TENDINOPATHY

Perspective

Emergency physicians may see a wide variety of patients with tendinopathies due to overuse and injury given the increased participation of people in athletics and fitness. Approximately half of all sports participants will be injured at some time, and of these injuries, up to half will involve a tendinopathy. Tendinopathies have been implicated in approximately 40% of tennis injuries, and between 37 and 79% of participants in running will suffer an injury, with most of these injuries related to overuse. In the workplace, the incidence of work-related musculoskeletal disorders is higher in occupations that involve repetitive motion, localized contact stress, awkward positions, vibrations, and forceful exertion. Ergonomic and medical intervention programs may reduce the incidence of work-related injuries.

Complicating the acute pain and functional limitations, tendinopathies often become chronic and can be disabling. Patients may have symptoms for extended periods despite appropriate therapy. The management of tendinopathy focuses on identification of the cause of discomfort; elimination of sources of primary tendinopathy; institution of treatment modalities, such as analgesic medication, protection, relative rest, application of ice, compression, and elevation as necessary; modification of behavior to minimize or to eliminate sources of continuing irritation; and, importantly, referral for appropriate follow-up care and early rehabilitation.

Principles of Disease

Tendons are collagenous structures that connect muscle to bone. They transmit the forces originating in the muscle to the bone, which enables joint motion. The diagnosis of tendinitis, a commonly used term implying an "inflammation of the tendon," has long been attached to many overuse injuries. Many practitioners now advocate use of the term tendinosis as a more accurate reflection of the pathologic process, given that histologic findings consistent with inflammation are not clearly evident in pathoanatomic studies. Although it has been noted that reliable, well-conducted epidemiologic studies have not been performed for most tendinopathies, the histopathologic substratum, in many cases, is degenerative. The term tendinopathy is used throughout this chapter to refer to the impaired tendon, as it encompasses the variety of pathologic processes.

Mechanical overload and repetitive microtrauma to the musculotendinous unit are thought to be the major precipitating causes of most tendinopathies. This is a result of extrinsic and intrinsic factors that modify the pathophysiologic state. Intrinsic factors, such as age, gender, malalignment, joint laxity, muscle weakness, and imbalance, can result in excessively high or frequent mechanical loads during normal activity. Extrinsic factors, such as ergonomics, abnormal movements, excessive duration of activity, and increased frequency or intensity of activity, can also contribute to the development of a tendinopathy. Many injuries have a multifactorial origin. An increased incidence of tendinopathy and tendon rupture, particularly of the Achilles tendon, is reported in patients taking fluoroquinolone antibiotics, particularly in individuals receiving steroid treatment or those with renal disease.

Under optimal conditions, such as appropriate athletic training, the musculotendinous units are able to adapt to tension overload because of the ability of bone to increase its load-bearing capacity and because of an increase in size and strength by hypertrophy of existing muscle fibers. An enhancement of tendon and ligament strength occurs by an increase in collagen content, collagen cross-linking, and mucopolysaccharide content. Unfortunately, many athletes may not allot sufficient time for this adaptive process to occur. For instance, a runner may increase mileage, intensity, or both with excessive alacrity, not allowing time for the cellular changes that are required to adapt to the increased stresses. Poor technique and improper equipment may also contribute to the development of an overuse syndrome.

The pathophysiologic mechanism of tendon healing has mainly been described in the literature in the context of acute injury (e.g., rupture), and correlation to the healing process in tendinopathies remains unclear. Acutely injured tendons go through several stages in the healing process, and it may take 6 to 12 weeks for structural organization and collagen cross-linking to return the tendon to its preinjured strength. As the healing process ensues, unrestricted activity is generally avoided. However, atrophy associated with immobilization should also be avoided because the strength in healing tendons and ligaments increases faster when controlled forces are applied. Consequently, flexibility forces, eccentric strength training, and a measured return to resistive exercises have been suggested as long as pain is not produced. Most patients with overuse tendinopathies fully recover within 3 to 6 months. In summary, a prescription for good follow-up with proper rehabilitation is important in caring for the patient with tendinopathy. Some areas with common presentations of tendinopathies are diagrammed in Figure 117-1.

Clinical Features

General Tendinopathy

The history of the patient presenting with a tendinopathy can be variable, although certain clinical aspects are characteristic. A
In the evaluation of the patient with a tendinopathy, a thorough, directed musculoskeletal examination yields important information. Inspection searching for signs of edema, effusion, erythema, atrophy, deformity, symmetry, or trauma can be helpful. Palpation of the tendon, noting warmth or evidence of crepitation on movement, is important. Evidence of tenderness over the tendon, especially localized, reproducing the patient's pain should be elicited. Underlying bone tenderness (and consideration of other differential diagnoses, including avulsion fracture and osteomyelitis) should be assessed as well. Motor function, particularly passive and active range of motion (and symptoms elicited during the examination), strength (and evidence of weakness or pain), and joint involvement and stability should be noted.

In narrowing the diagnosis, it is important to determine whether the source of pain is articular (within the joint capsule) or periarticular (around the joint capsule). In general, arthritis produces generalized joint pain, warmth, swelling, and tenderness. The discomfort of arthritis increases with both passive and active motion of the joint. In contrast, the pain of a tendinopathy tends to be more localized. Tenderness and swelling do not occur uniformly across the joint, and pain may be produced only with certain movements, most commonly with resisted active contraction or passive stretching of the affected muscles or tendons.
Specific Tendinopathies

Shoulder. Tendinopathies of the shoulder joint include impingement syndrome, which includes subacromial bursitis or rotator cuff tendinopathy, bicipital tendinopathy, calcific tendinopathy, and adhesive capsulitis.

Impingement Syndrome and Rotator Cuff Tendinopathies. The shoulder joint is predisposed to soft tissue injury because of its extensive range of motion and unique anatomical structure. Although it is inherently unstable, the muscles of the rotator cuff (supraspinatus, infraspinatus, teres minor, and subscapularis) and the glenohumeral ligaments serve to stabilize the joint. The muscles of the rotator cuff originate from the scapula (hence their nomenclature), and their tendinous insertion is found on the fibrous capsule of the glenohumeral joint after traversing through the subacromial space. The presence of the subacromial bursa, as for all bursae, serves to ensure fluidity of movement, but it may become inflamed as a part of an impingement syndrome. Impingement of the tendons occurs because of their unique position interposed between the humeral head and the acromion, which may predispose to a chronic tendinopathy. The functional arc of the elevated shoulder is forward and in the anterior plane. As a result of this position, the greater tuberosity of the humerus may compress (impinge) the tendons of the rotator cuff (usually the supraspinatus) against the undersurface of the anterior third of the acromion. Because of the insertion of the tendon of the long head of the biceps, it too may be involved as part of the impingement syndrome. Development of this tendinopathy may be a result of overuse of the extremity that leads to microtrauma of the tendinous fibers, or it may be due to individual anatomic differences (congenital or from the process of aging, such as osteophytic changes) that predispose to tendinopathy, or both. Other entities that may coexist and complicate an impingement syndrome include subacromial bursitis, bicipital tendinopathy, and calcific tendinopathy.

More than 30 years ago, Neer noted that 95% of rotator cuff tears are associated with impingement (excluding tears due to a one-time traumatic event). He described three progressive stages of the impingement syndrome as a result of overuse. The first stage is frequently seen in athletes younger than 25 years who participate in sports that require repetitive overhead motions of the shoulder (e.g., swimming and baseball). It is characterized by edema and hemorrhage within and around the tendon. The pain is usually described as a dull ache over the anterolateral shoulder. Extension from the shoulder to the middle upper arm, often occurring after an activity involving flexion and abduction of the arm. Point tenderness may be elicited over the greater tuberosity. No weakness or loss of motion is generally present. This condition is generally believed to be reversible with appropriate treatment. In the second stage, as mechanical trauma continues, fibrosis and thickening of the tendon and subacromial bursa can occur. This generally affects patients between 25 and 40 years of age. The pain becomes constant and may worsen at night. Active motion may be limited by pain, and any activity involving overhead movement exacerbates the symptoms. Passive range of motion should be preserved, and on physical examination, pain is more diffuse and intense. The third stage has symptoms similar to those of the second stage but may involve a prolonged history of shoulder problems. The range of motion of the shoulder is usually decreased because of either disuse or a partial rotator cuff tear. On pathologic examination, tendon degeneration and attrition may be present. Partial-thickness tears may occur or extend with minor trauma or stress. Complete tears of the rotator cuff, biceps tendon rupture, and osteophytic bone changes are sometimes seen.

Physical examination in the evaluation of a rotator cuff tendinopathy includes maneuvers that can exacerbate the symptoms of impingement. Because the supraspinatus tendon is most often involved, a physical examination sign (sometimes referred to as Jobe’s sign, after Frank Jobe, team physician of the Los Angeles Dodgers, or the empty can test, describing the position of emptying aluminum cans) is helpful in assessing the supraspinatus tendon with resistance testing. With the arms abducted at 90 degrees in the scapular plane (30 degrees anterior to the coronal plane), the arms are internally rotated with the thumbs pointed downward. The examiner places a downward force on the arms, and the patient is instructed to resist the examiner and to keep the arms parallel to the floor. Weakness or pain is considered a positive finding. If the patient is unable to resist the force of the examiner, supraspinatus weakness should be suspected.

Another sign of rotator cuff tendinopathy is elicited by the Neer test, which suggests mechanical impingement with decrease of the subacromial space. The examiner forward flexes the arm, which causes impingement of the greater tuberosity of the humerus with the anterior and inferior edge of the acromion. The patient’s shoulder should then be fully flexed to 180 degrees. A positive result occurs if there is pain produced at the end range of the arc.

The Hawkins test, also indicative of mechanical impingement, is performed by forcibly internally rotating the proximal humerus while the shoulder is forward flexed to 90 degrees and the elbow flexed to 90 degrees. Pain with this maneuver indicates a positive finding.

A complete rotator cuff tear is evaluated by the drop arm test, in which the arm is passively abducted at 90 degrees and the patient is asked to maintain the abduction. If the arm drops to the side, a large rotator cuff tear should be considered. The shrug sign is exhibited when a patient with acute macro-trauma to the rotator cuff is asked to abduct the arm at 90 degrees and appears to be giving a shrug with that side. This movement results from the scapula’s attempting to abduct the arm without the assistance of the rotator cuff. Although this has historically been associated with rotator cuff disease, it is somewhat nonspecific and can be associated with other shoulder disease. Patients with adhesive capsulitis (frozen shoulder) have limitation of active and passive range of motion.

Bicipital Tendinopathy. The tendon of the long head of the biceps, given its insertion into the humerus in proximity to the rotator cuff, can be associated with the impingement syndrome. The patient with bicipital tendinopathy may report pain in the anterior shoulder that radiates down to the radius. Discomfort occurs when the individual rolls on the shoulder at night or attempts to reach a hip pocket or a back zipper. Focal tenderness can be obtained by palpation in the groove between the greater and lesser tuberosities of the humerus while testing for Yergason’s sign, which is elicited by having the patient flex the elbow to 90 degrees with the arm against the body and resisting supination of the forearm. Pain in the area of the proximal tendon is considered to be a positive finding and indicative of bicipital tendinopathy.

Another physical examination tool in the diagnosis of bicipital tendinopathy is the Speed sign. With the elbow extended and the forearm supinated, the patient is instructed to resist forward flexion of the adducted shoulder at 60 degrees. Pain in the area of the proximal biceps tendon (bicipital groove) is indicative of a positive finding. This may also be suggestive, however, of labral disease.

Calcific Tendinopathy. Calcific tendinopathy is an acute or chronically painful condition associated with deposition of calcium crystals that occurs in or around the tendons of the rotator cuff. The cause is unknown but has been postulated to be related to tissue hypoxia and degeneration due to overuse. Although it can affect any of the rotator cuff tendons, it seems to have a predilection for the supraspinatus. The symptoms are similar to those of an impingement syndrome, and the condition generally affects people older than 40 years. Calcium deposition
Lateral Epicondylosis. Lateral epicondylosis (“tennis elbow”) is a painful elbow condition that occurs at the insertion of the common extensor tendon (extensor carpi radialis brevis) onto the lateral epicondyle of the humerus. Although it occurs in many tennis players, epidemiologic studies suggest that less than 5% of patients with such a syndrome actually play tennis. Activities such as driving in screws, use of a wrench, and repetitive work on an assembly line have also been implicated. Symptoms often begin as a dull ache on the outer (lateral) aspect of the elbow. The discomfort can be exacerbated by activities that involve extension or supination of the wrist, such as grasping and twisting. Cozen’s test is performed by having the patient keep the fist clenched while extending the wrist. The examiner grasps the forearm with the left hand while the right hand pulls the patient’s hand toward flexion against the patient’s resistance. A positive finding is pain at the lateral epicondyle, reproducing the patient’s symptoms.

Active extension of the long finger against resistance with the elbow in extension can also reproduce the pain over the lateral epicondyle at the insertion of the extensor carpi radialis brevis. In addition, patients will typically have tenderness to palpation just distal to the lateral epicondyle, over the origin of the extensor carpi radialis brevis.

Medial Epicondylosis. The pain of the less common medial epicondylosis (“pitcher’s elbow” or “golfer’s elbow”) can result from microtrauma at the site of the insertion of the flexor carpi radialis on the medial epicondyle. It is important to differentiate medial epicondylosis from other causes of medial elbow pain, including medial ulnar collateral ligament injury. As a result of repetitive valgus stress placed on the joint, microtraumatic injury and valgus instability at the ligament can occur. With disruption of the medial ulnar collateral ligament, abnormal stress is placed on the articular surfaces, which may lead to degenerative changes and the formation of osteophytes. In the case of medial epicondylosis, patients will generally have tenderness over the flexor-pronator origin slightly distal and anterior to the medial epicondyle. Pain can be reproduced by having the patient attempt wrist flexion and forearm pronation against resistance.

Radiographs can be helpful in cases with atypical or prolonged symptoms and to rule out other pathologic conditions. Approximately 20% of patients demonstrate tendon calcification or a reactive exostosis at the tip of the epicondyle.

The differential diagnosis of lateral epicondylosis includes posterior interosseous nerve entrapment (motor aspect of radial nerve in forearm). Other associated lesions include plica, synovitis, chondromalacia, and adolescent osteochondral defect.

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Wrist de Quervain’s Tenosynovitis. The wrist and hand comprise several tendons that pass through thick, fibrous retinacular tunnels. These help prevent subluxation of the tendons and act as a pulley system. Overuse syndromes are thought to result from changes of the synovial lining between these tendons and retinaculum. de Quervain’s tenosynovitis involves the synovial lining of the abductor pollicis longus and extensor pollicis brevis. Although the term tenosynovitis indicates an inflammation of the tendon sheath, it has been noted that there are many potential forms of tenosynovitis. Classic acute inflammatory changes that are characteristic of tenosynovitis may be related to systemic manifestations of disease (e.g., rheumatoid arthritis and gout); tenosynovitis related to de Quervain’s syndrome is referred to by some practitioners as tenosynovitis. The pathologic process of de Quervain’s tenosynovitis does not generally involve inflammation because the primary change is thickening of the extensor retinaculum covering the first dorsal compartment of the wrist. It has been suggested that de Quervain’s disease is a result of intrinsic degenerative mechanisms rather than extrinsic inflammatory ones.

The history may consist of chronic, repetitive trauma or unaccustomed repetitive efforts, such as firm grasping and movement of the hand in a radial direction. Direct trauma, such as a direct blow or fall, has occasionally been implicated. The discomfort of de Quervain’s tenosynovitis can be localized over the radial styloid process. Radiation of pain proximally to the forearm or distally down the thumb has been noted. The pain is generally constant but may be exacerbated by maneuvers that include grasping, abduction of the thumb, and ulnar deviation of the wrist. In most cases of de Quervain’s tenosynovitis, the onset is gradual and not associated with a history of acute trauma.

On physical examination, slight swelling may be seen over the radial styloid. Crepitation may be palpable over the tendons with flexion and extension of the thumb. An increase in the tensile load (passive stretching or active contraction) in the abductor pollicis longus or extensor pollicis brevis increases pain. The Finkelstein test, which exacerbates the discomfort of de Quervain’s tenosynovitis, is the most pathognomonic physical sign. The patient is instructed to hold the affected thumb in the palm by the fingers and then to ulnar deviate the wrist. Pain will occur near the radial styloid, which is also the point of tenderness. Pain with this maneuver is considered to be a positive finding. Radiographs are characteristically normal.
The differential diagnosis includes scaphoid fracture and osteoarthrosis of the carpometacarpal joint, which has pain caused by longitudinal traction and compression. Rarely, infections such as tuberculosis or disseminated gonococcal infections can be manifested as tenosynovitis.

**Knee**

**Patellar Tendinopathy.** Patellar tendinopathy or “jumper’s knee” occurs in sports that have jumping as a part of the activity, although it can also occur as a result of other sporting activities. Patients complain of pain at the inferior pole of the patella. The discomfort may abate with activity early in the tendinopathy but later progresses to the point of discomfort during exercise and at rest. With the knee flexed at 30 degrees, which relaxes the quadriceps, tenderness may be localized to the deep surface of the proximal attachment of the patellar tendon at the inferior pole of the patella. However, healthy active athletes sometimes have tenderness on examination as well.

The differential diagnosis includes patellofemoral syndrome, which arises from imbalances in the forces controlling tracking during knee flexion and extension. The patient usually complains of anterior knee pain, described as “behind” or “around” the patella, which is classically worse on ascending or descending stairs or on rising from a seated position. On occasion, there is tenderness of the medial or lateral retinacula or facets.

Imaging with ultrasonography and magnetic resonance imaging (MRI) may reflect collagen degeneration but is generally not efficacious in distinguishing tendinopathy from other conditions. It is noted that some asymptomatic jumping athletes have imaging appearances similar to those of affected individuals and that prognosis and outcome are not predicted by such imaging.

**Ankle**

**Achilles Tendinopathy.** Achilles tendinopathy is a common overuse syndrome that typically affects male athletes. The Achilles tendon, named for the mythological Achilles, whose heel was not immersed in the river Styx as his mother dipped him in the river for its protective powers, arises from the medial and lateral heads of the gastrocnemius muscle and inserts on the calcaneal tuberosity. A major function is plantar flexion of the foot. It is the strongest and largest tendon in the body and can withstand tensile loads more than 12 times the body’s weight during running.

The Achilles tendon is vulnerable to injury from either trauma or overuse. A tendinopathy can also develop as a result of systemic disease (e.g., ankylosing spondylitis, Reiter’s syndrome, gout, and pseudogout). The use of fluoroquinolones has also been related to Achilles tendinopathy.

The occurrence of Achilles tendinopathy is highest among individuals who participate in middle- and long-distance running, orienteering, track and field, tennis, badminton, volleyball, and soccer. Some studies have noted an almost 10% annual incidence of Achilles disorders in elite runners. The most common Achilles disorders are tendinopathy (55-66%) and insertional problems, such as retrocalcaneal bursitis and insertional tendinopathy (20-25%). Many cases of Achilles tendinopathy are thought to be multifactorial in origin. Body mechanics and environmental factors (e.g., uneven terrain) may apply valgus or varus stress to the tendon. Technique, equipment, and body mechanics can also contribute to the development of this tendinopathy. Anatomically, the vascular supply to the tendon creates a watershed area approximately 2 to 6 cm above the calcaneal insertion. This is thought to be responsible for clinical symptoms and the pathologic disruption commonly seen at this site.

The patient’s history provides most of the information necessary to make the diagnosis of Achilles tendinopathy. Pain, a cardinal symptom of Achilles tendinopathy, may lead the patient to seek medical help. Some practitioners have noted that the patient’s symptoms reflect the degree of the tendon abnormality. As with many tendinopathies, pain after strenuous activities is reported in the early phase, whereas pain in the later phase occurs during activity and even at rest. At this point, the patient is often unable to perform sporting activities.

On physical examination, inspect the contour of the muscle-tendon unit and note areas of swelling and erythema. In acute Achilles tendinopathy, the tendon may be diffusely swollen and exhibit tenderness on palpation, usually greatest in the middle third. Typically, in the patient who has acute symptoms of tendinopathy, the area of swelling and tenderness does not move with dorsiflexion of the ankle joint. On palpation, local heat, crepitation, and palpable tendon nodules or defects may be noted. Examination for ankle instability and biomechanical faults should be considered.

**Achilles Tendon Rupture.** Although rupture of the Achilles tendon most often occurs when it is preceded by tendon damage, it is possible for untrained athletes to apply excessive force and to rupture the tendon in the absence of prior changes of tendinopathy. Partial and complete rupture may occur, most commonly in 30- to 40-year-old men. Complete rupture is more common in the middle-aged recreational athlete. Historically, the patient may note a “popping” sensation followed by acute weakness and inability to continue the exercise or sport. The patient may report that it felt as though someone kicked him or her in the back of the ankle or, if playing a racket sport, as if he or she were struck on the calf by a ball.

On physical examination, a defect in the tendon can sometimes be palpated. If enough time has elapsed to allow hematoma formation, bogginess may be noted over the injured area of the tendon. The ability to plantar flex the foot is not incompatible with a complete rupture of the Achilles tendon. There are multiple plantar flexors of the foot and toes. Muscles such as the tibialis posterior, flexor digitorum longus, flexor hallucis longus, peroneus brevis, and peroneus longus can remain functional and therefore disguise a complete rupture with the ability to plantar flex the foot. The Simmonds (Thompson) test can be performed to evaluate for complete rupture. With the patient prone and feet hanging over the edge of the bed, the examiner squeezes the calf muscles at their widest point and looks for passive plantar flexion. The absence of plantar flexion is considered a positive finding, indicative of a complete tear of the Achilles tendon. The presence of plantar flexion does not, however, negate the possibility of a partial tear of the Achilles tendon.

**Diagnostic Strategies**

The diagnosis of tendinopathy is generally made on clinical grounds. Although plain radiographs may be helpful in excluding bone abnormalities, ultrasonography has been recommended by some practitioners as the modality of choice for evaluation of pathologic tendon conditions. Ultrasonography can be especially useful when other conditions (e.g., gouty arthritis) obscure the findings of concomitant tendinopathy. Although ultrasound imaging is employed in the emergency department (ED), the use of this imaging technique for soft tissue evaluation in this setting is relatively unstudied, and thus it is not frequently used.

In cases of acute or chronic tendinopathy, one or more of the following features can be seen: loss of the fibrillar echotexture, focal tendon thickening, diffuse thickening, focal hypoechoic areas, extended hypoechogeticity, irregular and ill-defined borders, microruptures, and peritendinous inflammatory edema. Hypoechoic areas surrounding tendons provide evidence for surrounding soft tissue inflammation. In addition to tendinopathy, tendon tears, both partial and complete, can be delineated by ultrasonography.

MRI has been used to visualize pathologic conditions of the tendon. It is able to provide high intrinsic tissue contrast, which
permits the distinction between normal tendons and abnormal tendons, and the high spatial resolution that permits detailed anatomic structures to be identified. MRI has superb resolution of the soft tissue structures and can aid in a variety of tendon disorders. Cost, availability, the need to have the patient be still during examination, and the loss of the interactive and dynamic component compared with ultrasound examination are disadvantages to use of this modality. 40

Differential Diagnosis

The differential diagnosis for tendinopathy includes tendon ruptures, bursitis, ligamentous injuries, septic and inflammatory arthritis, nerve entrapment syndromes, and bone abnormalities (such as fracture, foreign body, tumor, and osteomyelitis).

Management

General Tendinopathy

The management of tendinopathy focuses on identification of the cause of discomfort; elimination of sources of primary tendinopathy; institution of treatment modalities, such as analgesic medication, protection, relative rest, application of ice, compression, and elevation as necessary; modification of behavior to minimize or to eliminate sources of continuing irritation; and, importantly, referral for appropriate follow-up care. 6

Cryotherapy (cold treatments, 20 minutes at a time every several hours, for the first 24 to 48 hours) may be beneficial. Nonsteroidal anti-inflammatory drugs (NSAIDs) are also sometimes indicated to provide pain relief, although there is little evidence to support a beneficial effect with their use because tendinopathies tend to represent a degenerative rather than an inflammatory process. 41 Graduated range-of-motion exercises may be useful after the period of immobilization. Although corticosteroid injection is often used, its role for most tendinopathies is unclear. 42,43 The use of corticosteroid injection is controversial. Steroids should not be injected into a major tendon such as the Achilles tendon or the patellar tendon, which may be at risk for spontaneous rupture if it is already weakened.

Specific Tendinopathies

Impingement Syndrome and Rotator Cuff Tendinopathies. The treatment of rotator cuff tendinopathies and the impingement syndrome follows the treatment of tendinopathy in general. Emphasis is placed on physical rehabilitation and strengthening exercises. A significant proportion of patients will improve with conservative management. In those who do not, surgical intervention, such as acromioplasty or repair, may be indicated. 44,45

Calcific Tendinopathy. The initial treatment of calcific tendinopathy is mainly conservative and consists of analgesia and brief sling immobilization because prolonged immobilization may result in adhesive capsulitis. The use of corticosteroids is controversial. Ultrasonography and extracorporeal shock wave therapy have been used with some success. 42 A small percentage of patients who do not respond to these interventions may need surgery. As well as arthroscopic treatment, localized disruption in the resorptive phase by needle lavage under fluoroscopic guidance or in the operating room has been noted to be an efficacious therapy. 22,23 Follow-up care is important because calcific tendinopathy has been described as the best known cause of reactive cuff failure.

Lateral and Medial Epicondylitis. In up to 95% of patients, epicondylitis will improve with conservative therapy. 24 Initial efforts include making the patient more comfortable with the standard principles of protection, relative rest, cryotherapy, compression, elevation, medication (NSAIDs), and modalities of physical therapy. The term relative rest implies the avoidance of abuse and not the absence of activity. Activities that aggravate the pain should be eliminated, and an attempt to protect the tendon through such strategies as a reduction in playing time or intensity should be considered. Control of force loads by bracing, improved performance technique, and use of appropriate equipment should also be considered. 24 Follow-up evaluation should be ensured.

de Quervain’s Tenosynovitis. The initial treatment of de Quervain’s tenosynovitis consists of immobilization with a thumb spica splint, anti-inflammatory medications, and prompt referral. Corticosteroid injection has been shown to be an effective treatment of de Quervain’s disease, and failure to respond may be due to anatomic variation or poor technique. 29,46 Surgical decompression of the first dorsal compartment may be indicated if these treatments fail. 29

Achilles Tendinopathy and Rupture. In addition to routine conservative treatment, patients with Achilles tendinopathy should be referred for orthopedic evaluation and correction of limb malalignment with the use of orthotics or heel lifts. Eccentric loading exercise and low-energy shock wave therapy are effective therapies for Achilles tendinopathy. 47-49

The management of Achilles tendon rupture may be either operative or nonoperative, depending on the patient involved. Appropriate consultation with an orthopedist is essential. A conservative approach, including immobilization in a short or long leg cast in the equinus position, may be indicated. 50 Some authors note that complete ruptures in active athletes should be treated surgically in most cases. 51 Although other risks and benefits must be taken into consideration, some studies indicate that early surgery may be indicated because the risk for Achilles tendon re-rupture is less. 50,51

Disposition

Most patients with tendinopathy are safely discharged home with proper discharge instructions, relative rest of the tendon, analgesia, and appropriate follow-up. The exception is elderly or disabled patients who are rendered unable to perform the activities of daily living by the tendinopathy. Although appropriate rest and analgesia provide symptomatic relief, underlying causes should be sought and modified.

Bursitis

Perspective

Bursae are closed, round, flat sacs lined by synovium. 52 They occur at areas of friction between skin and underlying ligaments and bone. The bursa permits lubricated movement of soft tissues over areas of potential impingement (e.g., subacromial bursa) and friction (e.g., olecranon bursa and prepatellar bursa). Many bursae are nameless, and new bursae can form as a result of frequent irritation. The two most common identifiably inflamed bursae are the olecranon and the prepatellar. When these bursae are inflamed, they are generally recognizable over the extensor surface of the elbow or the knee, respectively.

Principles of Disease

Many cases of bursitis are idiopathic in nature, but common identifiable causes of inflammation include infection (most often due to Staphylococcus aureus), trauma (which may predispose to infection), rheumatologic disorders (e.g., gout, pseudogout, ankylosing spondylitis, and rheumatoid and psoriatic arthritis), and other systemic diseases.
Clinical Features

Olecranon and Prepatellar Bursitis

Less than half of patients who present with olecranon or prepatellar bursitis show signs of an infectious origin.53,54 Distinguishing septic from nonseptic bursitis is not always straightforward, either on clinical grounds or as a result of diagnostic testing. Patients with septic bursitis generally present earlier in the clinical course and have more pain, tenderness, erythema, and warmth compared with those who have an inflammatory process. The most important predisposing factor in septic bursitis is trauma, which precedes septic bursitis in up to 70% of cases.52 Other predisposing factors for septic bursitis include chronic illness such as diabetes mellitus or alcohol abuse, chronic skin conditions such as atop dermatitis, and previous noninfectious inflammation of the bursae such as rheumatoid arthritis or gout.52 It may also be seen more commonly in people in occupations in which repetitive knee or elbow trauma is common.

The olecranon bursa, on the extensor surface of the elbow, is the only bursa of the elbow joint and is easily traumatized, resulting in inflammation, pain, and swelling. Infection can occur from local trauma (e.g., puncture wound and laceration) but may also be present in the absence of visible trauma. Hematogenous bacterial seeding is rare and is probably due to the limited vascular supply of the bursal tissue.53

On physical examination, localized swelling and fluctuance are usually present. Evidence of trauma may or may not be present. Tenderness and warmth are seen in most patients with septic bursitis. Erythema with overlying cellulitis is also common in septic bursitis, and approximately 40% of patients will also have a fever. Whereas tenderness, erythema, and warmth can also be seen in patients with purely inflammatory causes of bursitis, both the frequency and degree of these findings are less, and patients with septic bursitis will usually present earlier in the clinical course.52,54

Passive range of motion should not produce much pain, with the exception of full flexion, at which point there may be discomfort as the inflamed bursa is compressed. Evidence of significantly diminished range of motion, generalized joint swelling, or other signs and symptoms of joint involvement (e.g., joint pain, warmth, and joint effusion) should raise a concern for septic arthritis. Although the olecranon and prepatellar bursae generally do not communicate with the joint space, one must consider this possibility in the differential diagnosis, especially if trauma is involved and the integrity of the underlying joint is disrupted. Arthrocentesis to rule out septic arthritis should then be considered.

Diagnostic Strategies

When signs of acute inflammation are present, aspiration of the bursa is indicated to exclude the presence of infection or crystal-induced disease.52,53 Although a discussion of the technique of bursal aspiration is beyond the scope of this chapter, it should be performed with sterile technique. An 18- to 20-gauge needle can be used to perform this procedure. A lateral approach has been recommended to lower the risk of iatrogenic sinus track formation, although the relation between the two is still somewhat unclear. A distal approach can also be used when the olecranon bursa is aspirated. In cases of septic bursitis, the aspirate usually appears purulent but can occasionally appear serosanguineous or straw colored. In the case of nonseptic bursitis, the aspirate may vary from bloody aspirate to straw colored. The aspirate of an inflamed bursa should be sent for evaluation of white blood cell (WBC) count with differential, microscopy for crystals (if crystalline disease is suspected), Gram’s stain, appropriate cultures and sensitivities, and glucose level. A bursal fluid glucose–to-serum glucose ratio of less than 50% is almost exclusively seen in septic bursitis.

Organisms found on either Gram’s stain or culture are diagnostic for septic bursitis. There are no definitive guidelines about use of the WBC count to distinguish between septic and nonseptic bursitis. A bursal fluid WBC count higher than 1000/µL is almost always seen, and one higher than 5000/µL suggests bursal fluid infection, even in the presence of a negative Gram’s stain, but counts can be much lower in septic bursitis and above this level in nonseptic cases. Culture is the definitive test but will not be available during the initial evaluation. S. aureus is by far the most common organism in bursal infection, followed by other staphylococcal and streptococcal species, and this bacteriology has not changed in the last several decades.56–58

Because most cases of septic bursitis occur in the olecranon and prepatellar bursae, the diagnosis is made clinically in conjunction with aspiration. MRI can be used to aid in the diagnosis of inflammation or infection of deep bursae.59,60 Ultrasonography has also been used as a modality for aspiration of deep bursae.61

Differential Diagnosis

Conditions that mimic bursitis include underlying fracture and osteomyelitis. Radiography and bone scan or MRI may be necessary to exclude these conditions. Other causes of the nontraumatic, nonseptic bursitis include rheumatoid arthritis, gout, pseudogout, scleroderma, ankylosing spondylitis, systemic lupus erythematosus, hypertrophic pulmonary osteoarthropathy, Whipple’s disease, oxalosis, and idiopathic hypereosinophilic syndrome.

Management

Septic Bursitis

The optimal treatment of septic bursitis is uncertain because large-scale clinical prospective trials in this area are lacking. Debate remains about the use of outpatient (oral) versus inpatient (intravenous) administration of antibiotics, duration of therapy, use of needle aspiration and incision and drainage, and use of operative intervention.54

Patients who have bursal inflammation with suspicion (clinical or laboratory) of infection should be treated with appropriate antibiotics. Empirical therapy (including coverage for S. aureus and Streptococcus species) may be indicated until definitive culture results are available. The frequency of methicillin-resistant S. aureus (MRSA) in septic bursitis is unclear, although recent literature suggests that rates are low.56–58 MRSA was found to be the most common cause of community-onset adult septic arthritis in one case series from an ED population, so empirical coverage for this organism in septic bursitis should be considered.52 A trial of oral antibiotic therapy (up to 14 days) and treatment on an outpatient basis in the patient with uncomplicated bursitis and no underlying disease is reasonable. However, failure rates up to 67% have been reported for outpatient treatment of septic bursitis.54 One of the largest observational studies on the successful outpatient treatment of septic bursitis showed an admission rate of only 1 of 118 patients, but all patients in this study received sequential intravenous antibiotics for approximately 4 days at an outpatient clinic followed by oral antibiotic therapy.54 Treatment in an observation unit or inpatient setting with intravenous antibiotics therefore is a consideration for patients with significant symptoms, with overlying cellulitis, or unlikely to receive close follow-up.

Needle aspiration is a commonly used technique for the management of septic bursae. Successful treatment of septic bursitis can be achieved in patients receiving outpatient oral antibiotics.
after initial needle aspiration. Laupland and Davies showed no difference between patients who received aspiration (51% of total patients) and those who did not, but the authors conceded that patients who had more severe disease were likely to be selected for drainage. Also, the diagnosis of septic bursitis was confirmed by culture in only 26% of these patients, potentially underestimating the importance of bursal drainage in patients with true septic bursitis. Because needle aspiration is used for the diagnosis of septic bursitis, initial drainage at the same time seems warranted. Those with a purulent aspirate may require repeated aspiration at 1- to 3-day intervals if the effusion persists. In all cases, appropriate follow-up to assess response to therapy should be arranged. Warm soaks and wound care are also indicated. Surgical incision and drainage or bursectomy may also be necessary in severe, recurrent, or refractory cases.

Nonseptic Bursitis
The optimal treatment of nonseptic bursitis has not been clarified. Most cases improve with conservative therapy, although complete recovery can take many months. Aspiration can be considered in the initial treatment of acute nonseptic bursitis. Further conservative therapy includes the administration of NSAIDs and application of a compression dressing with an elastic bandage to prevent recurrent swelling. Systemic causes of bursitis (e.g., crystalline disease) should be treated appropriately. Avoidance of local trauma is important for treatment and successful prevention of bursitis. Recurrent olecranon bursitis may be caused by underlying anatomic disorders such as bone spurs. Steroid injection into a nonseptic bursa has been used as a treatment modality, but multiple complications have been described, including skin atrophy over the bursa, chronic pain, development of septic bursitis, bleeding, postinjection flare as a result of release of microcrystals, and tendon rupture.

Disposition
Patients without underlying medical problems who present with uncomplicated septic bursitis can usually be discharged with appropriate oral antibiotics. Those with underlying diseases (e.g., immunocompromise, leukopenia, and diabetes) and those with systemic toxicity or severe bursal infection (e.g., purulent drainage) are candidates for intravenous antibiotics and inpatient therapy. Patients with a purulent aspirate may need repeated aspiration. Close follow-up is necessary to ensure response to therapy. Patients with presumed nonseptic bursitis require close follow-up as well.

Other Types of Bursitis
Subacromial Bursitis
The subacromial bursa lies between the supraspinatus tendon and the acromion. Subacromial bursitis is thought to be nearly synonymous with supraspinatus tendinopathy and may be involved in the stages of rotator cuff impingement. Pain and tenderness, localized to the lateral aspect of the shoulder, and signs of impingement may be noted on physical examination. Although it is uncommon, septic subacromial bursitis can occur.

Trochanteric Bursitis
The trochanteric bursa has both deep and superficial components. The deep bursa is located between the greater trochanter and the tensor fascia lata; the superficial bursa is located between the greater trochanter and the skin. In general, middle-aged or older women report acute or chronic pain over the bursal area as well as the lateral thigh. Lying on the hip and walking may exacerbate the pain. Trochanteric bursitis can occur as a complication of rheumatoid arthritis. On examination, the pain of superficial bursitis may be reproduced by hip adduction, and the pain of deep trochanteric bursitis may be reproduced with hip abduction. The hip joint has normal examination findings. Septic trochanteric bursitis has been described in the literature.

Ischiogluteal Bursitis
The ischiogluteal bursa is located adjacent to the ischial tuberosity and overlies the sciatic and posterior femoral cutaneous nerves. Inflammation, known as , is described as pain over the center of the buttocks with radiation down the back of the leg. Sitting on a hard surface exacerbates the pain, and palpation over the ischial tuberosity causes discomfort.

Iliopsoas Bursitis
The iliopsoas bursa is the largest bursa around the hip. It lies between the iliopsoas tendon and the lesser trochanter. The pain of iliopsoas bursitis usually is manifested as anterior hip pain that can radiate down the medial thigh to the knee and is increased on hip extension.

Anserine Bursitis
The anserine bursa lies deep to the three tendons (sartorius, gracilis, and semitendinosus) that form the pes anserinus (“foot of the goose”) and superficial to the medial collateral ligament. The patient with anserine bursitis usually complains of medial knee pain approximately 2 or 3 cm distal to the joint line. There is usually tenderness to palpation in this area and occasionally swelling. Diabetes mellitus and possibly obesity and osteoarthritis of the knee are risk factors for development of this condition.

**KEY CONCEPTS**

**Tendinopathy**
- Mechanical overload and repetitive microtrauma are key underlying mechanisms in the development of tendinopathy. Patients most often present with a history of progressively worsening localized pain after work- or sport-related activities that are repetitive in nature.
- Tendinopathy may also be associated with nonmechanical causes, including systemic manifestations of diseases and the use of fluoroquinolones.
- Most patients with tendinopathy can initially be treated with conservative measures, such as protection, relative rest, application of ice, medication, and elevation. Overuse syndromes can take at least 6 to 12 weeks to heal. Inform patients of this and provide appropriate referral for follow-up.
- Operative treatment may be indicated for selected cases of tendon injury that require primary repair (e.g., rupture of the Achilles tendon) or that have failed to respond to conservative treatment (e.g., impingement syndrome) and are amenable to surgical amelioration.

**Bursitis**
- Consider the possibility of an infectious cause in all cases of acute bursitis.
- The definitive diagnosis of bursitis is made by aspiration of the bursa and evaluation of the fluid.
- Septic bursitis is most commonly caused by *Staphylococcus aureus*. 

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Laupland and Davies: 63
Staphylococcus aureus: 1525
Nonseptic bursitis may be traumatic, rheumatologic (e.g., gout and pseudogout), or idiopathic in nature. Other conditions, such as septic arthritis, osteomyelitis, and underlying fracture, are in the differential diagnosis of bursitis.

The management of bursitis includes treatment with appropriate medication (antibiotics for septic bursitis, NSAIDs for nonseptic bursitis), rest, application of ice, compression, and elevation as well as prompt referral for appropriate follow-up. Hospitalization is considered for severe local infections, for patients who are immunosuppressed, and in the presence of high fever or systemic toxicity.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.