation injury of the deltoid ligament. A 36-year-old woman presented after ankle supination injury complaining of severe medial and lateral ankle pain. Routine ankle series (non–weight bearing because she could not bear weight) showed severe lateral soft tissue swelling, normal alignment, and no fractures. Because of the severity of her pain, magnetic resonance imaging was performed 1 week after the injury. **A,** Axial proton density fat-suppressed (PDFS) image at the level of the tibial plafond shows complete avulsion of the medial malleolar fascial sleeve, including the origin of the superficial deltoid ligament (*white arrow*), the medial periosteum (*black arrow*), and the flexor retinaculum (*black arrowheads*). **B,** Axial PDFS image at the level of the deep deltoid ligament shows amorphous, lax, discontinuous deep deltoid ligament (*white arrow*). The ankle joint effusion (*black arrow*) extends through the completely torn anterior talofibular ligament. The distal bands of the superficial deltoid ligament are torn (*black arrowhead*). The posterior tibial tendon is thinned and irregular in shape, indicating a partial tear (*white arrowhead*). **C,** Coronal fast spin echo T2FS image at the level of the deep deltoid ligament confirms complete deltoid ligament tear (*white arrow*), medial malleolar fascial sleeve avulsion (*black arrowhead*), and partial tear of the posterior tibial tendon (*white arrowhead*). In addition, a complete tear of the calcaneofibular ligament (*black arrow*) is visible. (From Crim J. Medial-sided ankle pain: deltoid ligament and beyond. *Magn Reson Imaging Clin N Am.* 2017;25(1):63–77.)

injected. Resistance to injection should be slight. If significant resistance is encountered, the needle is probably in the ligament and should be withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. The injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of deltoid ligament injuries.

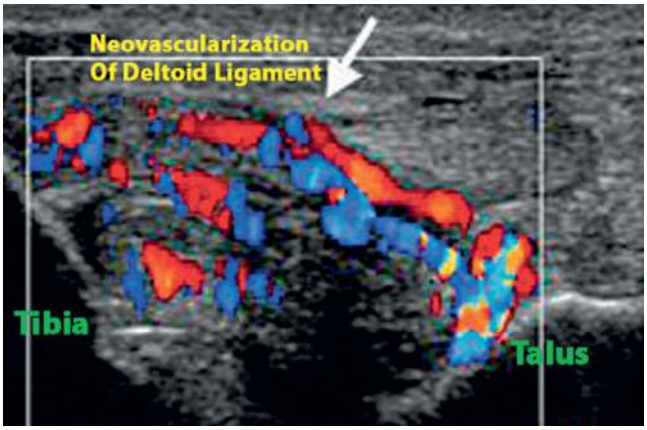

FIG 135.7 Color Doppler imaging may aid in the identification of neovascularization of the deltoid ligament following injury.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Failure to identify occult fractures of the ankle and foot may result in significant morbidity; therefore, radionuclide bone scanning and MRI should be performed in all patients with unexplained ankle and foot pain, especially if trauma is present. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility. A gentle technique should always be used when injecting around strained ligaments, to avoid further damage to the already compromised ligament.

CLINICAL PEARLS

Approximately 25,000 people are estimated to sprain an ankle every day. Although the public generally views this injury as minor, ankle sprains can result in significant permanent pain and disability. The injection technique described is extremely effective in treating the pain of deltoid ligament strain. Coexistent arthritis, bursitis, and tendinitis may contribute to medial ankle pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Cain JD, Dalmau-Pastor M. Anatomy of the deltoid-spring ligament complex. *Foot Ankle Clin*. 2021;26(2):237–247. https://doi.org/10.1016/j.fcl.2021.03.001.

Cao S, Wang C, Zhang C, et al. The effect of partial deltoid ligament injuries on the external rotation stability: a cadaveric study. *Foot Ankle Surg*. 2022. https://doi.org/10.1016/j.fas.2022.03.014.

Crim J. Medial-sided ankle pain: deltoid ligament and beyond. *Magn Reson Imaging Clin N Am*. 2017;25(1):63–77.

Gerstner GJB, Gerstner SJ, Rammelt S. Deltoid and spring ligament lesions in flat foot treatment. *Fuß & Sprunggelenk*. 2020;18(1):2–12. https://doi.org/10.1016/j.fuspru.2020.01.010.

Guerra-Pinto F, Fabian A, Mota T, et al. The tibiocalcaneal bundle of the deltoid ligament—prevalence and variations. *Foot Ankle Surg*. 2021;27(2):138–142. https://doi.org/10.1016/j.fas.2020.03.014.

Sogbein OA, Yi J, Sanders DW, et al. Early radiographic outcomes following deltoid ligament repair in bimalleolar equivalent ankle fractures. *Foot Ankle Surg*. 2021. https://doi.org/10.1016/j.fas.2021.08.007.

Waldman SD. The deltoid ligament. In: *Pain review*. Philadelphia: Saunders; 2009:157.

Waldman SD. Abnormalities of the deltoid ligament. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:905–910.

Waldman SD. Functional anatomy of the ankle and foot. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:149–150.

Waldman SD. Deltoid ligament injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:815–819.

Waldman SD. Intra-articular injection of the ankle and foot. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:794–797.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Waldman SD, Campbell RSD. Deltoid ligament tear. In: *Imaging of pain*. Philadelphia: Saunders; 2011:439–441.

136

Anterior Tarsal Tunnel Syndrome

ICD-10 CODE G57.50

THE CLINICAL SYNDROME

Anterior tarsal tunnel syndrome is caused by compression of the deep peroneal nerve as it passes beneath the superficial fascia of the ankle (Fig. 136.1). The most common cause of this compression is trauma to the dorsum of the foot. Severe, acute plantar flexion of the foot has been implicated in anterior tarsal tunnel syndrome, as has wearing tight shoes or squatting and bending forward, such as when planting flowers (Fig. 136.2). The syndrome has also been associated with hypertrophy of the extensor halluces brevis muscle in dancers (Fig. 136.3). Anterior tarsal tunnel syndrome is much less common than posterior tarsal tunnel syndrome.

SIGNS AND SYMPTOMS

This entrapment neuropathy manifests primarily as pain, numbness, and paresthesias in the dorsum of the foot that radiate into the first dorsal web space; these symptoms may also radiate proximal to the entrapment, into the anterior ankle. No motor involvement occurs unless the distal lateral division of the deep peroneal nerve is affected. Nighttime foot pain analogous to that of carpal tunnel syndrome is often present. Patients may report that holding the foot in the everted position decreases the pain and paresthesias.

Physical findings include tenderness over the deep peroneal nerve at the dorsum of the foot. A positive Tinel sign just medial to the dorsalis pedis pulse over the deep peroneal nerve as it passes beneath the fascia is usually present (Fig. 136.4). Active plantar flexion often reproduces the symptoms of anterior tarsal tunnel syndrome. Weakness of the extensor digitorum brevis may be present if the lateral branch of the deep peroneal nerve is affected.

TESTING

Electromyography (EMG) can distinguish lumbar radiculopathy and diabetic polyneuropathy from anterior tarsal tunnel syndrome. Plain radiographs are indicated in all patients who present with foot or ankle pain to rule out

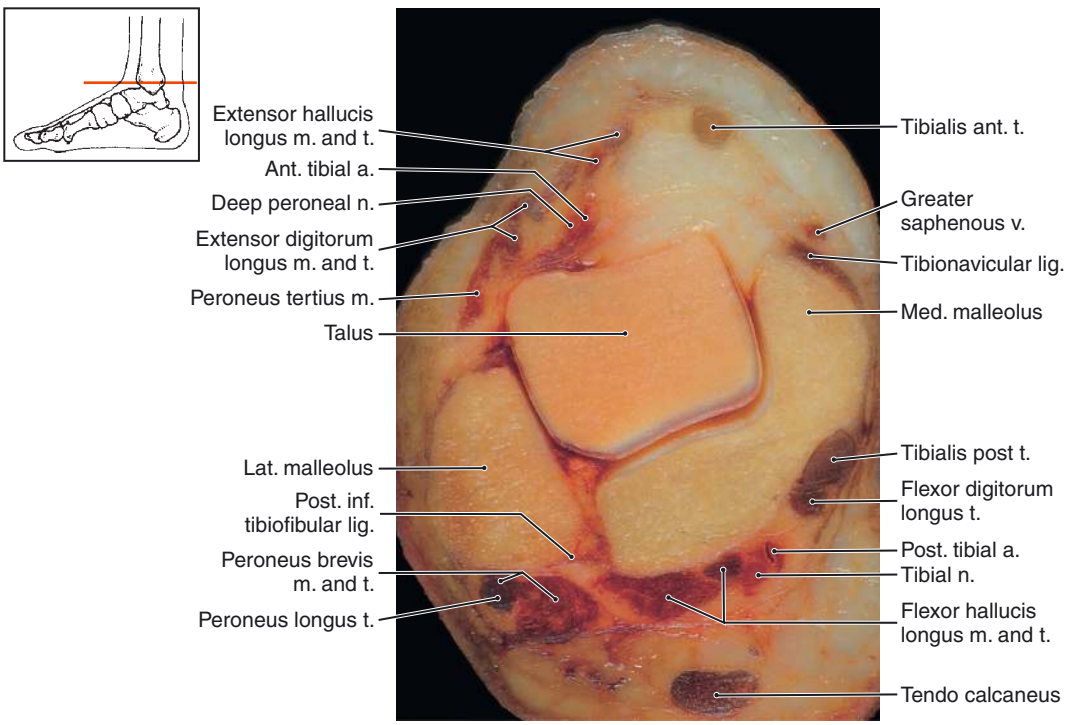

FIG 136.1 The relationships of the medial malleolus, the tibial artery and nerve, and the flexor tendons of the ankle. (From Kang HS, Ahn JM, Resnick D. *MRI of the extremities: an anatomic atlas*. 2nd ed. Philadelphia: Saunders; 2002:415.)

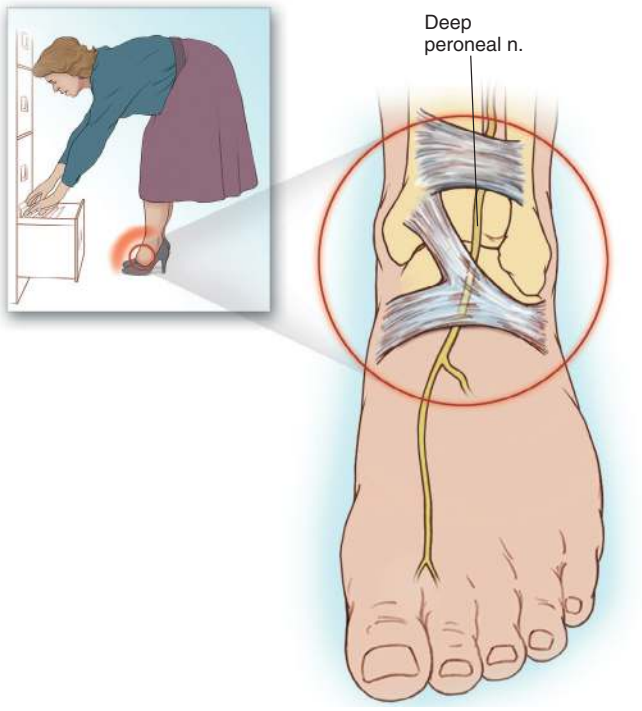

FIG 136.2 Anterior tarsal tunnel syndrome manifests as deep, aching pain in the dorsum of the foot, weakness of the extensor digitorum brevis, and numbness in the distribution of the deep peroneal nerve.

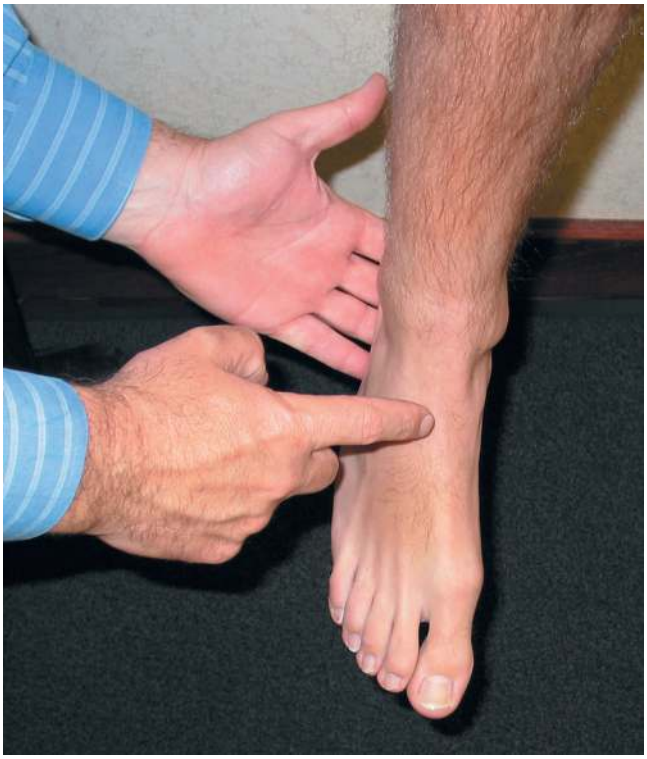

FIG 136.4 Eliciting Tinel sign for anterior tarsal tunnel syndrome. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms.* Philadelphia: Saunders; 2006:373.)

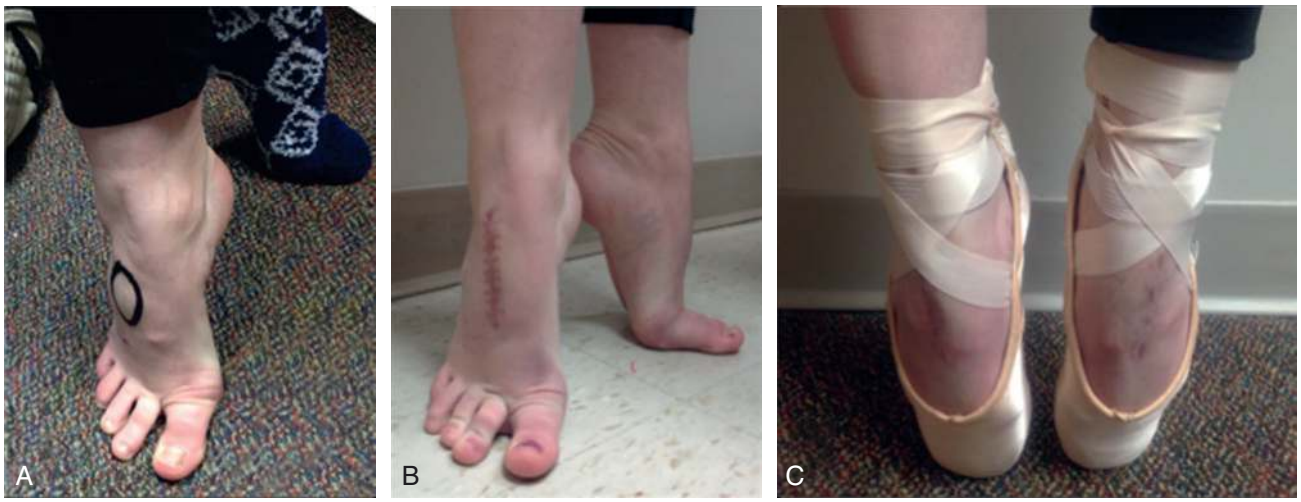

FIG 136.3 A, Preoperative right foot with identified dorsal hypertrophy of extensor hallucis brevis. B, Clinical photograph during *grand plié* at 2 (*left*) and 5 (*right*) months postoperatively and (C) clinical photograph *en pointe* after return to dance at seven (*left*) and 10 (*right*) months postoperatively. (From Tennant JN, Rungprai C, Phisitkul P. Bilateral anterior tarsal tunnel syndrome variant secondary to extensor hallucis brevis muscle hypertrophy in a ballet dancer: a case report. *Foot Ankle Surg.* 2014;20(4):e56–e58.)

occult bony disease (Fig. 136.5). Magnetic resonance imaging (MRI) and ultrasound imaging of the ankle and foot are indicated if joint instability or a space-occupying lesion is suspected (Figs. 136.6–136.8). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

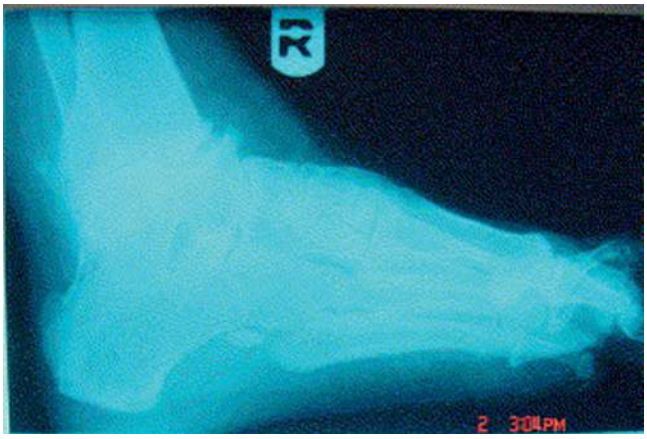

FIG 136.5 Radiograph shows tibial talar exostosis, which causes compression and anterior tarsal tunnel syndrome. (From DiDomenico LA, Masternick EB. Anterior tarsal tunnel syndrome. *Clin Podiatr Med Surg.* 2006;23(3):611–620.)

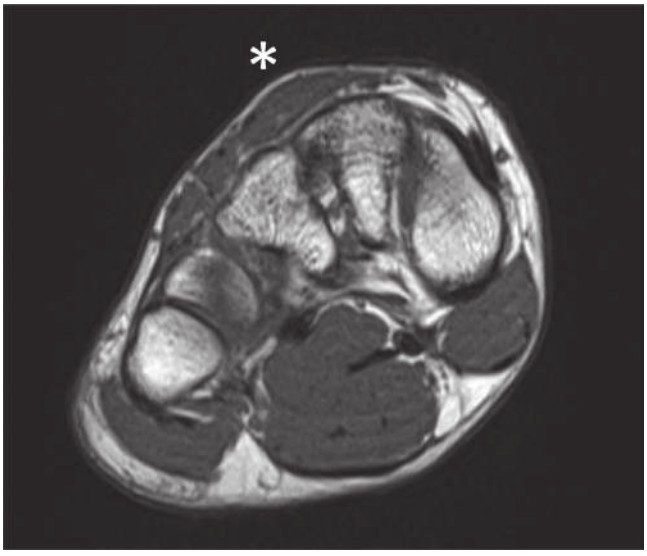

FIG 136.6 T1–weighted magnetic resonance image coronal cut at midfoot showing hypertrophy of the extensor hallucis brevis (EHB) in a ballet dancer with anterior tarsal tunnel syndrome (* immediately dorsal to EHB). (From Tennant JN, Rungprai C, Phisitkul P. Bilateral anterior tarsal tunnel syndrome variant secondary to extensor hallucis brevis muscle hypertrophy in a ballet dancer: a case report. *Foot Ankle Surg.* 2014;20(4):e56–e58.)

DIFFERENTIAL DIAGNOSIS

Anterior tarsal tunnel syndrome is often misdiagnosed as arthritis of the ankle joint, lumbar radiculopathy, or diabetic polyneuropathy. Patients with arthritis of the ankle, however, have radiographic evidence of arthritis. Most patients suffering from lumbar radiculopathy have reflex, motor, and sensory changes associated with back pain, whereas patients with anterior tarsal tunnel syndrome have no reflex changes or motor deficits, and sensory changes are limited to the distribution of the distal deep peroneal nerve. However, lumbar radiculopathy and deep peroneal nerve entrapment may coexist as the double-crush syndrome. Diabetic polyneuropathy generally manifests as a symmetric sensory deficit involving the entire foot, rather than a disorder limited to the distribution of the deep peroneal nerve. When anterior tarsal tunnel syndrome occurs in diabetic patients, diabetic polyneuropathy is usually also present.

TREATMENT

Mild cases of tarsal tunnel syndrome usually respond to conservative therapy; surgery should be reserved for severe cases. Initial treatment of tarsal tunnel syndrome consists of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors and splinting of the ankle. At a minimum, the splint should be worn at night, but wearing it for 24 hours a day is ideal. Avoidance of repetitive activities that may be responsible for the development of tarsal tunnel syndrome, such as prolonged squatting or wearing shoes that are too tight, can also ameliorate the symptoms. If patients fail to respond to these conservative measures, injection of the tarsal tunnel with local anesthetic and steroid is a reasonable next step.

Tarsal tunnel injection is performed by placing the patient in the supine position with the leg extended. The extensor hallucis longus tendon is identified by having the patient extend his or her big toe against resistance. A point just medial to the tendon at the skin crease of the ankle is identified and prepared with antiseptic solution. A 1½-inch, 25-gauge needle is advanced through this point very slowly toward the tibia until a paresthesia is elicited in the web space between the first and second toes, usually at a needle depth of ¼–½ inch. The patient should be warned to expect this paresthesia and instructed to say "There!" as soon as it is felt. If no paresthesia is elicited, the needle is withdrawn and is redirected slightly more posteriorly until a paresthesia is induced. The needle is then withdrawn 1 mm, and the patient is observed to ensure that he or she is not experiencing any persistent paresthesia. After careful aspiration, a total of 6 mL of 1% preservative-free lidocaine and 40-mg methylprednisolone is slowly injected. Care must be taken not to advance the needle into the substance of the nerve and inadvertently inject the solution intraneurally. After injection, pressure is applied to the injection site to decrease the incidence of ecchymosis and hematoma formation. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications (Fig. 136.9).

COMPLICATIONS AND PITFALLS

Failure to treat tarsal tunnel syndrome adequately can result in permanent pain, numbness, and functional disability; these problems can be exacerbated if coexistent reflex sympathetic dystrophy is not treated aggressively with sympathetic neural blockade. The main complications of deep peroneal nerve block are ecchymosis and hematoma, which can be avoided by applying pressure to the injection site.

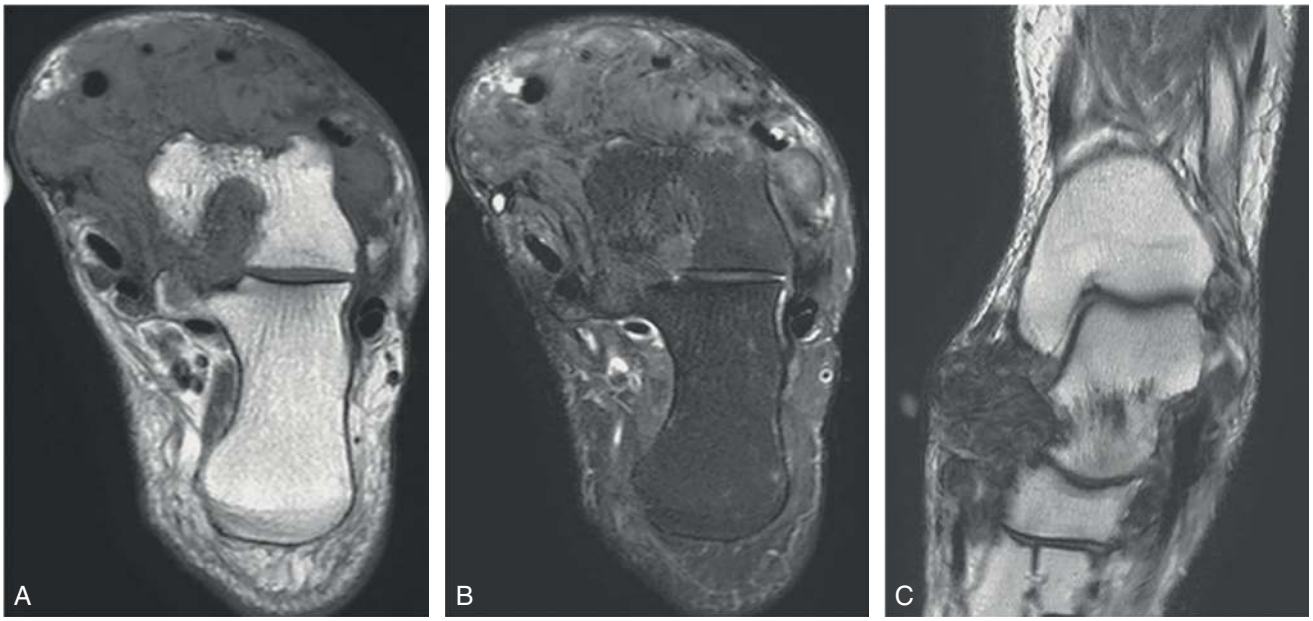

FIG 136.7 Axial T1–weighted **(A)** and fat-suppressed T2–weighted **(B)** magnetic resonance images of a patient with ankle pain and paresthesia over the dorsal aspect of the foot. A mass of synovitis arising from the ankle joint surrounds the extensor tendons and the anterior neurovascular bundle. The synovium has intermediate signal intensity on both images and also on the coronal T2–weighted image **(C).** There is also bony erosion. These appearances are typical of pigmented villonodular synovitis. (From Waldman SD, Campbell R. *Imaging of pain*. Philadelphia: Saunders; 2011: Fig. 164.3.)

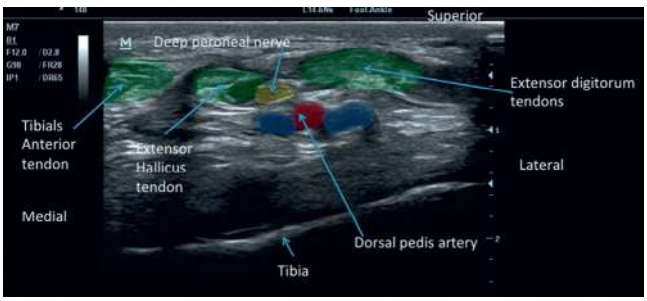

FIG 136.8 Transverse ultrasound image demonstrating the tibial artery and vein and the deep peroneal nerve just above and lateral to the vein.

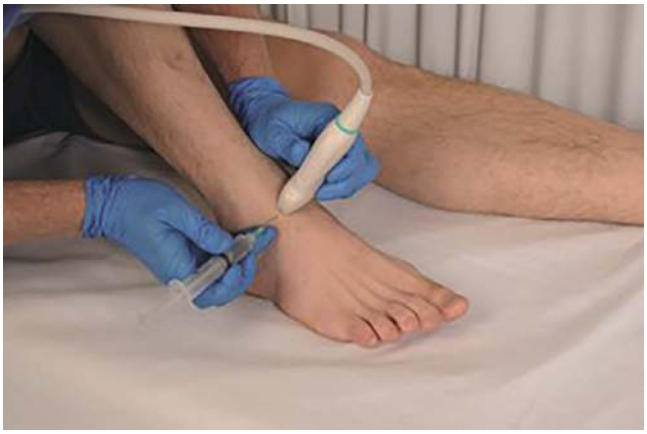

FIG 136.9 Proper in-plane needle position for performing injection technique for anterior tarsal tunnel syndrome.

Because a paresthesia is elicited with this technique, needle-induced trauma to the common peroneal nerve is a possibility. By advancing the needle slowly and then withdrawing it slightly away from the nerve, needle-induced trauma can be avoided.

CLINICAL PEARLS

The most common cause of pain radiating into the lower extremity is herniated lumbar disk or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the common or deep peroneal nerve. Lesions above the origin of the common peroneal nerve, such as lesions of the sciatic nerve, or lesions at the point where the common peroneal nerve winds around the head of the fibula, may be confused with deep peroneal nerve entrapment. EMG and MRI of the lumbar spine, combined with the clinical history and physical examination, can determine the cause of pain in the distal lower extremity and the foot. Diabetic patients and other patients with vulnerable nerve syndrome may be more susceptible to the development of anterior tarsal tunnel syndrome.

The injection technique described is useful in the treatment of anterior tarsal tunnel syndrome. Careful preinjection neurologic assessment is important to identify preexisting neurologic deficits that could later be attributed to the deep peroneal nerve block. These assessments are especially important in patients who have sustained trauma to the ankle or foot and in those suffering from diabetic neuropathy in whom deep peroneal nerve block is being used for acute pain control.

SUGGESTED READINGS

de Cesar Netto C, da Fonseca LF, Nascimento FS, et al. Diagnostic and therapeutic injections of the foot and ankle—an overview. *Foot Ankle Surg*. 2018;24(2):99–106.

DiDomenico LA, Masternick EB. Anterior tarsal tunnel syndrome. *Clin Podiatr Med Surg*. 2006;23(3):611–620.

Grant CRK, Raju PKBC. Lower limb nerve blocks. *Anaesth Intens Care Med*. 2016;17(4):182–186.

Ferkel E, Davis WH, Ellington JK. Entrapment neuropathies of the foot and ankle. *Clin Sports Med*. 2015;34(4):791–801.

Kennedy JG, Baxter DE. Nerve disorders in dancers. *Clin Sports Med*. 2008;27(2):329–334.

Tennant JN, Rungprai C, Phisitkul P. Bilateral anterior tarsal tunnel syndrome variant secondary to extensor hallucis brevis muscle hypertrophy in a ballet dancer: a case report. *Foot Ankle Surg*. 2014;20(4):e56–e58.

Waldman SD. Anterior tarsal syndrome and other abnormalities of the deep peroneal nerve. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:940–951.

Waldman SD. Anterior tarsal tunnel syndrome. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:309–310.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Posterior Tarsal Tunnel Syndrome

ICD-10 CODE G57.50

THE CLINICAL SYNDROME

Posterior tarsal tunnel syndrome is caused by compression of the posterior tibial nerve as it passes through the posterior tarsal tunnel. The posterior tarsal tunnel is made up of the flexor retinaculum, the bones of the ankle, and the lacunar ligament. In addition to the posterior tibial nerve, the tunnel contains the posterior tibial artery and certain flexor tendons that are subject to tenosynovitis. The most common cause of compression of the posterior tibial nerve at this location is trauma to the ankle, including fracture, dislocation, and crush injury (Fig. 137.1 and Box 137.1). Thrombophlebitis involving the posterior tibial artery has also been implicated in the development of posterior tarsal tunnel syndrome as has the wearing of tight high-heeled shoes with straps. Tumors of the posterior tibial nerve can also cause symptoms in the distribution of the posterior tibial nerve (Fig. 137.2). Patients with rheumatoid arthritis have a higher incidence of posterior tarsal tunnel syndrome than the general population. Posterior tarsal tunnel syndrome is much more common than anterior tarsal tunnel syndrome.

SIGNS AND SYMPTOMS

Posterior tarsal tunnel syndrome manifests in a manner analogous to carpal tunnel syndrome. Patients complain of pain, numbness, and paresthesias in the sole of the foot; these symptoms may also radiate proximal to the entrapment, into the medial ankle (Fig. 137.3). Patients may note weakness of the toe flexors and instability of the foot resulting from weakness of the lumbrical muscles. Nighttime foot pain analogous to that of carpal tunnel syndrome is often present.

Physical findings include tenderness over the posterior tibial nerve at the medial malleolus. A positive Tinel sign just below and behind the medial malleolus over the posterior tibial nerve is usually present (Fig. 137.4). Active inversion of the ankle often reproduces the symptoms of posterior tarsal tunnel syndromes. Medial and lateral plantar divisions of the posterior tibial nerve provide motor innervation to the intrinsic muscles of the foot; thus, weakness of the flexor digitorum brevis and the lumbrical muscles may be present if these branches of the nerve are affected.

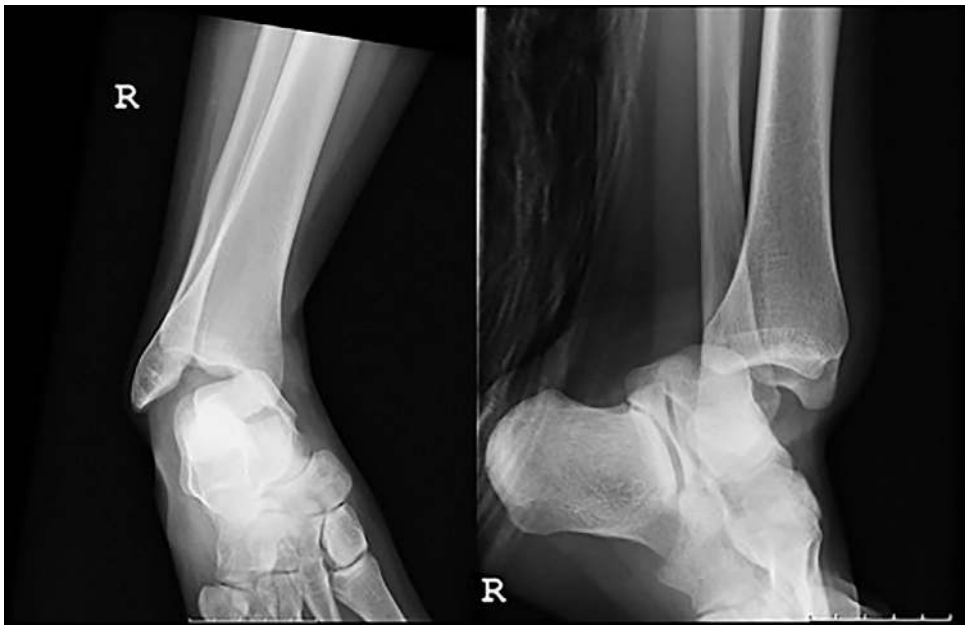

FIG 137.1 X-ray of a posteromedial pure ankle dislocation. No fracture is noted. (From Wight L, Owen D, Goldbloom D, et al. Pure ankle dislocation: a systematic review of the literature and estimation of incidence. *Injury*. 2017;48(10):20272034, Fig. 1.)

BOX 137.1 Conditions Associated With Posterior Tarsal Tunnel Syndrome

Structural/anatomic
- Lipoma
- Ganglion
- Neuroma
- Aneurysm
- Dislocation
- Fracture

Inflammatory
- Tenosynovitis
- Collagen vascular disease
  - Rheumatoid arthritis
  - Scleroderma
- Gout

Neuropathic/ischemic
- Diabetes
- Alcoholism
- Vitamin abnormalities
- Ischemic neuropathies
- Peripheral neuropathies
- Amyloidosis

Repetitive stress related
- Abnormal foot and ankle position
- Microtrauma
- Vibration

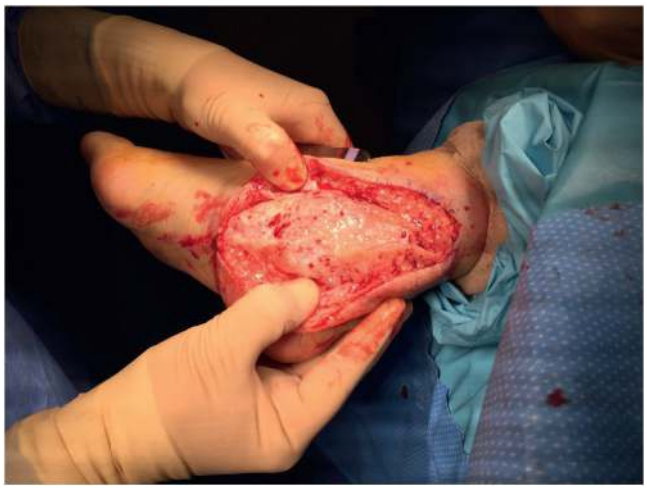

FIG 137.2 Intraoperative traumatic neuroma of the right foot extending from the inferior medial ankle to the plantar midfoot. (From Hassan MK, LaPolla JJ Jr., Traumatic neuroma of the posterior tibial nerve due to previous surgery presenting as a massive tumor in the midfoot: A case report. *Foot.* 2019;39:68–71.)

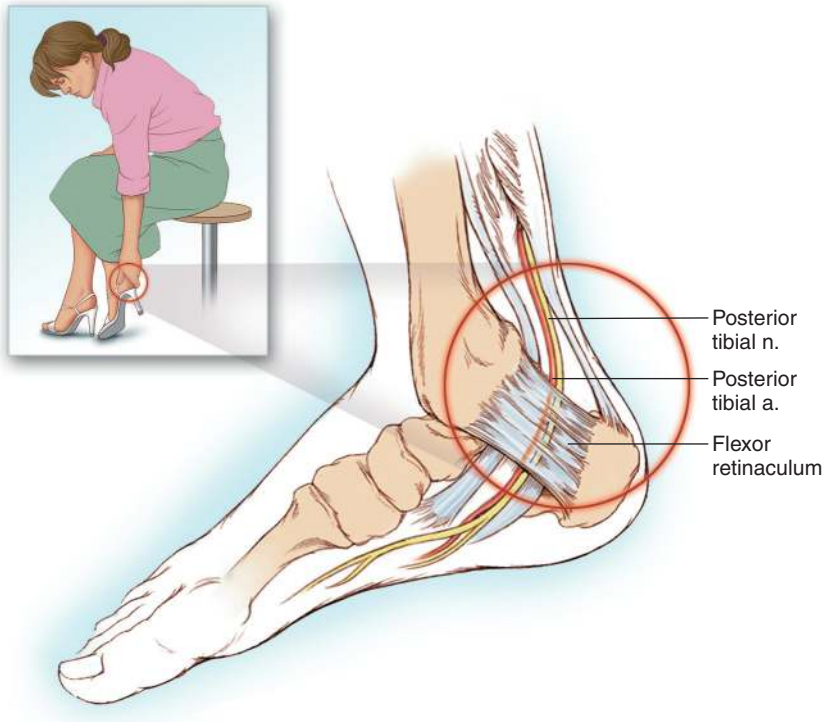

FIG 137.3 Posterior tarsal tunnel syndrome is characterized by pain, numbness, and paresthesias of the sole of the foot.

TESTING

Electromyography (EMG) can distinguish lumbar radiculopathy and diabetic polyneuropathy from posterior tarsal tunnel syndrome. Plain radiographs, magnetic resonance imaging (MRI), and ultrasound imaging are indicated for all patients who present with posterior tarsal tunnel syndrome, to rule out occult bony disease (Fig. 137.5). MRI and ultrasound imaging of the ankle and foot are also indicated if joint instability or a space-occupying lesion is suspected (Figs. 137.6–137.8). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, uric acid level, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

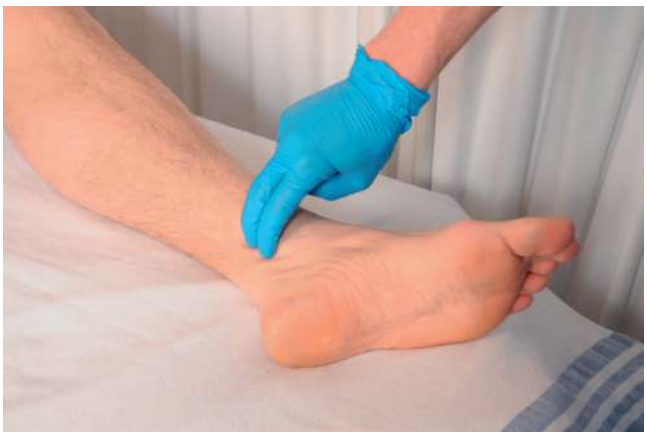

FIG 137.4 A positive Tinel sign just posterior to the medial malleolus over the posterior tibial nerve as it passes beneath the fascia is usually present in patients suffering from posterior tarsal tunnel syndrome.

DIFFERENTIAL DIAGNOSIS

Posterior tarsal tunnel syndrome is often misdiagnosed as arthritis of the ankle joint, lumbar radiculopathy, or diabetic polyneuropathy. Patients with arthritis of the ankle, however, have radiographic evidence of arthritis. Most patients suffering from lumbar radiculopathy have reflex, motor, and sensory changes associated with back pain, whereas those with posterior tarsal tunnel syndrome have no reflex changes, and motor and sensory changes are limited to the distribution of the distal posterior tibial nerve. However, lumbar

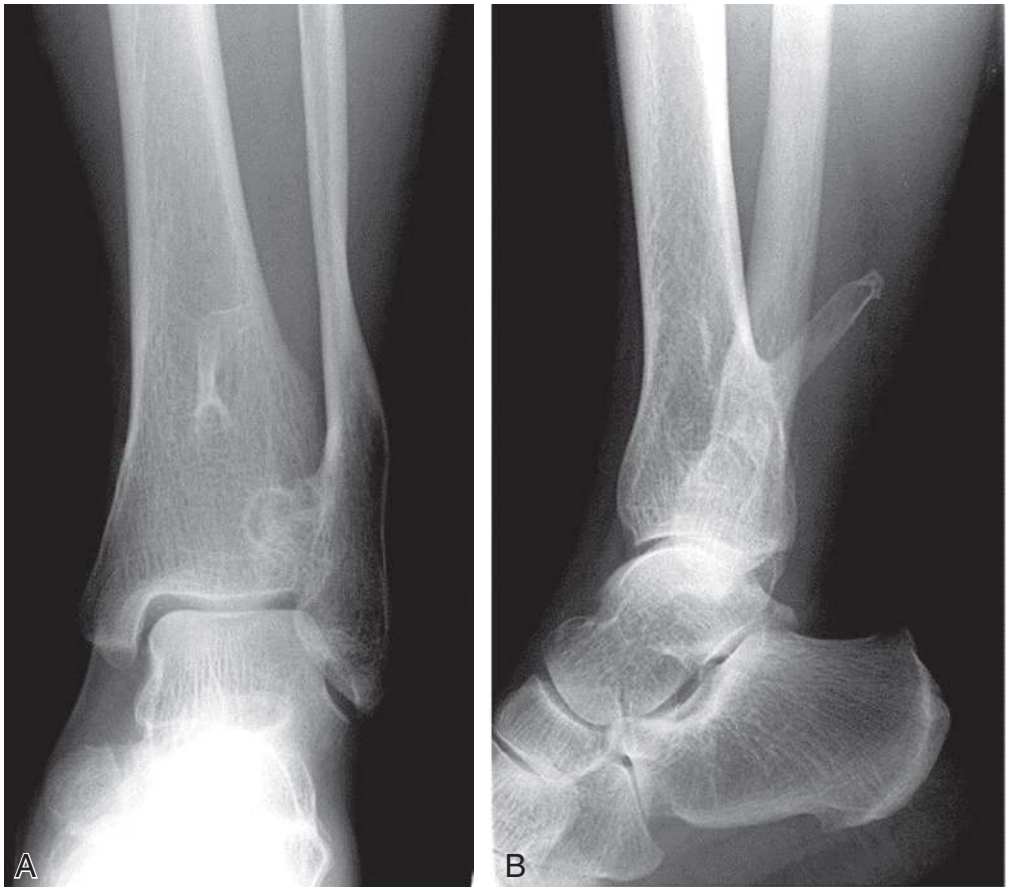

FIG 137.5 Anteroposterior **(A)** and lateral **(B)** radiographs of the left ankle of a 52-year-old woman presenting with symptoms consistent with posterior tarsal tunnel syndrome. Note the presence of osteochondromas on both the distal tibia and the fibula. (From Matsumoto K, Sumi H, Shimizu K. Tibial osteochondroma causing foot pain mimicking tarsal tunnel syndrome: a case report. *J Foot Ankle Surg.* 2005;44(2):159–162.)

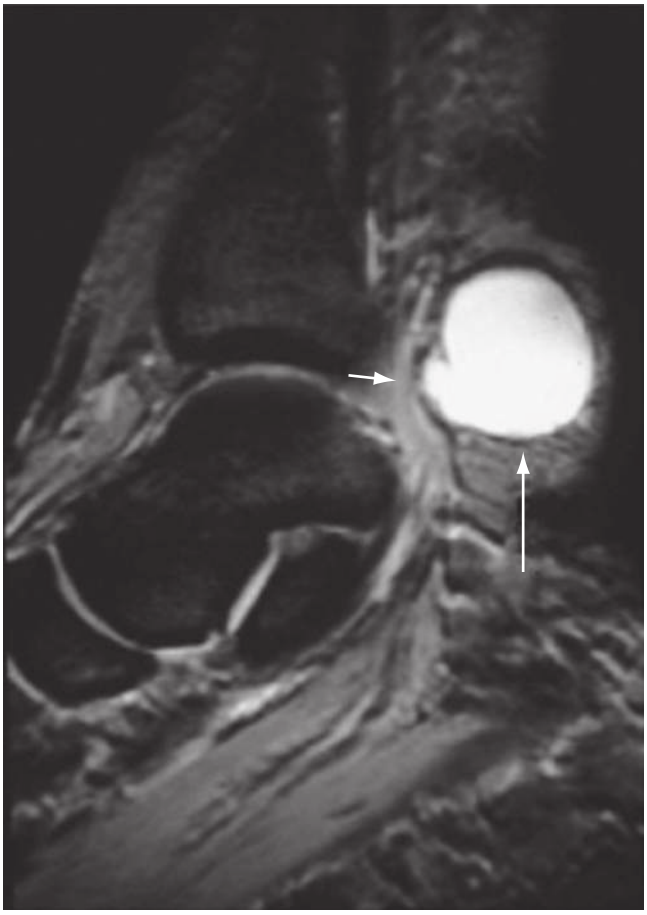

FIG 137.6 Tarsal tunnel syndrome. Sagittal short tau inversion recovery magnetic resonance image shows a ganglion cyst (*long arrow*) compressing the neurovascular bundle in the tarsal tunnel (*short arrow*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3454.)

radiculopathy and posterior tibial nerve entrapment may coexist as the double-crush syndrome. Diabetic polyneuropathy generally manifests as a symmetric sensory deficit involving the entire foot, rather than a condition limited to the distribution of the posterior tibial nerve. When posterior tarsal tunnel syndrome occurs in diabetic patients, diabetic polyneuropathy is usually also present.

TREATMENT

Mild cases of tarsal tunnel syndrome usually respond to conservative therapy; surgery should be reserved for severe cases. Initial treatment of tarsal tunnel syndrome consists of simple analgesics, nonsteroidal antiinflammatory drugs, or cyclooxygenase-2 inhibitors and splinting of the ankle. At a

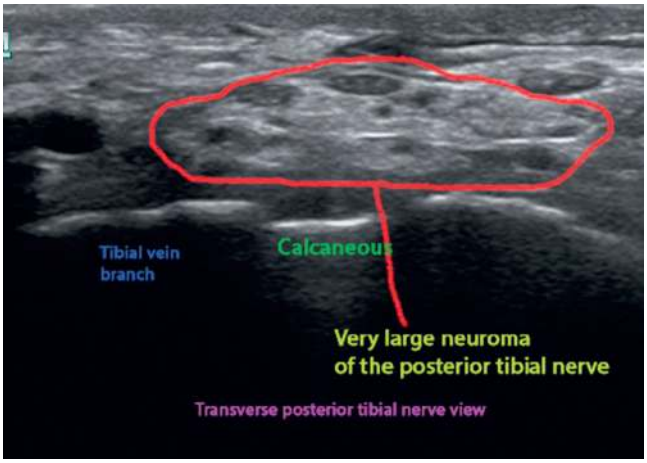

FIG 137.7 Transverse ultrasound image demonstrating a large neuroma of the posterior tibial nerve.

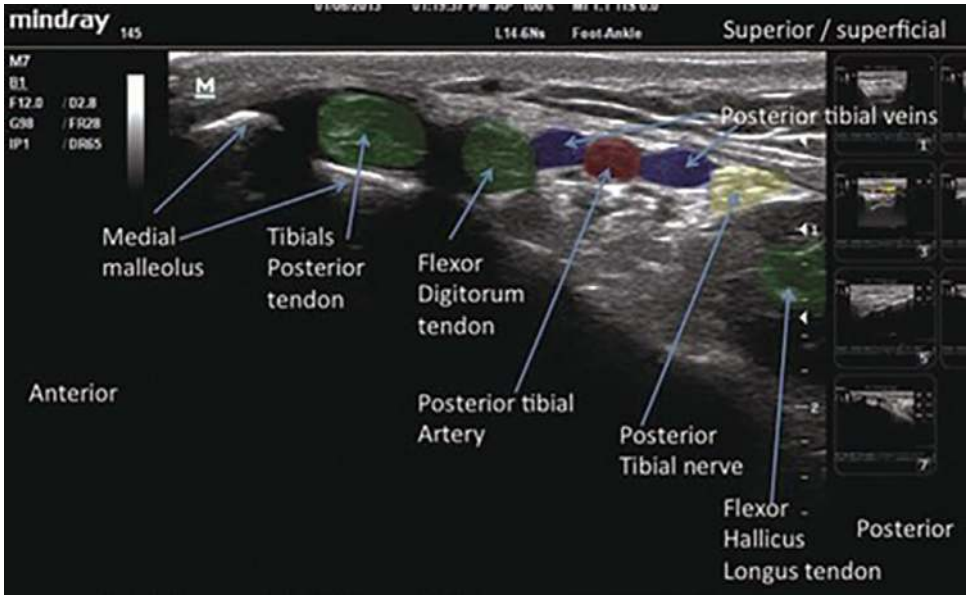

FIG 137.8 In addition to the posterior tibial nerve, the posterior tarsal tunnel contains the posterior tibial artery and vein as well as a number of flexor musculotendinous units.

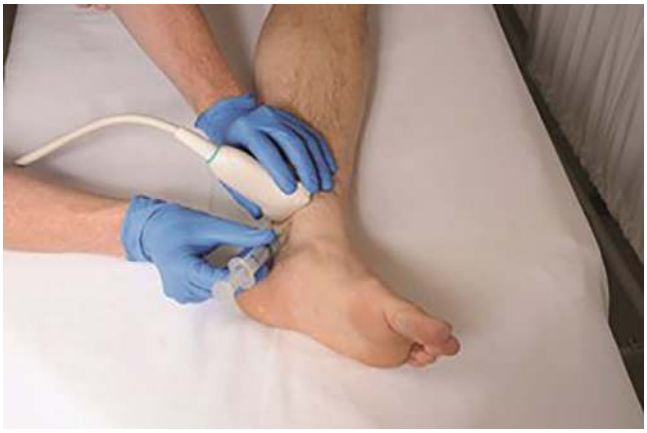

FIG 137.9 Proper out-of-plane needle position for performing posterior tibial nerve block at the ankle.

minimum, the splint should be worn at night, but wearing it for 24 hours a day is ideal. Avoidance of repetitive activities that may be responsible for the development of tarsal tunnel syndrome and can also ameliorate the symptoms. If patients fail to respond to these conservative measures, injection of the tarsal tunnel with local anesthetic and steroid is a reasonable next step.

Posterior tarsal tunnel injection is performed with the patient in the lateral position and the affected leg in the dependent position and slightly flexed (Fig. 137.9). The posterior tibial artery is palpated, and the area between the medial malleolus and the Achilles tendon is identified and prepared with antiseptic solution. A 1½-inch, 25-gauge needle is inserted at this level and is directed anteriorly toward the pulsation of the posterior tibial artery. If the arterial pulsation cannot be identified, the needle is directed toward the posterior superior border of the medial malleolus. The needle is advanced slowly toward the tibial nerve, which lies in the posterior groove of the medial malleolus, until a paresthesia is elicited in the distribution of the tibial nerve, usually after the needle is advanced ½–¾ inch. The patient should be warned to expect this paresthesia and instructed to say "There!" as soon as it is felt. If no paresthesia is elicited, the needle is withdrawn and is redirected slightly more cephalad until a paresthesia is induced. The needle is then withdrawn 1 mm, and the patient is observed to ensure that he or she is not experiencing any persistent paresthesia. After careful aspiration, a total of 6 mL of 1% preservative-free lidocaine and 40-mg methylprednisolone is slowly injected. Care must be taken not to advance the needle into the substance of the nerve and inadvertently inject the solution intraneurally. After injection, pressure is applied to the injection site to decrease the incidence of ecchymosis and hematoma formation. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

COMPLICATIONS AND PITFALLS

Failure to treat tarsal tunnel syndrome adequately can result in permanent pain, numbness, and functional disability; these problems can be exacerbated if coexistent reflex sympathetic dystrophy is not treated aggressively with sympathetic neural blockade. The main complications of injection are ecchymosis and hematoma, which can be avoided by applying pressure to the injection site. Because a paresthesia is elicited, needle-induced trauma to the nerve is a possibility. By advancing the needle slowly and then withdrawing it slightly away from the nerve, needle-induced trauma can be avoided.

CLINICAL PEARLS

The most common cause of pain radiating into the lower extremity is herniated lumbar disk or nerve impingement secondary to degenerative arthritis of the spine, not disorders involving the tibial, common, or deep peroneal nerve. Lesions above the origin of the tibial or common peroneal nerve may be confused with posterior tarsal tunnel syndrome. EMG and MRI of the lumbar spine, combined with the clinical history and physical examination, can determine the cause of distal lower extremity and foot pain.

SUGGESTED READINGS

Allen JM, Greer BJ, Sorge DS, et al. MR Imaging of neuropathies of the leg, ankle, and foot. *Magn Reson Imaging Clin N Am*. 2008;16(1):117–131.

Cancilleri F, Ippolito M, Amato C, et al. Tarsal tunnel syndrome: four uncommon cases. *J Foot Ankle Surg*. 2007;13(4):214–217.

Ferkel E, Davis WH, Ellington JK. Entrapment neuropathies of the foot and ankle. *Clin Sports Med*. 2015;34(4):791–801.

Fraser TW, Doty JF. Peripheral nerve blocks in foot and ankle surgery. *Orthop Clin North Am*. 2017;48(4):507–515.

Grant CRK, Raju PKBC. Lower limb nerve blocks. *Anaesth Intens Care Med*. 2016;17(4):182–186.

Kennedy JG, Baxter DE. Nerve disorders in dancers. *Clin Sports Med*. 2008;27(2):329–334.

Waldman SD. Posterior tarsal tunnel syndrome. In: *Pain review*. Philadelphia: Saunders; 2009:323–324.

Waldman SD. Posterior tarsal syndrome and other abnormalities of the posterior tibial nerve. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:952–963.

Waldman SD. Posterior tarsal tunnel syndrome. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:310–311.

Waldman SD. Posterior tibial nerve block at the ankle. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:837–842.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Williams TH, Robinson AH. Entrapment neuropathies of the foot and ankle. *J Orthop Trauma*. 2009;23(6):404–411.

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Achilles Tendinitis

ICD-10 CODE M65.879

THE CLINICAL SYNDROME

Achilles tendinitis has become more common as jogging has increased in popularity. The Achilles tendon is susceptible to the development of tendinitis both at its insertion on the calcaneus and at its narrowest part, a point approximately 5 cm above its insertion. The Achilles tendon is subjected to repetitive motion that may result in microtrauma, which heals poorly owing to the tendon's avascular nature. The appearance of Achilles tendinitis has been described as having a crabmeat appearance owing to the nonlinear orientation of the tendon fibers (Fig. 138.1). Running is often the inciting factor in acute Achilles tendinitis, which frequently coexists with bursitis and thus causes additional pain and functional disability. Calcium deposition around the tendon may occur if inflammation persists, and this complication makes subsequent treatment more difficult (Fig. 138.2). Continued trauma to the inflamed tendon may ultimately result in tendon rupture.

SIGNS AND SYMPTOMS

The onset of Achilles tendinitis is usually acute, occurring after overuse or misuse of the ankle joint. Inciting activities include running with sudden stops and starts, such as when playing tennis. Improper stretching of the gastrocnemius and Achilles tendon before exercise has also been implicated in Achilles tendinitis, as well as in acute tendon rupture. The pain of Achilles tendinitis is constant and severe and is localized in the posterior ankle (Fig. 138.3). Significant sleep disturbance is often reported. Patients may attempt to splint the inflamed Achilles tendon by adopting a flatfooted gait to avoid plantar flexing the tendon. Pain is induced with resisted plantar flexion of the foot, and a creaking or grating sensation may be palpated when the foot is passively plantar flexed (Fig. 138.4). A chronically inflamed Achilles tendon may suddenly rupture from stress or during injection into the tendon itself.

TESTING

Plain radiographs, ultrasound imaging, and magnetic resonance imaging (MRI) are indicated in all patients who present with posterior ankle pain (Figs. 138.5 and 138.6). MRI and ultrasound imaging of the ankle are also indicated if joint instability is suspected and to assess the amount of tendinosis and tendinopathy (Figs. 138.7–138.9). Radionuclide bone scanning is useful to identify stress fractures not seen on plain radiographs. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, comprehensive metabolic profile, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Achilles tendinitis is usually easily identified on clinical grounds. However, if the bursa located between the Achilles

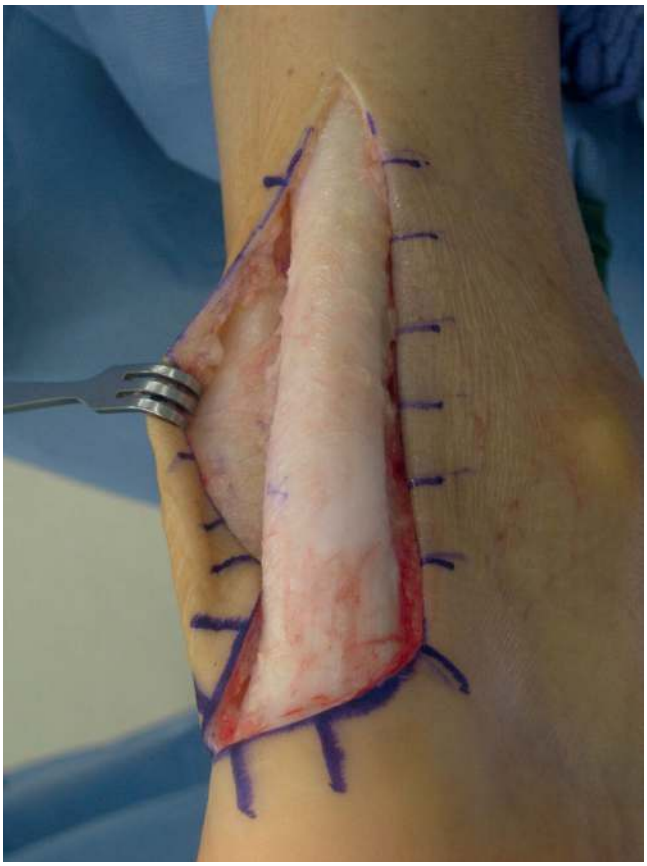


FIG 138.1 Bulbous "crabmeat" tendon without distinct orientation in the central aspect of the Achilles tendon. (From Sundararajan PP. Transosseous fixation in insertional Achilles tendonitis. *J Foot Ankle Surg.* 2012;51(6):806–812.)

tendon and the base of the tibia and the upper posterior calcaneus is inflamed, coexistent bursitis may confuse the diagnosis. Stress fractures of the ankle may also mimic Achilles tendinitis.

TREATMENT

Initial treatment of the pain and functional disability associated with Achilles tendinitis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Repetitive activities thought to be responsible for the development of tendinitis, such as jogging, should be avoided. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

Injection for Achilles tendinitis is carried out by placing the patient in the prone position with the affected foot hanging off the end of the table. The foot is gently dorsiflexed to facilitate identification of the margin of the tendon, because injection directly into the tendon should be avoided. The tender point at the tendinous insertion or at its narrowest part approximately 5 cm above the insertion is identified and marked with a sterile marker. The skin overlying this point is prepared with antiseptic solution. A sterile syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40-mg

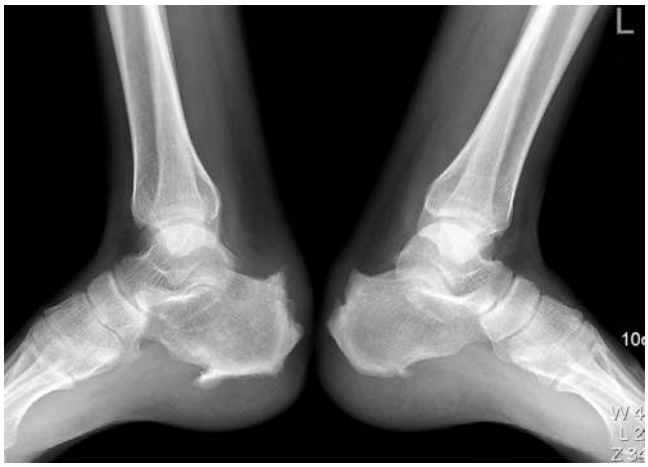


FIG 138.2 Radiograph of a significantly calcified insertional Achilles tendon with Haglund deformity. (From Miao XD, Jiang H, Wu YP, Tao HM, Yang DS, Hu H. Treatment of Calcified Insertional Achilles Tendinopathy by the Posterior Midline Approach. *J Foot Ankle Surg.* 2016 May-Jun;55(3):529–34. Fig. 1. ISSN 1067-2516.)

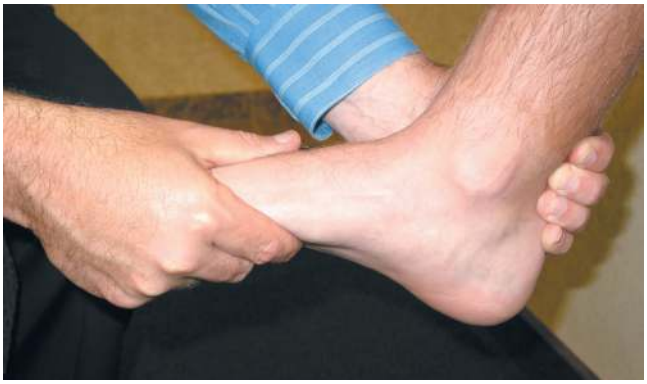


FIG 138.4 Eliciting the creak sign for Achilles tendinitis. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms.* Philadelphia: Saunders; 2006:377.)

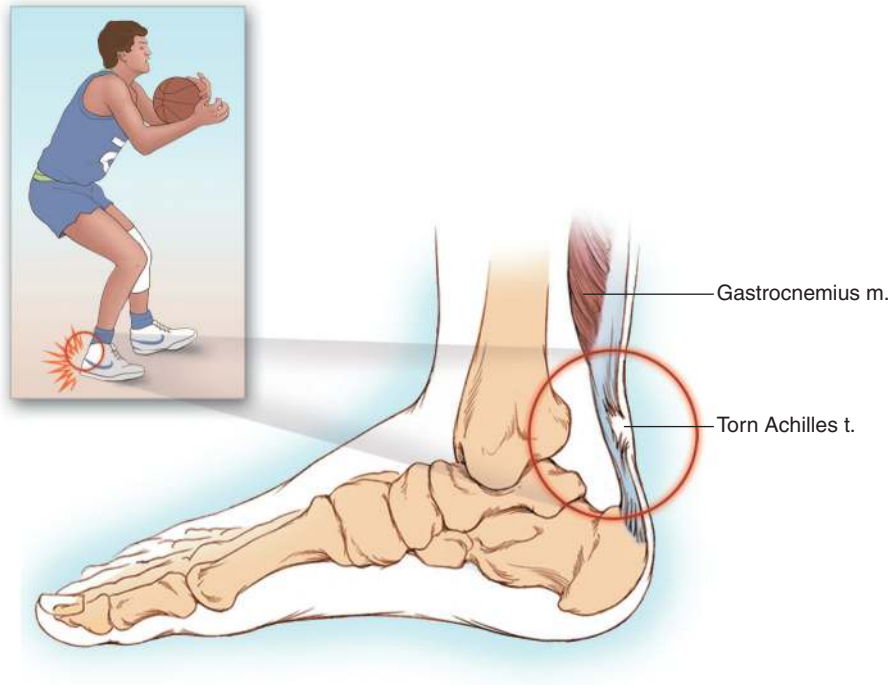


FIG 138.3 The pain of Achilles tendinitis is constant and severe and is localized to the posterior ankle.

FIG 138.5 Magnetic resonance imaging of chronic, degenerative tendinosis with thickening of the tendon in the sagittal (A) and coronal (B) planes compared with the opposite Achilles tendon. (From Lesic A, Bumbasirevic M. Disorders of the Achilles tendon. *Curr Orthop*. 2004;18(1):63–75.)

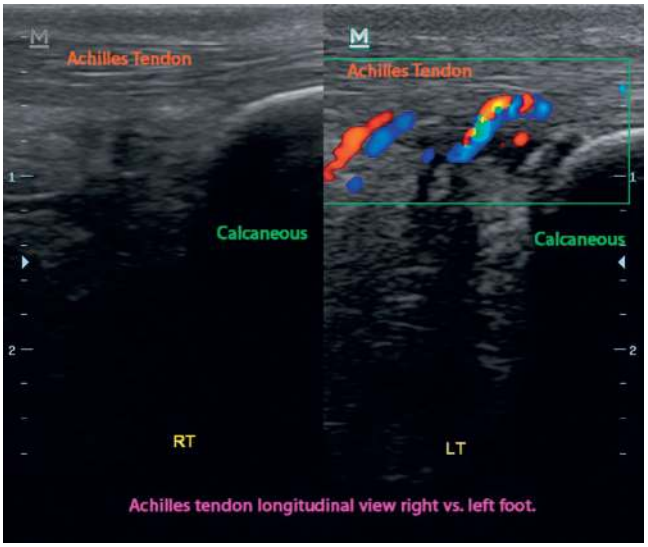


FIG 138.6 Longitudinal color Doppler image of tendinitis of the left Achilles tendon. Note the neovascularity when compared with the right.

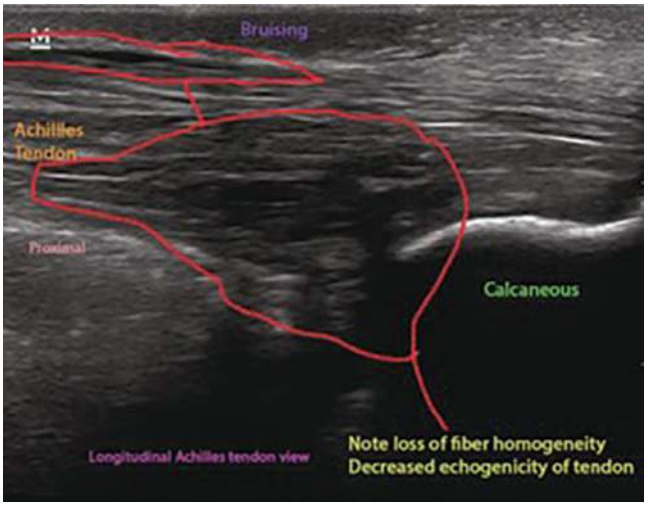


FIG 138.7 Longitudinal ultrasound image demonstrating bruising of the Achilles tendon from direct trauma as well as extensive tendinosis with significant tearing of the tendon substance.

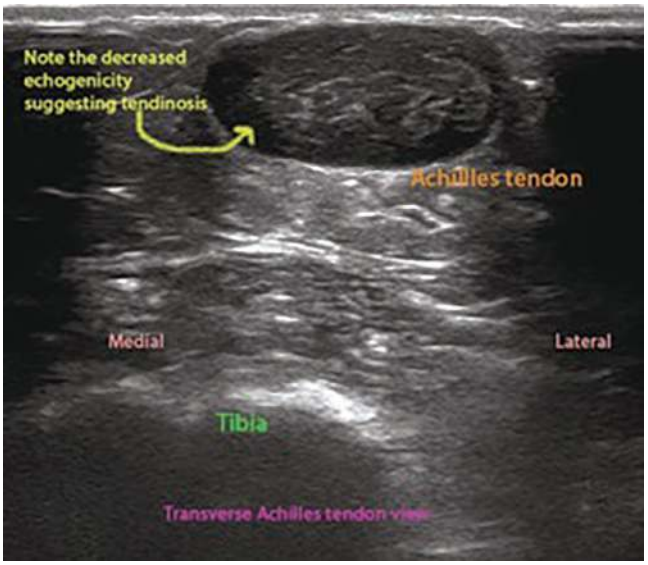


FIG 138.8 Transverse ultrasound image demonstrating tendinosis of the Achilles tendon. Note decreased echogenicity.

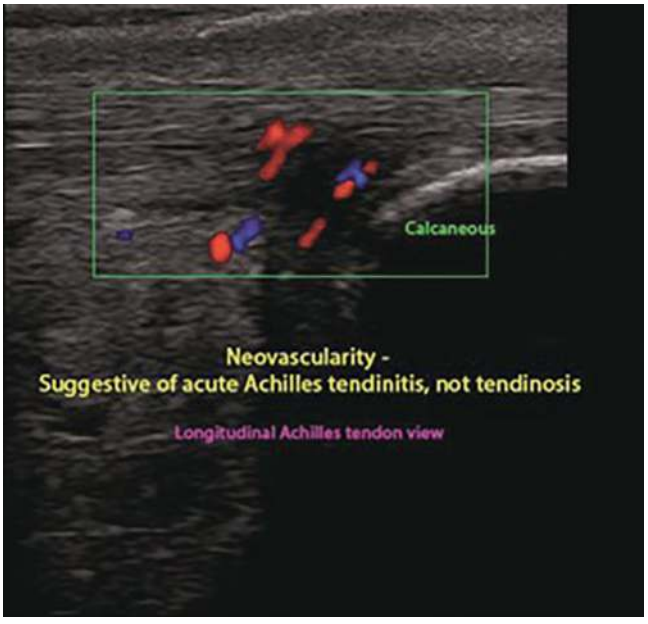


FIG 138.9 Color Doppler may help identify neovascularity of the tendon that is highly suggestive of tendinitis.

FIG 138.10 Proper position of the ultrasound transducer for the injection of Achilles tendinitis.

methylprednisolone is attached to a 1½-inch, 25-gauge needle using strict aseptic technique. The previously marked point is palpated, and the needle is carefully advanced at this point along the tendon and through the skin and subcutaneous tissues, with care taken not to enter the substance of the tendon. The contents of the syringe are gently injected while the clinician slowly withdraws the needle. Resistance to injection should be minimal. If resistance is significant, the needle tip is probably in the substance of the Achilles tendon and should be withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Simple analgesics and NSAIDs can be used concurrently with this injection technique. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications (Fig. 138.10). The injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of Achilles tendinitis.

COMPLICATIONS AND PITFALLS

Trauma to the Achilles tendon from the injection itself is an ever-present possibility. Tendons that are highly inflamed or previously damaged are subject to rupture if they are injected directly. This complication can be avoided if the clinician uses gentle technique and stops injecting immediately if significant resistance is encountered. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Although the Achilles tendon is the thickest and strongest tendon in the body, it is susceptible to inflammation or even rupture. It begins at the midcalf and continues downward, narrowing as it goes, to attach to the posterior calcaneus; it becomes narrowest approximately 5 cm above its calcaneal insertion. At these two points, tendinitis is most likely to develop. The injection technique described is extremely effective in treating Achilles tendinitis. Coexistent bursitis and arthritis may contribute to the patient's symptoms, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

SUGGESTED READINGS

Kim YC, Ahn JH, Kim MS. Infectious Achilles tendinitis after local injection of human placental extracts: a case report. *J Foot Ankle Surg*. 2015;54(6):1193–1196.

Tenforde AS, Yin A, Hunt KJ. Foot and ankle injuries in runners. *Phys Med Rehabil Clin N Am*. 2016;27(1):121–137.

Waldman SD. Achilles tendinitis and other abnormalities of the Achilles tendon. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:986–987.

Waldman SD. Achilles tendon. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:153–154.

Waldman SD. Achilles tendon injection. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:848–851.

Waldman SD, Campbell RSD. Achilles tendinitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:427–429.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Weinfeld SB. Achilles tendon disorders. *Med Clin N Am*. 2014;98(2):331–338.

139

Achilles Tendon Rupture

ICD-10 CODE M66.369

THE CLINICAL SYNDROME

Achilles tendon rupture most often occurs following an injury after acute push-off during jumping or sprinting as the result of extreme ankle dorsiflexion. Occurring in otherwise healthy adults, it is a disease of the third to fifth decades and has a male predominance. Rupture of the Achilles tendon most often occurs in the left leg because right-handed individuals usually push off with the left leg when they jump.

The Achilles tendon is most susceptible to rupture at its narrowest part, a point approximately 5 cm above its insertion. The Achilles tendon is subjected to repetitive motion that may result in microtrauma, which heals poorly owing to the tendon's avascular nature. Repeated microtrauma leads to tendinitis and tendinopathy that may predispose the tendon to rupture. Achilles tendinitis frequently coexists with bursitis, which causes additional pain and functional disability.

In addition to traumatic rupture of the Achilles tendon, sudden, nontraumatic rupture may occur. Factors that predispose the patient to traumatic and nontraumatic rupture of the Achilles tendon include steroid use, dialysis, gout, rheumatoid arthritis, systemic lupus erythematosus, diabetes, endocrinopathies, renal transplant, hyperlipidemias, and the use of fluoroquinolones (Box 139.1).

SIGNS AND SYMPTOMS

The onset of Achilles tendon rupture is usually acute, occurring after acute push-off during jumping or sprinting as the result of extreme ankle dorsiflexion. Improper stretching of the gastrocnemius and Achilles tendon before exercise has also been implicated in the development of Achilles tendinitis and acute tendon rupture. The pain of Achilles tendon rupture is constant and severe and is localized in the posterior ankle. The patient often complains of a feeling like being kicked in the ankle. Significant ecchymosis, swelling, and hematoma are frequently present. Palpation of the ruptured Achilles tendon may reveal a lack of tendon continuity. The patient suffering from Achilles tendon rupture exhibits positive results of the toe raise and Thompson squeeze tests (Fig. 139.1). The knee flexion test can also help identify a ruptured Achilles tendon (Fig. 139.2).

BOX 139.1 Factors Associated With Rupture of the Achilles Tendon

- Steroid use
- Dialysis
- Gout
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Diabetes
- Endocrinopathies
- Renal transplantation
- Hyperlipidemias
- Fluoroquinolone use

TESTING

Plain radiographs, ultrasound imaging, and magnetic resonance imaging (MRI) are indicated in all patients who present with posterior ankle pain and who are suspected of suffering from Achilles tendon rupture (Figs. 139.3–139.6). MRI of the ankle is also indicated if joint instability, bursitis, or occult tumor are suspected. Radionuclide bone scanning is useful to identify stress fractures not seen on plain radiographs. Ultrasound imaging may also help assess the integrity of the Achilles tendon (Fig. 139.7). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, comprehensive metabolic panel, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Achilles tendon rupture is usually easily identified on clinical grounds. However, if the bursa located between the Achilles tendon and the base of the tibia and the upper posterior calcaneus is inflamed, coexistent bursitis may confuse the diagnosis. Stress fractures of the ankle may also mimic the pain of Achilles tendon rupture (Fig. 139.8).

TREATMENT

Initial treatment of the pain and functional disability associated with Achilles tendon rupture includes elevation, relative rest, and ice. A combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and short-acting opioid analgesics, such as hydrocodone, may be necessary

FIG 139.1 A, To perform the toe raise test for Achilles tendon rupture, the patient is asked to stand in a comfortable position and then raise himself or herself on tiptoe. B, To perform the Thompson squeeze test for Achilles tendon rupture, the examiner grasps the calf on the patient's affected side just below the point of the calf's maximum girth and firmly squeezes the calf. The absence of plantar flexion on the affected side provides a presumptive diagnosis of rupture of the Achilles tendon. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. 2nd ed. Philadelphia: Saunders; 2010:344–346.)

FIG 139.2 The knee flexion test is performed with the patient prone and ankles extending past the edge of the table. The patient is asked to actively flex the knee to 90 degrees. During this movement, the foot on the affected side falls into neutral or dorsiflexion, and a rupture of the Achilles tendon can be diagnosed.

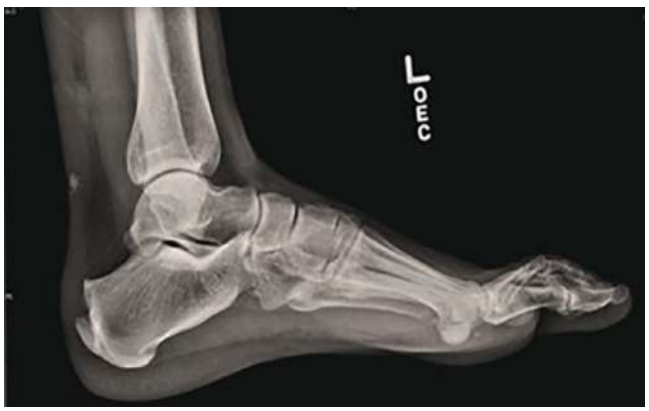


FIG 139.3 Lateral radiograph showing calcification within the Achilles tendon and at the insertion. Note the lack of equinus in this non–weight bearing view. (From Saxena A, Hofer D. Triple Achilles tendon rupture: case report. *J Foot Ankle Surg*. 2018;57(2):404–408, Fig. 1.)

to manage the acute pain associated with this condition. Although some specialists recommend conservative therapy, most believe that surgical repair of the tendon with postoperative immobilization is the best option in otherwise healthy patients (Fig. 139.9). Clinical experience suggests that the injection of platelet-rich plasma and/or stem cells may improve tendon healing.

COMPLICATIONS AND PITFALLS

Repeat rupture of the affected Achilles tendon represents a real risk whether conservative or operative treatment is pursued. Care must be taken to avoid immobilization with casts until the acute swelling associated with tendon rupture is resolved or nerve compression and pressure ulcers may result. Gentle physical therapy during the healing phase is essential if functional ability is to be maintained.

CLINICAL PEARLS

Although the Achilles tendon is the thickest and strongest tendon in the body, it is susceptible to inflammation or even rupture. It begins at the midcalf and continues downward, narrowing as it goes, to attach to the posterior calcaneus; it becomes narrowest approximately 5 cm above its calcaneal insertion. Coexistent bursitis and arthritis may contribute to the patient's symptoms, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone.

FIG 139.4 A magnetic resonance image with *arrows* pointing to the ends of the Achilles tendon ends. The area between the *arrows* represents the gap between the tendon ends. (From Padanilam TG. Chronic Achilles tendon ruptures. *Foot Ankle Clin*. 2009;14(4):711–728.)

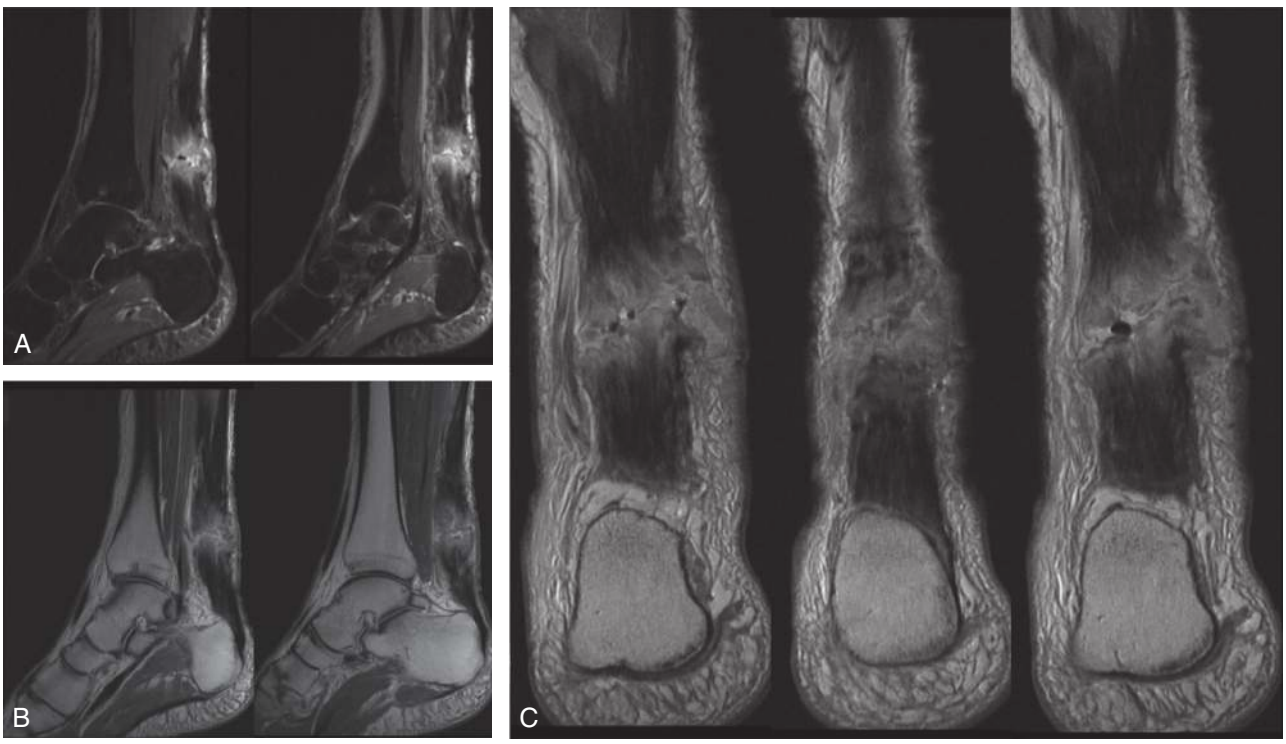
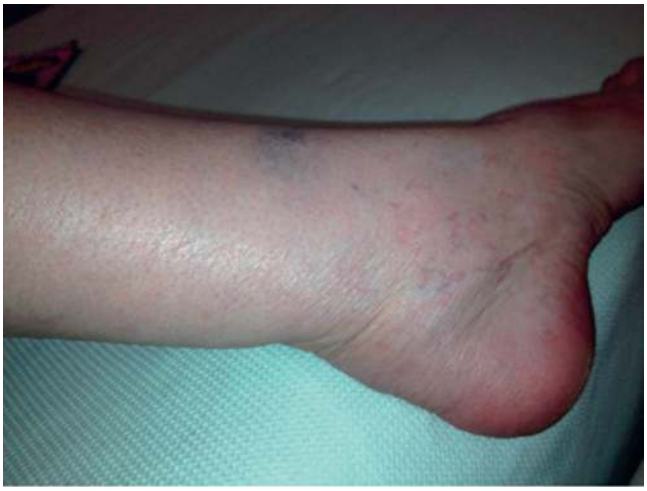
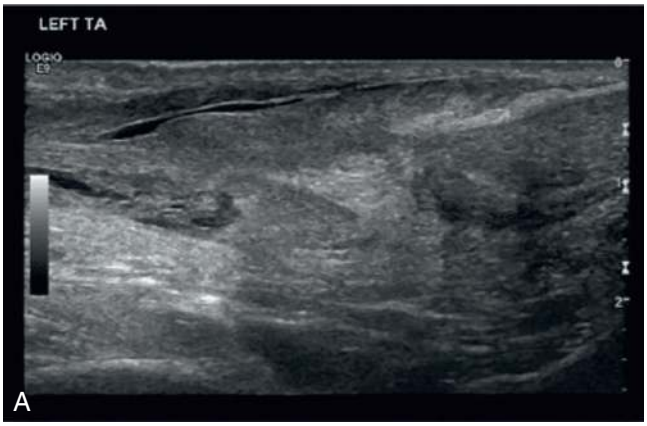


FIG 139.5 Magnetic resonance scans of the right lower leg of the patient (postcontrast fat-suppressed T2-weighted sagittal views in) **(A),** T1-weighted sagittal views in **(B),** and T1-weighted coronal views in **(C)** demonstrating a complete tendon discontinuity 5.5 cm proximally to the insertion with a 1.3-cm gap, and extensive postcontrast peripheral enhancement of soft tissues around the rupture site, consistent with deep infection. (From de Cesar Netto C, Bernasconi A, Roberts L, et al. Open re-rupture of the Achilles tendon following minimally invasive repair: a case report. *J Foot Ankle Surg*. 2018;57(6):1272–1277, Fig. 3.)





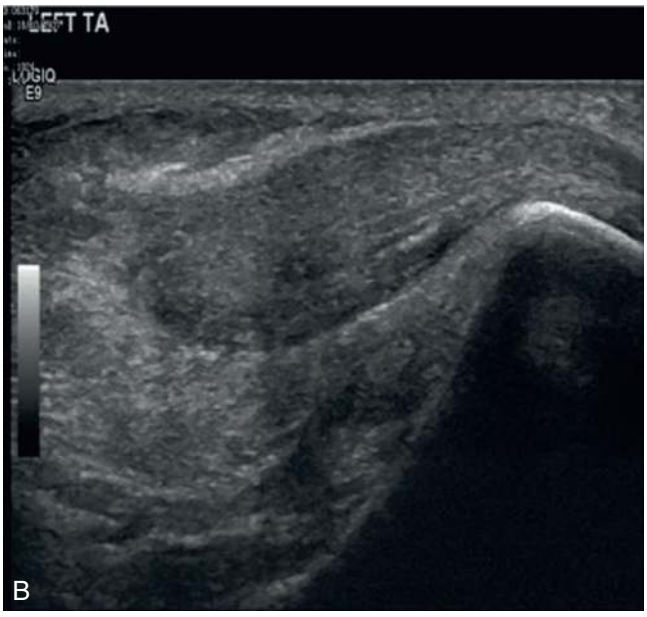
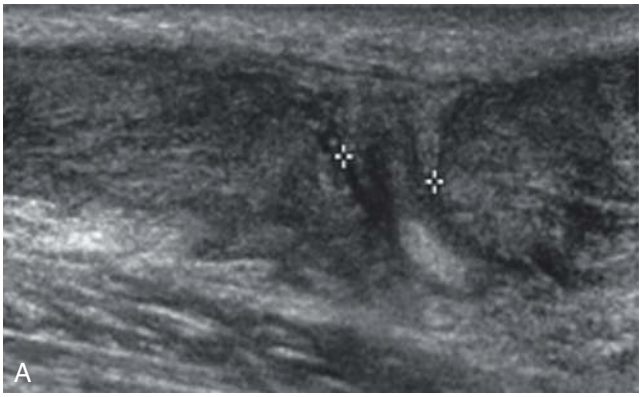


FIG 139.6 A, Photograph of ankle in patient with complete rupture of the Achilles tendon. Note the obvious defect in the tendon. B, Longitudinal ultrasound images demonstrating complete rupture of the Achilles tendon. (From AL-Saadi S, Michael A. Levofloxacin-induced Achilles tendinitis and tendon rupture. *Eur Geriatr Med*. 2012;3(6):380–381, Fig. 1.)



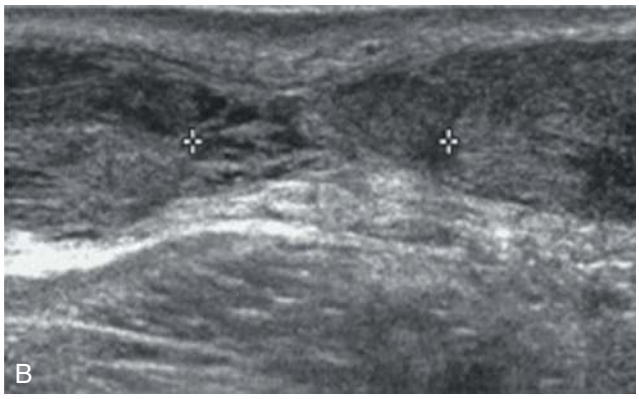


FIG 139.7 A, Longitudinal ultrasound scan of a ruptured Achilles tendon during plantar flexion shows less than 1 cm of separation between the torn tendon ends. The patient was successfully treated with casting in plantar flexion. B, Same patient with longitudinal scanning in dorsiflexion shows increased separation and better delineation of the complete tendon rupture. (From Fessell DP, Jacobson JA. Ultrasound of the hindfoot and midfoot. *Radiol Clin North Am*. 2008;46(6):1027–1043.)

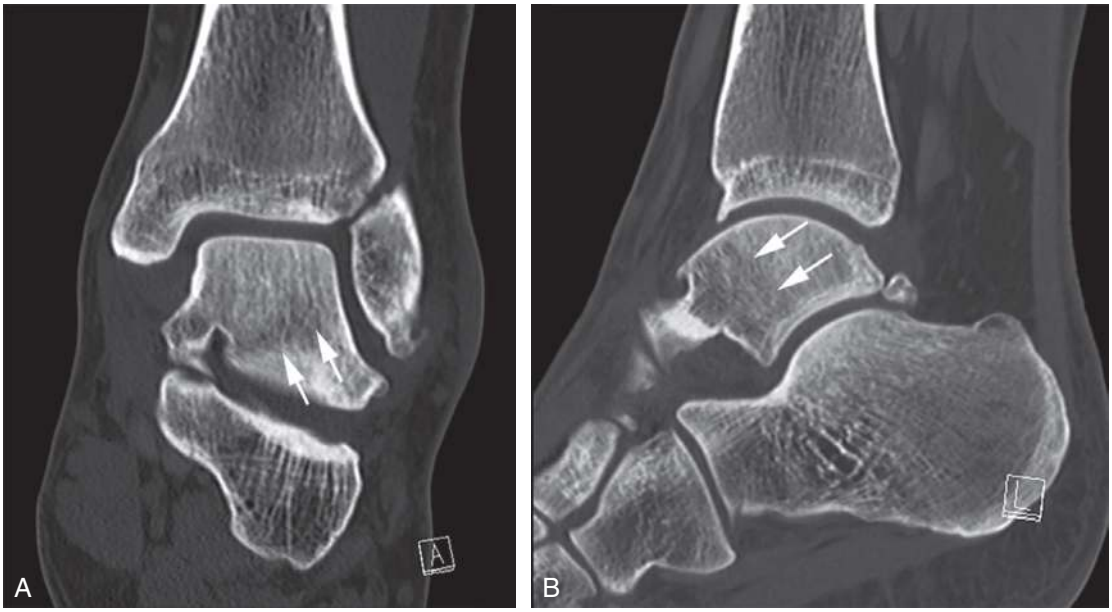


FIG 139.8 Stress fractures of the ankle can mimic the pain of Achilles tendon rupture. A, B, Two-dimensional reconstruction computed tomography image showing a vertical radiolucent line (*arrow*) at the lateral region of the talar body. (From Kim YS, Lee HM, Kim JP, et al. Fatigue stress fracture of the talar body: an uncommon cause of ankle pain. *J Foot Ankle Surg.* 2016;55(5):1113–1116, Fig. 4.)

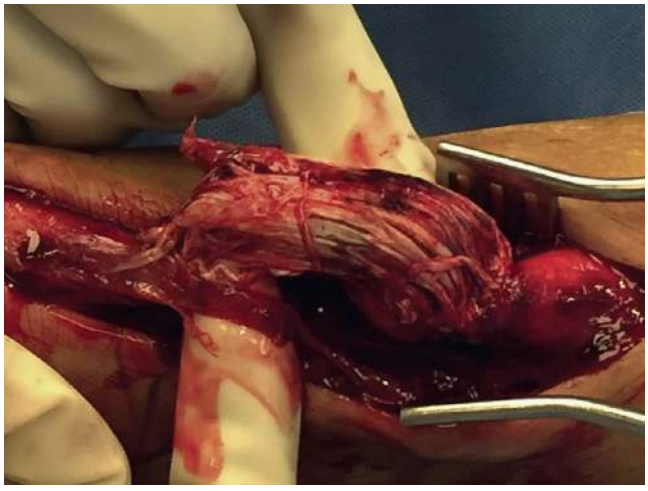


FIG 139.9 Intraoperative view showing the watershed region ruptures. Calcification within the main body was palpable. (From Saxena A, Hofer D. Triple Achilles tendon rupture: case report. *J Foot Ankle Surg.* 2018;57(2):404–408, Fig. 3.)

SUGGESTED READINGS

AL-Saadi S, Michael A. Levofloxacin-induced Achilles tendinitis and tendon rupture. *Eur Geriatr Med*. 2012;3(6):380–381.

Maffulli N, Via AG, Oliva F. Chronic Achilles tendon disorders: tendinopathy and chronic rupture. *Clin Sports Med*. 2015;34(4):607–624.

Malagelada F, Clark C, Dega R. Management of chronic Achilles tendon ruptures—a review. *Foot*. 2016;28:54–60.

Ribbans WJ, Henman PD, Bliss WH. Achilles tendon ruptures in teenagers involved in elite gymnastics. *Sports Orthop Traumatol*. 2016;32(4):375–379.

Tenforde AS, Yin A, Hunt KJ. Foot and ankle injuries in runners. *Phys Med Rehabil Clin N Am*. 2016;27(1):121–137.

Waldman SD. Achilles tendinitis and other abnormalities of the Achilles tendon. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:986–987.

Waldman SD, Campbell RSD. Achilles tendon rupture. In: *Imaging of pain*. Philadelphia: Saunders; 2011:431–432.

Weinfeld SB. Achilles tendon disorders. *Med Clin North Am*. 2014;98(2):331–338.

140

Arthritis Pain of the Toes

ICD-10 CODE M19.90

THE CLINICAL SYNDROME

The toe joint is susceptible to the development of arthritis from various conditions that can damage the joint cartilage. Osteoarthritis is the most common form of arthritis that results in toe joint pain; rheumatoid arthritis and posttraumatic arthritis are also frequent causes of toe pain (Fig. 140.1 and Box 140.1). Less common causes include the collagen vascular diseases, infection, and Lyme disease. Acute infectious arthritis is usually accompanied by significant systemic symptoms, including fever and malaise, and should be easily recognized; it is treated with culture and antibiotics rather than injection therapy. Collagen vascular disease generally manifests as polyarthropathy rather than as monoarthropathy limited to the toe joint, although toe pain secondary to collagen vascular disease responds exceedingly well to the intraarticular injection technique described later. Gout often affects the first interphalangeal joint of the foot and the clinical syndrome is known as podagra (Fig. 140.2 and Box 140.2).

SIGNS AND SYMPTOMS

Most patients present with pain localized to the affected joint of the foot, most commonly the great toe. Activity, especially flexion of the toe joints, makes the pain worse (Fig. 140.3),

BOX 140.1 Metabolic Causes of Osteoarthritis

- Acromegaly
- Hyperparathyroidism
- Hypothyroidism
- Diabetes mellitus (may relate to obesity)
- Haemochromatosis
- Wilson's disease
- Gaucher's disease
- Ochronosis (alkaptonuria)
- Kashin–Beck disease
- Hemoglobinopathies/avascular necrosis

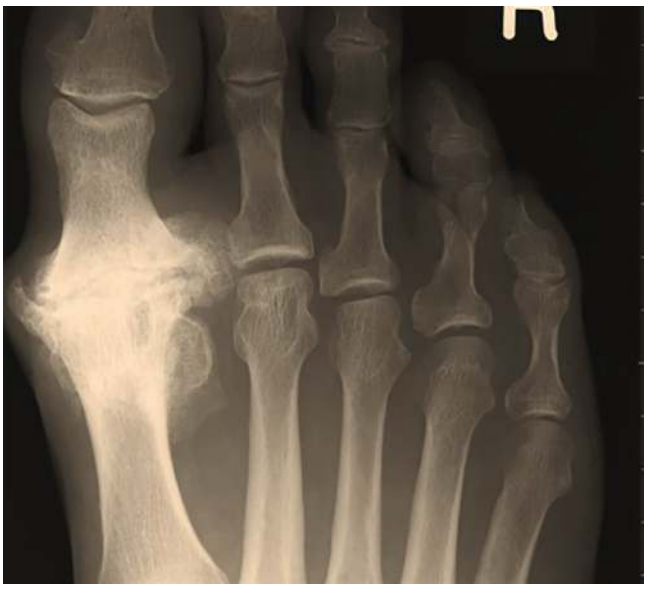


FIG 140.1 Osteoarthritis of the great toe. Note joint narrowing of the metatarsophalangeal joint of the great toe.

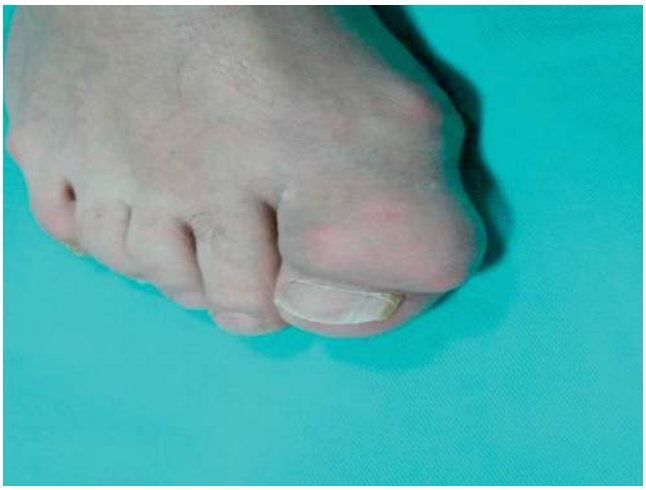


FIG 140.2 Podagra. Swelling over dorsomedial aspect of first interphalangeal joint of right foot in a patient with acute gouty arthritis. (Reprinted with permission from Elsevier, Dalbeth N, Merriman TR, Stamp LK, Gout. *Lancet*. 2016;388(10055):2039–2052.)

BOX 140.2 Risk Factors for the Development of Gout

Genetic
- Male sex
- Ancestry
- *SLC2A9*
- *ABCG2*
- *SLC17A1/SLC17A3*
- *GCKR*

Drugs
- Diuretics
- Cyclosporin
- Tacrolimus
- Angiotensin-converting enzyme inhibitors
- Nonlosartan angiotensin II receptor blockers
- β-Blockers
- Pyrazinamide
- Ritonavir

Dietary
- Red meat
- Seafood
- Beer
- Spirits
- Sugar-sweetened beverages

Other
- Increasing age
- Menopause
- Chronic kidney disease
- Overweight, obesity, or weight gain
- Hypertension
- Hyperlipidemia
- Hypertriglyceridemia
- Congestive cardiac failure
- Obstructive sleep apnea
- Anemia
- Psoriasis
- Sickle cell anemia
- Hematological malignancy
- Lead exposure

Reprinted with permission from Elsevier, Dalbeth N, Merriman TR, Stamp LK, Gout, *Lancet*. 2016;388(10055):2039–2052.

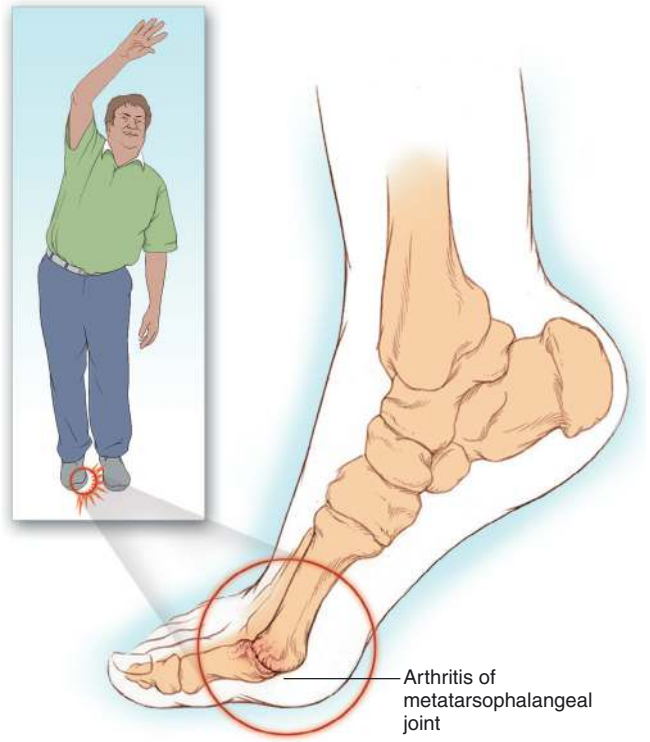


FIG 140.3 Arthritis of the toe manifests as pain that is made worse with weight-bearing activity.

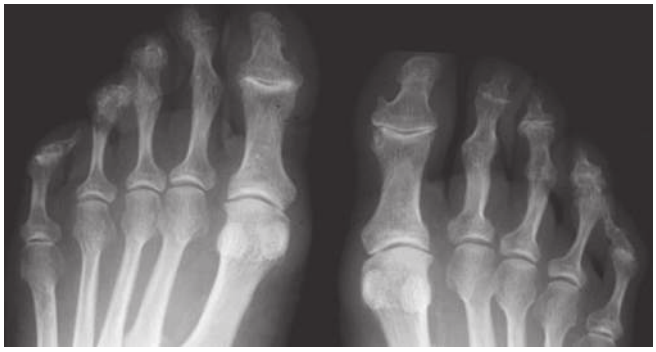


FIG 140.4 Feet radiographs showing joint narrowing, bone proliferation, and ankylosis. (From Mas AJ, Rotés-Querol J. Erosive osteoarthritis of the feet: description of two patients. *Jt Bone Spine*. 2007;74(3):296–298.)

whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with the use of the joint, and crepitus may be present on physical examination. In addition to pain, patients often experience a gradual decrease in functional ability because of reduced toe range of motion that makes simple everyday tasks such as walking, standing on tiptoes, and climbing stairs quite difficult.

TESTING

Plain radiographs are indicated in all patients who present with toe joint pain (Figs. 140.4–140.6). Magnetic resonance imaging, computerized tomography, and ultrasound imaging of the toe are indicated if joint instability, an occult mass, or a tumor are suspected (Figs. 140.7–140.10). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Bursitis and tendinitis of the foot, as well as entrapment neuropathies such as tarsal tunnel syndrome, may confuse the

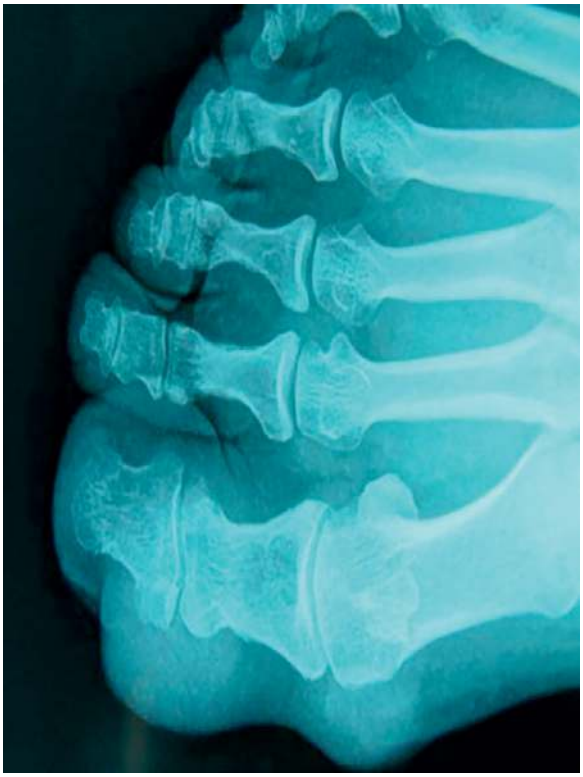


FIG 140.5 Podagra. Plain radiography of right foot showing joint effusion, and soft tissue swelling around first interphalangeal joint. (From Alici T, Imren Y, Erdil M, Gundes H. Gouty arthritis at interphalangeal joint of foot after sildenafil use: A case report. *Int J Surg Case Rep*. 2013;4(1):11–4. doi: 10.1016/j.ijscr.2012.08.014. Epub 2012 Sep 28. PMID: 23088905; PMCID: PMC3537936.)

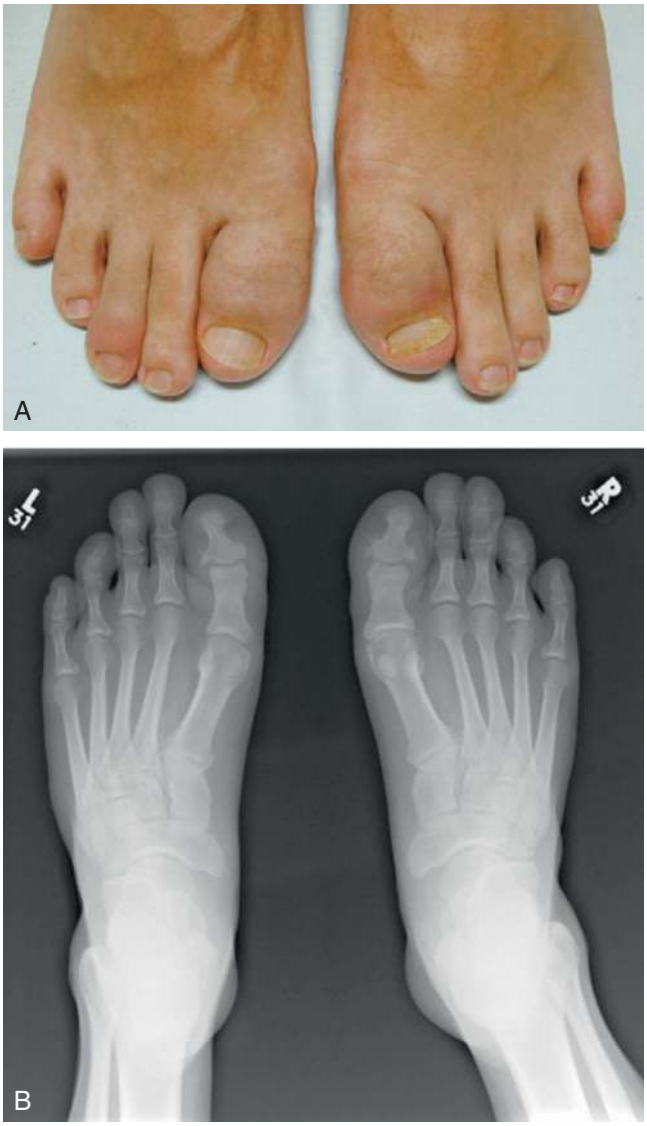


FIG 140.6 **A,** Patient with psoriatic arthritis. Clinical photograph showing dactylitis of both big toes and swelling and redness over the third right toe distal interphalangeal joint. **B,** Case 1, both feet, radiographs demonstrating erosions and mild periosteal reaction. (From Gladman DD. Management of psoriatic arthritis. In: Weisman MH, Weinblatt ME, Louie JS, et al., eds. *Targeted treatment of the rheumatic diseases*. Philadelphia: Saunders; 2010:55–69.)

diagnosis; these conditions may coexist with arthritis of the toes. Primary and metastatic tumors of the foot, occult fractures of the tarsals and metatarsals, and fractures of the sesamoid bones of the foot may manifest in a manner similar to arthritis of the toes.

TREATMENT

Initial treatment of the pain and functional disability associated with arthritis of the toes includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, as well as short-term immobilization of the toe joints, may provide relief. For patients who do not respond to these treatment modalities, intraarticular injection with local anesthetic and steroid is a reasonable next step.

To perform intraarticular injection of the toes, the patient is placed in the supine position, and the skin overlying the affected toe joint is prepared with antiseptic solution. A sterile syringe containing 1.5 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a ⅝-inch, 25-gauge needle using strict aseptic technique. The affected toe is distracted to open the joint space, which is identified. At this point, the needle is carefully advanced perpendicular to the joint space next to the extensor tendons through the skin, subcutaneous tissues, and joint capsule and into the joint. If bone is encountered, the needle is withdrawn into the subcutaneous tissues and is redirected superiorly. Once the needle is in the joint space, the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced slightly into the joint space until the injection can proceed without significant

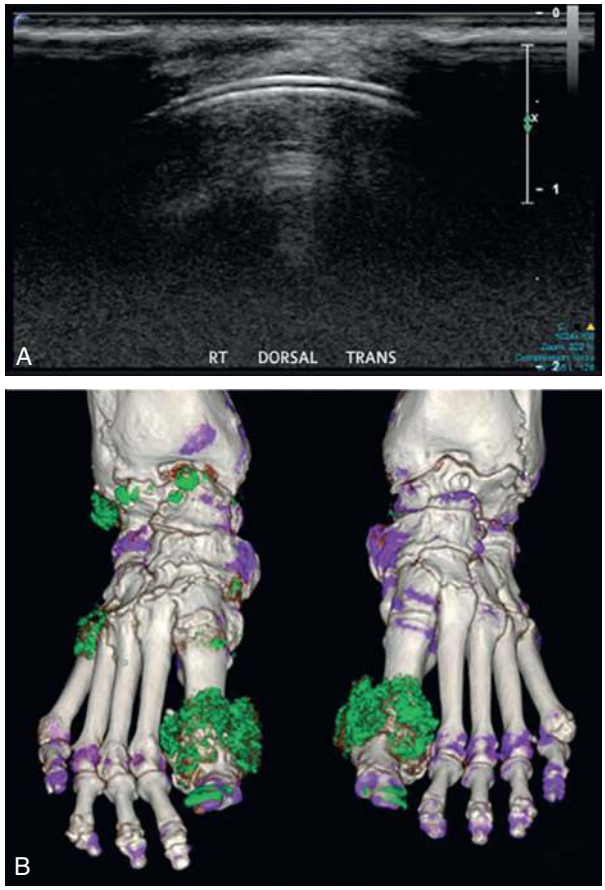


FIG 140.7 Crystal deposition involving the joints of the feet. Imaging features of monosodium urate crystal deposition. **(A)** The top image shows the double contour sign in the first metatarsophalangeal joint on ultrasonography (transverse view of the dorsal surface of the joint), defined as hyperechoic enhancement over the surface of the hyaline cartilage. **(B)** The bottom image shows a dual-energy CT of a patient with tophaceous gout. Urate deposition (color coded in green) can be seen at characteristic sites including the first metatarsophalangeal joint, midfoot, and ankle. Green signal at the nails of the big toes is an artifact commonly observed at this site. (Reprinted with permission from Elsevier, Dalbeth N, Merriman TR, Stamp LK, Gout. *Lancet*. 2016;388(10055):2039–2052.)

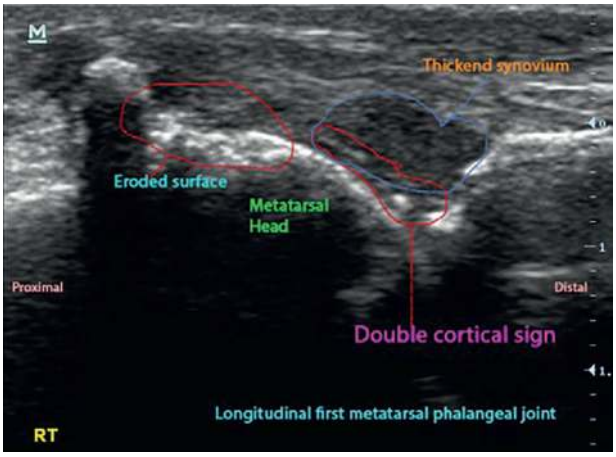


FIG 140.8 Ultrasound image of the metatarsophalangeal joint of the first toe in a patient with gouty arthritis. Note the double cortical sign which is highly suggestive for crystal deposition disease.

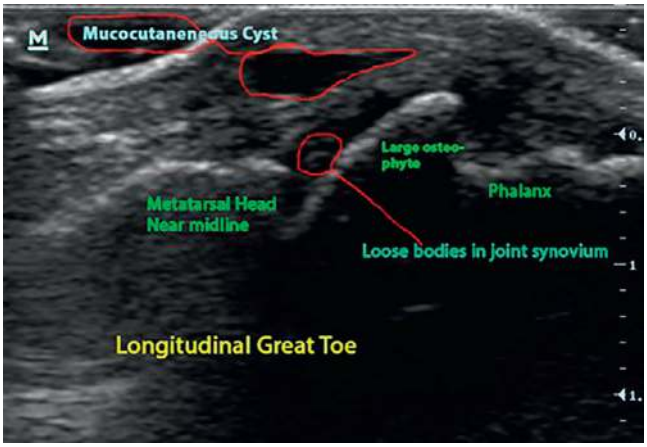


FIG 140.9 Longitudinal ultrasound image demonstrating joint mice in the metatarsophalangeal joint of the great toe.

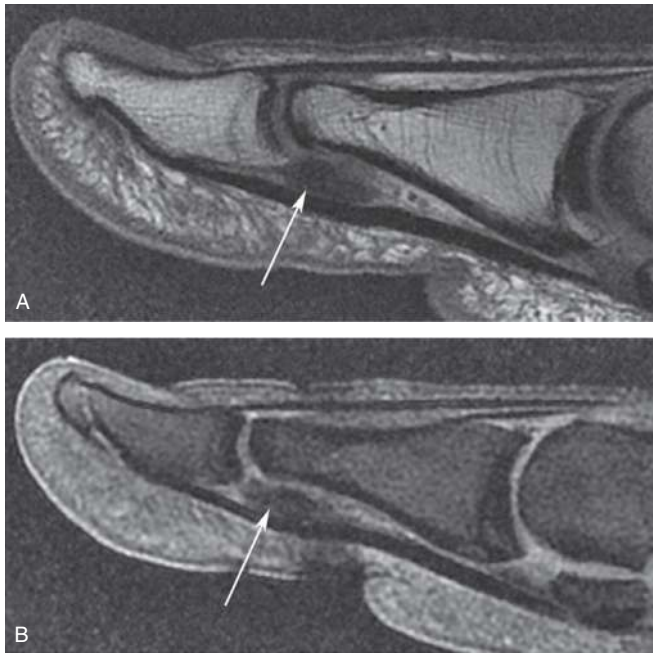


FIG 140.10 Turf toe. A 23-year-old man with first interphalangeal plantar plate injury. Sagittal T1W **(A)** and proton density fat suppression **(B)** images show thickening and indistinctness of the first interphalangeal plantar plate (*arrow*). (From Schein AJ, Skalski MR, Patel DB, et al. Turf toe and sesamoiditis: what the radiologist needs to know. *Clin Imaging*. 2015;39(3):380–389.)

resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications (Fig. 140.11). The injection of platelet-rich plasma and/or stem cells may reduce the pain and functional disability of arthritis of the toes.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms.

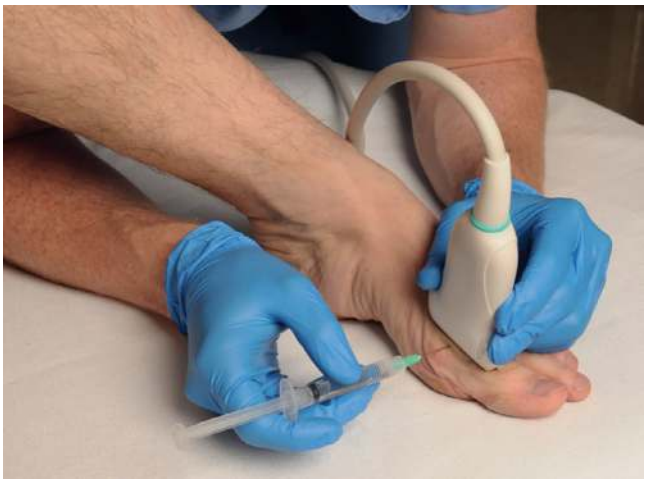


FIG 140.11 Proper needle position for ultrasound-guided out-of-plane injection of the metatarsophalangeal joint.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of arthritis of the toe joints.

SUGGESTED READINGS

Alici T, Imren Y, Erdil M, et al. Gouty arthritis at interphalangeal joint of foot after sildenafil use: a case report. *Int J Surg Case Rep*. 2013;4(1):11–14.

Schein AJ, Skalski MR, Patel DB, et al. Turf toe and sesamoiditis: what the radiologist needs to know. *Clin Imaging*. 2015;39(3):380–389.

Waldman SD. Arthritis and other abnormalities of the metatarsophalangeal and interphalangeal joints. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1030–1042.

Waldman SD. Intra-articular injection of the interphalangeal joints of the toes. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:794–797.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

141

Bunion Pain

ICD-10 CODE M20.10

THE CLINICAL SYNDROME

Bunion, also known as hallux valgus, is one of the most common causes of foot pain. The term *bunion* refers to soft tissue swelling over the first metatarsophalangeal joint associated with abnormal angulation of the joint that results in a prominent first metatarsal head and overlapping of the first and second toes, called the hallux valgus deformity. The first metatarsophalangeal joint may ultimately subluxate and cause the overlapping of the first and second toes to worsen (Fig. 141.1). An inflamed adventitious bursa may accompany bunion formation (Fig. 141.2). The most common cause of bunions is the wearing of narrow-toed shoes, and high heels may exacerbate the problem (Fig. 141.3); thus bunions are more common in women.

SIGNS AND SYMPTOMS

Most patients present with pain localized to the affected first metatarsophalangeal joint and complain of being unable to get shoes to fit. Walking makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with the use of the joint, and crepitus may be present on physical examination. In addition to pain, patients with bunions develop the characteristic hallux valgus deformity, with a prominent first metatarsal head, improper angulation of the joint, and overlapping first and second toes.

TESTING

Plain radiographs are indicated in all patients who present with bunion pain (Figs. 141.4 and 141.5). Magnetic resonance imaging and ultrasound imaging of the toe are indicated if joint instability, an occult mass, or a tumor is suspected (Fig. 141.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

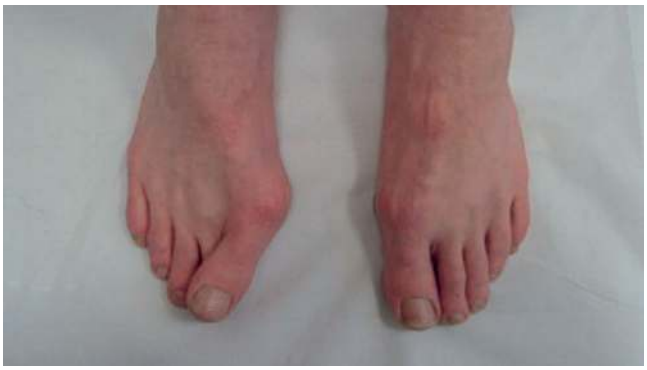


FIG 141.1 Hallux valgus deformity. (From Johal S, Sawalha S, Pasapula C. Post-traumatic acute hallux valgus: a case report. *Foot (Edinb)*. 2010;20(2–3):87–89.)

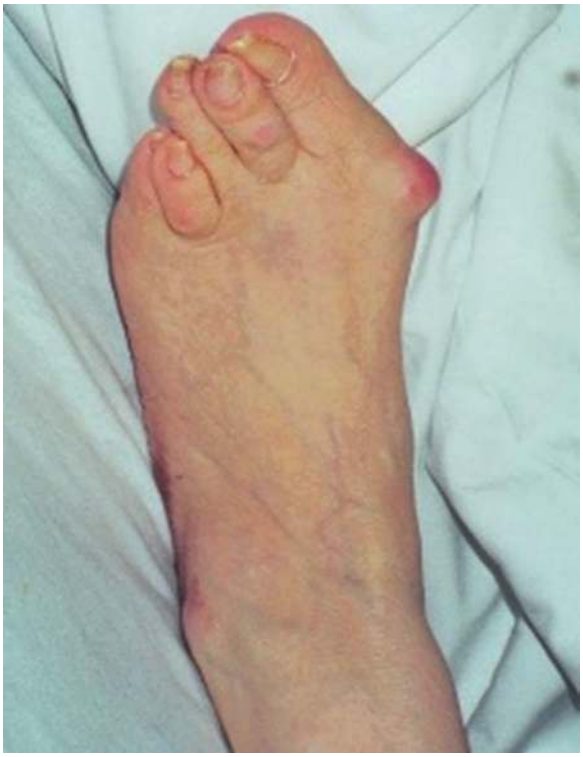


FIG 141.2 An inflamed adventitial bursa frequently accompanies the pain and functional disability associated with bunion. (From Waldman SD. *Chapter 9, in ankle and foot: pain medicine*. Philadelphia: Elsevier, 2023:128–140.)

DIFFERENTIAL DIAGNOSIS

The diagnosis of bunion is usually obvious on clinical grounds alone. Bursitis and tendinitis of the foot and ankle often coexist with bunion pain. In addition, stress fractures of the metatarsals, phalanges, or sesamoid bones may confuse the diagnosis and require specific treatment (Box 141.1). Joplin's neuroma, which is perineural fibrosis of the medial plantar digital nerve, is sometimes seen following bunion surgery and may confuse the clinical picture (Fig. 141.7).

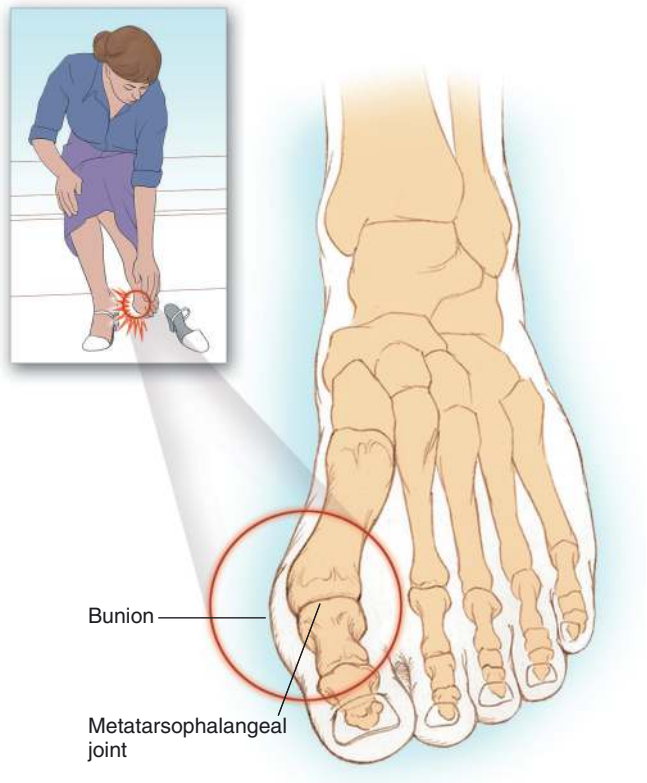


FIG 141.3 Narrow-toed shoes are implicated in the development of bunions.

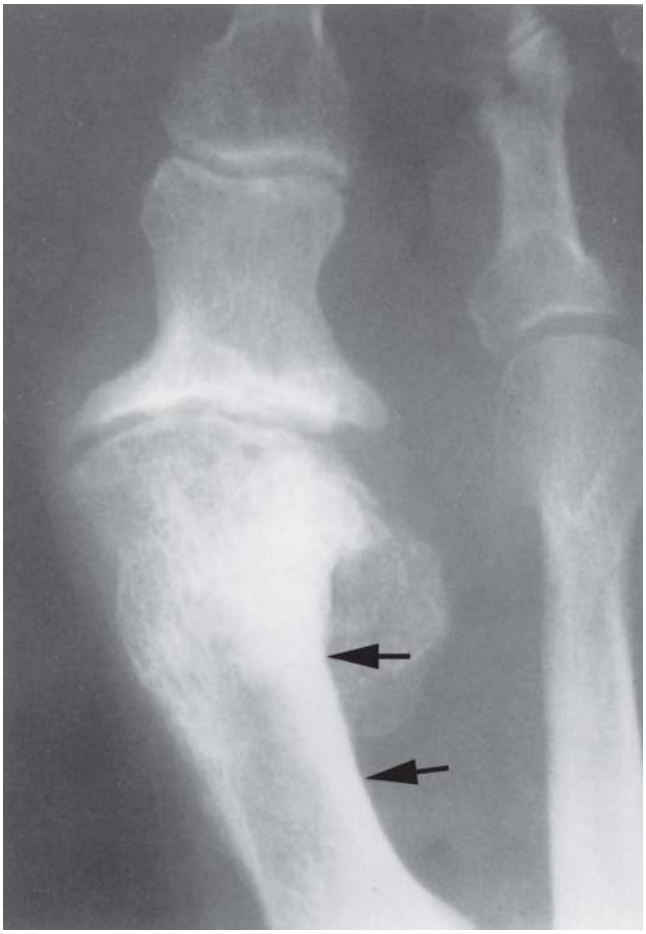


FIG 141.4 Osteoarthritis of the first metatarsophalangeal joint in a patient with hallux valgus deformity. The sesamoids are lateral to the metatarsal head. The radiograph shows narrowing of the joint space, with subchondral bone and osteophyte formation. Marked thickening of the lateral cortex of the metatarsal shaft (*arrows*) is evident. (From Brower AC, Flemming DJ. *Arthritis in black and white*. 2nd ed. Philadelphia: Saunders; 1997.)

BOX 141.1 Differential Diagnosis of Hallux Valgus

- Ganglion cyst
- Inflamed bursa
- Synovitis
- Fracture callus
- Exuberant synovium
- Fibroma
- Giant cell tumor
- Aneurysmal bone cyst
- Unicameral bone cyst
- Lipoma
- Neural tumors
- Interosseous ganglions
- Osteoid osteoma
- Osteochondroma
- Osteosarcoma
- Metastatic disease

From Waldman SD. *Chapter 9, in ankle and foot: pain medicine*. Philadelphia: Elsevier, 2023:128–140.

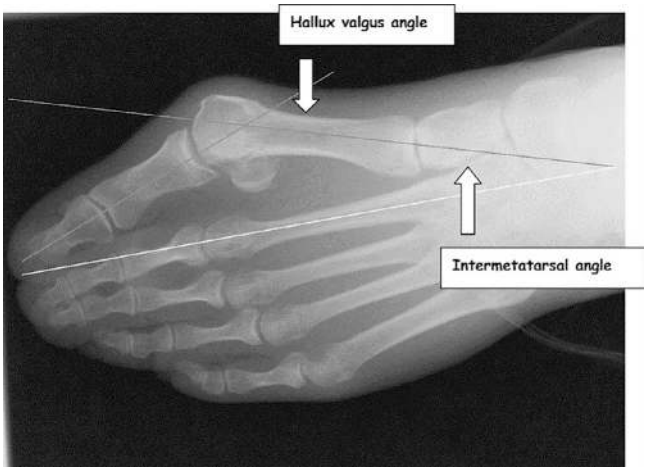


FIG 141.5 Radiographic assessment of hallux valgus (bunion). (From Thomas S, Barrington R. Hallux valgus. *Curr Orthop*. 2003;17(4):299–307.)

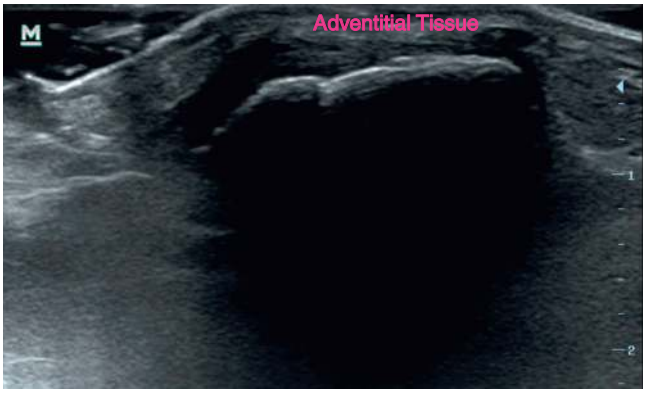


FIG 141.6 Longitudinal ultrasound view of hallux valgus. Note the significant adventitial tissue over the lateral great toe.

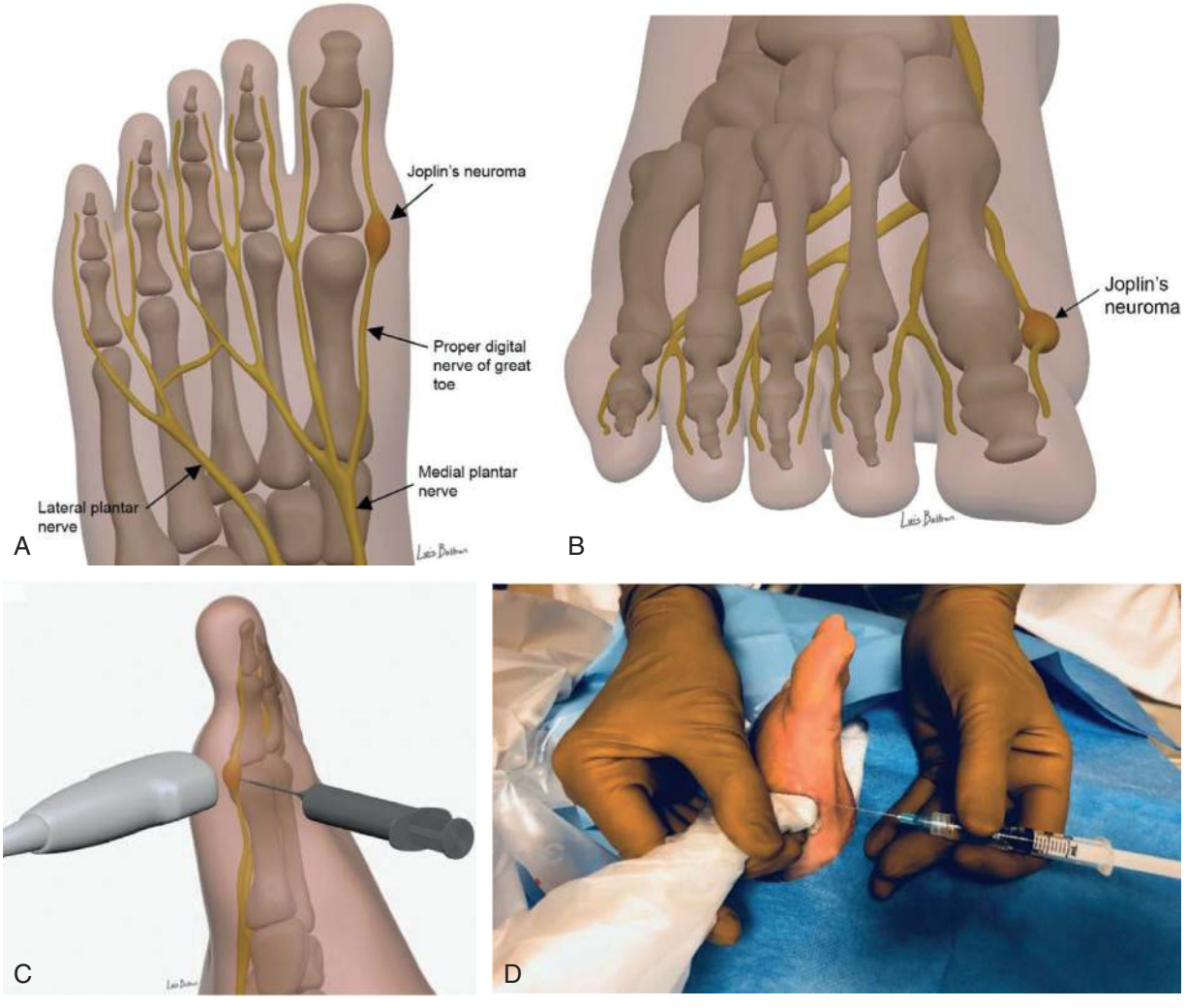


FIG 141.7 Joplin's neuroma. **A–C,** Schematic images demonstrating the anatomic course of medial plantar proper digital nerve (MPPDN) of the great toe and associated Joplin's neuroma. The MPPDN is the terminal medial branch arising from the medial plantar nerve, passing medially along the hallux where a Joplin's neuroma can form. **D,** Photograph demonstrating the needle trajectory utilized to inject Joplin's nerve. (From Burke CJ, Sanchez J, Walter WR, et al. Ultrasound-guided therapeutic injection and cryoablation of the medial plantar proper digital nerve (Joplin's nerve): sonographic findings, technique, and clinical outcomes. *Acad Radiol*. 2020;27(4):518–527.)

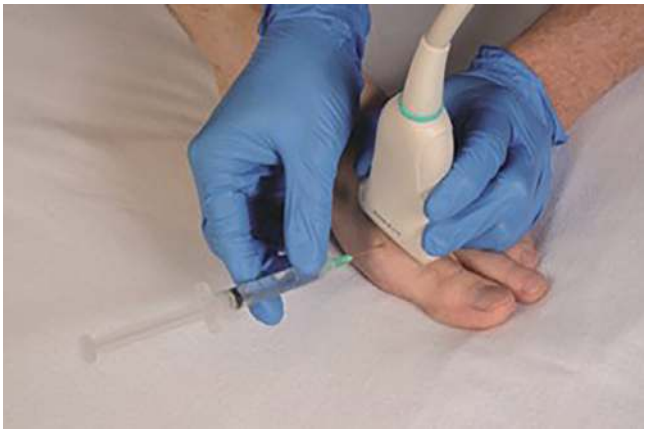


FIG 141.8 Proper needle position for ultrasound-guided out-of-plane injection of the metatarsophalangeal joint.

TREATMENT

Initial treatment of the pain and functional disability associated with bunion includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of narrow-toed or high-heeled shoes, and short-term immobilization of the affected toes may also provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To inject the bunion deformity, the patient is placed in the supine position, and the skin overlying the bunion is prepared with antiseptic solution. A sterile syringe containing 1.5 mL of 0.25% preservative-free bupivacaine and 40-mg methylprednisolone is attached to a ⅝-inch, 25-gauge needle using strict aseptic technique. The bunion is identified, and the needle is carefully advanced against the first metatarsal head. The needle is then withdrawn slightly out of the periosteum, and the contents of the syringe are gently injected. Little resistance to injection should be felt. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications (Fig. 141.8).

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The injection technique is safe if careful attention is paid to the clinically relevant anatomy. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's foot pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating bunion pain. Narrow-toed and high-heeled shoes should be avoided, because they will exacerbate the patient's symptoms.

SUGGESTED READINGS

Dayton P, Kauwe M, Feilmeier M. Clarification of the anatomic definition of the bunion deformity. *J Foot Ankle Surg*. 2014;53(2):160–163.

Dayton P, Kauwe M, Feilmeier M. Is our current paradigm for evaluation and management of the bunion deformity flawed? A discussion of procedure philosophy relative to anatomy. *J Foot Ankle Surg*. 2015;54(1):102–111.

Mann R. Bunion deformity in elite athletes. In: Porter DA, Schon LC, eds. *Baxter's the foot and ankle in sport*. 2nd ed. St Louis: Mosby; 2008:435–443.

Melendez MM, Patel A, Lee Dellon A. The diagnosis and treatment of Joplin's neuroma. *J Foot Ankle Surg*. 2016;55(2):320–323.

Waldman SD. Arthritis and other abnormalities of the metatarsophalangeal and interphalangeal joints. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1043–1048.

Waldman SD. Hallux valgus deformity. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolthers Kluwer; 2016:1030–1042.

Waldman SD. Injection technique for bunion pain syndrome. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:885–888.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

142

Morton's Neuroma

ICD-10 CODE G57.60

THE CLINICAL SYNDROME

Morton's neuroma is one of the most common pain syndromes affecting the forefoot. It is characterized by tenderness and burning pain in the plantar surface of the forefoot, with painful paresthesias in the two affected toes. This pain syndrome is thought to be caused by perineural fibrosis of the interdigital nerves (Fig. 142.1). Although the nerves between the third and fourth toes are affected most commonly, the second and third toes and, rarely, the fourth and fifth toes can be affected as well (Fig. 142.2). Patients may feel like they are walking with a stone in the shoe. The pain of Morton's neuroma worsens with prolonged standing or walking for long distances and is exacerbated by poorly fitting or improperly padded shoes. As with bunion and hammer toe deformities, Morton's neuroma is associated with wearing tight, narrow-toed shoes.

SIGNS AND SYMPTOMS

On physical examination, pain can be reproduced by performing Mulder's maneuver: firmly squeezing the two metatarsal heads together with one hand while placing firm pressure on the interdigital space with the other (Fig. 142.3).

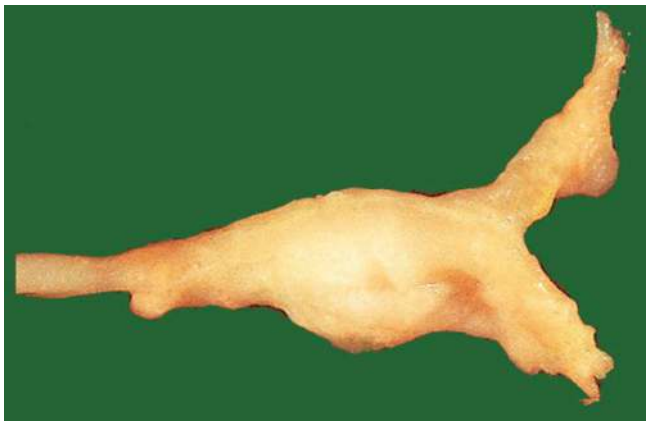


FIG 142.1 Morton's neuroma. Gross photograph of a segment of the plantar interdigital nerve resected from the space between the third and fourth metatarsal heads in a patient with Morton's neuroma shows fusiform swelling of the neurovascular bundle just proximal to the bifurcation. (From Benign soft tissue tumors. In: Bullough PG, ed. *Orthopaedic pathology*. 5th ed. Philadelphia: Mosby; 2010:497–532.)

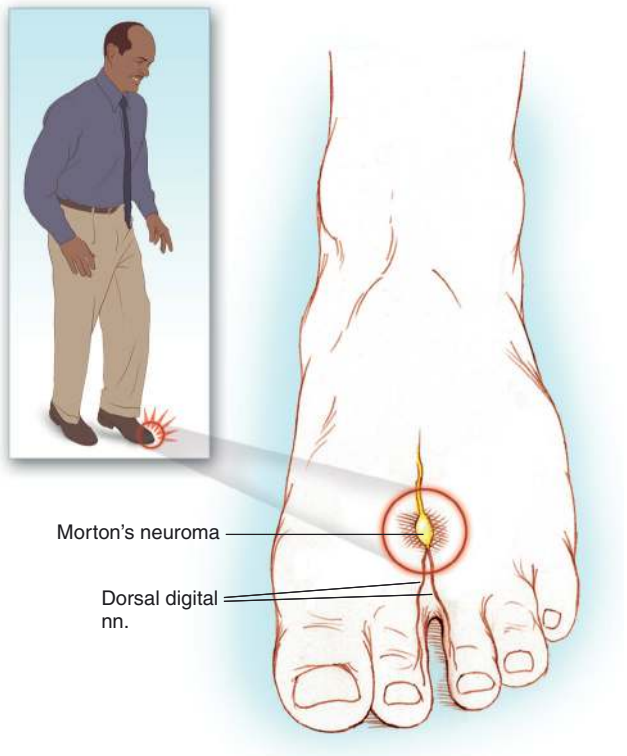


FIG 142.2 The pain of Morton's neuroma is made worse with prolonged standing or walking.

FIG 142.3 Eliciting Mulder sign for Morton neuroma. Mulder sign is elicited by firmly squeezing the metatarsal heads together with one hand while placing firm pressure on the interdigital space with the other. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:381.)

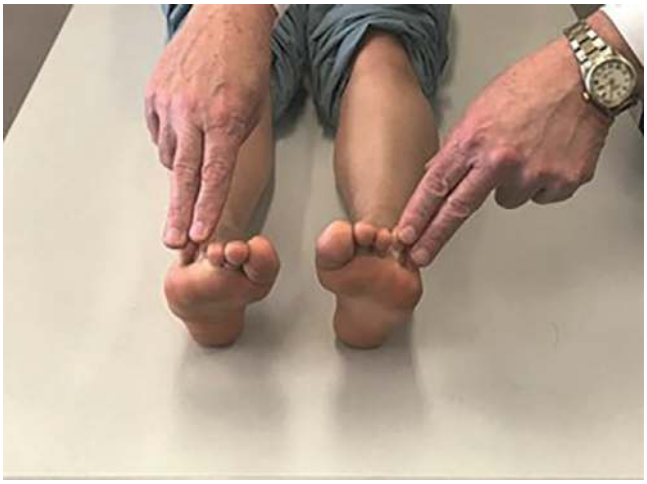


FIG 142.4 To perform the digital nerve stretch test for Morton neuroma, the patient is placed in the supine position. The patient is then asked to bring both ankles into full dorsiflexion. The examiner then fully extends the toes on each side of the webspace suspected of harboring the Morton neuroma. The test is positive if this maneuver reproduces the patient's symptoms.

The digital nerve stretch test may help confirm the diagnosis (Fig. 142.4). In contrast to metatarsalgia, in which the tender area is over the metatarsal heads, with Morton's neuroma, the tender area is localized to only the plantar surface of the affected interspace, with paresthesias radiating into the two affected toes. Patient may also exhibit a positive Tinel sign when the interdigital nerve is percussed from the plantar surface of the affected foot. Patients with Morton's neuroma often exhibit an antalgic gait in an effort to reduce weight bearing during walking.

TESTING

Plain radiographs, ultrasound imaging, computerized tomography, and magnetic resonance imaging (MRI) are indicated in all patients who present with Morton's neuroma, to rule out fractures and to identify sesamoid bones that may have become inflamed (Figs. 142.5 and 142.6). MRI of the metatarsal bones is also indicated if joint instability, an occult mass, or a tumor is suspected. Ultrasound imaging may also aid in the diagnosis of Morton's neuroma (Fig. 142.7). Radionuclide bone scanning may be useful to identify stress fractures of the metatarsal or sesamoid bones that may be missed on plain radiographs. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

Fractures of the sesamoid bones of the foot are often confused with Morton's neuroma; although the pain of sesamoid fracture is localized to the plantar surface of the foot, it is less

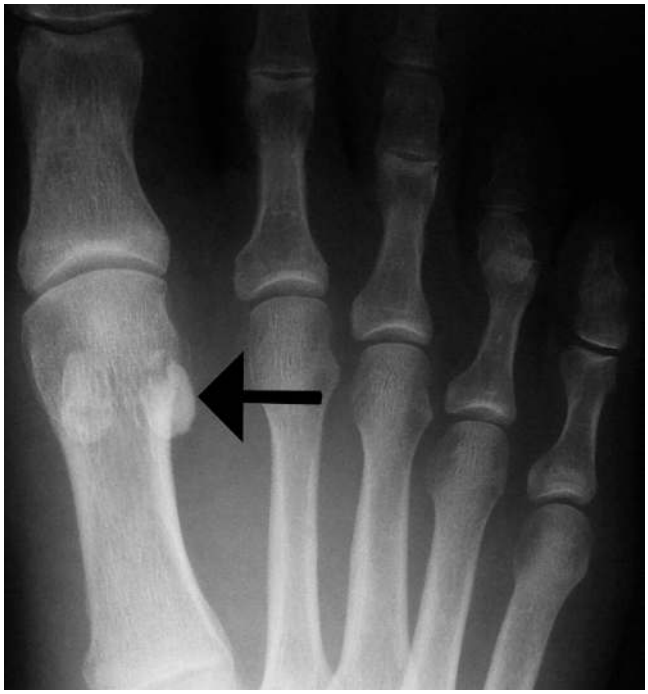


FIG 142.5 Anteroposterior radiographic view demonstrating irregular shape and sclerosis of the fibular sesamoid (*arrow*). (From Williams G, Kenyon P, Fischer B, et al. An atypical presentation of hallucial sesamoid avascular necrosis: a case report. *J Foot Ankle Surg.* 2009;48(2):203–207, Figure 1. ISSN 1067–2516.)

neuritic than is that of Morton's neuroma. Tendinitis, bursitis, and stress fractures of the foot can also mimic the pain of Morton's neuroma.

TREATMENT

Initial treatment of the pain and functional disability associated with Morton's neuroma includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of narrow-toed or high-heeled shoes, and short-term immobilization of the affected foot may also provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To inject Morton's neuroma, the patient is placed in the supine position with a pillow under the knee to slightly flex the leg. A total of 3 mL non–epinephrine-containing local anesthetic and 40 mg methylprednisolone is drawn up in a 12-mL sterile syringe. The affected interdigital space is identified, the dorsal surface of the foot at this point is marked with a sterile marker, and the skin is prepared with antiseptic solution. At a point proximal to the metatarsal head, a 1½-inch, 25-gauge needle is inserted between the two metatarsal bones in the area to be blocked (Fig. 142.8). While the clinician is slowly injecting, the needle is advanced from the dorsal

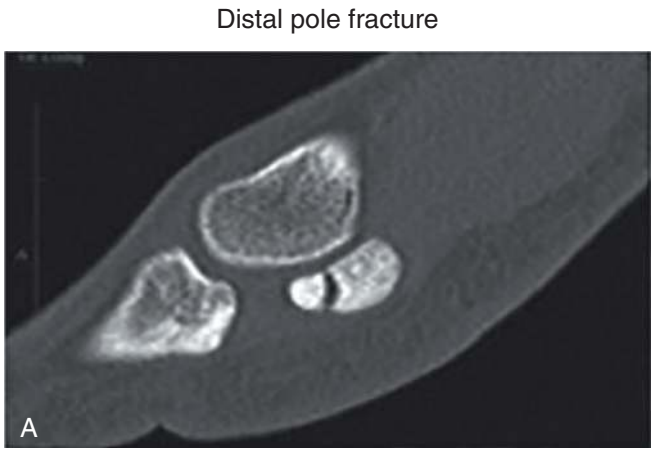


FIG 142.6 A–D, Computed tomography imaging of sesamoid fractures. (From Ribbans WJ, Hintermann B. Hallucal sesamoid fractures in athletes: diagnosis and treatment. *Sport Orthop Traumatol*. 2016;32(3): 295–303.)

surface of the foot toward the palmar surface. Because the plantar digital nerve is situated on the dorsal side of the flexor retinaculum, the needle must be advanced almost to the palmar surface of the foot. The needle is removed, and pressure is applied to the injection site to avoid hematoma formation. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. Ultimately, the patient may require surgical extirpation of the neuroma (Fig. 142.9).

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The major complication of injection is infection, although

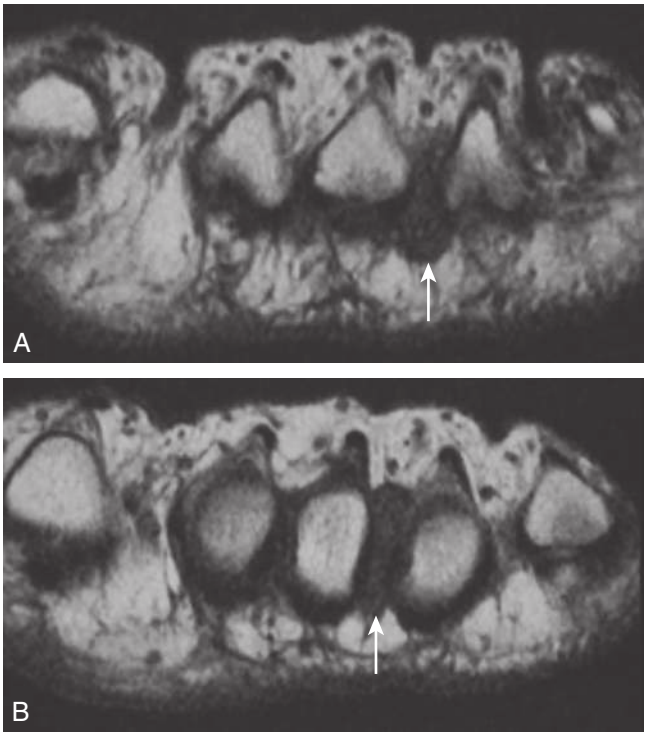


FIG 142.7 A, B, Consecutive coronal T1W magnetic resonance images of a low-SI Morton's neuroma (*white arrows*) arising between the third and fourth metatarsal heads. (From Waldman SD, Campbell RS. *Imaging of pain*. Elsevier; 2011.)

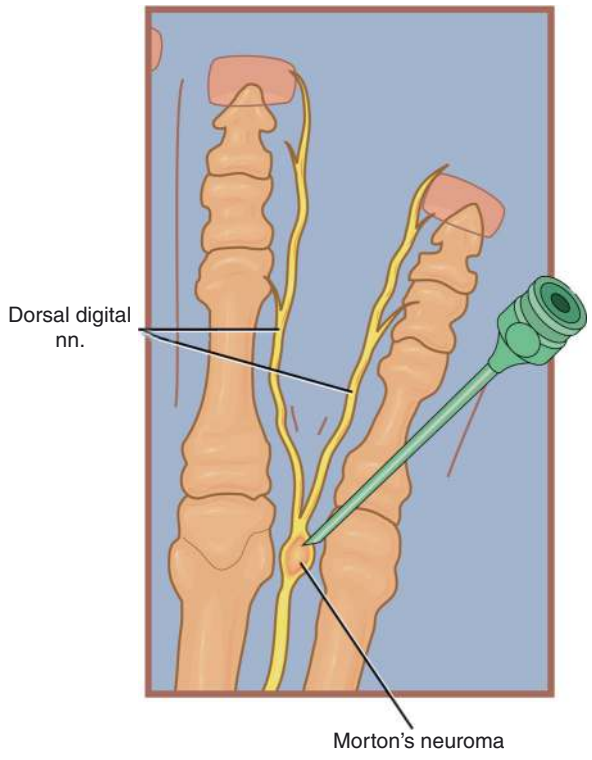


FIG 142.8 Proper needle placement for injection of Morton's neuroma. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000.)

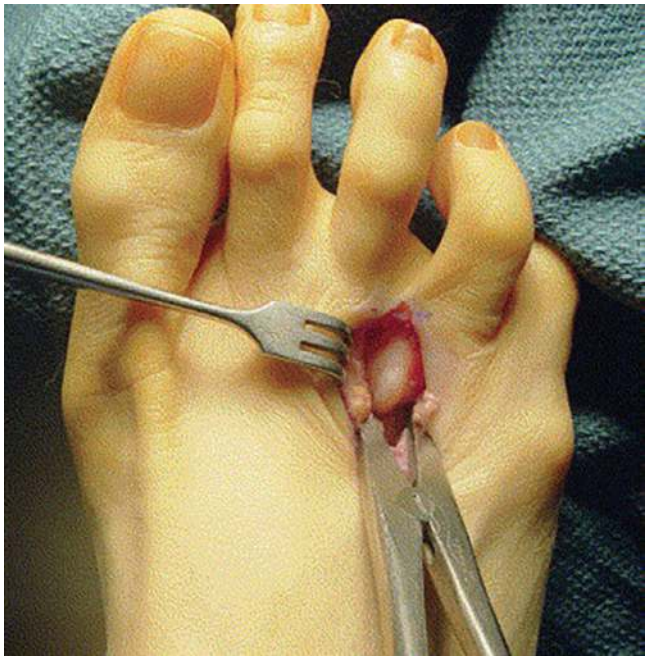


FIG 142.9 Surgical extirpation of a Morton neuroma using the dorsal approach. Note the large neuroma in situ. (From Singh SK, Ioli JP, Chiodo CP. The surgical treatment of Morton's neuroma. *Curr Orthop*. 2005;19(5):379–384, Figure 2. ISSN 0268-0890.)

this should be exceedingly rare if strict aseptic technique is followed. Because of the confined space of the soft tissues surrounding the metatarsals and digits, mechanical compression of the blood supply after injection is a possibility. To prevent vascular insufficiency and gangrene from occurring, the clinician must avoid rapidly injecting a large volume of solution into these confined spaces; epinephrine-containing solutions should not be used, for the same reasons. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's foot pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of Morton's neuroma; however, patients often require shoe orthoses and shoes with a wider toe box to take pressure off the affected interdigital nerves.

SUGGESTED READINGS

Arshad Z, Alshahwani A, Bhatia M. The management of multiple Morton's neuromas in the same foot: a systematic review. *J Foot Ankle Surg*. 2022;61(1):163–169. https://doi.org/10.1053/j.jfas.2021.08.003.

Bhatia M, Thomson L. Morton's neuroma – current concepts review. *J Clin Orthop Trauma*. 2020;11(3):406–409. https://doi.org/10.1016/j.jcot.2020.03.024.

Clinical Practice Guideline Forefoot Disorders Panel, Thomas JL, Blitch EL IV, et al. Diagnosis and treatment of forefoot disorders. Section 3. Morton's intermetatarsal neuroma. *J Foot Ankle Surg*. 2009;48(2):251–256.

Ferkel E, Davis WH, Ellington JK. Entrapment neuropathies of the foot and ankle. *Clin Sports Med*. 2015;34(4):791–801.

George VA, Khan AM, Hutchinson CE, et al. Morton's neuroma: the role of MR scanning in diagnostic assistance. *Foot*. 2005;15(1):14–16.

Mahadevan D, Venkatesan M, Bhatt R, et al. Diagnostic accuracy of clinical tests for Morton's neuroma compared with ultrasonography. *J Foot Ankle Surg*. 2015;54(4):549–553.

Mak MS, Chowdhury R, Johnson R. Morton's neuroma: review of anatomy, pathomechanism, and imaging. *Clin Radiol*. 2021;76(3):235.e15–235.e23. https://doi.org/10.1016/j.crad.2020.10.006.

Richardson DR, Dean EM. The recurrent Morton neuroma: what now? *Foot Ankle Clin*. 2014;19(3):437–449.

Waldman SD. Morton neuroma syndrome. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1063–1070.

Waldman SD. Injection technique for Morton neuroma syndrome. In: *Atlas of pain management injection techniques*. 5th ed. Philadelphia: Elsevier; 2023:900–903.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Waldman SD, Campbell RSD. Morton neuroma. In: *Imaging of pain*. Philadelphia: Saunders; 2011:459–460.

Williams TH, Robinson AH. Entrapment neuropathies of the foot and ankle. *Orthop Trauma*. 2009;23(6):404–411.

Wu KK. Morton's interdigital neuroma: a clinical review of its etiology, treatment, and results. *J Foot Ankle Surg*. 1996;35(2):112–142.

Intermetatarsal Bursitis

ICD-10 CODE M71.572

THE CLINICAL SYNDROME

Bursae are formed from synovial sacs whose purpose is to allow easy sliding of muscles and tendons across one another at areas of repeated movement. These synovial sacs are lined with a synovial membrane that is invested with a network of blood vessels that secrete synovial fluid. Inflammation of the bursa results in an increase in the production of synovial fluid with swelling of the bursal sac. With overuse or misuse, these bursae may become inflamed, enlarged, and on rare occasions infected. Although significant intrapatient variability as to the number, size, and location of bursae is seen, anatomists have identified a number of clinically relevant bursae, including the intermetatarsal bursae. The intermetatarsal bursa lies between the metatarsophalangeal joints in a position that is just dorsal to the deep transverse intermetatarsal ligament. The bursae extend approximately 1 cm beyond the distal border of the ligament in the web spaces between the second and third and third and fourth digits.

SIGNS AND SYMPTOMS

Patients with intermetatarsal bursitis experience pain and tenderness over the affected intermetatarsal spaces, with the pain made worse by wearing high-heeled shoes or shoes that are too narrow (Fig. 143.1). Obesity may also predispose to this condition. The pain may radiate distally into the toes, especially if the adjacent interdigital nerve is involved. Often the patient is unable to stand on tiptoes or walk upstairs. Activity worsens the pain. The pain is constant, is characterized as sharp, and may interfere with sleep. Coexistent neuritis, neuropathy, Morton's neuroma, stress fractures, metatarsalgia, and synovitis may confuse the clinical picture. As the bursitis worsens, the affected intermetatarsal bursae tend to expand, surrounding the adjacent interdigital nerves and making the patient's clinical presentation indistinguishable from the pain of Morton's neuroma (Fig. 143.2). If the inflammation of the intermetatarsal bursae becomes chronic, calcification of the bursae and fibrosis of the surrounding interdigital space may occur.

On physical examination, pain can be reproduced by squeezing the affected web space between the index finger and thumb. If the interdigital nerve is involved or if Morton's neuroma has developed, a positive Mulder sign can be elicited by firmly squeezing the two metatarsal heads together with one hand while placing firm pressure on the interdigital space with the other hand (Fig. 143.3).

The patient with intermetatarsal bursitis often exhibits an antalgic gait in an effort to reduce weight bearing during walking.

TESTING

Plain radiographs are indicated for all patients with intermetatarsal bursitis to rule out fractures and identify sesamoid bones that may have become inflamed. On the basis of the patient's clinical presentation, additional testing may be indicated, including complete blood cell count, sedimentation rate, and antinuclear antibody testing. Magnetic resonance imaging and ultrasound imaging of the metatarsal bones are indicated if Morton's neuroma, joint instability, occult mass, or tumor is suggested (Figs. 143.4–143.6). Radionuclide bone scanning may be useful in identifying stress fractures of the

FIG 143.1 Patients with intermetatarsal bursitis experience pain and tenderness over the affected intermetatarsal spaces, with the pain worsening by wearing high-heeled shoes or shoes that are too narrow.

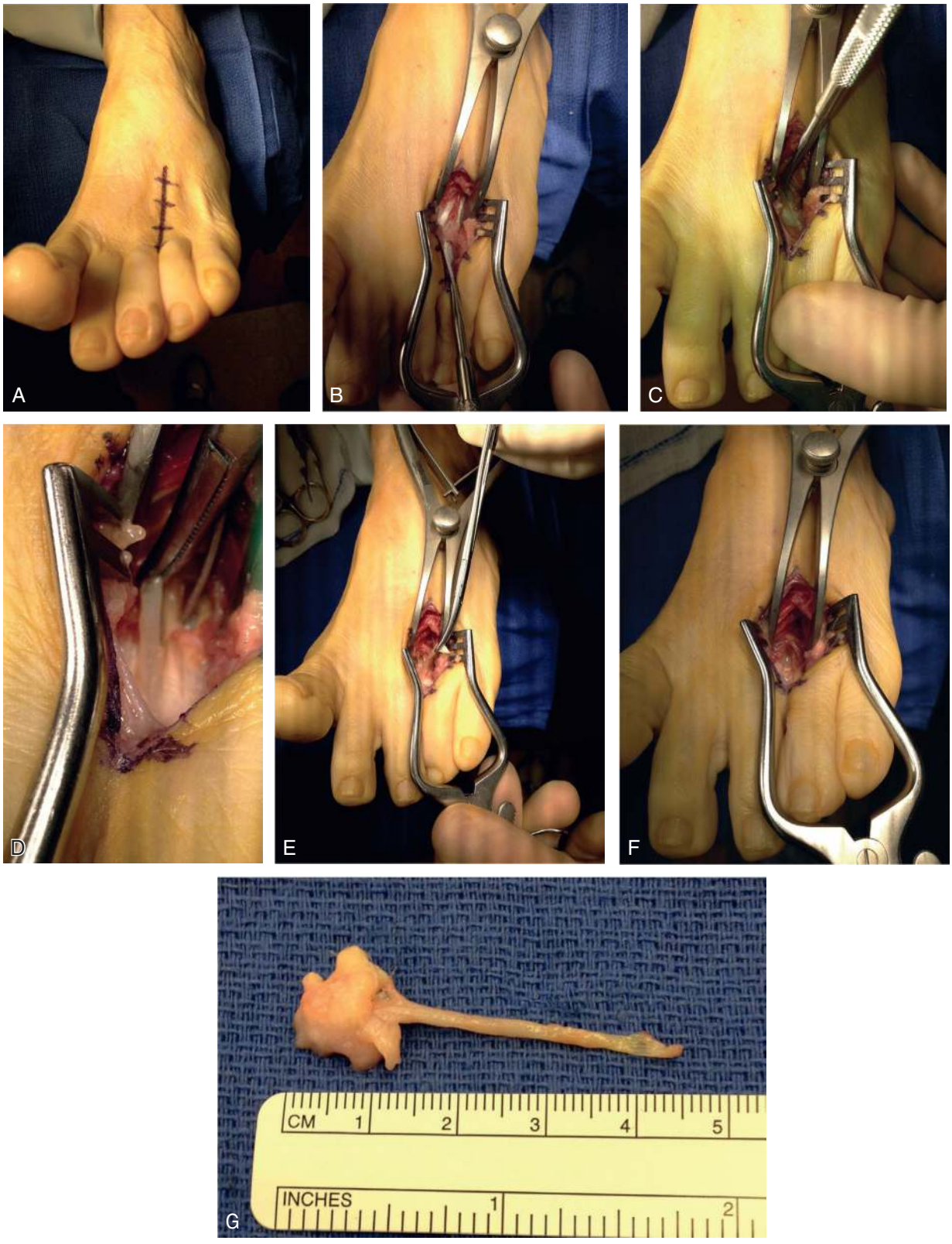


FIG 143.2 Surgical removal of combined Morton's neuroma and inflamed intermetatarsal bursa. **A,** Dorsal longitudinal incision. **B,** Reformed transverse metatarsal ligament after previous dorsal approach. **C,** Normal interdigital nerve is identified proximal to the stump neuroma. **D,** Hemostat is placed as far proximally on the nerve as possible to apply gentle traction before **(E)** transection of the nerve. **F** and **G,** The neuroma and surrounding bursa are dissected free from the interspace. (From Richardson DR, Dean EM. The recurrent Morton neuroma: what now? *Foot Ankle Clin.* 2014;19(3):437–449.)

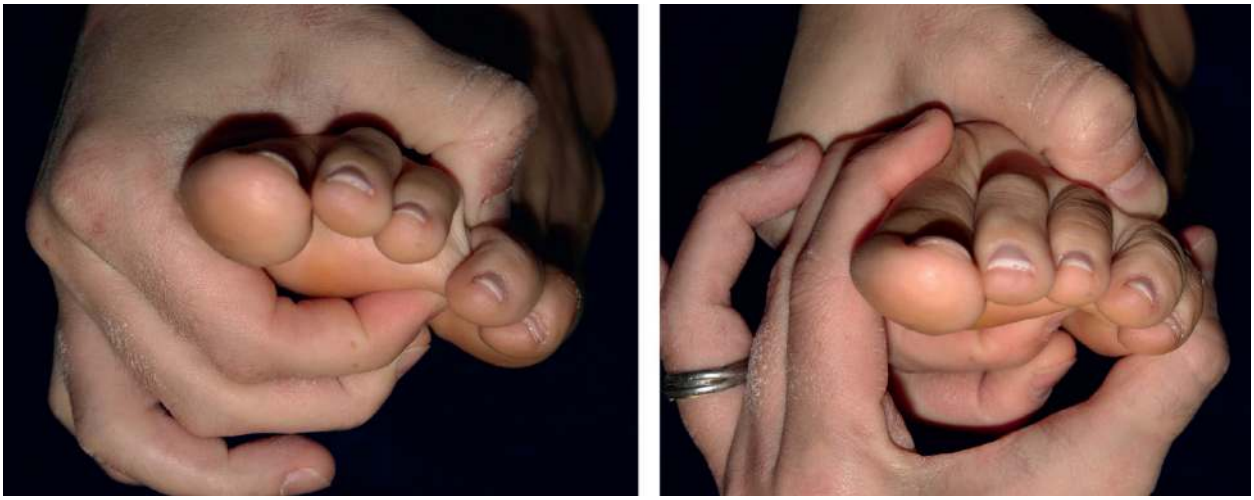


FIG 143.3 The pain of intermetatarsal bursitis may be reproduced with compression of the metatarsals. (From Richardson DR, Dean EM. The recurrent Morton neuroma: what now? *Foot Ankle Clin*. 2014;19(3):437–449.)

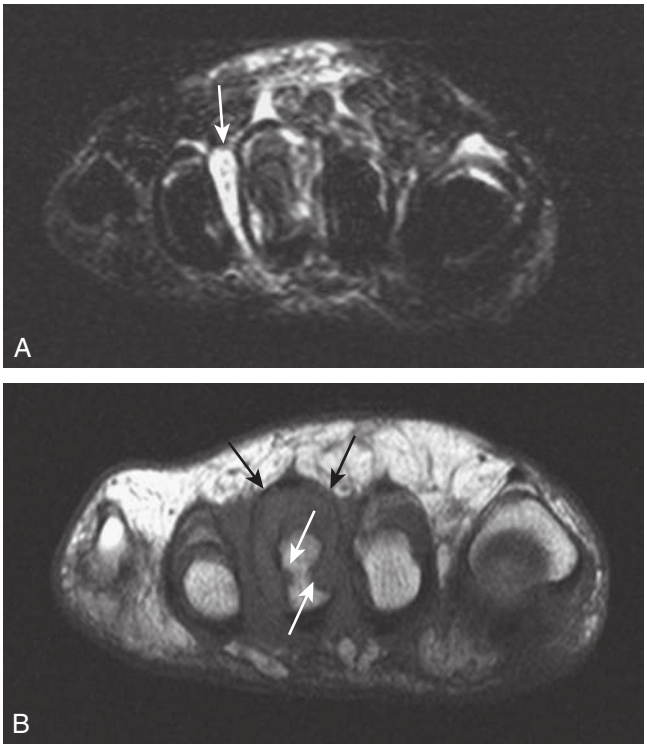


FIG 143.4 A, Coronal T2-weighted magnetic resonance (MR) image of a patient with rheumatoid arthritis demonstrating an inflammatory bursa in the intermetatarsal space (*white arrow*). There is associated synovitis in the third metatarsophalangeal joint. B, The corresponding T1-weighted MR image shows the synovial thickening (*black arrows*) and the associated bony erosions (*white arrows*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

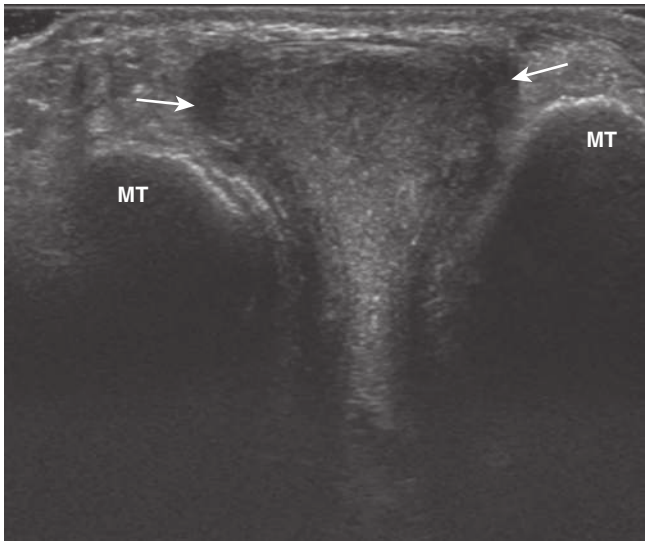


FIG 143.5 Transverse ultrasound image of an echogenic intermetatarsal bursa (*white arrows*) arising between the metatarsal necks. The mass was easily compressible with sonopalpation. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011:160.)

metatarsal bones or sesamoid bones that may be missed on plain radiographs of the foot.

DIFFERENTIAL DIAGNOSIS

Morton's neuroma and fractures of the sesamoid bones of the foot are often confused with intermetatarsal bursitis; although the pain of sesamoid fracture is localized to the plantar surface of the foot, it is less neuritic than that of Morton's neuroma. Tendinitis, infections, foreign bodies, plantar warts, synovial cysts, and stress fractures of the foot can also mimic the pain of intermetatarsal bursitis.

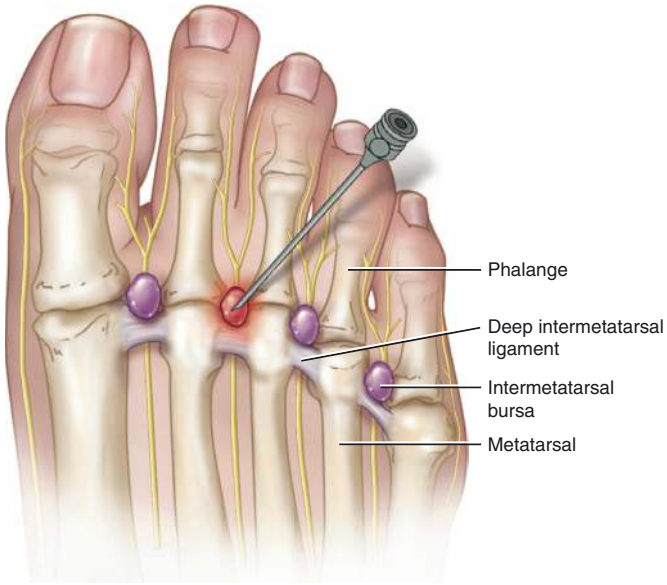


FIG 143.7 Proper needle placement for injection of intermetatarsal bursitis. (From Waldman SD. *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:676.)

FIG 143.6 Forefoot foreign-body granuloma. A 35-year-old agricultural worker presented with 1-month history of forefoot pain with unclear traumatic history. Proton density fat-saturated short-axis images (A, B) and sagittal image (C) showed a thin signal-void structure that is linear on sagittal image and rounded on short-axis images. It is surrounded by a hyperintense halo and a hyperintense signal of the surrounding soft tissues of the plantar aspect of the first intermetatarsal space. A palm thorn was retrieved on surgical exploration. (From Nouh MR, Abd El-Gawad EA, Abdulsalam SM. MRI utility in patients with non-traumatic metatarsalgia: a tertiary musculoskeletal center observational study. *Egypt J Radiol Nucl Med*. 2015;46(4):1057–1064.)

TREATMENT

Initial treatment of the pain and functional disability associated with intermetatarsal bursitis includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of narrow-toed or high-heeled shoes, and short-term immobilization of the affected foot may also provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To inject patients with intermetatarsal bursitis, the patient is placed in the supine position with a pillow under the knee to slightly flex the leg. A total of 3 mL non-epinephrine-containing local anesthetic and 40 mg methylprednisolone is drawn up in a 12-mL sterile syringe. The affected interdigital space is identified, the dorsal surface of the foot at this point is marked with a sterile marker, and the skin is prepared with antiseptic solution. At a point proximal to the metatarsal head, a 1½-inch, 25-gauge needle is inserted between the two metatarsal bones in the area to be blocked (Fig. 143.7). While slowly injecting, the clinician advances the needle from the dorsal surface of the foot toward the palmar surface. Because the plantar digital nerve is situated on the dorsal side of the flexor retinaculum, the needle must be advanced almost to the palmar surface of the foot. The needle is removed, and pressure is applied to the injection site to avoid hematoma formation. The use of ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Because of the confined space of the soft tissues surrounding the metatarsals and digits, mechanical

compression of the blood supply after injection is a possibility. To prevent vascular insufficiency and gangrene from occurring, the clinician must avoid rapidly injecting a large volume of solution into these confined spaces; epinephrine-containing solutions should not be used, for the same reasons. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's foot pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of intermetatarsal bursitis; however, patients often require shoe orthoses and shoes with a wider toe box to take pressure off the affected interdigital nerves.

SUGGESTED READINGS

Besse JL. Metatarsalgia. *Orthop Traumatol Surg Res*. 2017; 103(suppl 1):S29–S39.

Ferkel E, Davis WH, Ellington JK. Entrapment neuropathies of the foot and ankle. *Clin Sports Med*. 2015;34(4):791–801.

Mahadevan D, Venkatesan M, Bhatt R, et al. Diagnostic accuracy of clinical tests for Morton's neuroma compared with ultrasonography. *J Foot Ankle Surg*. 2015;54(4):549–553.

Richardson DR, Dean EM. The recurrent Morton neuroma: what now? *Foot Ankle Clin*. 2014;19(3):437–449.

Waldman SD. Intermetatarsal bursitis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1063–1070.

Waldman SD. Intermetatarsal bursitis injection. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:674–677.

Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.

Waldman SD, Campbell RSD. Morton neuroma. In: *Imaging of pain*. Philadelphia: Saunders; 2011:459–460.

Freiberg Disease

ICD-10 CODE M72.90

THE CLINICAL SYNDROME

Freiberg disease is an often missed clinical diagnosis. The disease may be identified as a result of the characteristic radiographic findings of collapse of the second and, less commonly, third metatarsal head or heads. Like the scaphoid, the second metatarsal joint is extremely susceptible to this disease because of the tenuous blood supply of the articular cartilage. This blood supply is easily disrupted, and this often leaves the proximal portion of the bone without nutrition, which leads to osteonecrosis. Although Freiberg disease can occur at any age, it is a disease of adolescence through the second decade. Freiberg disease is five times more common in female patients.

Although the exact cause of Freiberg disease remains elusive, many investigators believe that this condition is the result of repetitive microtrauma to the second and third metatarsal heads. Investigators believe that the relative immobility of the second and third metatarsals, combined with the extreme load transmission, makes these bones particularly susceptible to the development of avascular necrosis (Fig. 144.1). High heels, which increase the load on the metatarsal heads, may raise the risk of the development of Freiberg disease, as well as of any disease that impairs the blood supply to the foot (e.g., diabetes, vasculitis, and human immunodeficiency virus infection). Steroids have also been implicated as a cause of Freiburg disease.

The patient with Freiberg disease complains of pain over the affected metatarsal head or heads that may radiate into the adjacent toes. The pain is deep and aching, and the patient often complains of increased pain on weight bearing and a limp when walking. The patient may or may not have a clear history of foot trauma that can be identified as the inciting incident for the disease.

SIGNS AND SYMPTOMS

Physical examination of patients suffering from Freiberg disease reveals pain on deep palpation of the affected metatarsal joints. The pain can worsen by passive and active range of motion. Subtle swelling over the affected joint or joints may

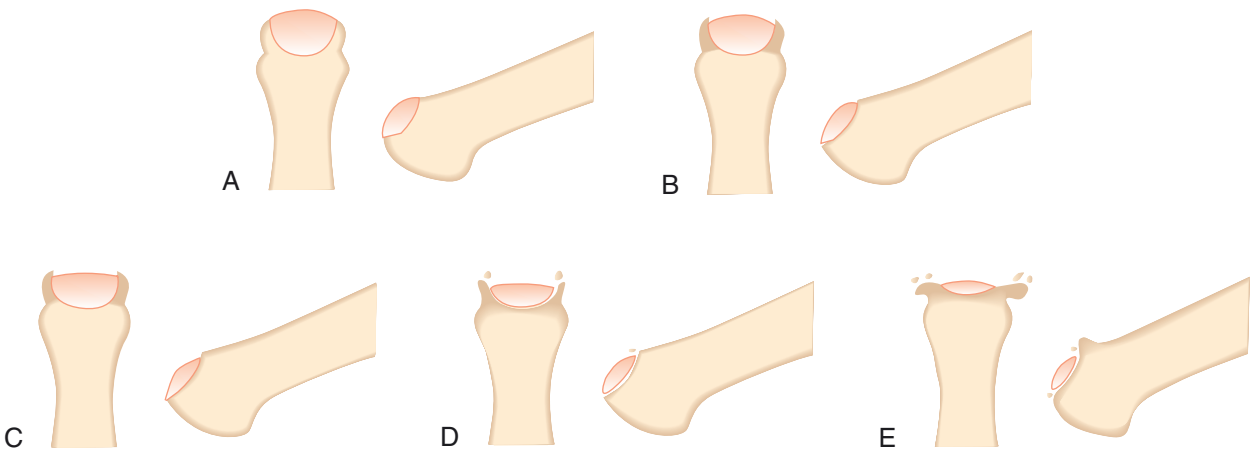


FIG 144.1 Levels of progression of Freiberg disease. **A,** Early fracture of the subchondral epiphysis. **B,** Early collapse of the dorsal central portion of the metatarsal with flattening of the articular surface. **C,** Further flattening of the metatarsal head with continued collapse of the central portion of the articular surface with medial and lateral projections; the plantar articular cartilage remains intact. **D,** Loose bodies form from fractures of lateral projections and separation of a central articular fragment. **E,** End-stage degenerative arthrosis with marked flattening of the metatarsal head and joint space narrowing. (Redrawn from Katcherian DA. Treatment of Freiberg's disease. Orthop Clin North Am. 1994;25:69–81.)

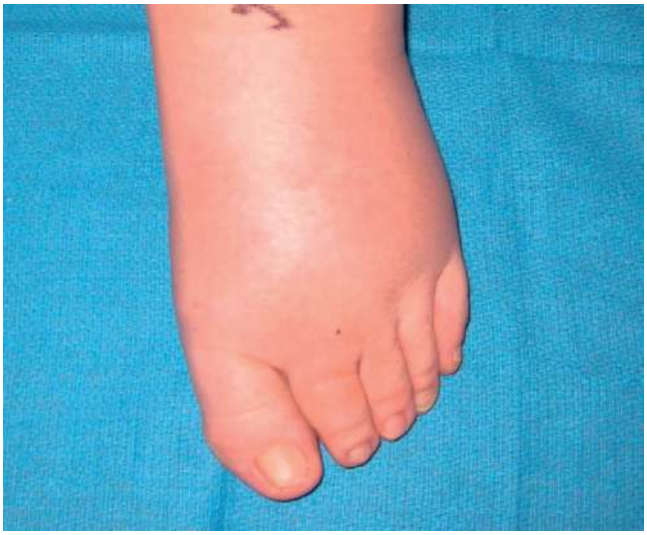


FIG 144.2 Swelling of the second toe in patient with Freiberg disease. (From Cerrato RA. Freiberg's disease. *Foot Ankle Clin*. 2011;16(4):647–658.)

be appreciated on careful physical examination (Fig. 144.2). An antalgic gait is invariably present.

TESTING

Plain radiographs are indicated in all patients who present with Freiberg disease to confirm the diagnosis as well as to rule out underlying occult bony disease (Fig. 144.3). Early subtle sclerotic changes and joint space narrowing are often attributed to degenerative arthritis. Magnetic resonance imaging (MRI) may reveal articular changes before significant changes are evident on plain radiographs (Fig. 144.4). Based on the patient's clinical presentation, additional testing, including complete blood cell count, uric acid, sedimentation rate, and antinuclear antibody testing, may also be indicated. MRI and computerized tomography of the foot are indicated in all patients suspected of suffering from Freiberg disease, as well as when other causes of joint instability, infection, or tumor are suspected, or if the plain radiographs are nondiagnostic (Figs. 144.5 and 144.6). Administration of gadolinium followed by postcontrast imaging may help delineate the adequacy of blood supply; contrast enhancement of the metatarsal joint is a good prognostic sign. Electromyography is indicated if coexistent lumbar radiculopathy or lumbar plexopathy is suspected. A very gentle intraarticular injection of the affected joint with small volumes of local anesthetic provides immediate levitation of the pain and helps demonstrate that the nidus of the patient's pain is, in fact, the metatarsal joint. Ultimately, joint replacement is required in most patients suffering from Freiberg disease.

DIFFERENTIAL DIAGNOSIS

Coexistent arthritis and gout of the metatarsal joints, bursitis, synovitis, and tendinitis may also coexist with Freiberg disease and exacerbate the patient's pain and disability. Morton's neuroma can mimic the pain of Freiberg disease. Tears of the ligaments, bone cysts, bone contusions, and fractures may also mimic the pain of Freiberg disease, as can occult metastatic disease.

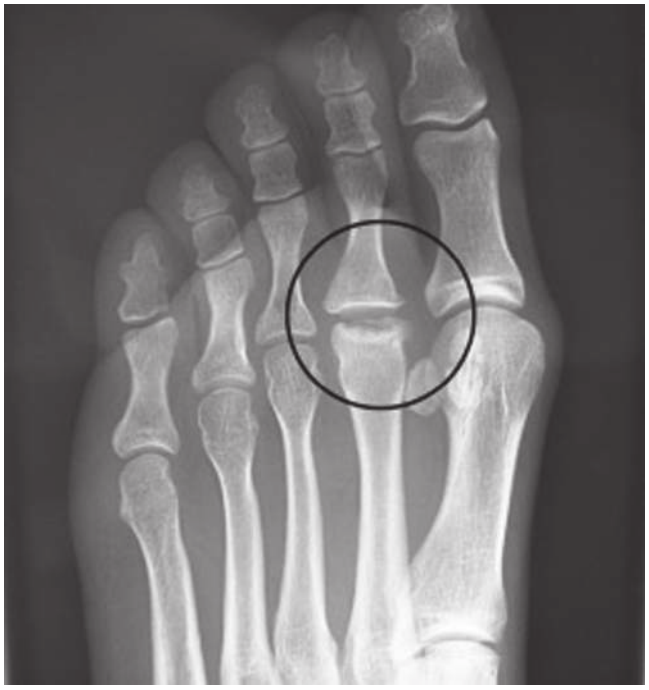


FIG 144.3 Radiographic appearance of Freiberg infarction (*circled area*). (From Miller MD. *Review of orthopaedics*. 6th ed. Philadelphia: Saunders; 2012.)

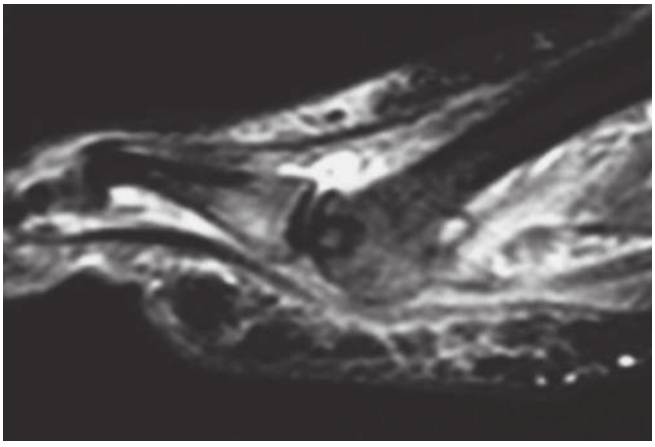


FIG 144.4 Sagittal short tau inversion recovery magnetic resonance image demonstrating higher juxta-articular marrow edema with a well-delineated low-signal defect of the second metatarsal head. (From Dolce M, Osher L, McEneany P, et al. The use of surgical core decompression as treatment for avascular necrosis of the second and third metatarsal heads. *Foot*. 2007;17(3):162–166.)

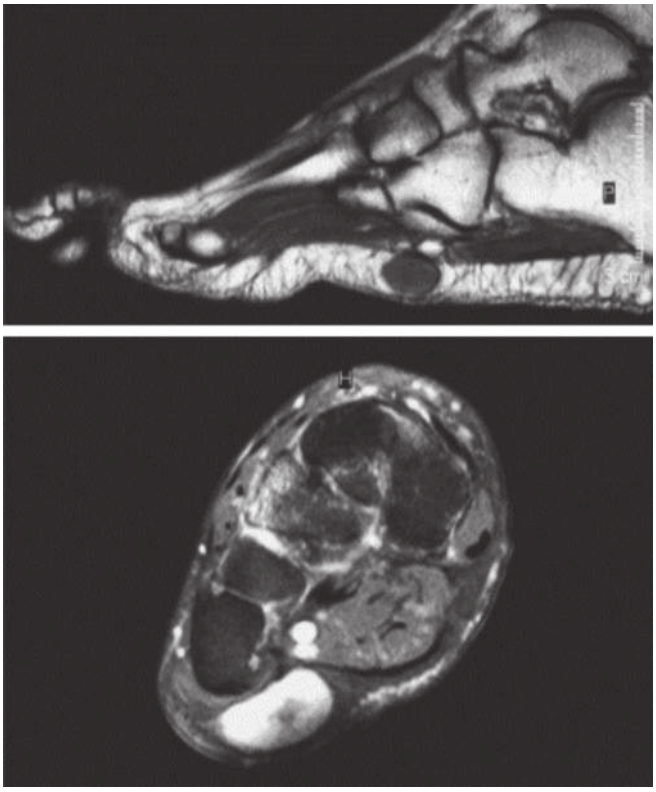


FIG 144.5 Sagittal and axial magnetic resonance images showing a lesion arising from the sole and not involving the deeper structures. This lesion was later shown to be a fibroma of the flexor tendon sheath. (From Vasconez HC, Nisanci M, Lee EY. Giant cell tumour of the flexor tendon sheath of the foot. *J Plast Reconstr Aesthet Surg.* 2008;61(7):815–818.)

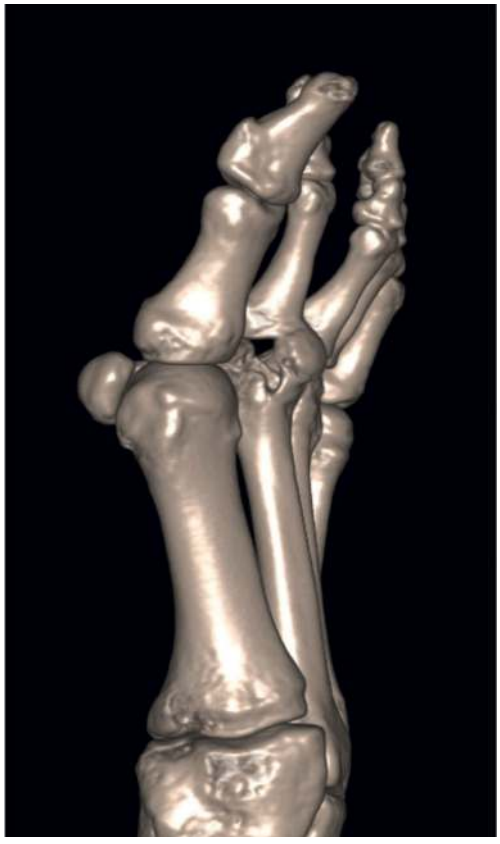


FIG 144.6 Three-dimensional reconstruction of computed tomography scan of the right foot showing displaced fracture in a patient with steroid-induced Freiberg disease. (From Kenny L, Purushothaman B, Teasdale R, El-Hassany M, Parvin B. Atypical presentation of acute Freiberg disease. *J Foot Ankle Surg.* 2017;56(2):385–389.)

TREATMENT

Initial treatment of the pain and functional disability associated with Freiberg disease should include a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and decreased weight bearing of the joint or joints. The local application of heat and cold may also be beneficial. For patients who do not respond to these treatment modalities, an injection of a local anesthetic and steroid into the joint may be a reasonable next step to provide palliation of acute pain. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Ultimately, surgical repair in the form of total joint arthroplasty is the treatment of choice.

COMPLICATIONS AND PITFALLS

Failure to treat significant Freiberg disease surgically usually results in continued pain and disability and, in most patients, leads to ongoing damage to the affected joints. A failure to diagnose diseases that may mimic Freiberg disease may result in significant pain and functional disability.

CLINICAL PEARLS

Freiberg disease is a diagnosis that is often missed, thus leading to much unnecessary pain and disability. The clinician should include Freiberg disease in the differential diagnosis in all patients complaining of forefoot pain. Coexistent arthritis, tendinitis, and gout may also contribute to the pain and may require additional treatment. The use of physical modalities, including local heat and cold, as well as decreased weight bearing, may provide symptomatic relief. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms and may cause further damage to the foot. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

SUGGESTED READINGS

Alhadhoudn MA, Alsiri NF, Daniels TR, Glazebrook MA. Surgical interventions of Freiberg's disease: a systematic review. *Foot Ankle Surg.* 2021;27(6):606–614. https://doi.org/10.1016/j.fas.2020.08.005.

Blaise Williams DS III, Hertel J, Ingersoll CD, et al. Rehabilitation of leg, ankle, and foot injuries. In: *Pathology and intervention in*

musculoskeletal rehabilitation. 2nd ed. W.B. Saunders; 2016:851–880.
Cerrato RA. Freiberg's disease. *Foot Ankle Clin*. 2011;16(4): 647–658.
Hoggett L, Nanavati N, Cowden J, et al. A new classification for Freiberg's disease. *Foot*. 2022;51:101901. https://doi.org/10.1016/j.foot.2021.101901.
Kenny L, Purushothaman B, Teasdale R, et al. Atypical presentation of acute Freiberg disease. *J Foot Ankle Surg*. 2017;56(2):385–389.
Parvizi J, Kim GK. Freiberg's disease. In: *High yield orthopaedics*. Philadelphia: W.B. Saunders; 2010:197–198.
Seybold JD, Zide JR. Treatment of Freiberg disease. *Foot Ankle Clin*. 2018;23(1):157–169. https://doi.org/10.1016/j.fcl.2017.09.011.
Waldman SD. Functional anatomy of the ankle and foot. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:149–150.
Waldman SD, Campbell RSD. Anatomy: special imaging considerations of the ankle and foot. In: *Imaging of pain*. Philadelphia: Saunders; 2011:417–420.
Waldman SD, Campbell RSD. Freiberg's disease. In: *Imaging of pain*. Philadelphia: Saunders; 2011:447–448.
Wax A, Leland R. Freiberg disease and avascular necrosis of the metatarsal heads. *Foot Ankle Clin*. 2019;24(1):69–82.

145

Plantar Fasciitis

ICD-10 CODE M72.9

THE CLINICAL SYNDROME

Plantar fasciitis is characterized by pain and tenderness over the plantar surface of the calcaneus. It is twice as common in women as in men. Plantar fasciitis is thought to be caused by inflammation of the plantar fascia, which can occur alone or as part of a systemic inflammatory condition such as rheumatoid arthritis, Reiter's syndrome, or gout. Obesity seems to predispose patients to the development of plantar fasciitis, as does going barefoot or wearing house slippers for prolonged periods (Fig. 145.1). High-impact aerobic exercise has also been implicated as a causative factor.

SIGNS AND SYMPTOMS

The pain of plantar fasciitis is most severe when first walking after a period of non–weight bearing and is made worse by prolonged standing or walking. On physical examination, patients exhibit a positive calcaneal jump sign, which consists of point tenderness over the plantar medial calcaneal tuberosity (Fig. 145.2). The windlass test may also help make the diagnosis (Fig. 145.3). Patients may also have tenderness along the plantar fascia as it moves anteriorly. Pain is increased by dorsiflexing the toes, which pulls the plantar fascia taut, and then palpating along the fascia from the heel to the forefoot.

TESTING

Plain radiographs, magnetic resonance, and ultrasound imaging are indicated in all patients who present with pain thought to be caused by plantar fasciitis, to rule out occult bony disorders and tumor (Figs. 145.4–145.7). Although characteristic radiographic changes are lacking in plantar fasciitis, radionuclide bone scanning may show increased uptake where the plantar fascia attaches to the medial calcaneal tuberosity; it can also rule out stress fractures not visible on plain radiographs. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

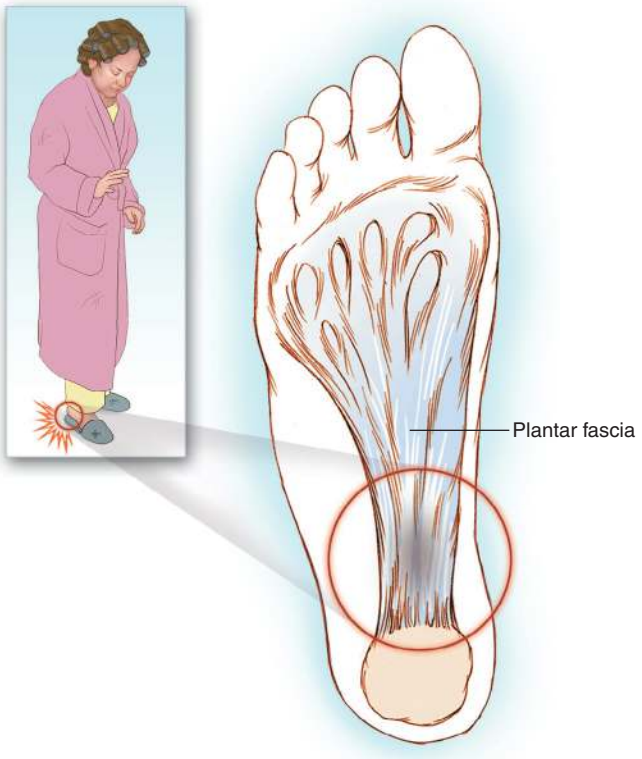


FIG 145.1 The pain of plantar fasciitis, which is localized to the hindfoot, can cause significant functional disability.

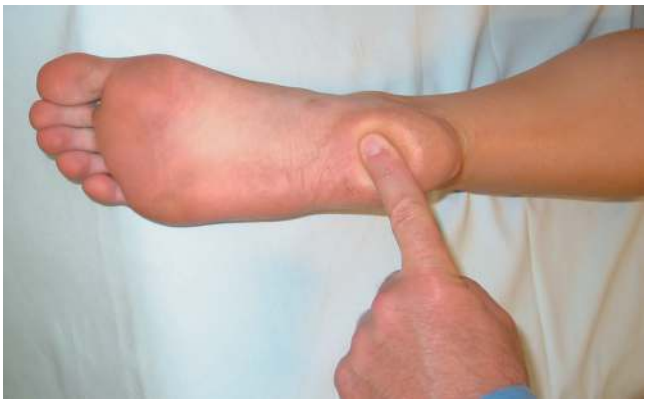


FIG 145.2 Eliciting the calcaneal jump sign for plantar fasciitis. (From Waldman SD. *Physical diagnosis of pain: an atlas of signs and symptoms*. Philadelphia: Saunders; 2006:379.)

FIG 145.3 The windlass test for plantar fasciitis. To perform the windlass test for plantar fasciitis, the patient is placed in the supine position with the knee flexed to 90 degrees and the affected foot in neutral position. The examiner then stabilizes the head of the first metatarsal and dorsiflexes the great toe. The test is positive if it reproduces or exacerbates the patient's pain.

DIFFERENTIAL DIAGNOSIS

Common causes of heel pain are listed in Box 145.1. The pain of plantar fasciitis may be confused with many diseases including the pain of Sever's disease, Morton's neuroma, or sesamoiditis. However, the characteristic pain on dorsiflexion of the toes associated with plantar fasciitis should help distinguish these conditions. Stress fractures of the metatarsal or sesamoid bones, bursitis, and tendinitis may also confuse the clinical picture.

TREATMENT

Initial treatment of the pain and functional disability associated with plantar fasciitis includes a combination of nonsteroidal anti-inflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of walking barefoot or with shoes that do not provide good support, and short-term immobilization of the affected foot may provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To perform an injection for plantar fasciitis, the patient is placed in the supine position. The medial aspect of the heel

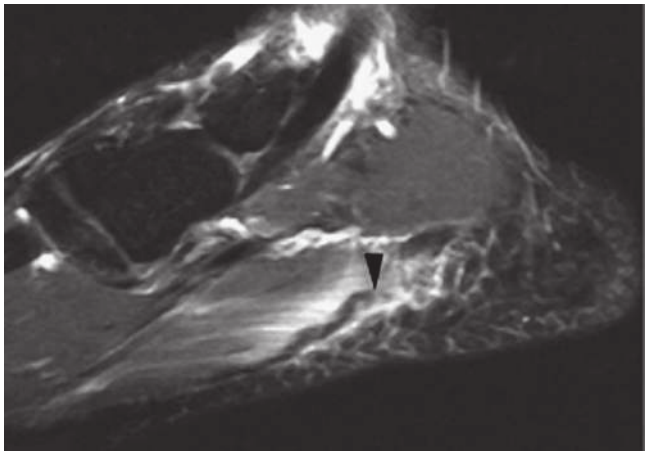


FIG 145.4 Rupture of the central cord of the plantar fascia. This sagittal short tau inversion recovery magnetic resonance image demonstrates discontinuity of the plantar fascia, with extensive edema of the flexor digitorum brevis muscle (*arrowhead*). (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3456.)

is identified by palpation, and the skin overlying this point is prepared with antiseptic solution. A syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge needle. The needle is slowly advanced through the previously identified point at a right angle to the skin, directly toward the center of the medial aspect of the calcaneus, until the needle impinges on bone. The needle is then withdrawn slightly out of the periosteum, and the contents of the syringe are gently injected as the needle is slowly withdrawn. The operator should feel slight resistance to injection, given the closed nature of the heel. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications. Clinical experience suggests that the injection of platelet-rich plasma and/or stem cells may provide symptomatic relief and aid in healing.

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Stretching exercises may be of particular benefit (Fig. 145.8). Heel pads or molded orthotic devices may also be of value. Low-energy extracorporeal shockwave therapy may also provide symptomatic relief in resistant cases. Simple analgesics, NSAIDs, and antimyotonic agents, such as tizanidine, can be used concurrently with this injection technique.

COMPLICATIONS AND PITFALLS

Most complications of injection are related to needle-induced trauma at the injection site and in the underlying tissues. Many patients complain of a transient increase in pain after injection, which can be minimized by injecting gently and slowly. Infection, although rare, may occur if sterile technique is not followed.

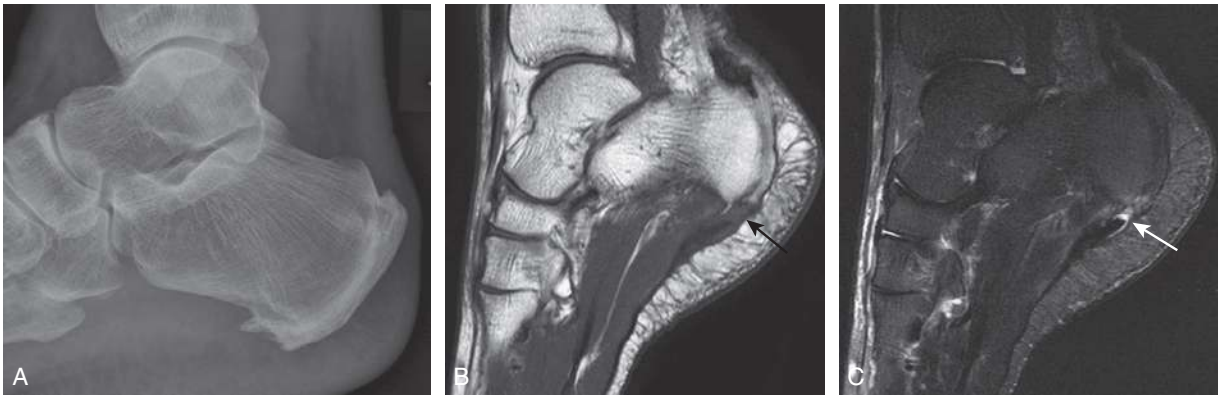


FIG 145.5 A, Lateral radiograph of a plantar spur on the calcaneus. B, The sagittal T1W magnetic resonance (MR) image demonstrates thickening and increased signal intensity (SI) within the plantar fascia origin (*black arrow*). There is high-SI fatty marrow within the bony spur. C, High-SI fluid (*white arrow*) is seen within the plantar fascia origin on the sagittal fat-suppressed T2-weighted MR image. The appearances are consistent with plantar fasciitis and partial tearing of the origin of the fascia. (From Waldman RS, Campbell RSD. *Imaging of pain*. Philadelphia: Elsevier; 2011.)

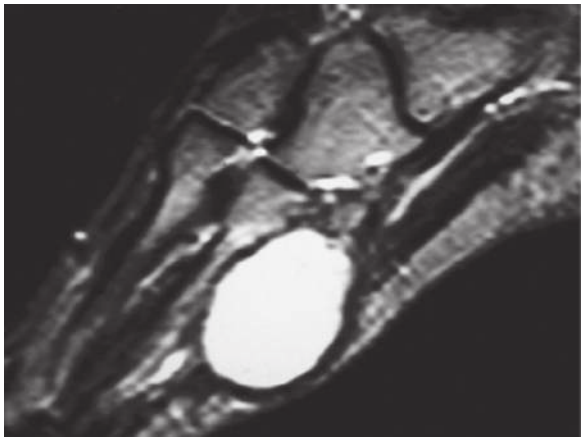


FIG 145.6 Synovial sarcoma. This sagittal T2-weighted magnetic resonance image demonstrates a large soft tissue mass in the plantar aspect of the foot. The mass is homogeneous and exhibits a thick capsule, simulating a fluid collection. (From Edelman RR, Hesselink JR, Zlatkin MB, et al., eds. *Clinical magnetic resonance imaging*. 3rd ed. Philadelphia: Saunders; 2006:3456.)

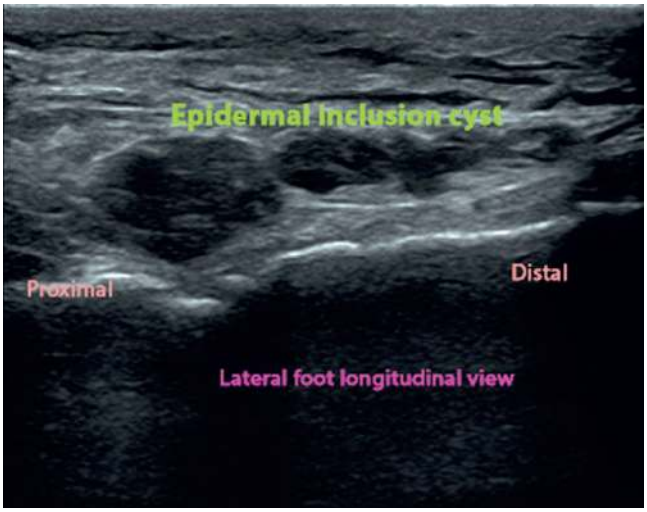


FIG 145.7 Longitudinal ultrasound image of the plantar surface demonstrating an epidermoid inclusion cyst.

BOX 145.1 Common Causes of Heel Pain

Skeletal
Calcaneal epiphysitis (Sever's disease)
Calcaneal stress fracture
Tarsal stress fractures
Infections, osteomyelitis
Inflammatory arthropathies (e.g., Reiter's syndrome, psoriatic arthritis)
Subtalar arthritis

Soft Tissue
Achilles tendinitis
Achilles tendon rupture
Fat pad atrophy
Heel pad contusion
Plantar fascia rupture
Posterior tibial tendinitis
Retrocalcaneal bursitis

Neurologic
Abductor digiti quinti nerve entrapment
Lumbar spine disorders
Problems with medial calcaneal branch of the posterior tibial nerve
Neuropathies
Tarsal tunnel syndrome

Miscellaneous
Metabolic disorders
Neuromas
Osteomalacia
Paget's disease
Sickle cell disease
Tumors
Vascular insufficiency

Modified from Karabat N, Toros T, Hurel C. Ultrasonographic evaluation in plantar fasciitis. *J Foot Ankle Surg*. 2007;46(6):442–446.

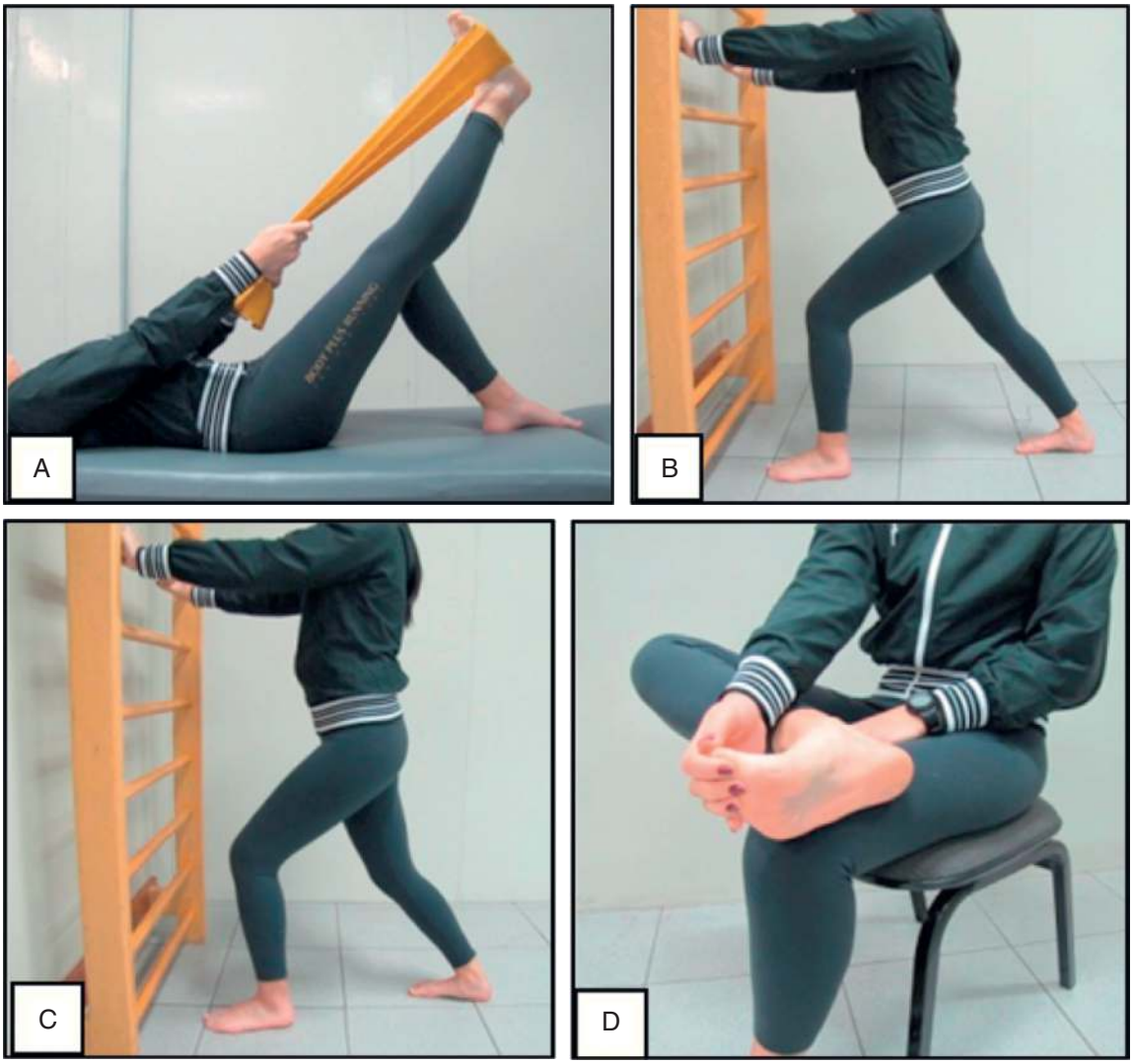


FIG 145.8 Stretching alone exercise group. **A,** Straight leg raise in supine position. **B,** Plantar flexor muscles stretch with knee extended. **C,** Plantar flexor muscles stretch with knee flexed. **D,** Plantar fascia stretch. (From Kamonseki AH, Gonçalves GA, Yi LC, et al. Effect of stretching with and without muscle strengthening exercises for the foot and hip in patients with plantar fasciitis: a randomized controlled single-blind clinical trial. *Manual Therapy*. 2016;23:76–82.)

CLINICAL PEARLS

The injection technique described is extremely effective in treating the pain of plantar fasciitis. It is a safe procedure if careful attention is paid to the clinically relevant anatomy, sterile technique is used to avoid infection, and universal precautions are implemented to minimize any risk to the operator.

SUGGESTED READINGS

Dinneen AJ, Shafafy R, Carne A, et al. Posterior heel pain. *OrthopTrauma*. 2016;30(1):8–17.

Gogna P, Gaba S, Mukhopadhyay R, et al. Plantar fasciitis: a randomized comparative study of platelet rich plasma and low dose radiation in sportspersons. *Foot (Edinb)*. 2016;28:16–19.

Huffer D, Hing W, Newton R, et al. Strength training for plantar fasciitis and the intrinsic foot musculature: a systematic review. *Phys Ther Sport*. 2017;24:44–52.

Rose B, Singh D. Inferior heel pain. *Orthop Trauma*. 2016;30(1):18–23.

Waldman SD. Plantar fasciitis and other abnormalities of the plantar fascia. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1005–1019.

Waldman SD. Injection technique for plantar fasciitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:648–651.

Waldman SD. Plantar fasciitis. In: *Pain review*. 2nd ed. Philadelphia: Elsevier; 2017:314–315.

Waldman SD, Campbell RSD. Plantar fasciitis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:457–458.

Metatarsalgia

ICD-10 CODE M77.40

THE CLINICAL SYNDROME

Along with sesamoiditis, metatarsalgia is another painful condition of the forefoot being seen with increasing frequency in clinical practice because of the increased interest in jogging and long-distance running. Metatarsalgia is characterized by tenderness and pain over the metatarsal heads. The patient often feels as if he or she is walking with a stone in the shoe. The pain of metatarsalgia worsens with prolonged standing or walking for long distances and is exacerbated by improperly fitting or padded shoes. Often a patient with metatarsalgia develops hard callus over the heads of the second and third metatarsals when trying to shift the weight off the head of the first metatarsal to relieve the pain (Fig. 146.1). This callus increases the pressure on the metatarsal heads and exacerbates the patient's pain and disability.

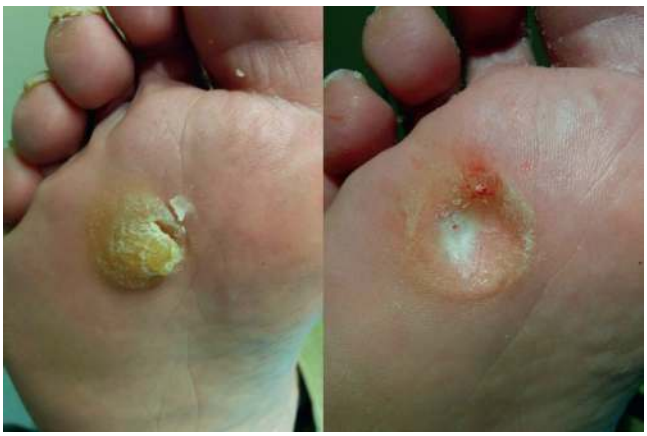


FIG 146.1 Plantar keratosis over the metatarsal heads can cause pain and abnormalities of gait. (From Araguas Garcia C, Corbi Soler F. The effect of plantar hyperkeratosis debridement on self-perception of pain levels in older people. *Int. J. Gerontol.* 2018;12(4):314–318.)

SIGNS AND SYMPTOMS

On physical examination, pain can be reproduced by pressure on the metatarsal heads (Fig. 146.2). Callus often is present over the heads of the second and third metatarsal heads and can be distinguished from plantar warts by the lack of thrombosed blood vessels that appear as small dark spots through the substance of the wart when the surface

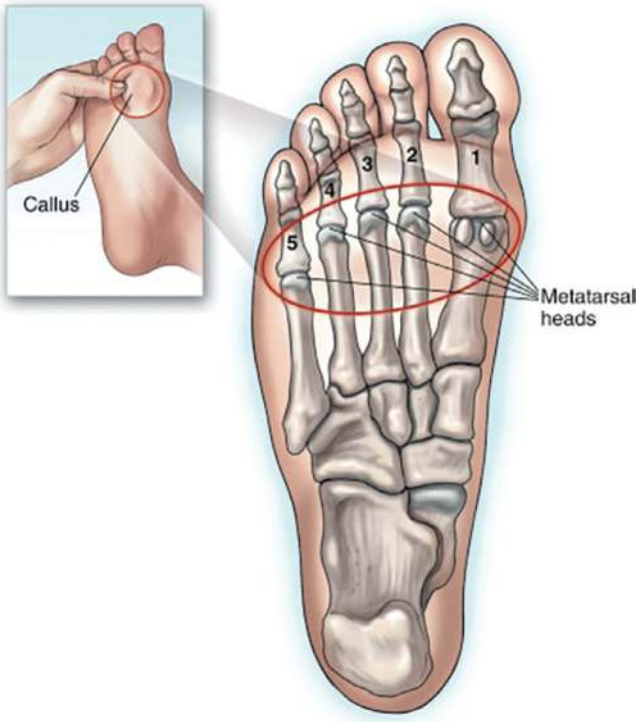


FIG 146.2 On physical examination, pain can be reproduced by pressure on the metatarsal heads. (From Waldman SD. *Atlas of uncommon pain syndromes*. 4th ed. Philadelphia: Saunders; 2023.)

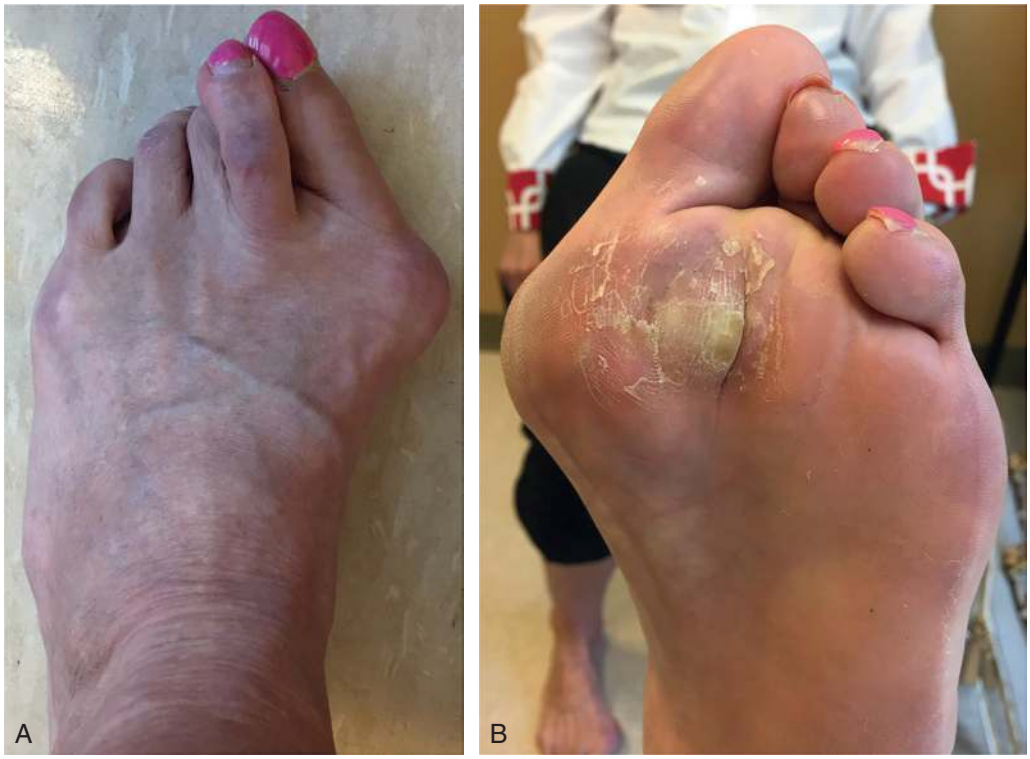


FIG 146.3 Clinical photographs of a patient suffering from metatarsalgia, including dorsal **(A)** and plantar **(B)** views, of a patient with a severe hallux valgus and lesser toe deformities, particularly a dorsally dislocated second metatarsophalangeal joint and resulting hammer toe deformity that presented with transfer metatarsalgia pain caused by overload at the plantar aspect of the second toe, evidenced by the heavy, callus formation at plantar forefoot. (From Federer AE. Tainter DM, Adams SB, Schweitzer KM. Conservative management of metatarsalgia and lesser toe deformities. *Foot Ankle Clin.* 2018;23(1):9–20.)

is trimmed (Fig. 146.3). A patient with metatarsalgia often exhibits an antalgic gait in an effort to reduce weight bearing during the static stance phase of walking. Ligamentous laxity and flattening of the transverse arch also may be present, giving the foot a splayed-out appearance.

TESTING

Plain radiographs are indicated in all patients with metatarsalgia to rule out fractures and to identify sesamoid bones that may have become inflamed (Figs. 146.4 and 146.5). Based on the patient's clinical presentation, additional tests, including complete blood cell count, erythrocyte sedimentation rate, and antinuclear antibody testing, may be indicated. Magnetic resonance imaging (MRI) and ultrasound imaging of the metatarsal bones are indicated if joint instability, occult mass, or tumor is suspected (Figs. 146.6 and 146.7). Radionucleotide bone scanning may be useful in identifying stress fractures that may be missed on plain radiographs of the foot.

DIFFERENTIAL DIAGNOSIS

Primary pathology of the foot, including gout and occult fractures, may mimic the pain and disability associated with metatarsalgia. Entrapment neuropathies such as tarsal tunnel syndrome, bursitis, and plantar fasciitis of the foot also may confuse the diagnosis; bursitis and plantar fasciitis may coexist with sesamoiditis. Sesamoid bones beneath the heads of the metatarsal bones are present in some individuals and are subject to the development of inflammation termed *sesamoiditis* as well as occult stress fractures (Fig. 146.8). Sesamoiditis is another common cause of forefoot pain and may be distinguished from metatarsalgia by the fact that the pain of metatarsalgia is centered over the patient's metatarsal heads and does not move when the patient actively flexes his or her toes, as is the case with sesamoiditis. The muscles of the metatarsal joints and their attaching tendons are susceptible to trauma and wear and tear from overuse and misuse and may contribute to forefoot pain. Primary and metastatic tumors of the foot also may manifest in a manner analogous to that of arthritis of the midtarsal joints.

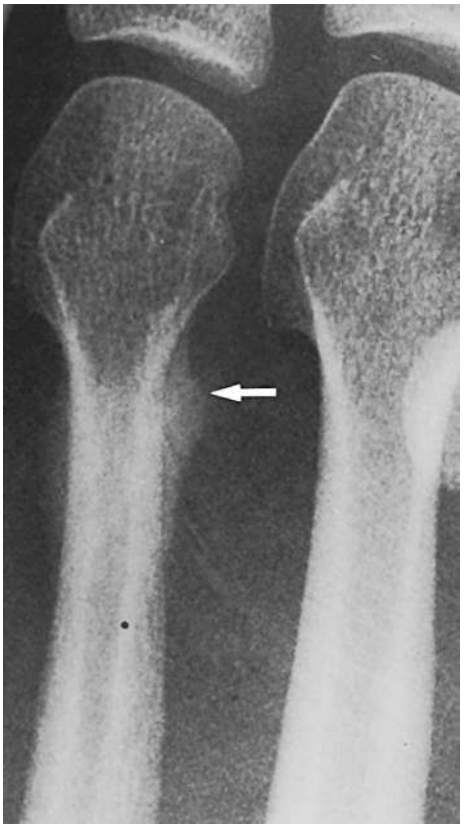


FIG 146.4 Stress fracture of the metatarsal (march fracture). Anteroposterior radiograph shows fluffy periosteal new bone along the distal shaft of the third metatarsal (*arrow*); the patient had foot pain for 16 days. (From Grainger RG, Allison D. *Grainger and Allison's diagnostic radiology: a textbook of medical imaging*. 3rd ed. New York: Churchill Livingstone; 1997:1610.)

TREATMENT

Initial treatment of the pain and functional disability associated with metatarsalgia should include a careful evaluation of the patient's footwear. Shoes should be well fitting with adequate length and a wide toe box. A firm rocker bottom sole may provide symptomatic relief. A combination of nonsteroidal anti inflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. Local application of heat and cold may be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms and short-term immobilization of the midtarsal joint also may provide relief. For patients who do not respond to these treatment modalities, injection of the affected metatarsal heads with a local anesthetic and steroid may be a reasonable next step. Ultrasound guidance may improve the accuracy of needle placement and decrease the incidence of needle-related complications.

COMPLICATIONS AND PITFALLS

The major complication of injection of the metatarsal heads is infection. This complication should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients report a transient increase in pain after injection of the metatarsal heads, and patients should be warned of this possibility. Another potential risk of this injection technique is trauma to the associated tendons from the injection.

CLINICAL PEARLS

Pain emanating from the forefoot is a common problem encountered in clinical practice. Metatarsalgia must be distinguished from stress fractures of the metatarsal bones, Morton's neuroma, and sesamoiditis. Although the previously mentioned injection technique provides palliation of the pain of metatarsalgia, the patient often also requires shoe orthoses, including metatarsal bars and padded insoles, to help remove pressure from the metatarsal heads. Coexistent bursitis and tendinitis may contribute to metatarsal pain and may require additional treatment with more localized injection of a local anesthetic and depot steroid. Injection of the metatarsal heads with a local anesthetic and steroid is a safe procedure if careful attention is paid to the clinically relevant anatomy in the areas to be injected. The use of physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes this injection technique for metatarsalgia pain. Vigorous exercises should be avoided because they would exacerbate the patient's symptoms. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

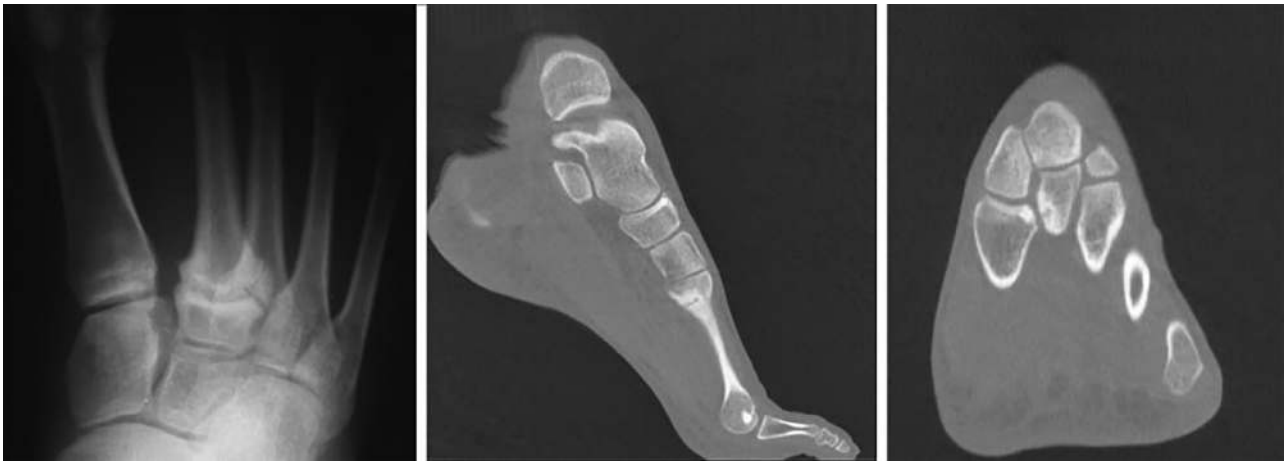


FIG 146.5 Stress fracture of the base of the second metatarsal in dancers. Radiographic anterior–posterior view and computed tomography scan, sagittal and transverse plane views demonstrating the transverse fracture line. (From de Cesar Netto C, Kennedy JG, Hamilton WG, O'Malley M. 24—Foot and ankle injuries in dancers. In: Porter DA, Schon LC, eds. *Baxter's the foot and ankle in sport*. 3rd ed. 2020:436–453.e1.)

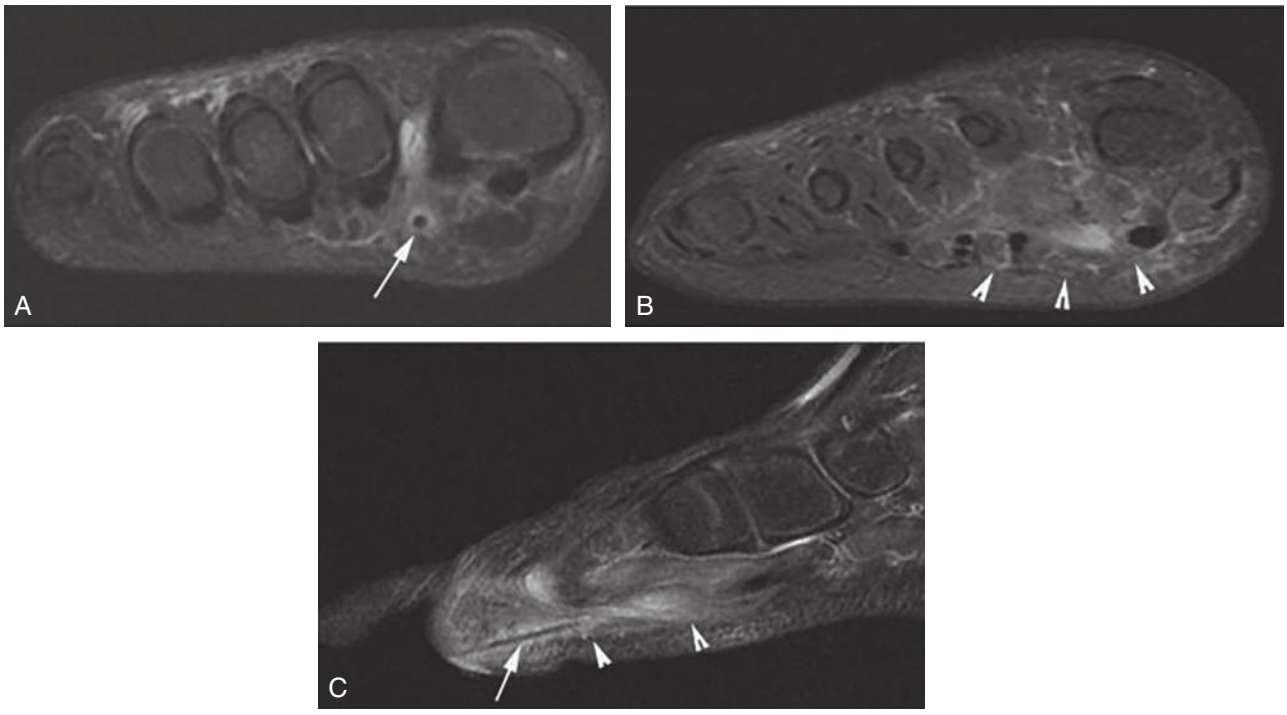


FIG 146.6 Forefoot foreign-body granuloma. A 35-year-old agricultural worker presented with 1-month history of metatarsalgia with unclear traumatic history. PD fat-saturated short-axis images **(A and B)** and sagittal image **(C)** showed a thin signal-void structure that is linear on sagittal image and rounded on short-axis images. It is surrounded by hyperintense halo and hyperintense signal of the surrounding soft tissues of the plantar aspect of first inter metatarsal space. A palm thorn was retrieved on surgical exploration. (From Nouh MR, El-Gawad EAA, Abdulsalam SM. MRI utility in patients with non-traumatic metatarsalgia: a tertiary musculoskeletal center observational study. *Egypt J Radiol Nucl Med*. 2015;46(4):1057–1064.)

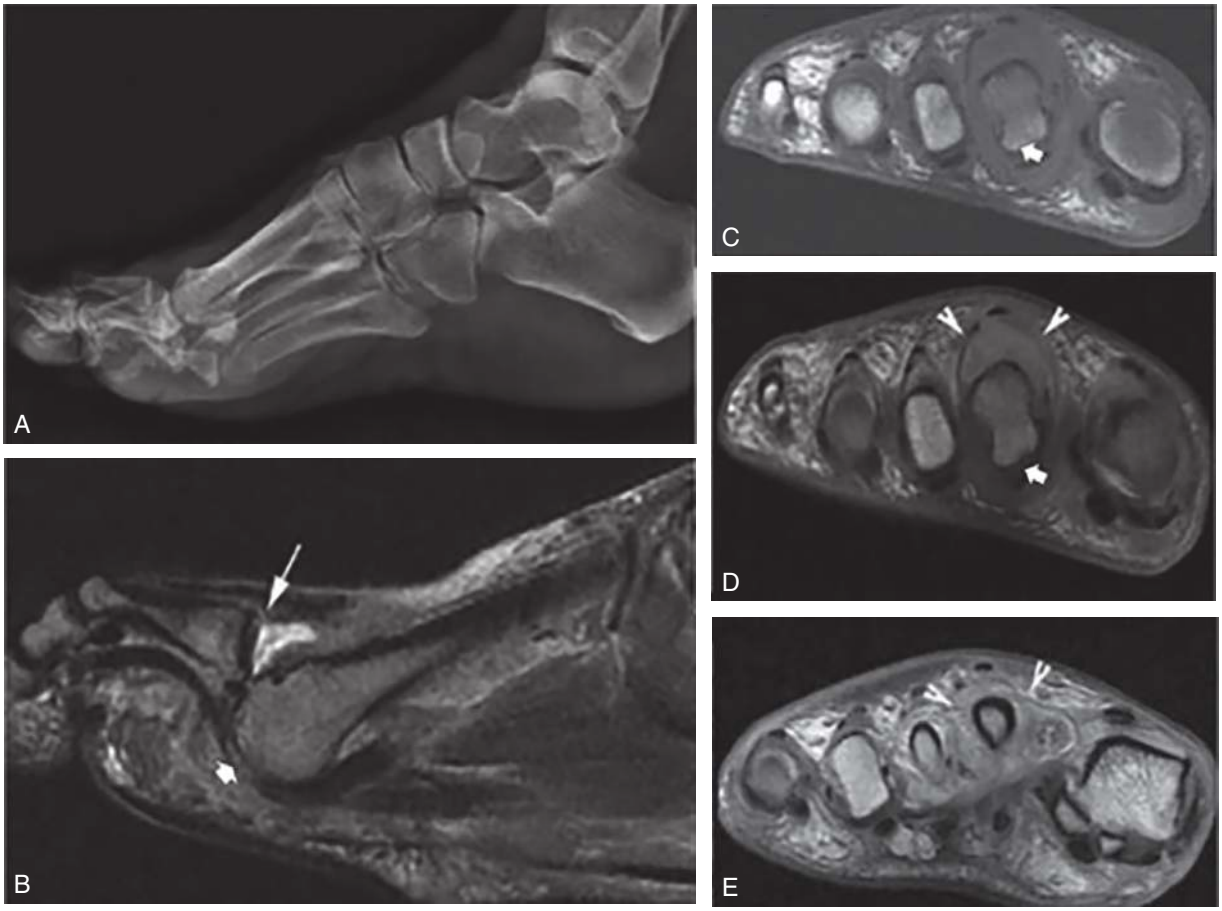


FIG 146.7 Plantar plate degeneration of the second metatarsophalageal (MTP) joint with subsequent second metatarsal ray dislocation syndrome. Lateral X-ray of the foot **(A),** sagittal T2-weighted image with fat saturation **(A),** short-axis T1-weighted images **(C)** of the forefoot; as well as postcontrast serial short-axis T1W images **(D and E)**; show nonvisualized plantar plate of the second MTP (*short-thick arrow*) due to its chronic degeneration with dorsal subluxation of the proximal phalanx, marked arthritic changes of the MTP (*long arrow* on **B**) with small dorsal osteophyte, joint effusion and edematous enhanced synovium as well as peri-articular soft tissues (*arrowheads* in **D** and **E**). (From Nouh MR, El-Gawad EAA, Abdulsalam SM. MRI utility in patients with non-traumatic metatarsalgia: a tertiary musculoskeletal center observational study. *Egypt J Radiol Nucl Med*. 2015;46(4):1057–1064.)

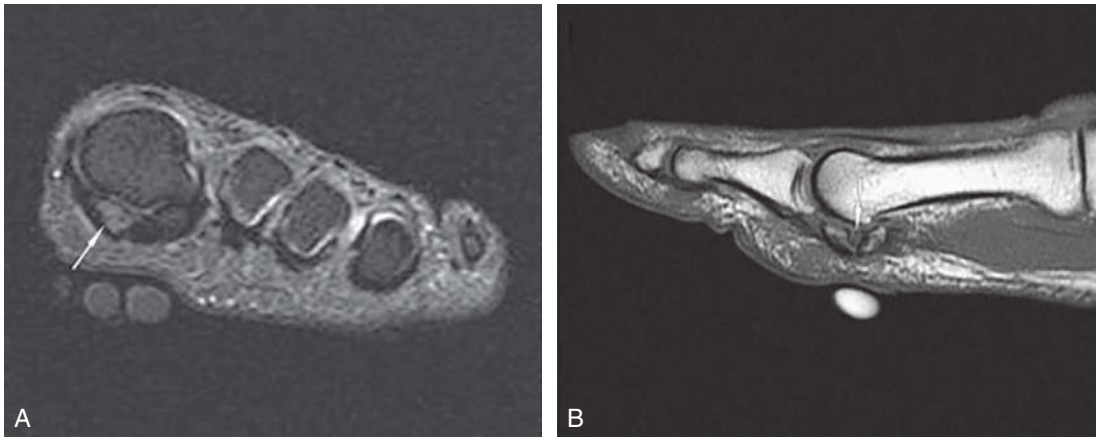


FIG 146.8 Nonunion of a tibial sesamoid fracture. Short-axis T2-weighted image with fat saturation **(A)** shows marrow edema *(arrow)* within the tibial sesamoid in a patient with chronic pain in this region. Sagittal T1-weighted image **(B)** shows a vertical fracture (*arrow*) with sclerotic margins of both fracture fragments indicating nonunion. (From Sanders TG, Rathur SK. Imaging of painful conditions of the hallucal sesamoid complex and plantar capsular structures of the first metatarsophalangeal joint. *Radiol Clin N Am*. 2008;46(6):1079–1092.)

SUGGESTED READINGS

Besse J-L. Metatarsalgia. *Orthop Traumatol Surg Res*. 2017;103 (suppl 1):S29–S39.

Federer AE, Tainter DM, Adams SB, Schweitzer KM. Conservative management of metatarsalgia and lesser toe deformities. *Foot Ankle Clin*. 2018;23(1):9–20.

Finney FT, Cata E, Holmes JR, Talusan PG. Anatomy and physiology of the lesser metatarsophalangeal joints. *Foot Ankle Clin*. 2018;23(1):1–7.

Gregg JM, Schneider T, Marks P. MR imaging and ultrasound of metatarsalgia: the lesser metatarsals. *Radiol Clin N Am*. 2008;46:1061–1078.

Umans HR. Imaging sports medicine injuries of the foot and toes. *Clin Sports Med*. 2006;25:763–780.

Welck MJ, Hayes T, Pastides P, et al. Stress fractures of the foot and ankle. *Injury*. 2017;48(8):1722–1726.

Waldman SD. Metatarsalgia. In: Waldman SD, ed. *Pain review*. 2nd ed. Philadelphia: Elsevier; 2016:317–318.

147

Sesamoiditis

ICD-10 CODE M25.871

THE CLINICAL SYNDROME

Sesamoiditis is one of the most common pain syndromes that affects the forefoot. It is characterized by tenderness and pain over the metatarsal heads. Although the first sesamoid bone of the first metatarsal head is affected most commonly, the sesamoid bones of the second and fifth metatarsal heads also are subject to the development of sesamoiditis (Fig. 147.1). The patient often feels that he or she is walking with a stone in his or her shoe (Fig. 147.2). The pain of sesamoiditis worsens with prolonged standing or walking for long distances and is exacerbated by improperly fitting or padded shoes. Sesamoiditis is most often associated with pushing-off injuries during football or repetitive microtrauma from running or dancing. The sesamoid bones are small, rounded structures that are embedded in the flexor tendons of the foot, usually in close proximity to the joints. Sesamoid bones of the first metatarsal occur in almost all patients, with sesamoid bones being present in the flexor tendons of the second and fifth metatarsals in a significant number of patients. These sesamoid bones decrease friction and pressure of the flexor tendon as it passes in proximity to a joint.

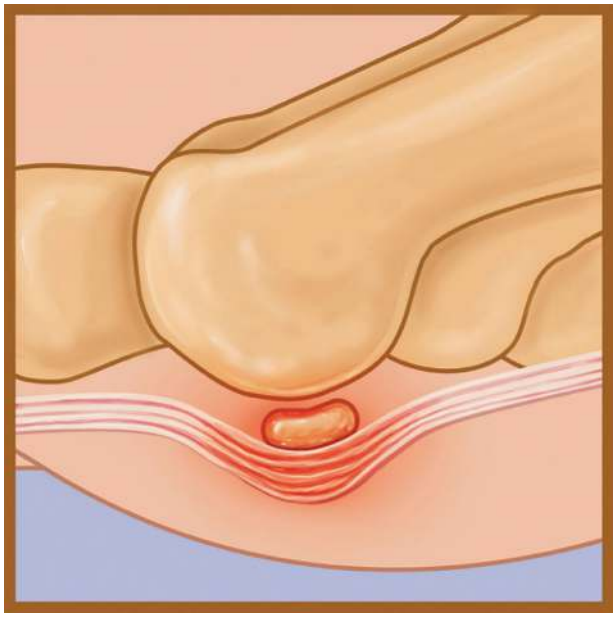


FIG 147.1 The sesamoid bones are small, rounded structures that are embedded in the flexor tendons of the foot and usually are in close proximity to the joints. Sesamoid bones of the first metatarsal occur in almost all patients, with sesamoid bones being present in the flexor tendons of the second and fifth metatarsals in a significant number of patients. (From Waldman S. *Atlas of pain management injection techniques*. 4th ed. St. Louis: Elsevier; 2017, Figure 181.1.)

SIGNS AND SYMPTOMS

The patient suffering from sesamoiditis will experience pain with any weight bearing that worsens with prolonged standing and walking. Stair climbing may become increasingly difficult as the inflammation increases. Wearing high heels, poorly fitting, and/or inadequately padded shoes will exacerbate the patient's functional disability and pain. Often, the patient with sesamoiditis will experience the sensation of walking on a stone in his or her shoe.

On physical examination, pain can be reproduced by pressure on the sesamoid bone. In contradistinction to metatarsalgia, in which the tender area remains over the metatarsal heads, with sesamoiditis the tender area moves with the flexor tendon when the patient actively flexes his or her toe. A callus overlying the affected sesamoid bone may be present (Fig. 147.3). The patient with sesamoiditis often exhibits an antalgic gait in an effort to reduce weight bearing during walking. With acute trauma to the sesamoid bone, ecchymosis over the plantar surface of the foot may be present.

TESTING

Plain radiographs, magnetic resonance, and ultrasound imaging are indicated in all patients who present with pain thought to be caused by sesamoiditis, to rule out occult bony disorders, fractures, and tumor (Figs. 147.4–147.6). Radionuclide bone scanning may rule out stress fractures not visible on plain radiographs (Fig. 147.7). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, comprehensive metabolic profile, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

Morton's neuroma and intermetatarsal bursitis are often confused with the inflammation and fractures of the sesamoid

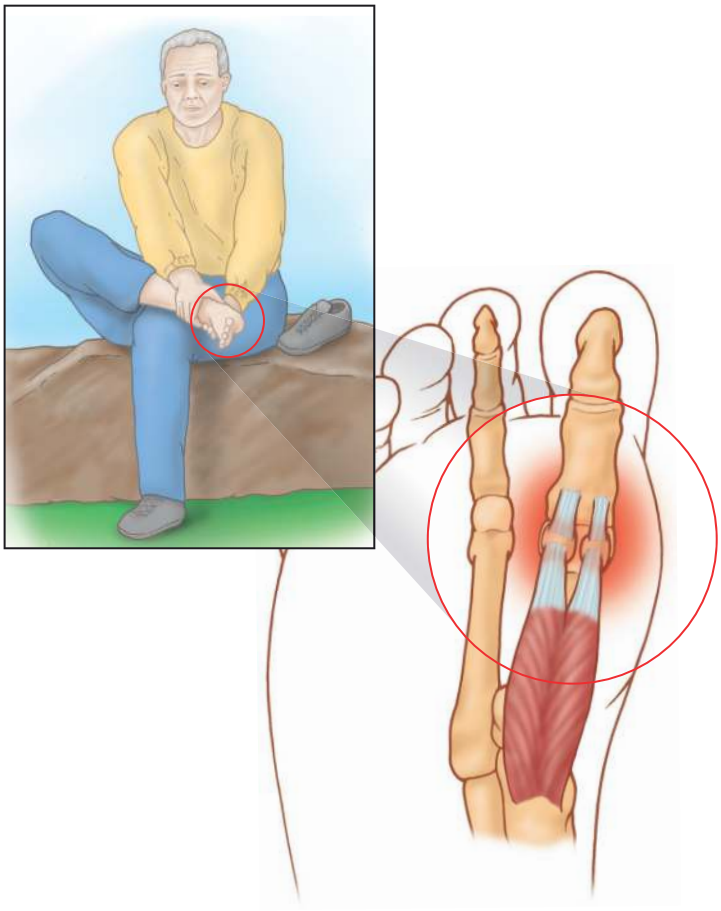


FIG 147.2 Patients with sesamoiditis often feel like they are walking with a stone in their shoe.

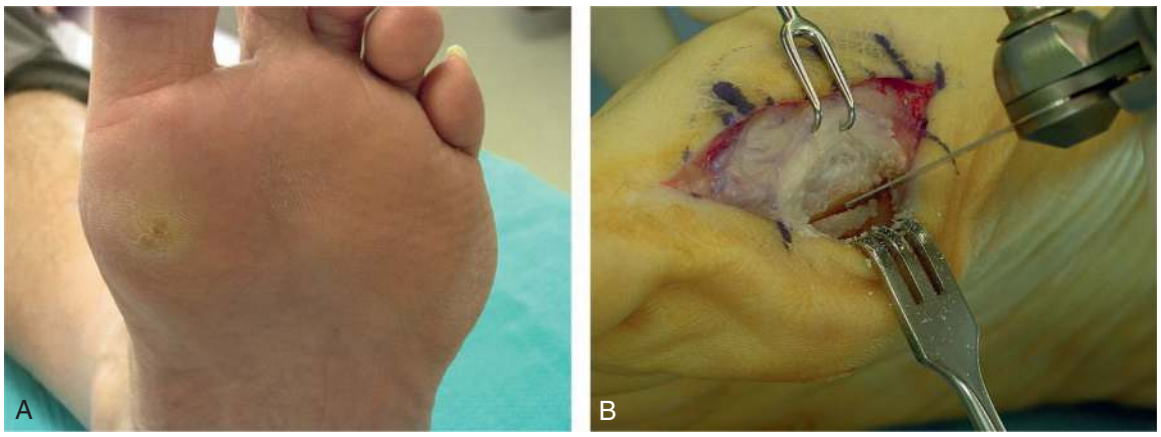


FIG 147.3 A, Clinical photograph of intractable plantar keratosis from tibial sesamoid. B, Operative photograph demonstrating technique of sesamoid shaving. A saw is used to resect a portion of prominent sesamoid plantar surface. (From Cohen BE. Hallux sesamoid disorders. *Foot Ankle Clin.* 2009;14(1):91–104.)

bones of the foot, although the pain of sesamoid fracture is localized to the plantar surface of the foot and is less neuritic than is that of Morton's neuroma. Tendinitis and metatarsal stress fractures of the foot can also mimic the pain of Morton's neuroma.

TREATMENT

Initial treatment of the pain and functional disability associated with sesamoiditis includes a combination of nonsteroidal antiinflammatory drugs (NSAIDs) or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat

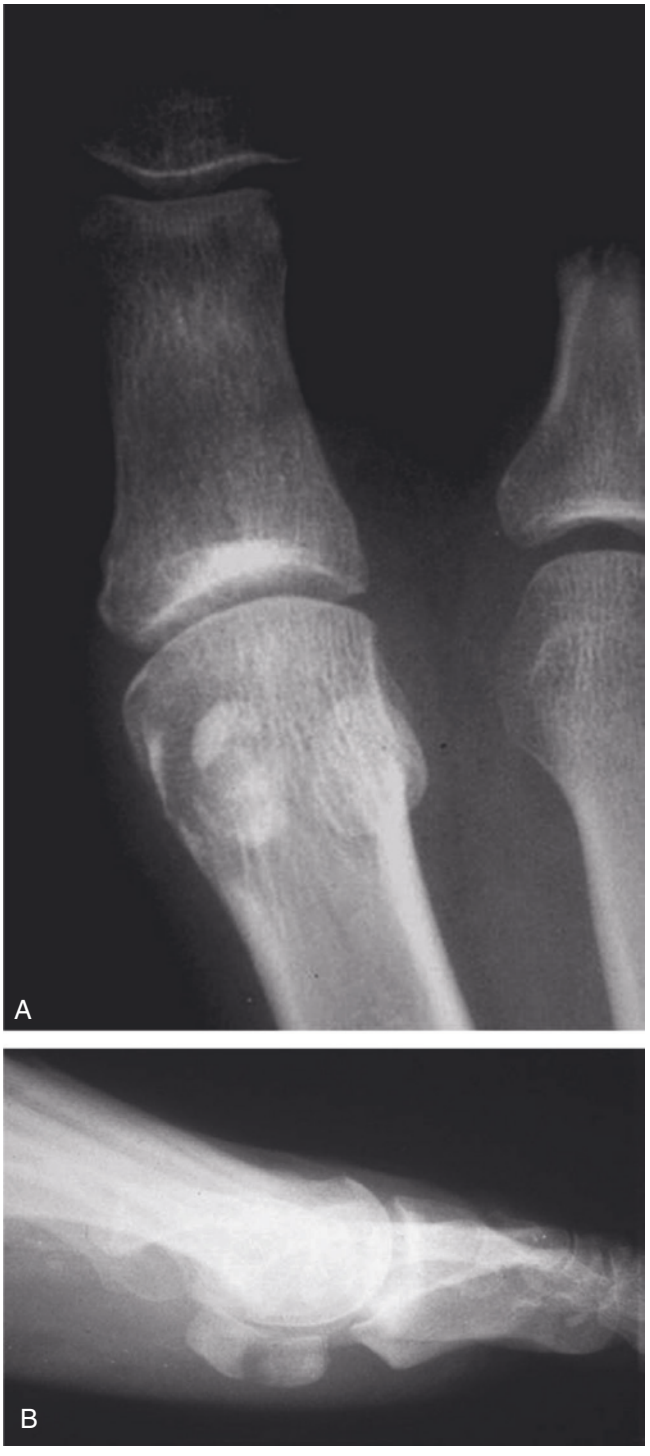


FIG 147.4 A, Sesamoid fractures. Anteroposterior radiograph of stress fracture of tibial sesamoid with diastasis. B, Lateral radiograph of stress fracture of tibial sesamoid with diastasis. (From Cohen BE. Hallux sesamoid disorders. *Foot Ankle Clin.* 2009;14(1):91–104.)

and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of walking barefoot or with shoes that do not provide good support, and short-term immobilization of the affected foot may provide relief. For patients who do not respond to these treat-

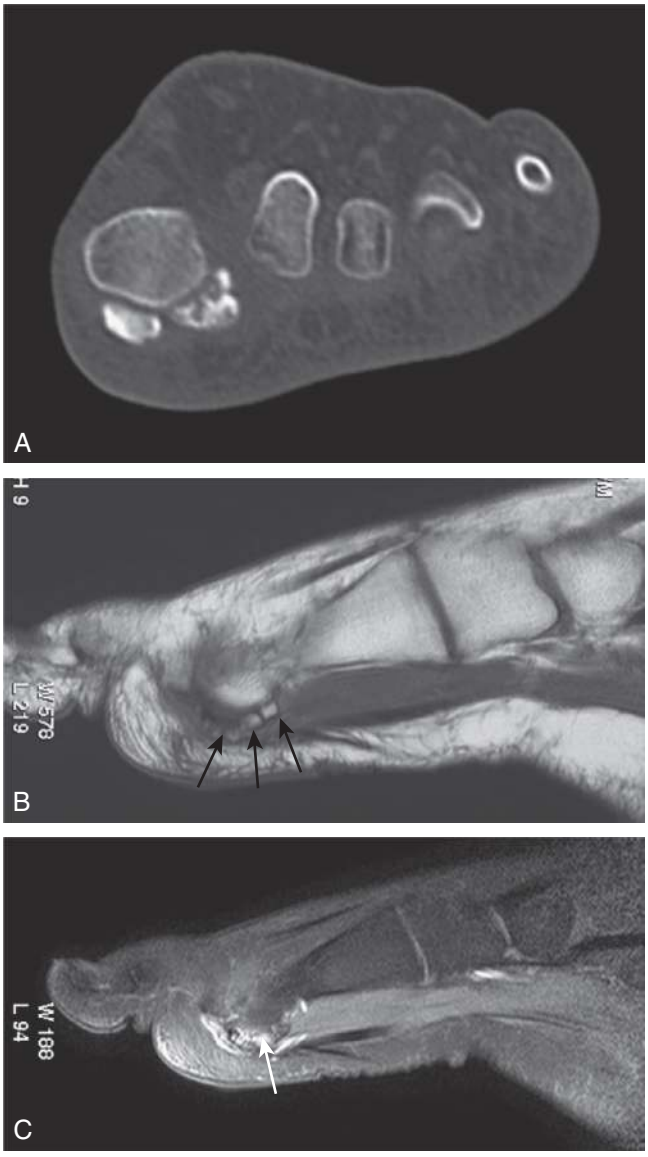


FIG 147.5 A, Coronal computerized tomography scan of a patient with sesamoiditis, demonstrating fragmentation and sclerosis of the lateral sesamoid. B, The sagittal T1W magnetic resonance (MR) image also demonstrates the bony fragmentation (*black arrows*). C, High-signal intensity (SI) reactive edema is present on the corresponding fat-suppressed T2-weighted MR image (*white arrow*). (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia, Elsevier; 2011.)

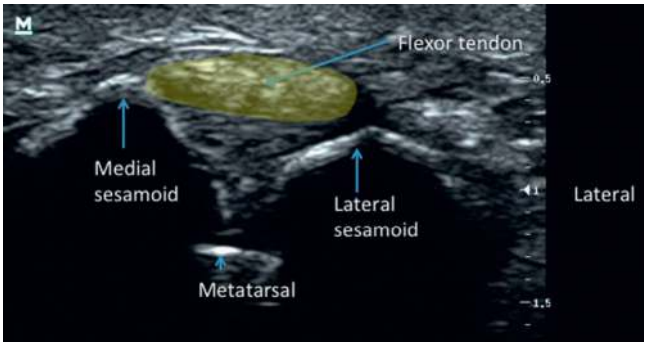


FIG 147.6 Ultrasound image demonstrating the lateral and medial sesamoids overlying the metatarsals.

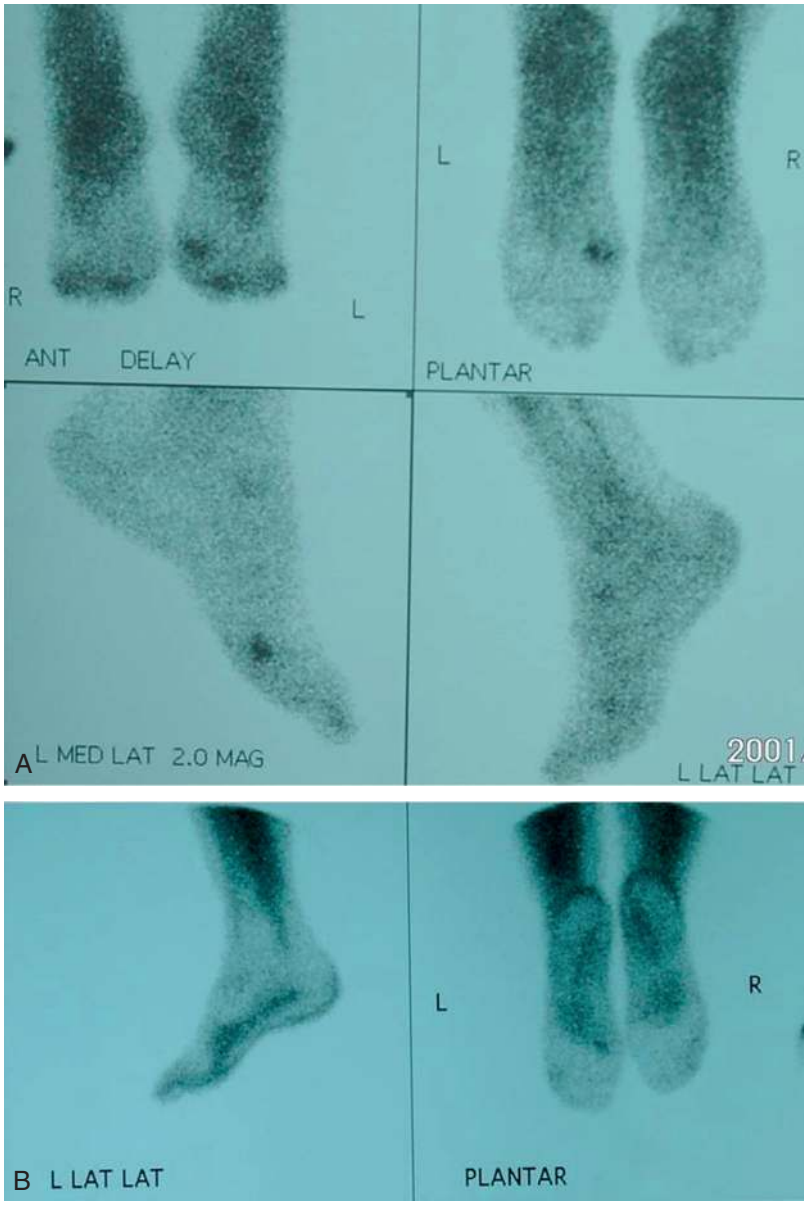


FIG 147.7 A, B. Sesamoiditis. Bone scan with technetium 99m-methyl diphosphonate with localized views of the forefoot demonstrating sesamoiditis.

ment modalities, injection with local anesthetic and steroid is a reasonable next step.

To perform an injection for plantar fasciitis, the patient is placed in the supine position, and the skin overlying the tender sesamoid bone is properly prepared with antiseptic solution. A sterile syringe containing 2.0 mL of 0.25% preservative-free bupivacaine and 40 mg of methylprednisolone is attached to a ⅝-inch, 25-gauge needle using strict aseptic technique. With strict aseptic technique, the affected sesamoid bones are identified. At this point, the needle is carefully advanced through the plantar surface of the foot until the needle tip rests against the sesamoid bone (Fig. 147.8). The needle is withdrawn slightly out of the periosteum and substance of the tendon. After the needle is in the correct position next to the affected sesamoid bone and aspiration for blood is negative, the contents of the syringe are gently injected. There may be slight resistance to injection, given the closed nature of the space. If significant resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection proceeds without significant resistance. The needle is then removed, and a sterile pressure dressing and ice pack are placed at the injection site. The ice should not be left on for more than 10 minutes to avoid freezing injuries. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle stretching exercises, should be introduced several days after the patient undergoes injection. Vigorous exercises should be avoided, because they will exacerbate the patient's symptoms. Metatarsal pads or molded orthotic devices may also be of value. Low-energy extracorporeal shockwave therapy may also provide symptomatic relief in resistant cases. Simple analgesics, NSAIDs, and antimyotonic agents, such as tizanidine, can be used concurrently with this injection technique.

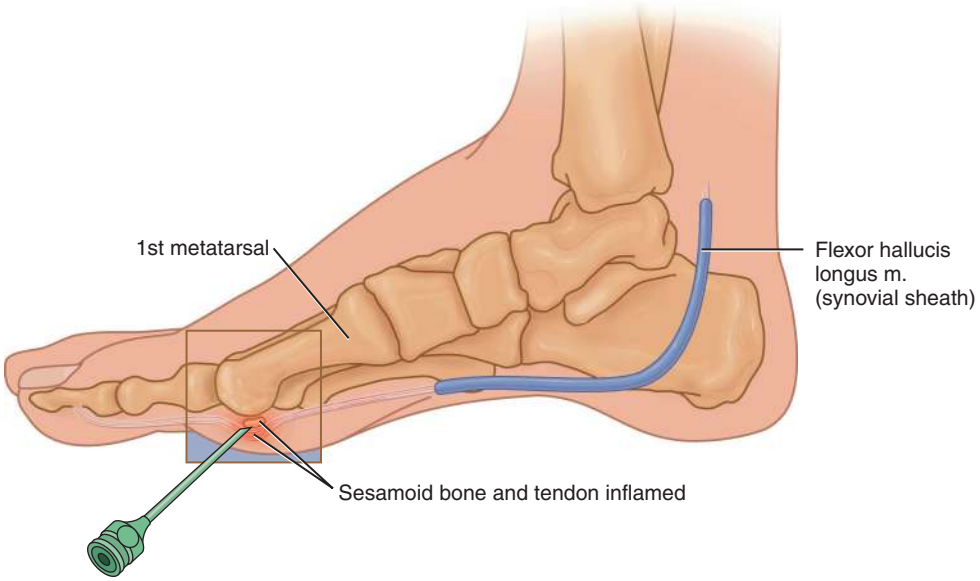


FIG 147.8 Proper needle position for sesamoid bone injection of the foot. (From Waldman SD. *Atlas of pain management injection techniques*. 5th ed. Philadelphia, Elsevier; 2017:680.)

COMPLICATIONS AND PITFALLS

Most complications of injection are related to needle-induced trauma at the injection site and in the underlying tissues. Many patients complain of a transient increase in pain after injection, which can be minimized by injecting gently and slowly. Infection, although rare, may occur if sterile technique is not followed.

CLINICAL PEARLS

Pain emanating from the forefoot is a common problem that is encountered in clinical practice. Sesamoiditis must be distinguished from stress fractures of the metatarsal bones, metatarsalgia, Morton's neuroma, and fractures of the sesamoid bones. Although the just-described injection technique provides palliation of the pain of sesamoiditis, the patient often also requires shoe orthoses, including padded insoles, to help remove pressure from the affected sesamoid bones. Coexistent bursitis and tendinitis also may contribute to metatarsal pain and may require additional treatment with more localized injection of local anesthetic and depot corticosteroid preparation. This technique is safe if careful attention is paid to the clinically relevant anatomy in the areas to be injected. Care must be taken to use sterile technique to avoid infection; universal precautions should be used to avoid risk to the operator. The incidence of ecchymosis and hematoma formation can be decreased if pressure is placed on the injection site immediately after injection. The use of physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient has undergone this injection technique for sesamoiditis pain. Vigorous exercise should be avoided because it exacerbates the patient's symptoms. Simple analgesics and NSAIDs may be used concurrently with this injection technique.

SUGGESTED READINGS

Cohen BE. Hallux sesamoid disorders. *Foot Ankle Clin*. 2009;14(1):91–104.

Kulemann V, Mayerhoefer M, Trnka HJ, et al. Abnormal findings in hallucal sesamoids on MR imaging—associated with different pathologies of the forefoot? An observational study. *Eur J Radiol*. 2010;74(1):226–230.

Schein AJ, Skalski MR, Patel DB, et al. Turf toe and sesamoiditis: what the radiologist needs to know. *Clin Imaging*. 2015;39(3):380–389.

Waldman SD. Sesamoiditis. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1179–1186.

Waldman SD. Injection technique for sesamoiditis pain. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:678–681.

Waldman SD, Campbell RSD. Sesamoiditis. In: *Imaging of pain*. Philadelphia: Saunders; 2011:454–456.

Calcaneal Spur Syndrome

ICD-10 CODE M77.30

THE CLINICAL SYNDROME

Calcaneal spurs are a common cause of heel pain. They can occur anywhere along the calcaneal tuberosity but are most frequently found at the insertion of the plantar fascia (Fig. 148.1). Calcaneal spurs are usually asymptomatic, but when they are painful, the condition is generally the result of inflammation of the insertional fibers of the plantar fascia at the medial tuberosity. Symptomatic calcaneal spurs are often found in association with plantar fasciitis. Like plantar fasciitis, calcaneal spurs can occur alone or may be part of a systemic inflammatory condition such as rheumatoid arthritis, Reiter's syndrome, or gout. In some patients, the cause seems to be entirely mechanical, and such patients often exhibit an abnormal gait with excessive heel strike. High-impact aerobic exercise has also been implicated in the development of calcaneal spur syndrome (Fig. 148.2).

SIGNS AND SYMPTOMS

The pain of calcaneal spurs is most severe when first walking after a period of non–weight bearing and is made worse by prolonged standing or walking. On physical examination, patients exhibit point tenderness over the plantar medial calcaneal tuberosity; they may also have tenderness along the plantar fascia as it moves anteriorly. The pain of calcaneal spurs is increased by weight bearing and is relieved by padding the affected heel.

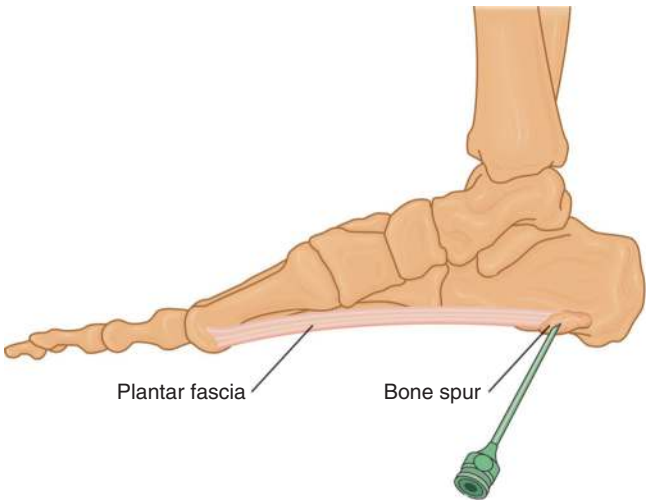


FIG 148.1 Calcaneal spurs commonly form at the insertion of the plantar fascia on the medial calcaneal tuberosity. (From Waldman SD. *Atlas of pain management injection techniques.* 2nd ed. Philadelphia: Saunders; 2007:554.)

TESTING

Plain radiographs are indicated in all patients who present with pain thought to be caused by calcaneal spurs, to confirm the diagnosis, as well as to rule out fracture of the spur, occult bony disorders, and tumor. Characteristic radiographic changes are lacking, but radionuclide bone scanning may show increased uptake at the point where the plantar fascia attaches to the medial calcaneal tuberosity (Fig. 148.3). Magnetic resonance imaging (MRI) and ultrasound imaging of the foot are indicated if calcaneal spurs, an occult mass, or a tumor is suspected (Figs. 148.4 and 148.5). MRI, ultrasound imaging, and radionuclide bone scanning may also be useful to exclude stress fractures and other occult findings not visible on plain radiographs (Fig. 148.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, prostate-specific antigen level, erythrocyte sedimentation rate, and antinuclear antibody testing. The injection technique described later serves as both a diagnostic and a therapeutic maneuver.

DIFFERENTIAL DIAGNOSIS

The pain of calcaneal spurs is often confused with that of plantar fasciitis, although the characteristic pain on dorsiflexion of the toes associated with plantar fasciitis should distinguish between these conditions. Stress fractures of the calcaneus, bursitis, and tendinitis can also confuse the clinical picture.

TREATMENT

Initial treatment of the pain and functional disability associated with calcaneal spur syndrome includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, combined with short-term immobilization of the affected heel, may provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

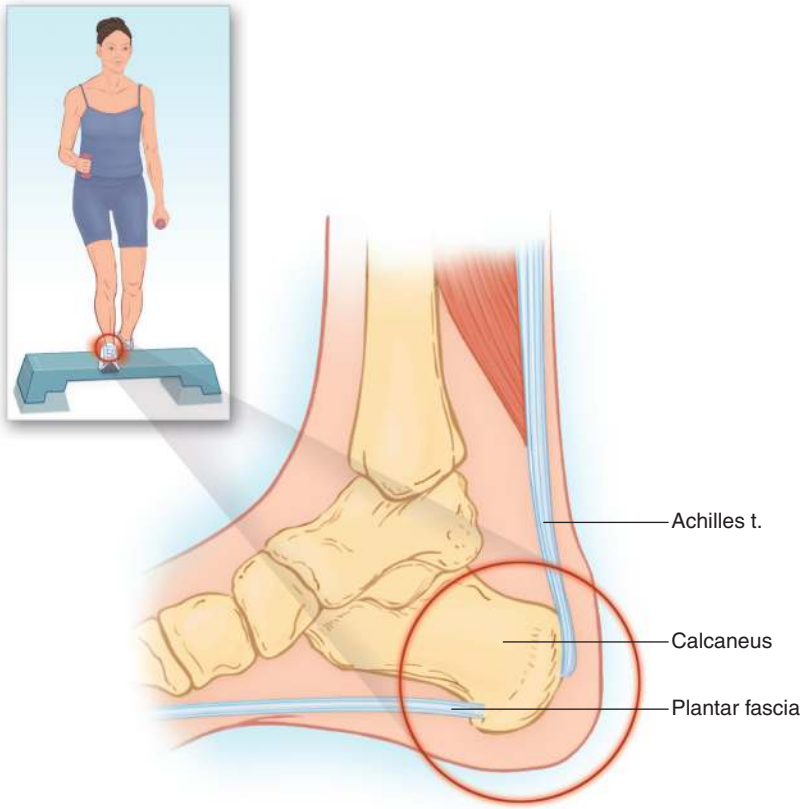


FIG 148.2 High-impact aerobic exercise has been implicated in the development of calcaneal spurs.

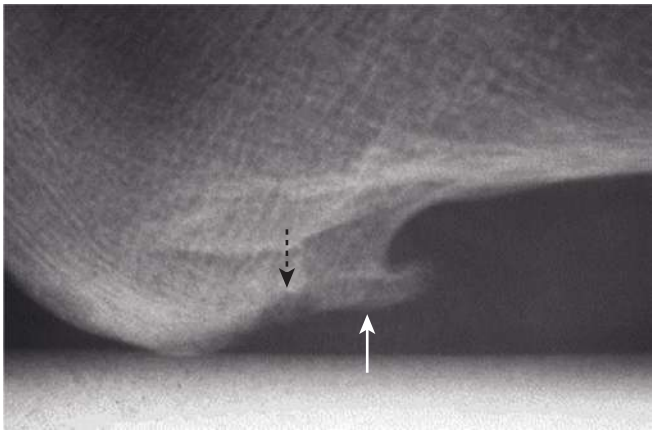


FIG 148.3 Close-up view of a heel spur demonstrated on a lateral weight-bearing radiograph. The *dashed arrow* indicates a small "saddle injury." The *solid arrow* indicates a subtle fracture line. (From Smith S, Tinley P, Gilheany M, et al. The inferior calcaneal spur: anatomical and histological considerations. *Foot*. 2007;17(1):25–31.)

To inject the calcaneal spur, the patient is placed in the supine position. The painful area of the heel overlying the calcaneal spur is identified by palpation, and the skin overlying this point is prepared with antiseptic solution. A syringe containing 2 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a 1½-inch, 25-gauge

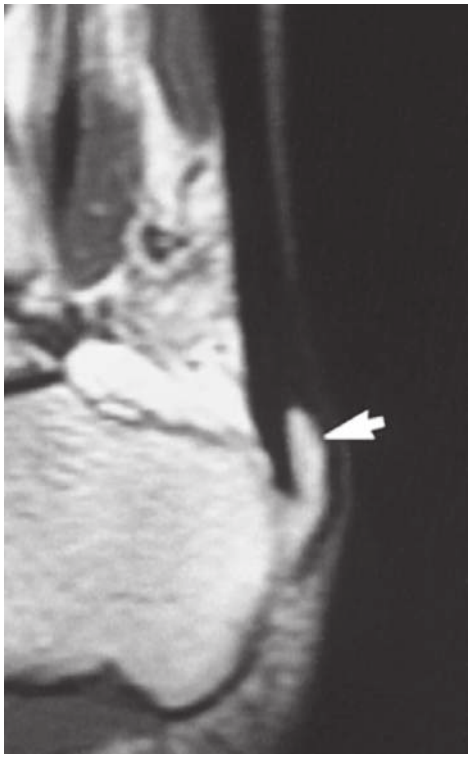


FIG 148.4 Sagittal T1-weighted spin-echo magnetic resonance image shows a large marrow-containing calcaneal enthesophyte arising at the insertion of the Achilles tendon (*arrow*). (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:555.)

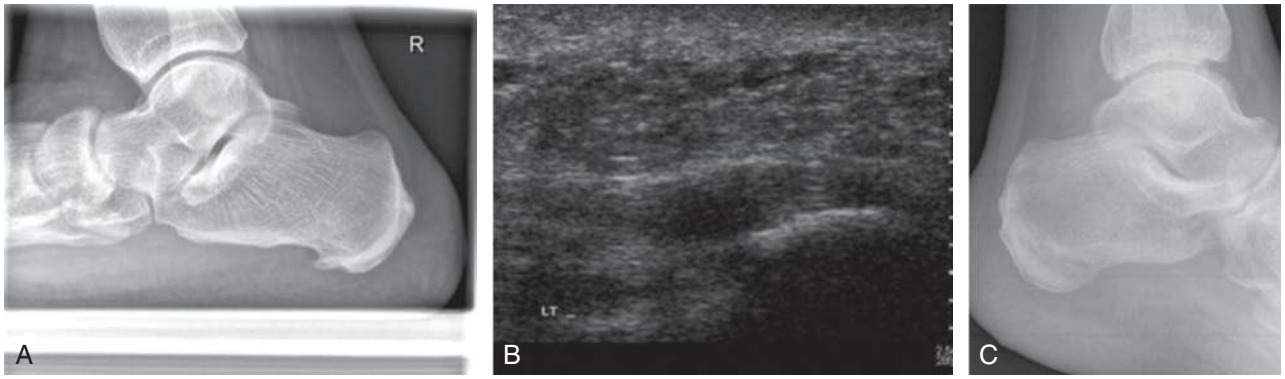


FIG 148.5 The assessment of chronic heel pain. **A,** A lateral calcaneal radiograph demonstrating a calcaneal spur. Although this is associated with plantar fasciitis, it can occur in asymptomatic individuals; if conformation of the diagnosis is required, it is much better to demonstrate the plantar fascia directly with ultrasound (US). **B,** Thickening of the plantar fascia in plantar fasciitis. US can be used to direct a steroid injection into the affected area. **C,** Lateral calcaneal radiograph showing a sclerotic area in a typical site for a calcaneal stress fracture. If a radiograph is performed in chronic heel pain, it should be done to exclude other causes of pain, such as a stress fracture, rather than to demonstrate a calcaneal spur. (From Rankine JJ. Imaging of foot and ankle disorders. *Orthop Trauma*. 2009;23(6):412–419.)

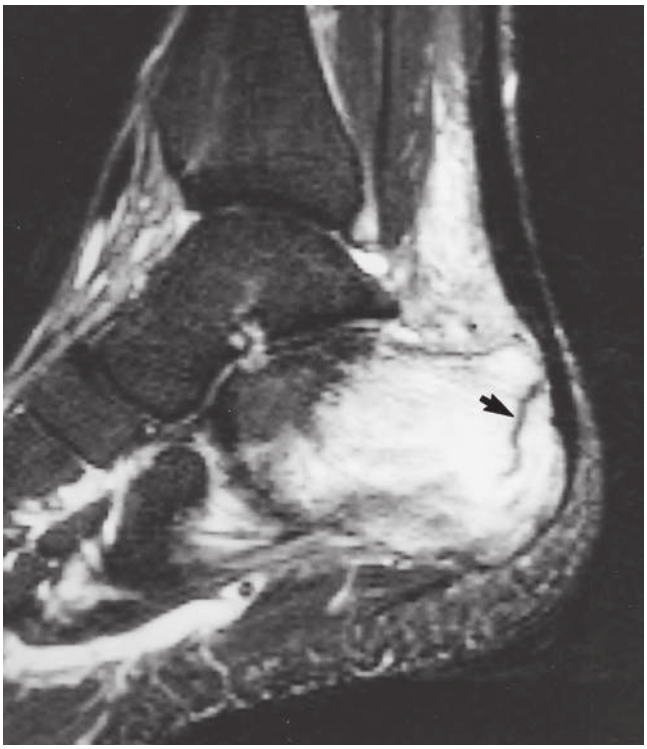


FIG 148.6 Calcaneal stress fracture. In a marathon runner, a sagittal short tau inversion recovery magnetic resonance image shows a fatigue fracture (*arrow*) of the calcaneus, with extensive marrow edema and slight thickening of the Achilles tendon. Abnormal high signal intensity anterior to this tendon indicates peritendinitis. (From Resnick D. *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: Saunders; 2002:2661.)

needle. The needle is slowly advanced through the previously identified point at a right angle to the skin, directly toward the center of the painful area, until it impinges on bone. The needle is then withdrawn slightly out of the periosteum, and the contents of the syringe are gently injected as the needle is slowly withdrawn. The operator should feel slight resistance to injection, given the closed nature of the heel. Ultrasound needle guidance will improve the accuracy of needle placement and decrease the incidence of needle-related complications.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection. A heel cup and gel padding of the posterior foot may also provide symptomatic relief.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis, plantar fasciitis, and tendinitis may contribute to the patient's foot pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of calcaneal spur syndrome, and it is safe if careful attention is paid to the clinically relevant anatomy.

SUGGESTED READINGS

Alatassi R, Alajlan A, Almalki T. Bizarre calcaneal spur: a case report. *Int J Surg Case Rep*. 2018;49:37–39. https://doi.org/10.1016/j.ijscr.2018.06.006.

Chang CD, Wu JS. MR imaging findings in heel pain. *Magn Reson Imaging Clin N Am*. 2017;25(1):79–93.

Johal KS, Milner SA. Plantar fasciitis and the calcaneal spur: fact or fiction? *Foot Ankle Surg*. 2012;18(1):39–41.

Rankine JJ. Imaging of foot and ankle disorders. *Orthop Trauma*. 2009;23(6):412–419.

Waldman SD. Calcaneal spurs. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1020–1029.

Waldman SD. Injection technique for plantar fasciitis. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:652–654.

Mallet Toe

ICD-10 CODE G57.00

THE CLINICAL SYNDROME

Mallet toe is a painful flexion deformity of the distal interphalangeal joint (Fig. 149.1). The second toe is affected most commonly, although the deformity may occur in all toes (Fig. 149.2). Mallet toe is usually the result of a jamming injury to the toe. However, as with bunion and hammer toe, the wearing of tight, narrow-toed shoes has also been implicated (Fig. 149.3); also like bunion, mallet toe occurs more commonly in female patients than in male patients. An inflamed adventitious bursa may accompany mallet toe and contribute to the patient's pain. A callus or ulcer overlying the tip of the affected toe may be present as well. High-heeled shoes may exacerbate the problem.

SIGNS AND SYMPTOMS

Most patients complain of pain localized to the distal interphalangeal joint and an inability to get shoes to fit. Walking makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching. Some patients complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical

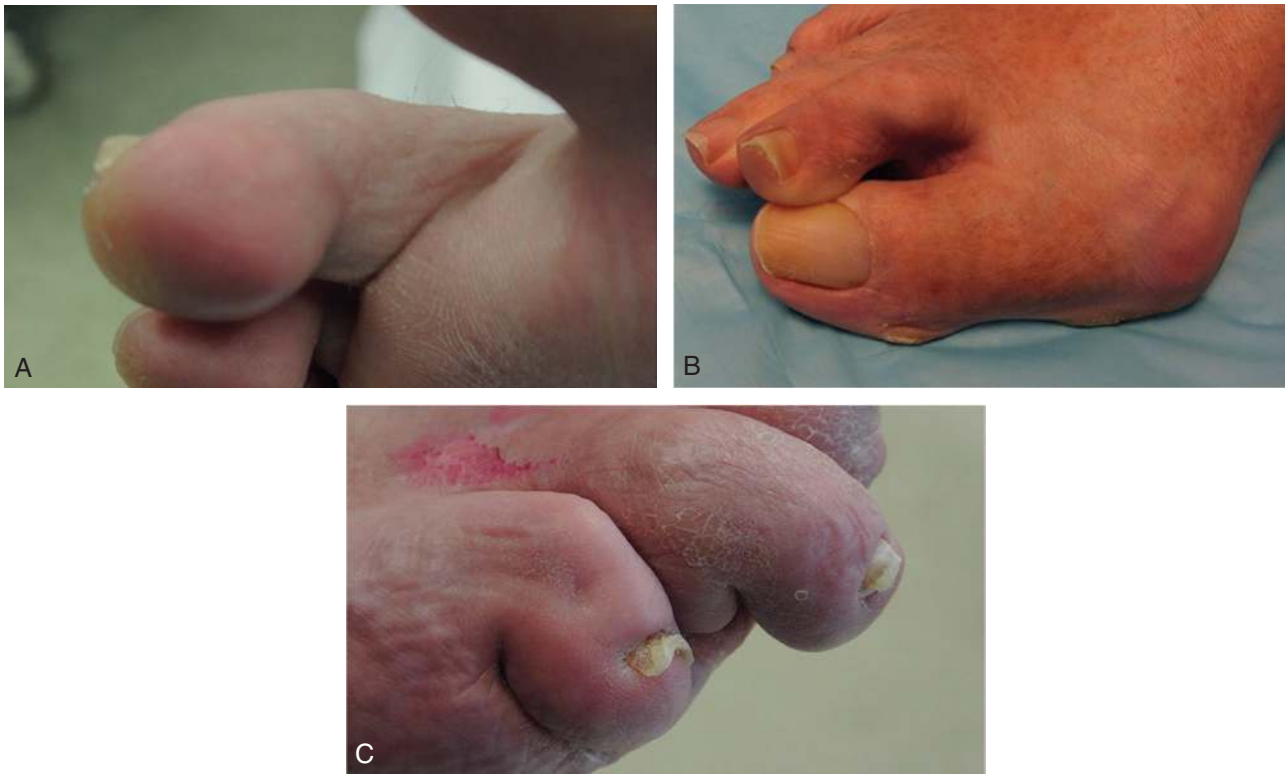


FIG 149.1 **A,** Clinical appearance of a mallet toe. There is a flexion deformity at the distal interphalangeal (DIP) joint. The proximal interphalangeal (PIP) and metatarsophalangeal (MTP) joints are in a neutral position. **B,** Clinical appearance of a hammer toe. There is extension of the MTP joint, flexion at the PIP joint, and extension of the DIP joint. It is associated with a hallux valgus deformity. **C,** Clinical appearance of claw toes in a patient with diabetes mellitus. There is extension of the MTP joint and flexion at the PIP and DIP joints. (From DiPreta JA. Metatarsalgia, lesser toe deformities, and associated disorders of the forefoot. *Med Clin N Am.* 2014;98(2):233–251.)

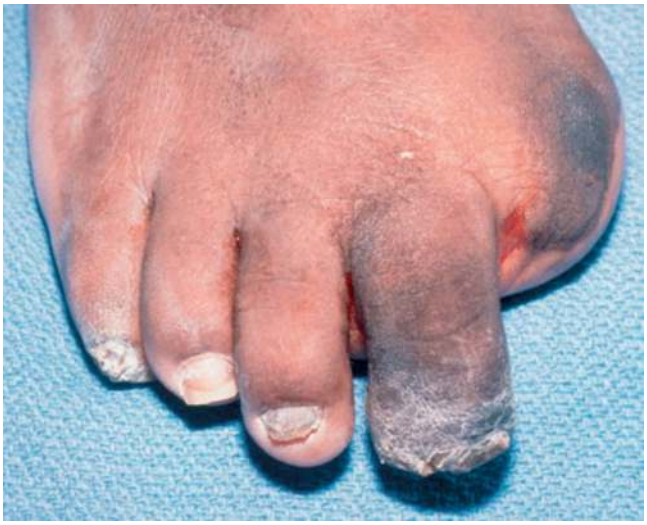


FIG 149.2 Foot of a diabetic patient with osteomyelitis of the distal phalanx of an insensate second mallet toe (distal interphalangeal [DIP] joint contracture). Disarticulation at the DIP joint removed the infective focus and shortened the prominent toe. Previous metatarsophalangeal joint disarticulation of the great toe had exposed the second toe to distal trauma from a shoe. (From Bowker JH, Pfeifer MA, eds. *Levin and O'Neal's the diabetic foot.* 7th ed. Philadelphia: Mosby; 2008:403–428.)

examination. In addition to pain, patients with mallet toe develop a characteristic flexion deformity of the distal interphalangeal joint. Unlike with bunion, alignment of the toes is relatively normal.

FIG 149.4 Mallet toe. Lateral radiograph depicting severe flexion of the distal interphalangeal joint causing tip of nail unit to be traumatized. (From Ceccarini P, Ceccarini A, Rinonapoli G, Caraffa A. Correction of hammer toe deformity of lateral toes with subtraction osteotomy of the proximal phalanx neck. *J Foot Ankle Surg*. 2015;54(4):601–606.)

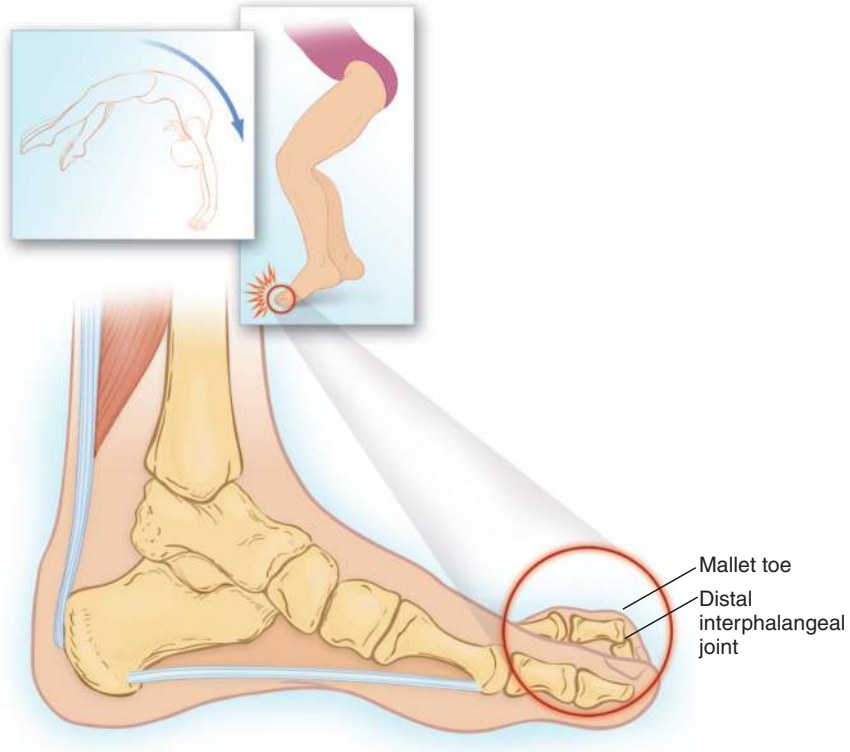


FIG 149.3 Mallet toe is usually the result of a jamming injury to the second toe. It is often seen in gymnasts, although the wearing of tight, narrow-toed shoes has also been implicated in its development.

TESTING

Plain radiographs and ultrasound imaging are indicated in all patients who present with mallet toe (Figs. 149.4 and 149.5). Magnetic resonance imaging of the toe is indicated if joint instability, an occult mass, or a tumor is suspected (Fig. 149.6). Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

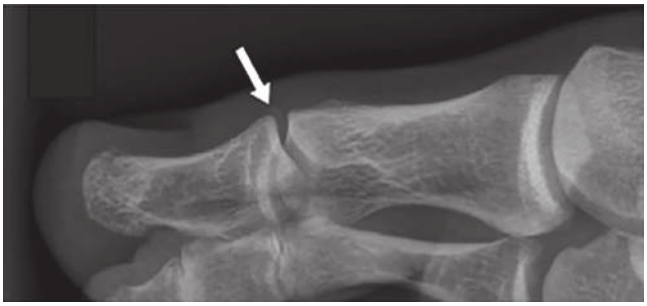


FIG 149.5 Magnified oblique radiograph of the foot demonstrates faint soft tissue mineralization at the dorsomedial base of the first distal phalanx (*arrow*) suggestive of an avulsion fracture or periosteal new bone formation related to a high-grade tendinous avulsion. (From Biondetti P, Dalstrom DJ, Ilfeld B, Smitaman E. Mallet hallux injury: a case report and literature review. *Clin Imaging*. 2020;62:33–36.)

DIFFERENTIAL DIAGNOSIS

The diagnosis of mallet toe is usually obvious on clinical grounds alone. Bursitis and tendinitis of the foot and ankle frequently coexist with mallet toe. In addition, stress fractures of the metatarsals, phalanges, or sesamoid bones may confuse the clinical diagnosis and require specific treatment.

TREATMENT

Initial treatment of the pain and functional disability associated with mallet toe includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of narrow-toed or high-heeled shoes, and short-term immobilization of the affected toes may provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

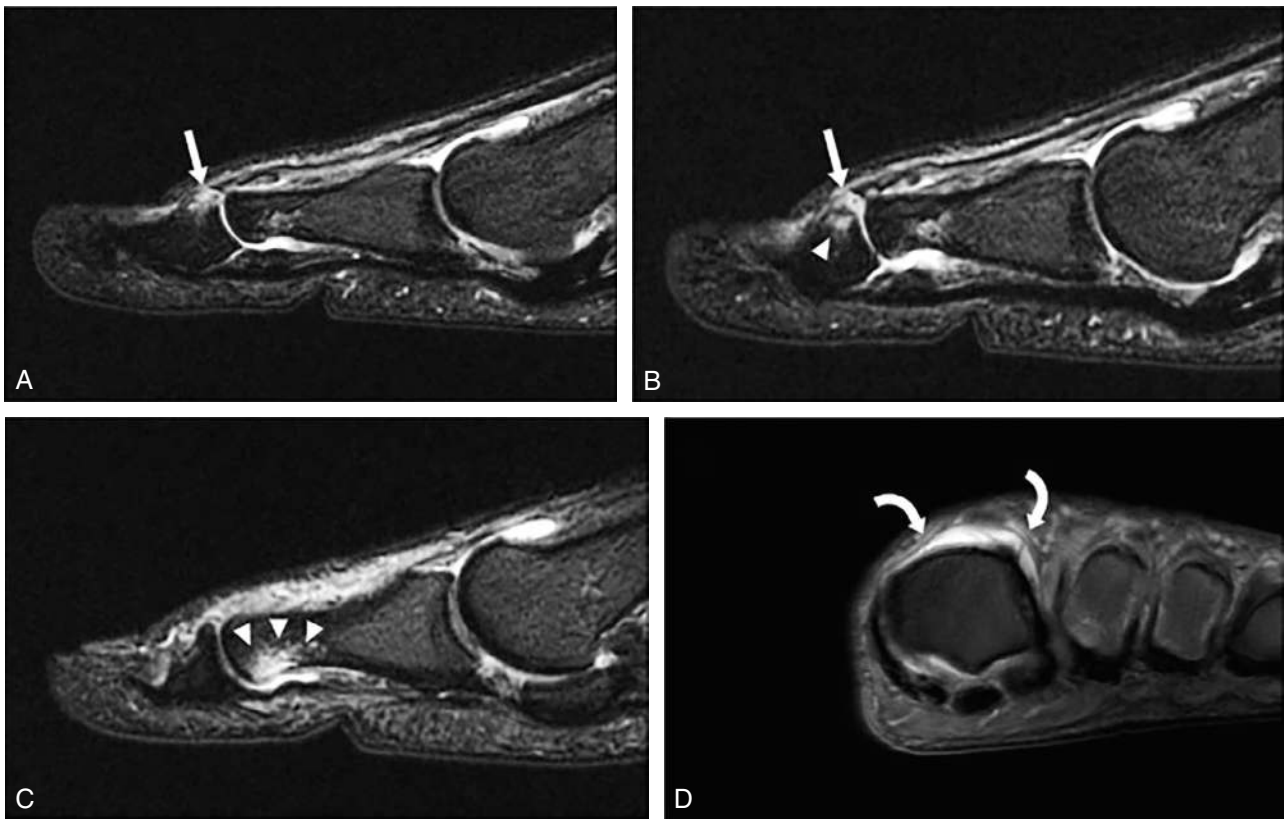


FIG 149.6 Sagittal (**A–C**) and short axis (**D**) T2-weighted fat-suppressed images of the great toe demonstrate extensor hallucis longus (EHL) tendon tear (*arrows* in **A** and **B**) with approximately 2 mm retraction of the torn tendon fibers, foci of marrow edema (*arrowheads*) at the dorsal base of the first distal phalanx (**B**) and plantar neck of the first proximal phalanx (**C**) supportive of a hyperflexion mechanism of injury, and disruption of the dorsal capsule at the first interphalangeal joint (*curved arrows* in **D**). (From Biondetti P, Dalstrom DJ, Ilfeld B, Smitaman E. Mallet hallux injury: a case report and literature review. *Clin Imaging*. 2020;62:33–36.)

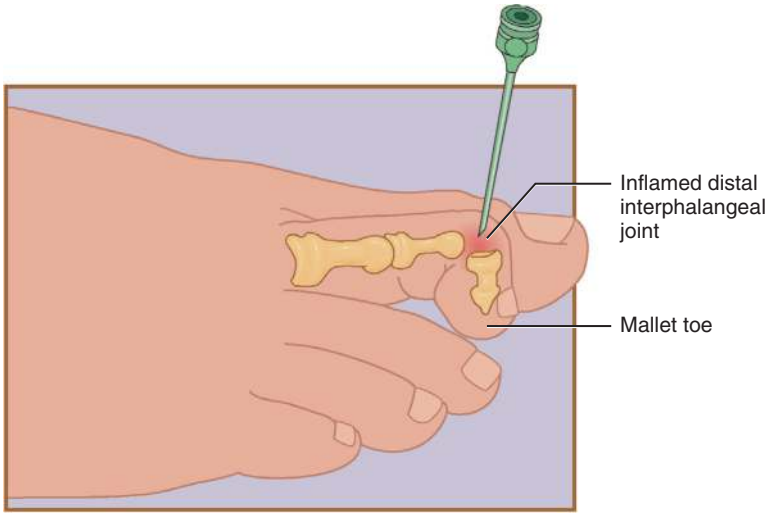


FIG 149.7 Injection technique for mallet toe. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:357.)

To inject mallet toe, the patient is placed in the supine position, and the skin overlying the affected toe is prepared with antiseptic solution. A sterile syringe containing 1.5 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a ⅝-inch, 25-gauge needle using strict aseptic technique. The mallet toe is identified, and the needle is carefully advanced against the affected distal phalanges (Fig. 149.7). The needle is then withdrawn slightly out of the periosteum, and the contents of the syringe are gently injected. The operator should feel little resistance to injection. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site.

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's foot pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of mallet toe, and it is safe if careful attention is paid to the clinically relevant anatomy. Narrow-toed and high-heeled shoes should be avoided, because they will exacerbate the patient's symptoms.

SUGGESTED READINGS

DiPreta JA. Metatarsalgia, lesser toe deformities, and associated disorders of the forefoot. *Med Clin North Am*. 2014;98(2):233–251.

Molloy A, Shariff R. Mallet toe deformity. *Foot Ankle Clin*. 2011;16(4):537–546.

Montgomery HC, Davies MB. Common disorders of the adult foot and ankle. *Surgery (Oxford)*. 2016;34(9):475–481.

Waldman SD. Hammer toe, claw toe, and mallet toe pain syndromes. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1055–1062.

Waldman SD. Injection technique for mallet toe. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:666–667.

Hammer Toe

ICD-10 CODE M20.40

THE CLINICAL SYNDROME

Hammer toe is a painful flexion deformity of the proximal interphalangeal joint in which the middle and distal phalanges are flexed down onto the proximal phalange (Fig. 150.1). The second toe is affected most often, and the condition is usually bilateral. Like hallux valgus deformity, hammer toe is usually the result of wearing shoes that are too tight in the toe box, although trauma has also been implicated (Fig. 150.2). As with bunion, hammer toe deformity occurs more commonly in females than in males. An inflamed adventitious bursa may accompany hammer toe and contribute to the patient's pain (Fig. 150.3). A callus overlying the plantar surface of these bony prominences is usually present as well. High-heeled shoes may exacerbate the problem.

SIGNS AND SYMPTOMS

Most patients complain of pain localized to the proximal interphalangeal joint and an inability to get shoes to fit. Walking makes the pain worse, whereas rest and heat provide some relief. The pain is constant and is characterized as aching; it may interfere with sleep. Some patients complain of a grating or popping sensation with use of the joint, and crepitus may be present on physical examination. In addition to pain, patients with hammer toe develop a characteristic flexion deformity of the proximal interphalangeal joint.

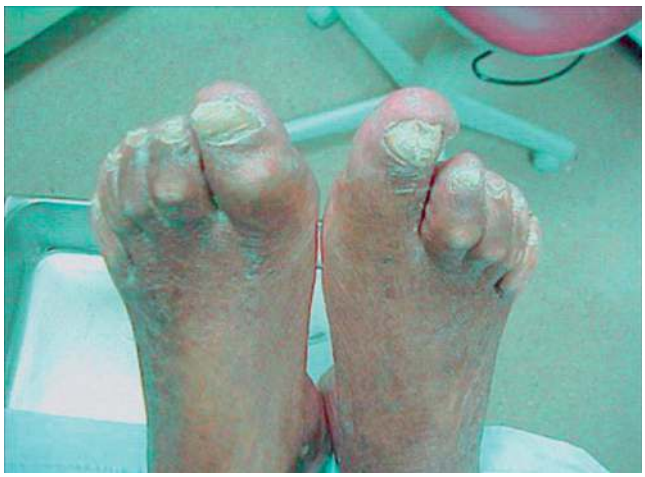


FIG 150.1 Patient with multiple semi-rigid hammertoes. Note mycotic nail changes whose causation may have been contributed to by constant trauma at the tips of the toes. (From Markinson BC, Wernick J, Gibbs RC. Disorders of the nail unit due to podiatric biomechanical considerations. In: Scher RK, Tosti A, Elewski BE, et al., eds. *Nails*. 3rd ed. Edinburgh: W.B. Saunders; 2005:215–220.)

TESTING

Plain radiographs are indicated in all patients who present with hammer toe (Fig. 150.4). Magnetic resonance imaging and ultrasound imaging of the toe are indicated if joint instability, an occult mass, or a tumor is suspected. Based on the patient's clinical presentation, additional testing may be warranted, including a complete blood count, erythrocyte sedimentation rate, and antinuclear antibody testing.

DIFFERENTIAL DIAGNOSIS

The diagnosis of hammer toe is usually obvious on clinical grounds alone. Bursitis and tendinitis of the foot and ankle frequently coexist with hammer toe. In addition, stress fractures of the metatarsals, phalanges, or sesamoid bones may confuse the clinical diagnosis and require specific treatment.

TREATMENT

Initial treatment of the pain and functional disability associated with hammer toe includes a combination of nonsteroidal antiinflammatory drugs or cyclooxygenase-2 inhibitors and physical therapy. The local application of heat and cold may also be beneficial. Avoidance of repetitive activities that aggravate the patient's symptoms, avoidance of narrow-toed or high-heeled shoes, and short-term immobilization of the affected toes may provide relief. For patients who do not respond to these treatment modalities, injection with local anesthetic and steroid is a reasonable next step.

To inject hammer toe, the patient is placed in the supine position, and the skin overlying the affected toe is prepared with antiseptic solution. A sterile syringe containing 1.5 mL of 0.25% preservative-free bupivacaine and 40 mg methylprednisolone is attached to a ⅝-inch, 25-gauge needle using strict aseptic technique. The hammer toe is identified, and the needle is carefully advanced against the second metatarsal head (Fig. 150.5). The needle is then withdrawn slightly out

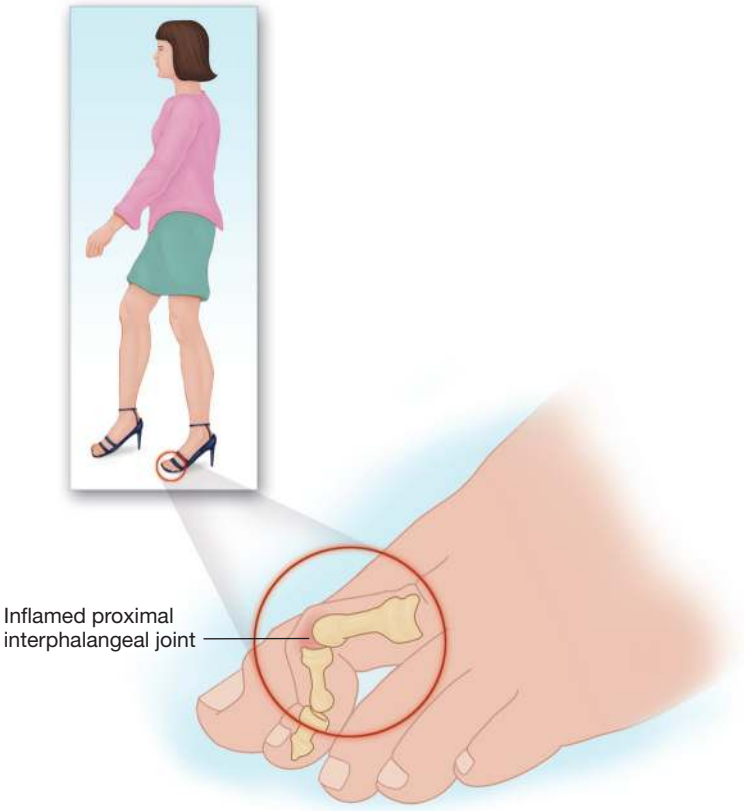


FIG 150.2 Hammer toe deformity is usually the result of wearing shoes that are too tight in the toe box, although trauma has also been implicated.

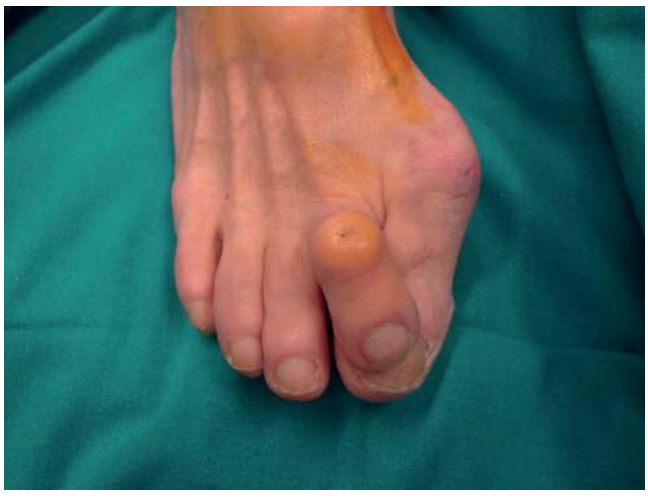


FIG 150.3 Hammer toe deformity. Note adventitious bursa. (From Ceccarini P, Ceccarini A, Rinonapoli G, Caraffa A. Correction of hammer toe deformity of lateral toes with subtraction osteotomy of the proximal phalanx neck. *J Foot Ankle Surg.* 2015;54(4):601–606.)

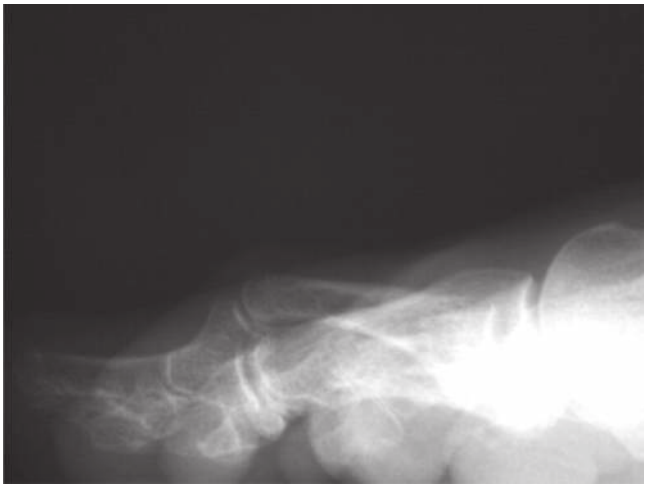


FIG 150.4 Lateral x-ray view of hammer toe deformity. (From Kwon JY, De Asla RJ. The use of flexor to extensor transfers for the correction of the flexible hammer toe deformity. *Foot Ankle Clin.* 2011;16(4):573–582.)

of the periosteum, and the contents of the syringe are gently injected. The operator should feel little resistance to injection. If resistance is encountered, the needle is probably in a ligament or tendon and should be advanced or withdrawn slightly until the injection can proceed without significant resistance. The needle is removed, and a sterile pressure dressing and ice pack are applied to the injection site.

Physical modalities, including local heat and gentle range-of-motion exercises, should be introduced several days after the patient undergoes injection.

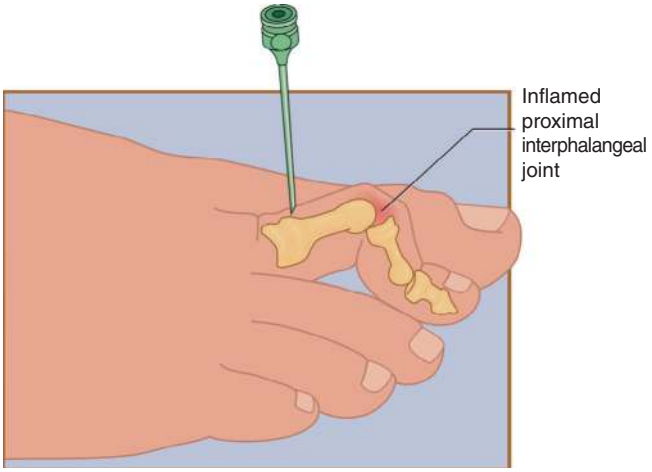


FIG 150.5 Correct needle placement for injection of hammer toe deformity. (From Waldman SD. *Atlas of pain management injection techniques*. Philadelphia: Saunders; 2000:359.)

COMPLICATIONS AND PITFALLS

Failure to identify primary or metastatic tumor of the foot that is causing the patient's pain can have disastrous results. The major complication of injection is infection, although this should be exceedingly rare if strict aseptic technique is followed. Approximately 25% of patients complain of a transient increase in pain after injection, and patients should be warned of this possibility.

CLINICAL PEARLS

Coexistent bursitis and tendinitis may contribute to the patient's foot pain, thus necessitating additional treatment with more localized injection of local anesthetic and methylprednisolone. The injection technique described is extremely effective in treating the pain of hammer toe, and it is safe if careful attention is paid to the clinically relevant anatomy. Narrow-toed and high-heeled shoes should be avoided, because they will exacerbate the patient's symptoms.

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Waldman SD. Hammer toe, claw toe, and mallet toe pain syndromes. In: *Waldman's comprehensive atlas of diagnostic ultrasound of painful conditions*. Philadelphia: Wolters Kluwer; 2016:1055–1062.

Waldman SD. Injection technique for hammer toe. In: *Atlas of pain management injection techniques*. 4th ed. Philadelphia: Elsevier; 2017:668–669.