Tendon Disorders of the Foot and Ankle

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Abstract

Attritional and traumatic injuries to the tendons around the foot and ankle are not uncommon. Treatment of overuse-type injuries (tendinitis) remains straightforward. However, surgical treatment of peroneal subluxation, Achilles tendon ruptures, and posterior tibial tendon insufficiency remains somewhat controversial. Generally speaking, soft-tissue reconstruction of the superior peroneal retinaculum is superior to bony procedures for peroneal dislocation. Open repair of a torn Achilles tendon is more predictable than closed treatment. Good clinical judgment is needed in determining the best treatment for posterior tibial tendon problems. The painful os peroneum syndrome is a newly described spectrum of posttraumatic conditions that may be the cause of lateral foot pain, which is frequently difficult to identify.

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Every tendon around the foot and ankle can cause symptoms as the result of overuse or injury. Most frequently affected are the anterior tibial, flexor hallucis longus, Achilles, peroneal, and posterior tibial tendons. In this article I will review the relevant anatomy, discuss the clinical syndromes involving these tendons, and outline the appropriate treatment.

Anterior Tibial Tendon

The anterior tibial muscle originates from the proximal two thirds of the tibia, the lateral tibial condyle, and the interosseous membrane, with insertion onto the navicular, the first metatarsal base, and the medial cuneiform. This musculotendinous unit supplies 80% of the dorsiflexion power of the ankle. The straight course of the tendon under the superior extensor retinaculum results in minimal mechanical demands; therefore, an overuse syndrome is less common than would be expected in a muscle of this size and power.

Localized swelling, tenderness, and crepitus over this tendon indicate a diagnosis of anterior tibial tenosynovitis. Treatment includes ice, rest, and, on rare occasions, immobilization. Spontaneous ruptures are rare and usually painless. An incomplete rupture with minimal dorsiflexion weakness does not require repair. A complete rupture may be overlooked or confused with a foot drop from a lumbosacral radiculopathy or peroneal palsy. Such ruptures usually occur in the sixth and seventh decades. In cases of complete rupture with foot drop, end-to-end surgical repair should be performed, although elderly patients may choose to use a dorsiflexion-arrestive brace. Early diagnosis and treatment are important. The proximal end of the tendon retracts to the superior retinaculum but can be reapproximated if rupture is diagnosed early.[1]

In cases diagnosed late, half of the thickened proximal end of the tendon may be used as a free graft and routed under the cruciate ligament, or a free extensor tendon graft may be utilized.[2]

Flexor Hallucis Longus Tendon

The flexor hallucis longus tendon is most frequently affected in athletes and other individuals who are involved in repetitive push-off maneuvers (e.g., ballet dancers, in whom the *sur les pointes* position is frequently implicated). These activities transmit tremendous forces across the tendon and its sheath, resulting in irritation and tenosynovitis.

The flexor hallucis longus tendon originates from the lower part of the posterior surface of the fibula lateral to the medial crest, with a portion of the origin arising from the covering fascia and the adjacent fascial septum that it shares with other muscles. At the ankle, the tendon lies in the most posterior lateral compartment of the flexor retinaculum, whence it travels distally to insert on the distal phalanx of the great toe. As the tendon courses behind the medial malleolus, it passes through a fibro-osseous tunnel located on the posterior aspect of the talus, bordered anteriorly by the body of the talus, medially by the medial tubercle of the talus, laterally by the lateral tubercle of the talus, and posteriorly by the flexor retinacu-

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lum. The associated tendon sheath courses behind the medial malleolus and forms a separate compartment.

Because the tendon runs through the confined space in the fibro-osseous tunnel, it is particularly predisposed to mechanical irritation and inflammation. Prolonged inflammation or stenosis of the fibrous tunnel may cause the tendon to develop a partial rupture, sometimes accompanied by snapping or triggering. Patients complain of pain, tenderness, and a snapping sensation posteromedially. Occasionally, inability to flex the great toe is reported as well. A useful clinical test is to compare the amount of passive extension of the great toe metatarsal joint with the foot in the neutral and plantar-flexed positions. Contracture or triggering is suggested when the patient is unable to extend the metatarsophalangeal joint beyond neutral with the foot and ankle in the neutral position, but passive extension is possible with the ankle plantar-flexed.

Conservative treatment consists of ice, nonsteroidal anti-inflammatory agents, strapping of the foot, and longitudinal arch supports placed in firm-sole shoes. These measures will frequently alleviate pain and over time facilitate restoration of function. Surgery is required when severe stenosis of the fibro-osseous tunnel is present, accompanied by pain, triggering, and tendon contracture. The procedure consists of release of the constrictive flexor retinaculum and resultant decompression of the flexor hallucis tendon. Even after successful surgical release, the patient should be informed that there is a possibility of future complete or partial rupture of the central fibers of the tendon.

Peroneal Tendons

The peroneal tendons (i.e., those of the peroneus brevis and longus muscles) pass posterior to the fibula and are restrained by the superior peroneal retinaculum (SPR). Most fibulae have a definite sulcus, but significant anatomic variations do exist.[3] The sulcus width ranges from 5 to 10 mm; 7% of these grooves are convex, 11% are flat, and 82% are concave. Regardless of the sulcus, peroneal tendon stabilization primarily depends on the SPR. Most anatomy texts illustrate the SPR as a single band originating from the posterior ridge of the fibula and inserting onto the lateral wall of the calcaneus. However, insertions onto the Achilles tendon and the lateral calcaneus sometimes are present.

Most of the attention concerning peroneal pathology has been directed toward subluxation and dislocation of the peroneal tendons. Recently, however, incomplete tears of the peroneus brevis and the painful os peroneum syndrome[4] have been recognized as important clinical entities.

Subluxation or Dislocation

Subluxation or dislocation of the peroneal tendons can be an occult event and is probably often overlooked. Either condition can be difficult to diagnose but should always be considered a possibility following any injury that results in sudden and forceful contraction of the peroneal muscles in association with rapid plantar flexion and inversion of the foot and ankle.

Although acute subluxation of the peroneal tendons is frequently confused with ankle sprain, the examiner can differentiate them by the location of tenderness. While ankle sprains cause tenderness over the anterior talofibular ligament, subluxation of the peroneal tendons results in tenderness over the retromalleolar area. Because of the early traumatic swelling, palpating the tendons during dislocation is quite difficult. Radiographs are seldom helpful. However, if an avulsion fracture of the lateral ridge of the distal fibula is present on an x-ray film, the diagnosis of subluxation or dislocation of the peroneal tendons can be made with certainty. Ankle arthrograms are seldom beneficial, and radiographic views that show a shallow peroneal groove are seldom of diagnostic significance.

Treatment of acute dislocation of the peroneal tendons remains controversial. Some orthopaedists believe that conservative treatment is of benefit, while others recommend early surgical intervention, particularly for active, competitive athletes.

Conservative treatment consists of a compression dressing fabricated from a felt pad cut in the shape of a keyhole and strapped over the lateral malleolus. Gentle pressure is placed on the peroneal tendons and the SPR. This is reinforced with a plaster splint. Once the acute symptoms have resolved, a well-molded cast is applied for a total of 6 weeks. After cast removal, an aggressive ankle rehabilitation program emphasizes both strengthening and proprioception education.

If surgery is selected, numerous procedures have been described for stabilizing the peroneal tendons.[5] The procedures fall into two general categories, bony procedures and soft-tissue procedures. Bony procedures are of historical interest only and include a variety of methods to increase the depth of the peroneal groove.[6-9] Soft-tissue procedures include that of Bonnin,[10] which plicates the attenuated retinaculum; that of Eckert and Davis,[11] who reattach the retinaculum to the malleolar ridge; and that of Jones,[12,13] wherein a sling is fabricated from a small strip of the adjacent Achilles tendon. Sarmiento and Wolf[14] describe rerouting the peroneal tendons beneath the calcaneal fibular ligament.

Two types of pathologic lesions are most frequently found when

surgery is being performed. The first lesion is simple attenuation of the SPR. If this is the only abnormality, surgical treatment consists of advancing the SPR to the posterior edge of the fibula, where it is sutured through drill holes. Anatomic reconstruction of the attenuated SPR is the desired goal. Failure to advance the stretched-out SPR adequately allows continued partial subluxation of the peroneus brevis tendon, while overtightening the SPR can lead to painful stenosis.

On other occasions, a "Bankart-type" lesion[15] is found (Fig. 1). The SPR is lifted from its posterolateral fibular attachment, creating a pouch that allows anterior subluxation of the peroneal tendons. The repair consists of reattaching the SPR to the posterolateral aspect of the fibula, thereby obliterating the offending pouch.

Rarely, an anomalous peroneus brevis muscle will cause subluxation. The peroneus muscle belly may extend into the fibular groove, causing encroachment, or there may be a bifid tendon.[16,17]

Longitudinal Tears of the Peroneus Brevis

Historically, little mention has been made about tears of the peroneus brevis tendon. However, this condition has now been recognized as a distinct clinical entity. Tears of the peroneus brevis occur when the anterior portion of the tendon slips forward, out of the groove, and over the sharp posterior ridge of the fibula. A complete or incomplete longitudinal tear can measure from 2 to 5 cm (Fig. 2). In all cases, the central portion of the longitudinal split is centered over the distal tip of the fibula. The tear usually involves the middle or anterior portion of the tendon.

Patients generally present with retromalleolar pain and tenderness. They may have a history of multiple ankle sprains or chronic ankle instability.

In patients with documented tears of the peroneus brevis, conservative treatment is generally unsuccessful. Surgical repair is accomplished through a curved 7-cm incision along the posterior third of the fibula. The competence of the SPR is assessed. The SPR is then opened in such a way that it can be tightened if attenuated.

If the split is through the anterior third of the tendon and the smaller portion of the tear is frayed in any way, I excise the anterior third. If the tear is in the middle third and both fragments are without degenerative change, I repair the tendon with buried nonabsorbable suture. If, however, the entire width of the peroneus brevis tendon is involved and there is significant fraying, the degenerated segment of the tendon is excised in toto, and tenodesis of the proximal and distal stumps to the peroneus longus tendon is performed (Fig. 3)

Following surgery on the peroneus brevis tendon, the SPR is advanced and imbricated onto a fresh bony bed. If there is associated ankle instability, this should be repaired as well.

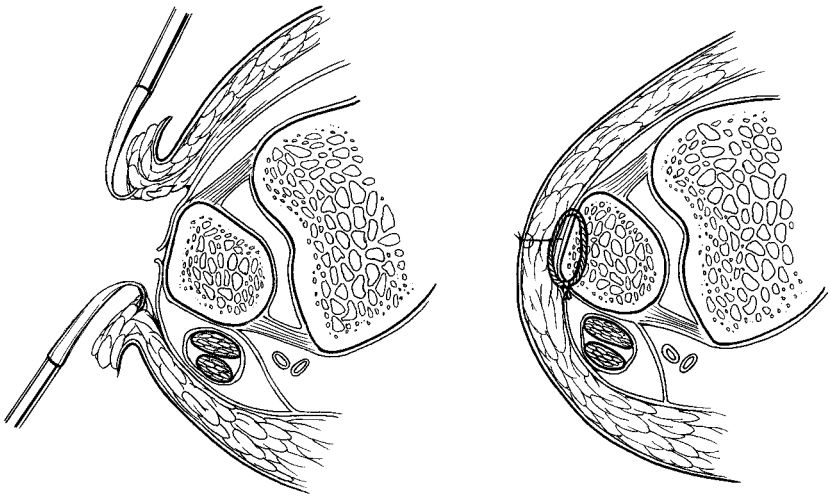

Fig. 1 Repair of a Bankart-type lesion. **Left**, Elevation of the SPR from the posterior fibula creates a pouch. Peroneal tendons subluxate or dislocate into this pouch. **Right**, Reattachment of the SPR to the fibula obliterates the pouch, stabilizing the peroneal tendons.

Painful Os Peroneum Syndrome

The painful os peroneum syndrome is a spectrum of posttraumatic conditions, including one or more of the following[4]: (1) an acute os peroneum fracture or diastasis of a multipartite os peroneum; (2) a chronic os peroneum fracture or diastasis of a multipartite os peroneum fracture associated with stenosing peroneus longus tenosynovitis; (3) attrition or partial rupture of the peroneus longus tendon proximal or distal to the os peroneum; (4) frank rupture of the peroneus longus tendon; or (5) the presence of a gigantic peroneal tubercle on the lateral wall of the calcaneus that traps the peroneus longus tendon and its os peroneus during peroneus longus tendon excursion.

Patients with the painful os peroneum syndrome have a history of either direct trauma to the lateral side of the foot or a supination-inversion ankle injury. Symptoms usually include tenderness along the

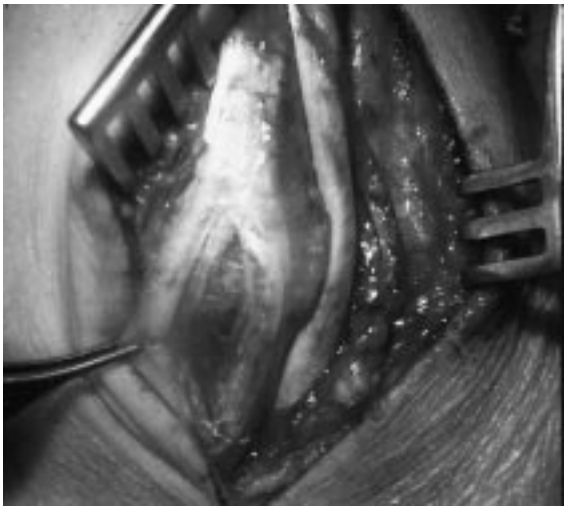

Fig. 2 Complete longitudinal tear of the peroneus brevis. The peroneus longus remains reduced, while the torn peroneus brevis dislocates over the tip of the fibula.

peroneus longus tendon distal to the fibula. Pain is usually exacerbated by resisted plantar flexion of the first ray and the heel-rise phase of gait. Patients also have weakness or pain with forced foot eversion.

Diagnosis may be based on radiographic or magnetic resonance (MR) imaging data or the findings on exploration motivated by a high degree of suspicion.

Conservative treatment consists of cast immobilization, with or without corticosteroid injections. I always cast the extremity at least once for 4 to 6 weeks. Corticosteroid administration is optional; if palpable synovitis is present, however, I routinely perform an injection. Surgical treatment consists of (1) excision of the os peroneum and the giant peroneal tubercle with primary repair of the peroneus longus tendon; (2) excision of the os peroneum and degenerated peroneus longus tendon with tenodesis of the peroneus brevis to the peroneus longus tendon; or (3) excision of the os peroneum with primary repair of the peroneus longus tendon.

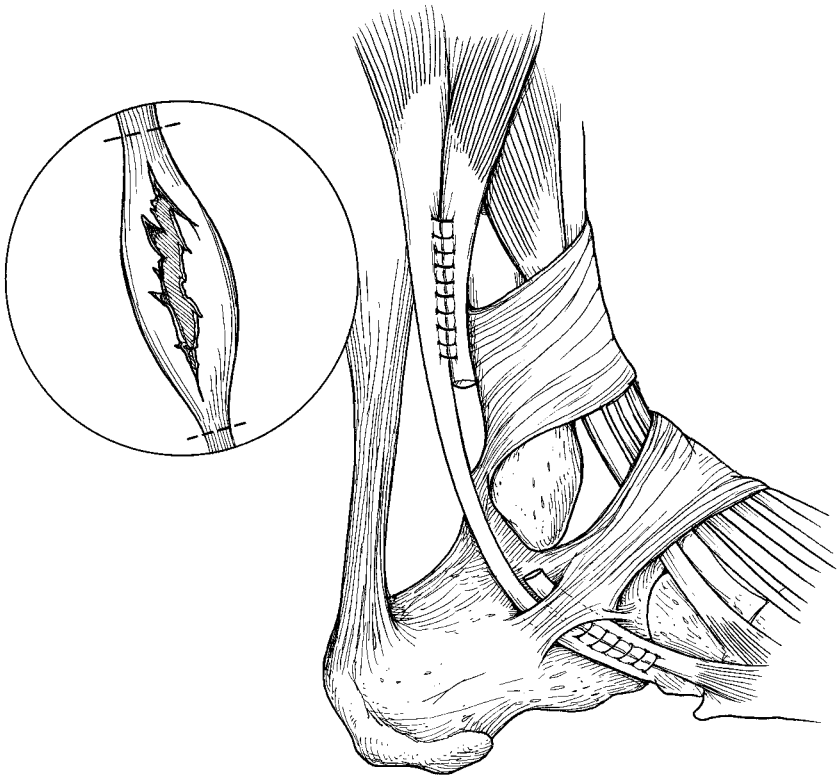

Fig. 3 Irreparable tear of the peroneus brevis necessitates excision of the tear and tenodesis to the adjacent peroneus longus.

Achilles Tendon

The gastrocnemius originates from the lateral and medial femoral condyles, while the soleus originates from the posterior surface of the tibia and the fibula. The soleus and gastrocnemius contribute separately to the formation of the Achilles tendon, with the gastrocnemius segment measuring 11 to 26 cm and the soleus portion measuring 3 to 11 cm. The blood supply to the Achilles tendon comes from both proximal and distal sources (Fig. 4). The least vascular area is 2 to 6 cm above the tendon insertion into the calcaneus, which is the usual location of chronic inflammation and rupture.

The primary etiologic factor resulting in damage to the Achilles tendon is training errors, such as a sudden increase in training mileage, a single severe competitive session (a 10-km race or a marathon), a sudden increase in training intensity, repetitive heel running, recommencement of training after an extended period of inactivity, and

running on uneven or slippery terrain. Hindfoot and leg malalignments may also contribute.

Peritendinitis

Peritendinitis is inflammation within the peritenon without associated Achilles tendinosis (Fig. 5). The symptoms consist primarily of pain, which is aggravated by activity and relieved by rest. Tenderness is present several centimeters proximal to the insertion of the Achilles tendon into the calcaneus.

Fig. 4 Blood supply to the Achilles tendon. Note that longitudinal vessels supply the tendon proximally and distally, while transverse vessels vascularize the middle portion.

Initially, treatment includes ice, massage, contrast baths, and nonsteroidal anti-inflammatory medications. If hindfoot alignment problems are present, an orthosis is prescribed. In more advanced or chronic cases, the peritenon of the Achilles tendon becomes fibrotic and stenosed. While the use of steroidal injections for tendon injuries is generally considered quite hazardous, we have found that subperitenon infiltration of lidocaine is effective in relieving symptoms. Mechanical lysis of adhesions can be achieved by rapid injection of 15 ml of local anesthetic into the subperitenon space.

If conservative means fail, open lysis of adhesions is performed through a medial incision exposing the involved area of tendon. One should be very careful to protect the anterior fatty tissue, as this is a source of the blood supply of the tendon. However, the medial, lateral, and posterior peritenon can be excised.

Tendinosis

The pathology of Achilles tendinosis is interstitial microscopic failure or obvious central tissue necrosis with subsequent mucoid degeneration. This is usually the result of accumulated repetitive microtrauma and evolves pathologically in a relatively predictable manner. Initially, the peritenon sheath becomes inflamed. If the overuse continues, the tendon itself may become inflamed or hypovascular secondary to restriction of blood flow through the scarred peritenon. Degenerative changes in the tendon then follow. With tendinosis, the tendon has a noninflammatory histologic appearance with collagen fiber disorientation, hypocellularity, scattered vascular ingrowth, and occasional areas of necrosis or calcification. Despite these changes, the condition can be asymptomatic. Patients will frequently note a palpable but painless mass in the Achilles tendon approximately 4 to 6 cm proximal to the insertion of the tendon. Those patients who become symptomatic usually have peritenous inflammation along with the intratendinous mucoid degeneration.

Nonsurgical treatment includes (1) a 1- to 2-week period in a nonweight-bearing cast if the symptoms are severe, (2) anti-inflammatory agents and ice, (3) heel-cord stretching within limits of comfort, and (4) careful assessment of the foot and leg alignment, with orthotic correction if necessary. Conservative treatment is provided for 6 months. If

Fig. 5 Thickened inflamed Achilles peritenon.

symptoms persist, surgery is recommended.

Surgery consists of first debriding the overlying inflamed peritenon. The tendon is then palpated in the area of fusiform thickening or nodular enlargement. If there are only minor changes on palpation but significant preoperative symptoms, several longitudinal incisions are made into the tendon. The purposes of these longitudinal incisions are to visualize areas of central tendon necrosis, which should be excised, and to stimulate a healing reaction. If a significant area of degeneration, characterized by a glossy homogeneous appearance (Fig. 6), is found, it is excised. The area of elliptical excision is then closed. If the defect is large and the excision extensive, the Achilles tendon is reinforced using the plantaris tendon, the flexor digitorum communis, or a turndown flap.

The period of postoperative immobilization depends on the size of the defect. If a small defect is excised, the patient is immobilized for 2 weeks. If a larger defect is excised, 4 to 6 weeks of immobilization may be necessary.

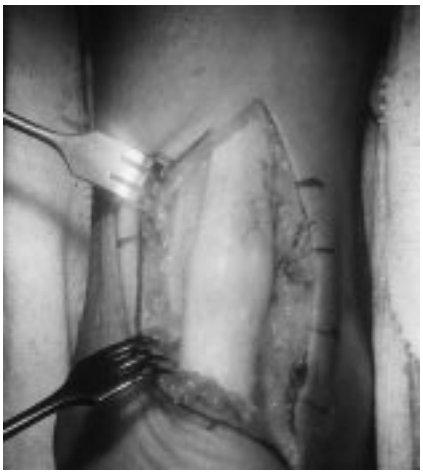

Fig. 6 Nodular Achilles mucoid degeneration. Note the smooth, glossy appearance of the involved tendon.

Partial Rupture

Partial ruptures of the Achilles tendon were thought to be rare until Ljungqvist described 24 cases in 1968.[18] Unlike total ruptures, which tend to occur in middle-aged deconditioned persons, partial ruptures occur in well-trained athletes. Partial tears usually involve the lateral aspect of the Achilles and may be longitudinal, transverse, or both. Diagnosis of partial tears, although frequently difficult, has been enhanced considerably through the use of MR imaging (Fig. 7).

If a large partial tear is identified, immediate repair should be undertaken. However, if a small defect is present, conservative treatment consisting of heel lifts, ice, and rest is usually adequate. If a small defect fails to respond to conservative treatment, surgical excision of the involved area or repair of the tear is undertaken.

Complete Rupture

Complete rupture of the Achilles tendon occurs most frequently in the middle-aged, competitive male involved in intermittent athletic activities. There may be a history of prerupture intermittent heel pain suggestive of long-standing mild chronic Achilles tendinosis. More frequently, however, rupture occurs without preexisting complaints.

Two theories are suggested to explain the cause of acute Achilles tendon rupture: (1) chronic tendon degeneration and (2) acute mechanical overload.[19] In fact, both of these factors are usually involved.

The symptoms of rupture are fairly classic. The middle-aged athlete will often hear or feel a pop while experiencing minimal discomfort. Immediate weakness in push-off is noted, followed by pain and swelling.

On physical examination, the Thompson test is positive (squeezing the calf does not cause passive ankle plantar flexion), and the patient is usually unable to perform a single heel rise. However, the patient is frequently able to plantarflex the foot when it is not bearing weight because of the plantarflexion action of the posterior tibial, toe flexor, and peroneal tendons.

The main objective of treatment is to provide the patient with a tendon as close to normal in length and strength as possible. Nonoperative measures can achieve this objective provided the length of treatment is sufficient to allow the tendon to reestablish adequate intrinsic strength and to avoid elongation with future activities.

The controversy of closed versus open treatment of Achilles tendon ruptures has been ongoing for years. When deciding between operative and nonoperative treatment, the physician and the patient should weigh carefully the risks

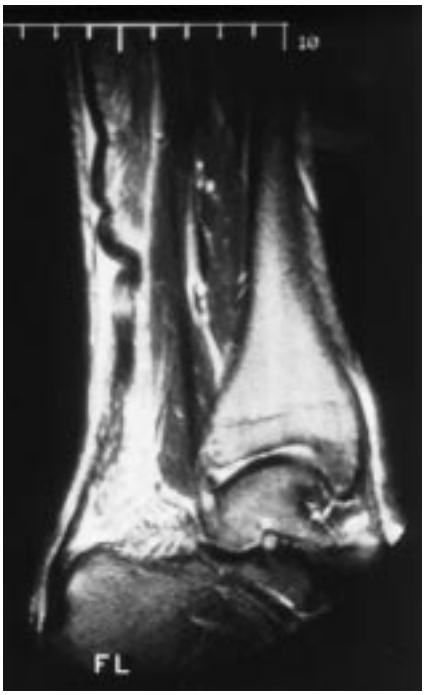

Fig. 7 Magnetic resonance image of a partial longitudinal tear of the Achilles tendon.

and benefits. The risks of closed treatment utilizing prolonged non-weight-bearing cast immobilization are decreased strength, rerupture, and stiffness.[20] The strength of the tendon is approximately 30% of normal in the conservatively treated patient group, compared with near normal in the surgical group.[21] The average rerupture rate in the conservatively treated patient is 18%, while the patient who undergoes open surgical treatment has a 2% rerupture rate.[22,23] The risks of surgery include pulmonary embolism, sural nerve injury, suture granulomas, skin problems, stiffness, and infection.

A compromise between open and closed treatment is the technique of Ma and Griffith.[24] They plantar-flex the ankle, bring the Achilles tendon ends together, and percutaneously repair the tendon. They have reported their results as excellent. The advantage of open repair is that it provides direct visualization of the disrupted tendon ends and allows restoration of the tendon to its normal length.

Postoperatively, two methods of treatment are available. One is the standard rigid immobilization. This is accomplished by using a short-leg cast for 6 to 8 weeks, followed by wearing a shoe with an elevated heel for 1 month. The second method is functional postoperative treatment, which is now gaining popularity. Mahan and Carter[21] have described the use of a postoperative functional orthosis rather than cast immobilization. Saltzman and Thermann[22] have described the use of a modified boxer's boot, which provides protection as well as functional treatment.

Posterior Tibial Tendon

Inflammation of the posterior tibial tendon is more common than anterior tibial tenosynovitis. As the tendon curves behind the medial malleolus, it functions much like a rope being pulled through a pulley. Attrition occurs at the bone-tendon points of contact. It should also be noted that during the pronation phase of running gait, the mechanical demands placed on this structure are quite high. As a result, microtrauma occurs, and the tendon may become inflamed and undergo degeneration and rupture. Posterior tibial tenosynovitis is also commonly seen in systemic inflammatory diseases such as rheumatoid arthritis.

The primary complaints of patients with posterior tibial tenosynovitis, partial rupture, or complete rupture are pain, weakness, and eventual deformity. The pain is aggravated by activity and is partially relieved by rest and anti-inflammatory agents. When the tendon ruptures completely, the pain may be referred from its usual medial location to the lateral aspect of the ankle in the sinus tarsi region. This type of pain is associated with the development of a valgus deformity. The anterior process of the talar articular surface of the posterior facet impinges on the superior aspect of the calcaneus. The eventual severe clinical deformity secondary to posterior tibial tendon rupture is a combination of hindfoot valgus with forefoot abduction and pronation.

Treatment is determined by the degree of involvement of the posterior tibial tendon. If the patient has swelling and inflammation of only a few weeks' duration, an aggressive conservative treatment program should be instituted. This program consists of 4 to 6 weeks of cast immobilization holding the foot slightly inverted and plantar-flexed. If immobilization is unsuccessful, one can consider a corticosteroid injection into the tendon sheath without injecting steroid into the tendon itself.

If the patient is unresponsive to conservative treatment after a few months, surgical treatment is indicated to prevent further damage to the tendon. At the time of surgery a thorough tenosynovectomy is undertaken. The tendon is also thoroughly inspected. If the tendon is intact but minimal longitudinal rents in the tendon are found, the rents are either sutured or debrided.

If the tendon is detached from the navicular, severely attenuated, or ruptured, the treatment plan should be based on the degree of deformity. If the patient has minimal or no deformity, attempts should be made either to reattach the tendon to the navicular through a bony tunnel or to augment the ruptured tendon. If augmentation is chosen, the tendon of choice is the flexor digitorum communis. If the deformity is severe and well established, the patient usually has had a moderate degree of pain for a period of years. A subtalar arthrodesis is used in this setting. An isolated talonavicular arthrodesis or a talonavicular arthrodesis in combination with a calcaneal cuboid arthrodesis may also be performed to stabilize the hindfoot.

An unusual problem is recurrent subluxation. The diagnosis is based on the patient's symptoms, physical examination findings, and MR imaging evaluation. Surgical repair is generally indicated.[25]

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