
MRI of Sports-Related Injuries of the Foot and Ankle: Part 2

José A. Narváez, MD,^a Luís Cerezal, MD,^b and Javier Narváez, MD^c

Tendon Lesions

The ankle tendons can be divided into four anatomic and functional groups. Medially, the posterior tibial, flexor digitorum longus (FDL), and flexor hallucis longus (FHL) assist in inverting the foot as well as flexing the toes. Laterally, the peroneus brevis and peroneus longus act to evert the foot. Anteriorly, the anterior tibial, extensor hallucis longus, extensor digitorum longus, and peroneus tertius are responsible for dorsiflexion of the foot and extension of the toes. The Achilles tendon is the primary posterior tendon and acts to plantar flex the foot. On magnetic resonance imaging (MRI), tendons demonstrate homogeneous low-signal intensity regardless of pulse sequence. To evaluate ankle and foot tendons, two imaging planes with at least two pulse sequences are required. Ankle tendons are best evaluated in oblique axial imaging perpendicular to the long axis of the tendon. Sagittal imaging allows a better depiction of the longitudinal extent of most tendon ankle injuries.

The spectrum of tendinous lesions includes tenosynovitis, stenosing tenosynovitis, tendinosis-tendinitis, rupture, and dislocation. Tenosynovitis is an inflammation of the tendon sheath, characterized by a considerable amount of synovial fluid that distends the tendon sheath. This condition demonstrates decreased signal intensity on T1-weighted MR images and increased signal intensity on T2-weighted images sur-

rounding low-signal intensity tendons. It is interesting to note that presence of small amounts of fluid in flexor tendon sheaths is considered a normal variant, especially in the FHL, which communicates with the ankle joint in 20% of patients.¹⁰ Repetitive acute tenosynovitis can progress to a stenosing tenosynovitis, a chronic condition characterized by the formation of fibrous or scar tissue in the tendinous sheath that interferes with tendon gliding. These fibrous changes appear as an intermediate signal-intensity ring surrounding the tendon on both T1- and T2-weighted images.

Tendinitis results from the inflammation within the tendon substance, whereas tendinosis is a structural degeneration of the tendon. These two terms are often used to designate an overuse tendon injury, which, in a chronic stage, is histologically characterized by minimal or absent inflammatory changes.¹¹⁻¹³ Thus, the term tendinosis is preferred by most authors. Tendinosis is characterized by variable tendinous thickening and increased signal intensity on T1-weighted or proton-density-weighted MRI.^{1,6} Tendinous signal intensity is normal or only slightly increased on T2-weighted images. Partial rupture appears as a tendon thickening with intratendinous areas of increased signal intensity on T1-weighted, proton-density, and T2-weighted images, similar to that seen in chronic tendinosis.^{1,6} Complete rupture is defined by the loss of continuity of the tendon fibers.

Achilles Tendon

The Achilles tendon is the longest tendon of the body. It is formed by the union of the tendons of the gastrocnemius and soleus muscles and inserts on the posterior aspect of the calcaneus. Distally, the tendon is separated from the calcaneus by the retrocalcaneal bursa. It is not invested by a synovial sheath; instead it is surrounded by loose connective tissue referred to as

From the ^aDepartment of CT and MR imaging, I.D.I. Ciutat Sanitària i Universitària de Bellvitge, Barcelona, Spain; ^bDepartment of Radiology, Instituto Cántabro de Radiología, Clínica Mompía, Santander, Spain; ^cDepartment of Rheumatology, Clínica Delfos, Barcelona, Spain.
Reprint requests: Dr. José Antonio Narváez, Department of MR imaging, Institut de Diagnòstic, Ciutat Sanitària i Universitària de Bellvitge, Hospital Duran i Reynals, Autovia de Castelldefels s/n, Hospitalet de Llobregat, 08907, Barcelona, Spain.
Curr Probl Diagn Radiol 2003;32:177-193.
© 2003 Mosby, Inc. All rights reserved.
0363-0188/2003/\$30.00 + 0
doi:10.1016/S0363-0188(03)00044-6

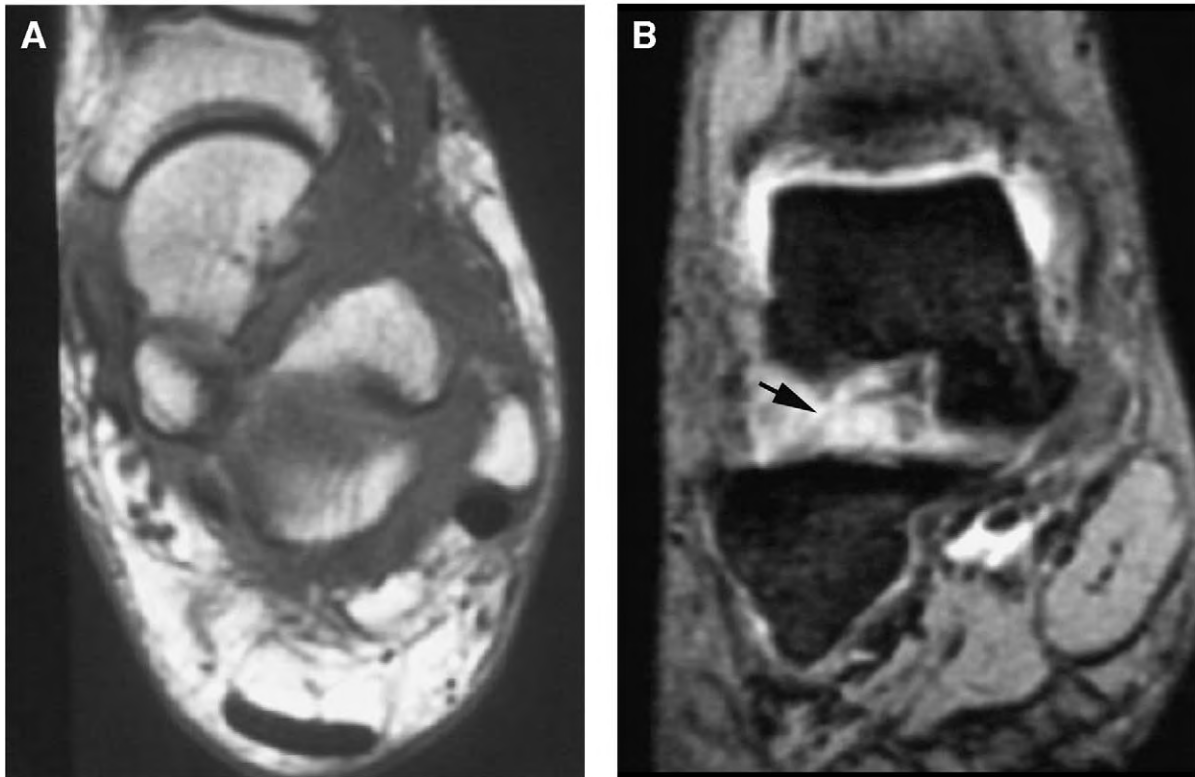


FIG 20. Sinus tarsi syndrome. Axial T1-weighted spin-echo (**A**) and coronal T2*-weighted (**B**) MRI show diffuse infiltration of the sinus tarsi consistent with chronic cynovitis and nonspecific inflammatory changes. The interosseous ligament is poorly defined (arrow).

the peritenon. The peritenon acts as an elastic sleeve that allows the tendon to move freely.

Achilles tendon injuries may be divided into insertional and noninsertional complaints.¹³ Insertional injuries occur near or at the insertion of the tendon and include insertional tendinosis and retrocalcaneal bursitis, which often coexist (Figs 21 and 22). Insertional injuries may be associated with a Haglund deformity, which is a prominence of the posterosuperior aspect of the calcaneus (Fig 22).^{11,13} Noninsertional injuries are usually located 2-6 cm from the Achilles tendon insertion in the calcaneus in an area characterized by decreased vascularization.^{11,13} These injuries include peritendinitis, tendinosis, and tendon rupture (Figs 23 and 24).

Achilles tendon injuries are common in the course of sports-related activities.^{11,13} Most of these sports-related injuries consist of peritendinitis, tendinosis and insertional complaints caused by overuse.^{11,13} These injuries are very common among runners and, less commonly, among dancers, tennis players, and basketball players.^{3,11,13} Clinical differentiation between achilleal tendinosis and peritendinitis is challenging, and both conditions may coexist in the same patient.¹²

The main symptom is pain associated with physical activity that improves with rest. Frequently, the pain is greater when initiating the activity and improves gradually with the exercise.

On MRI, Achilles peritendinitis shows a loss of normal signal intensity of peritendinous fat, with ill-defined reticulated areas of decreased signal intensity on T1-weighted images and increased signal on short tau inversion recovery (STIR) and T2-weighted images that represent inflammatory and edematous changes. Scarring of the preachilleal fat, with low-signal intensity, is seen in cases of chronic Achilles peritendinitis.¹ Tendinosis is characterized by tendon thickening and loss of the anterior concave or flat surface on axial images (Fig 23).^{12,14}

Intratendinous areas of increased signal intensity are often seen on T1-weighted and proton density-weighted images. Increased signal intensity on STIR and T2-weighted images is less commonly seen and is related to a more severe derangement of tendon fiber structure.¹² Insertional tendinosis may be associated with calcaneal marrow edema and retrocalcaneal bursitis (Figs 21 and 22).^{3,12,14} On MRI, a bursal fluid



FIG 21. Insertional Achilles tendinitis and retrocalcaneal bursitis. Sagittal STIR MRI demonstrates thickening of the distal Achilles tendon with increased intratendinous signal intensity (short arrows). Note also distention of the retrocalcaneal bursa by high-signal intensity fluid (long arrow), a finding that represents bursitis.

collection larger than 7 mm craniocaudally or 11 mm transversely is considered diagnostic of bursitis (Figs 21 and 22).^{4,15}

Treatment consists of rest, avoidance of the aggravating athletic activity, shoe modification, heel elevation to reduce tendon stretching during walking, nonsteroidal anti-inflammatory drugs, and physical therapy.

Achilles tendon ruptures, which are thought to be because of underlying tendinosis, are uncommon.¹¹ Most Achilles tendon ruptures are related to sports activities. These injuries usually occur in sports that require running, jumping, and quick turns.^{3,11} Partial ruptures usually occur in well-trained athletes, presenting as chronic overuse injuries.¹¹ Conversely, complete ruptures tend to occur in middle-aged, poorly conditioned men after abrupt calf muscle contraction.¹¹ In partial Achilles tendon rupture, active plantar flexion of the ankle may be preserved but painful. In those patients with complete Achilles tendon rupture, it is still possible to actively plantar-flex the ankle with the adjacent intact flexor tendons. However, squeezing the calf muscles with the patient sitting or kneeling on a chair produces little or no passive ankle plantar flexion (positive Thompson calf squeeze test). Clinical differentiation between partial and complete rupture in cases of chronic tendinosis

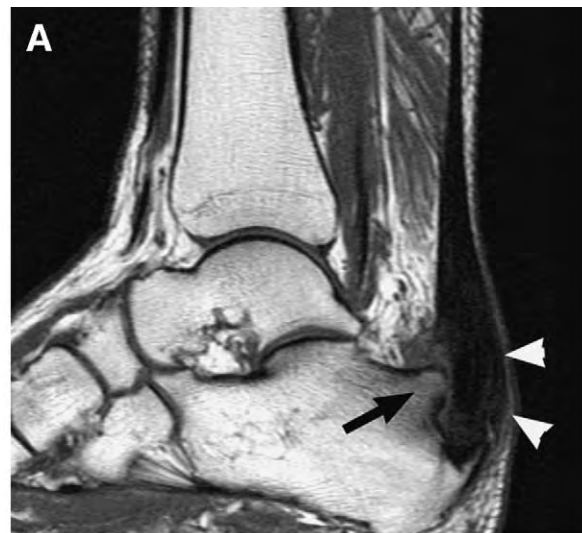


FIG 22. Haglund deformity. Sagittal T1-weighted (A) and STIR (B) MRI show a prominent posterior superior calcaneal tuberosity (arrow), associated with marked Achilleal thickening and intratendinous increased signal intensity (arrowheads). Note also associated retrocalcaneal bursitis.

might be complex. Moreover, clinical diagnosis of complete ruptures of the Achilles tendon is missed in about 25% of the cases.¹¹

MRI is useful to differentiate between partial and complete ruptures, to plan the surgical treatment in complete ruptures, and in the follow-up after surgical intervention.¹⁶ On MRI, partial ruptures appear as a tendon thickening with intratendinous areas of increased signal intensity on T2-weighted images. In cases of acute partial rupture, edematous and hemorrhagic changes in the peritenon are often seen.¹ Differential diagnosis with severe chronic

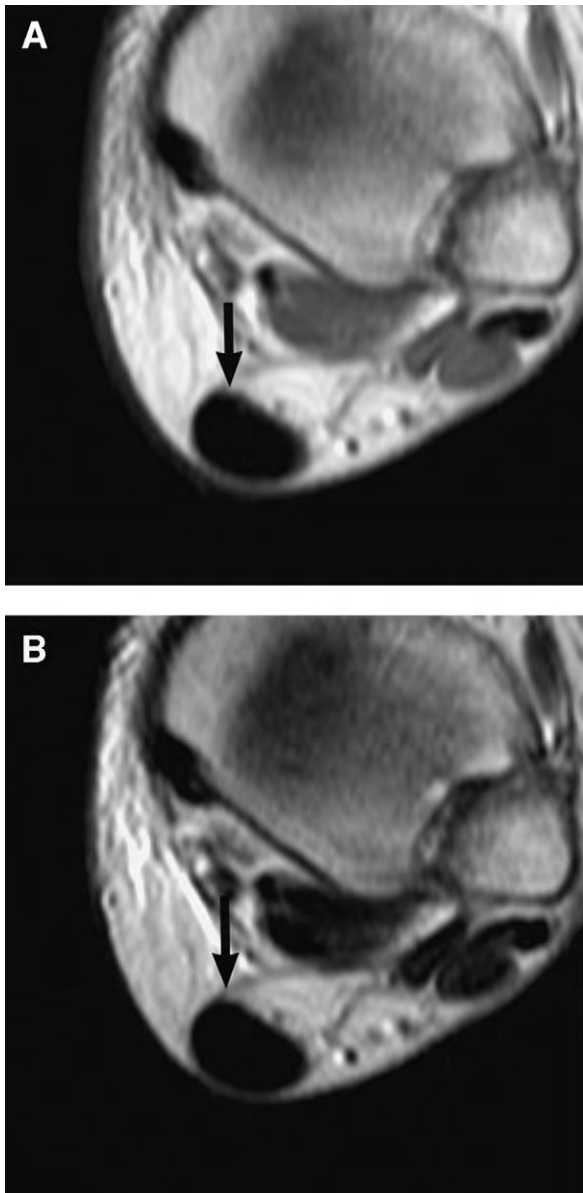


FIG 23. Achilles tendinitis. Axial proton-density-weighted (**A**) and T2-weighted (**B**) MRI show loss of the anterior concavity of the Achilles tendon (arrow) without evidence of intrasubstance increased signal intensity.

tendinosis only can be established on the basis of the clinical presentation of the patient.¹ Acute complete ruptures are seen in MRI as a complete disruption of the tendon fibers (Fig 24). The tendinous gap is filled by intermediate- to high-signal intensity on T1-weighted images and high-signal intensity on T2-weighted images indicative of edema or hemorrhage.¹⁶ Fraying or retraction of the tendon ends can be observed. In chronic complete rupture, the tendon gap may be occupied by

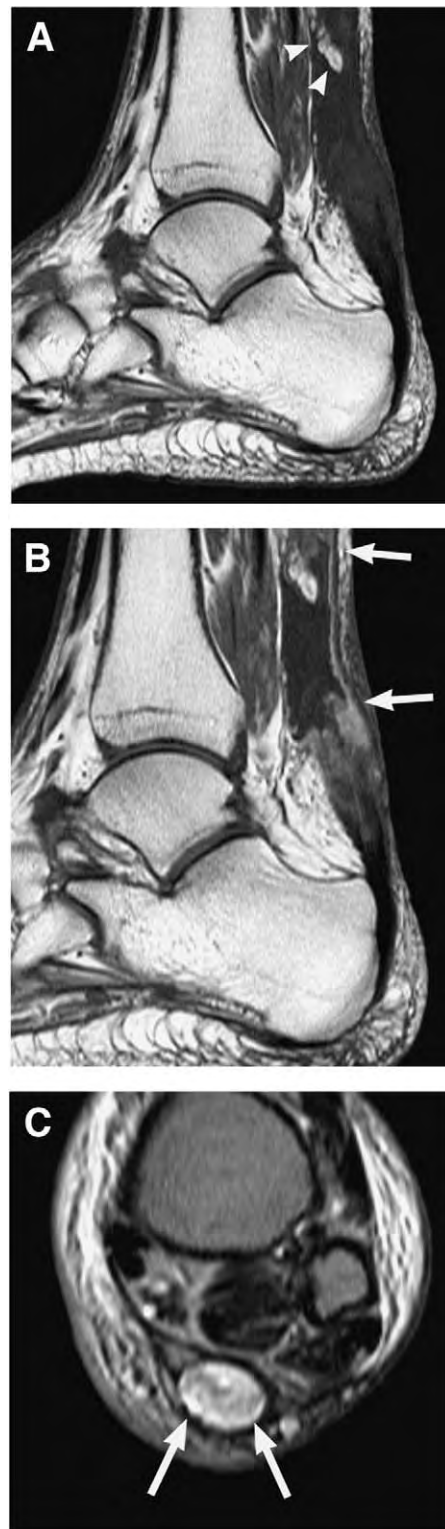


FIG 24. Acute complete tear of the Achilles tendon. **A**, Sagittal T1-weighted depicts complete disruption and retraction of the torn ends of the Achilles tendon with a tendon gap. Note small hyperintense foci corresponding to hemorrhage (arrowheads). **B**, Sagittal GD-enhanced T1-WI shows enhancement of the tendon ends, with better delineation of the tear (arrows). **C**, Axial T2-weighted shows the tendon gap filled with high signal intensity fluid (arrows).

fat and/or scar tissue, and hemorrhagic and edematous changes are absent.

The treatment of partial ruptures and, in some cases of complete ruptures, is conservative. Surgical treatment seems to be the treatment of choice for complete ruptures in athletes and young people and in cases of delayed ruptures.

Peroneal Tendons

The lateral compartment of the leg is formed by the muscles peroneus longus (PL) and peroneus brevis (PB). Their tendons pass behind the lateral malleolus within a fibro-osseous tunnel. The superior peroneal retinaculum forms the posterolateral border of this fibro-osseous tunnel. It extends inferiorly and posteriorly from the lateral malleolus to the lateral calcaneal surface, binding the PL and PB tendons. Distal to the lateral malleolus, the PB tendon runs adjacent to the lateral wall of the calcaneus within a separate osteo-aponeurotic canal created by the inferior peroneal retinaculum. The PB tendon inserts at the base of the fifth metatarsal. The PL tendon passes below the peroneal tubercle of the calcaneus and then it runs through a fibro-osseous tunnel formed by the long plantar ligament and the peroneal groove of the cuboid until it inserts onto the plantar face of the base of the first metatarsal and medial cuneiform.

Peroneal tenosynovitis is an overuse injury relatively common in athletic individuals.^{17,18} Displaced fractures of the calcaneus, enlarged peroneal tubercle of the calcaneus, and alterations of the mechanics of the foot (ie, flat foot, tarsal coalition) are considered predisposing factors.^{1,19} Clinical findings are pain and swelling in the inferolateral malleolar region, which can radiate to the external face of the leg. On physical examination, flexion and pronation of the foot are painful. Although a small amount of fluid in the peroneal tendon sheath is physiological,^{10,18} MR identification of a large volume of fluid indicates tenosynovitis (Fig 25). However, after an ankle sprain, the detection of a large amount of fluid in the peroneal tendon synovial sheath indicates the rupture of the calcaneofibular ligament.^{8,19}

Peroneal tendinosis represents a more advanced overuse injury that develops over a period of several weeks or months.¹⁹ MRI demonstrates tendon thickening and intratendinous signal abnormalities.¹⁶ In acute peroneal tendinosis, intratendinous signal abnormalities are less extensive than in chronic forms.¹⁸

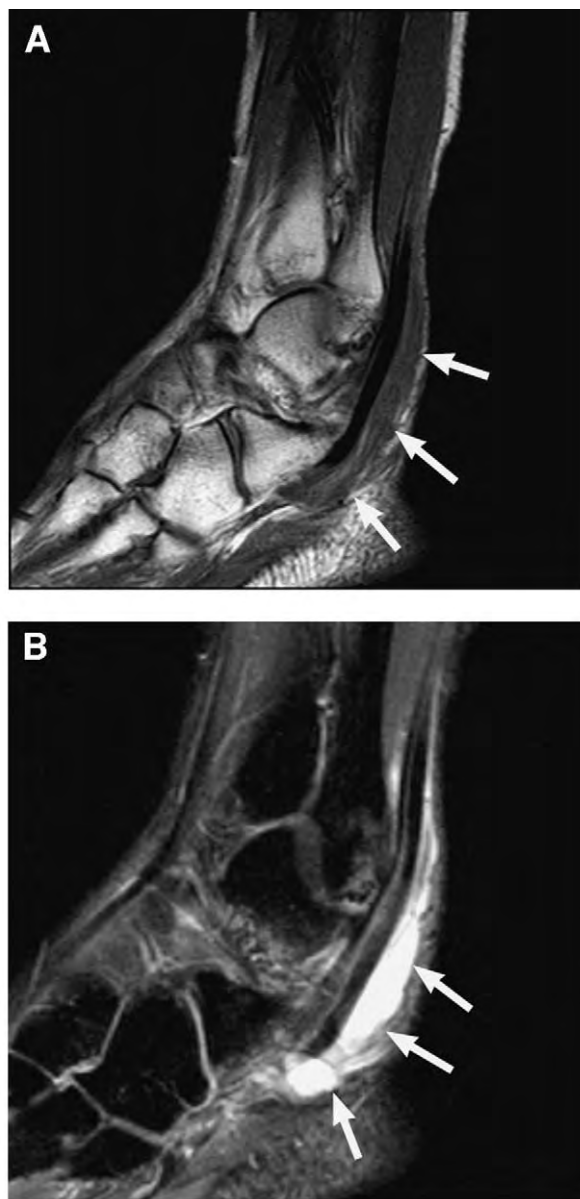


FIG 25. Peroneal tenosynovitis. Sagittal T1-weighted (A) and STIR (B) MRI show a fluid distending the synovial sheath of the peroneal tendons (arrows). Note mild intrasubstance increased signal signal in the PL tendon on STIR image.

Longitudinal ruptures of the peroneal tendons have been described more frequently in recent years.¹⁷⁻²² These injuries may occur as a result of overuse in dancers, runners, and competitive walkers.^{1,22} Longitudinal rupture or split lesion of the PB tendon occurs at the level of the lateral malleolus and can progress proximally or distally.^{21,22} It takes place as a result of the repeated compression of the PB tendon between the lateral malleolus and the PL tendon¹⁷⁻²² and is often associated with lateral ligamentous injuries,

insufficiency of the superior peroneal retinaculum, and repetitive peroneal tendon subluxation.^{1,19,24} A flattened or convex retromalleolar groove, a low-lying muscle belly of the PB tendon, and the existence of an accessory muscle, the peroneus quartus, are predisposing factors for longitudinal tearing of the peroneus brevis tendon.^{1,19,21,23} Clinically, it presents with long-standing retromalleolar pain and swelling, and a history of recurrent inversion injuries is often noted. Longitudinal ruptures of the PB tendon are often confused with chronic ankle instability syndrome and, indeed, both are frequently associated.^{19,21,23} These lesions can be difficult to diagnose, and MRI is an useful technique for demonstrating tendon tears as well as showing associated or predisposing abnormalities.¹⁸⁻²²

MRI findings include morphologic and signal intensity abnormalities of the PB tendon as well as marked peroneal tenosynovitis. The torn PB often wraps itself against the PL tendon, assuming a C-shaped configuration (Figs. 26 and 27).^{18,19,21,22} Some authors describe this morphologic abnormality as the *chevron sign*.²⁰ In advanced stages, the PB tendon may be completely divided in two portions (Fig 27).¹⁸⁻²² Intrastance areas of high-signal intensity on T2-weighted images in the PB tendon are commonly seen.^{22,24} About one third of PB tendon longitudinal ruptures are associated with PL tendon tears (Fig 27).¹⁹ When the PB tendon is bisected, the PL tendon comes into direct contact with the hard surface of the retromalleolar groove and becomes exposed to similar mechanical attrition, which causes a PB tendon tear.²⁹ On MRI, thickening and increased signal PL tendon are seen (Fig 27).

Ruptures of the PL tendon in the midfoot can develop as a result of sports-related trauma.^{17,25} These tears are typically located at the level of the calcaneocuboid joint, distal to the os peroneum.^{18,26} The lesional mechanism is an acute inversion or forced eversion of a supinated foot, causing forced contraction of the PL tendon.²⁶ Hypertrophied peroneal tubercle of the calcaneus and calcaneal and cuboid bone fractures are predisposing factors for PL tear.¹⁹ Clinical diagnosis is difficult, and MRI is often required to confirm the diagnosis.^{19,26} These injuries are better identified on oblique coronal MRI. MRI findings include an empty PL tendon sheath with retracted proximal and distal segments of the tendon in complete tears, and tendon thickening and increased signal intensity in partial tears.²⁶ Other associated MR find-

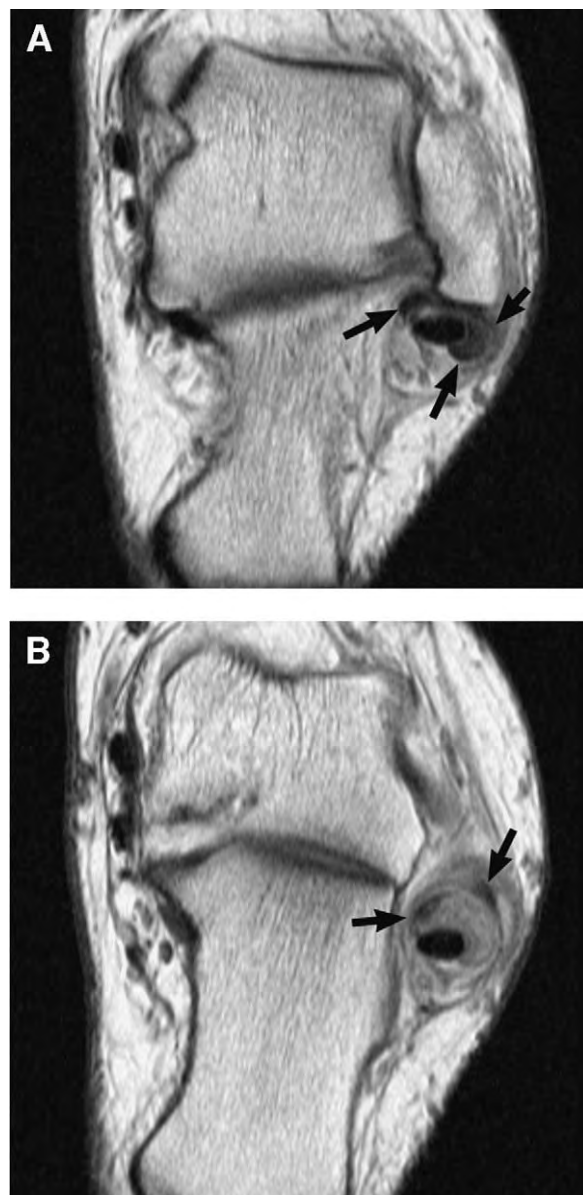


FIG 26. Longitudinal rupture of the PB tendon. **A, B,** Consecutive oblique axial proton-density-weighted images. At the level of the retromalleolar groove, the PB tendon presents a C-shaped configuration (arrows), wrapping against the adjacent the PL tendon. Distally to the lateral malleolus, the PB tendon is completely divided in two portions (arrows).

ings are increased tenosynovial fluid, enlarged peroneal tubercle, and bone marrow edema in the peroneal tubercle as well as along the lateral wall of the calcaneus.²⁶ Treatment of peroneal tendon tears in athletes is surgical in most cases.

Subluxation and dislocation of the peroneal tendons are common in skiers and to a lesser extent in ice skaters and basketball and soccer players.^{18,19} The lesional mechanism is traumatic detachment of supe-

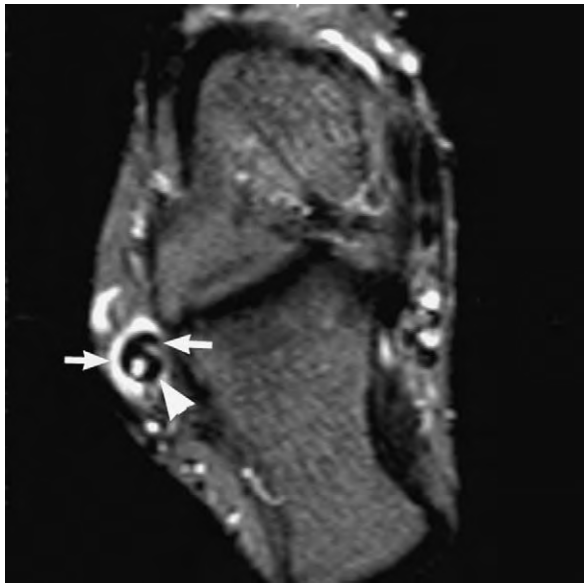


FIG 27. Longitudinal tear of the PB and PL. Axial T2-weighted shows a C-shaped split PB tendon (short arrow) partially enveloping the PL tendon that is thickened and presents a high signal intratendinous focus (long arrows). Synovial tendinous sheath is filled with high-signal intensity fluid.

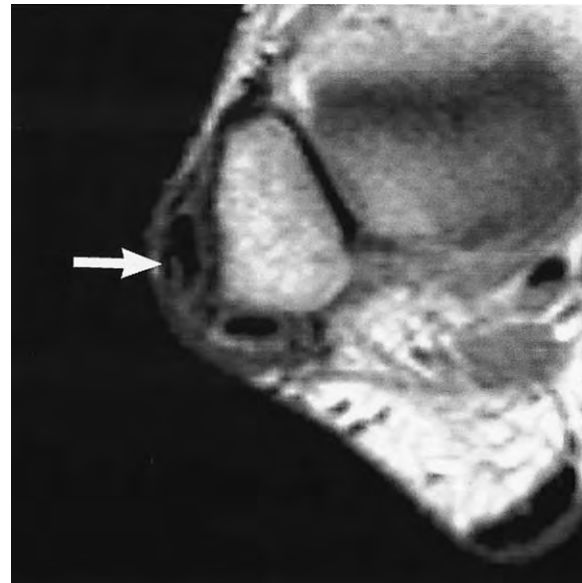


FIG 28. Dislocation of PB tendon. Consecutive axial proton density-weighted MRI shows the PB tendon (arrow) lateral to the peroneal malleolus. Note peroneus longus tendon normally located behind the malleolus.

rior peroneal retinaculum produced by a sudden dorsiflexion injury, with either inversion or eversion of the foot. Chronic ankle instability associated with superior peroneal retinaculum is considered a predisposing factor for chronic peroneal dislocation. Congenital absence of the superior peroneal retinaculum, low-lying PB muscle belly, peroneus quartus muscle, and flattened or convex retromalleolar grooves are also considered predisposing factors.^{18,19} Acute dislocation of the peroneal tendons may be clinically confused with lateral ankle ligament sprain. Physical examination demonstrates tenderness around the retromalleolar tunnel. Tendon luxation is elicited with foot plantar flexion and eversion. MRI reveals the peroneal tendons located anteriorly and laterally to the distal fibula (Fig 28). The tendon dislocation is best demonstrated on axial images that are also useful in identifying the torn or stripped-off superior peroneal retinaculum.^{18,19} Associated MR findings are tenosynovitis and longitudinal ruptures of the peroneal tendons.^{1,19} Treatment of peroneal dislocation in athletes with lateral ankle pain and instability is surgical.

FHL and FDL Tendons

The FDL and the FHL tendons course through the posteromedial ankle. The FDL tendon lies just posterolaterally to the posterior tibial tendon, running

posteriorly to the talus. The FHL tendon courses posterolaterally to the posterior tibial tendon and the FDL tendon, running through a shallow groove in the posteromedial aspect of the talus between the lateral and medial processes; it then continues distally under the sustentaculum tali. On the plantar aspect of the heel, the FHL tendon crosses deep to the FDL tendon, and both course along the plantar aspect of the sole. The FHL tendon inserts on the big toe. The FDL tendon inserts on the bases of the second through the fifth distal phalanges.

FHL tenosynovitis is common in athletes who perform extreme plantar flexion and push-off maneuvers from the forefoot, such as ballet dancers and soccer players.^{1-3,27} Repetitive full plantar flexion compresses the tendon within the talar tunnel.^{2,3} Repetitive episodes of acute tenosynovitis may progress to stenosing tenosynovitis, leading to fibrosis, tendon entrapment, and tendinosis.²⁷ Clinically, FHL tenosynovitis can mimic posterior impingement, and both often may coexist.²⁷

In acute tenosynovitis, synovial fluid surrounding the FHL tendon can be seen on MRI. Because synovial sheaths of the FHL and FDL tendons communicate with the ankle joint in 20% of individuals,¹⁰ the diagnosis of FHL tenosynovitis requires a disproportionate amount of fluid within the sheath.^{1,4,10} Fibrotic changes of stenosing tenosynovitis appear as an inter-

mediate-signal-intensity rind surrounding the tendon on both T1- and T2-weighted images.⁴ Other MR findings of FHL stenosing tenosynovitis are a FDL accessory muscle, distal FHL muscle edema, and os trigonum edema.²⁷

Treatment is conservative and includes rest, nonsteroidal anti-inflammatory drug therapy, and physical therapy. Surgical release of the FHL tendon is performed if conservative treatment fails.²⁷

Tenosynovitis may also affect the FHL tendon between the sesamoid bones of the first metatarsal, where it is subject to repetitive impact, and under the base of the first metatarsal bone in the region of Henry's knot, where the FDL crosses under the FHL tendon.¹ Rupture of the distal FHL tendon is a very uncommon injury that occurs as a result of acute or repetitive dorsiflexion injuries.

Posterior Tibial Tendon

The posterior tibial muscle forms part of the deep posterior compartment of the calf. It originates from the proximal third of the tibia and interosseous membrane and sends a tendon that runs directly behind the medial malleolus. The tendon then curves sharply toward its main insertion on the navicular tuberosity. The posterior tibial tendon is the most powerful inverter of the foot. It participates in maintaining the medial longitudinal arch and is one of the main stabilizers of the hindfoot against valgus deformity.

Posterior tibial tendon injuries in athletes are uncommon.²⁸ Acute tenosynovitis represents almost all of these sports-related injuries^{13,29} and has been described as an overuse injury in young athletes engaged in running and in sports that require rapid changes in direction, including ice hockey, soccer, basketball, and tennis.^{28,29} Clinically, tenosynovitis is characterized by medial ankle pain and swelling. On MRI, tenosynovial changes are seen along the course of the tendon behind or distally to the medial malleolus.

Tendon rupture of the posterior tibial tendon is rarely seen in athletes. Usually, these tendon tears occur in middle-aged athletes, but cases in young patients have been described.²⁹ The most common site of rupture is at the level of the medial malleolus. Surgical and MRI classification of posterior tibial tendon ruptures divides these injuries into three types. Partial tears are classified as hypertrophic (type I) or atrophic (type II). Type III consists of complete tears. Tendon rupture results in a progressive flatfoot deformity, with hindfoot valgus and forefoot abduction.



FIG 29. Posterior tibial tendon hypertrophic partial tear (type I). Oblique axial T2-weighted MRI. Note markedly enlarged tendon girth (arrow) and mild intrasubstance signal heterogeneity. There is also tenosynovial fluid surrounding the tendon.

Clinical diagnosis of posterior tibial tendon ruptures in athletes is often missed. Early diagnosis and treatment are important to avoid irreversible deformities and prolonged disabilities. In this setting, MRI is a useful diagnostic tool and helps to plan treatment.^{30,31} MRI can distinguish between partial and complete tears of the posterior tibial tendon.²⁸⁻³¹ In hypertrophic tears, the tendon is increased in size and loses its oval shape, appearing with a more rounded configuration (Fig 29). The signal intensity is often markedly increased, more so on proton density and T1-weighted than on T2-weighted images. In atrophic tears, the tendon shows thinning and an increased signal. Complete tears are delineated by a tendinous gap filled with fluid or granulation tissue, depending on the chronicity of the injury.

Most patients with tenosynovitis or type I tendon rupture respond to conservative treatment with anti-inflammatory medication, protected mobilization, and physical therapy.²⁹ Surgical treatment is reserved for cases in which conservative therapy fails, complete tendon rupture, or progressive deformity.

The presence of an accessory navicular bone is a predisposing factor for posterior tibial tendon tears.⁹ This ossicle, which is present in up to 14% of the population, is an anatomic variant that increases stress on the distal posterior tibial tendon, which predisposes it to rupture. Moreover, the accessory navicular bone itself may become symptomatic.³² This clinical condition has been termed painful accessory navicular bone.³² There are three types of accessory navicular: type I is a large sesamoid bone that is located in the posterior tibial tendon and is usually asymptomatic. Type II is a triangular or heart-shaped ossicle connected to the tarsal navicular by a cartilaginous or fibrocartilaginous bridge. Type III accessory navicular corresponds to a prominent navicular tuberosity.

Most cases presenting a painful accessory navicular bone have a type II variant. The lesional mechanism may be either acute or chronic repetitive trauma that damages the synchondrosis. Clinical symptoms include recurrent pain on the medial ankle that is aggravated by prolonged exercise. In cases of painful accessory navicular bone, MRI may show bone marrow edema changes in the ossicle and in the adjacent medial aspect of the navicular bone and surrounding increased signal intensity on STIR images reflecting inflammatory and edematous changes.³²

Anterior Tibial Tendon

The anterior tibial tendon is a dorsiflexor of the foot that originates on the anterior surface of the tibia. At the ankle joint, it passes under the superior and inferior extensor retacula and inserts on the medial cuneiform bone and base of the first metatarsal bone.

Injuries of the tendons on the anterior aspect of the ankle are uncommon.¹ The anterior tibial tendon may be injured in athletes engaged in downhill running or hiking.¹⁶ Tenosynovitis and tendinitis are more common than tendon rupture. Rupture of the anterior tibial tendon is a very rare injury in athletes. The tendon rupture is caused by a forced plantarflexion and most cases occur in middle-aged or elderly patients, who present with degenerative changes of the tendon. Main symptoms are inability or weakness of foot dorsiflexion, pain, and a palpable gap. Delayed diagnosis is common, and MRI can help to establish the diagnosis. The usual site for tendon rupture is 0.5 to 3 cm proximal to its bony insertion, where the tendon passes under the inferior extensor reticulum.³³ Complete discontinuity and retraction of the proximal tendon segment are seen on MRI as indicative of complete

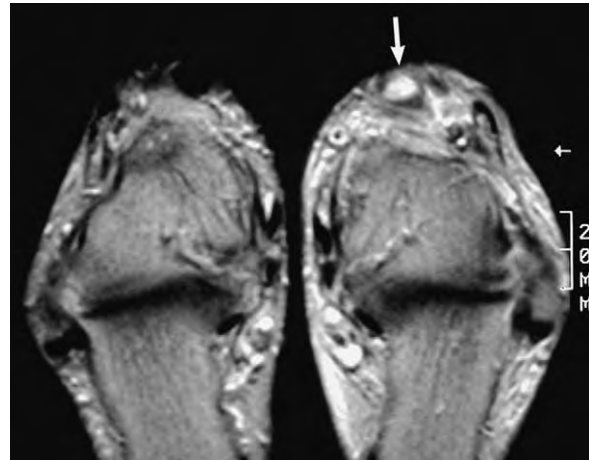


FIG 30. Acute complete tear of the tibialis anterior tendon in an older tennis player. Note the absence of left tendon with high signal fluid at the tear site (arrow) and surrounding edema changes on axial T2-weighted images. The right tibialis anterior tendon (ta) has normal size and intensity signal.

anterior tibial tendon rupture (Fig 30).³³ Treatment of anterior tibial tendon rupture is surgical.

Ankle Impingement Syndromes

Ankle impingement syndromes are painful entities caused by the friction of joint tissues, which is both the cause and the effect of altered joint biomechanics. The leading causes of impingement lesions are posttraumatic ankle injuries, usually ankle sprains, leading to chronic ankle pain.^{34,35}

From the anatomical and clinical viewpoints, these syndromes are classified as anterolateral, anterior, anteromedial, posteromedial, and posterior.³⁴ Careful analysis of patient history and features at physical examination can suggest a specific diagnosis in most cases. MRI and magnetic resonance arthrography (MRA) are the most useful imaging methods for detecting the osseous and soft-tissue abnormalities present in these syndromes and to rule out other potential causes of chronic ankle pain.³⁴

Anterolateral Impingement Syndrome (ALI)

ALI is a syndrome produced by entrapment of abnormal soft tissue in the anterolateral gutter of the ankle.³⁴⁻³⁹ ALI is a relatively uncommon cause of chronic lateral ankle pain and is frequently seen after single or multiple ankle inversion injuries. It is estimated that 3% of ankle sprains may lead to ALI.³⁴ ALI is thought to occur subsequent to relatively minor

trauma involving forced ankle plantar flexion and supination. The traumatic insult is usually sports related and is found most frequently in soccer, basketball, and football players. Such trauma may result in tearing of the anterolateral soft tissues and ligaments without substantial associated mechanical instability. Repeated microtrauma and soft-tissue hemorrhage can result in synovial scarring, inflammation, and hypertrophy in the anterolateral gutter of the ankle, with subsequent soft-tissue impingement.³⁴⁻³⁹ Other contributing factors are thought to include hypertrophy of the inferior portion of the anterior inferior tibiofibular (AITF) ligament and osseous spurs. First described by Bassett, a separate distal fascicle of the AITF ligament is a common variant.²⁰ It becomes pathologic when a tear of the anterior talofibular ligament results in anterolateral joint laxity. With increasing joint laxity, the talus extrudes anteriorly in dorsiflexion and comes into contact with the fascicle. Constant rubbing of the fascicle against the talus thickens the fascicle, which then develops into an impinging lesion in the anterolateral gutter.⁴⁰

The clinical diagnosis of ALI can be established based on the combined presence of the following signs and symptoms: chronic ankle pain after an ankle sprain, anterolateral ankle joint tenderness, recurrent joint swelling, anterolateral pain with forced ankle dorsiflexion and eversion, pain during the single-leg squat, and lack of lateral ankle stability.³⁴⁻³⁹ However, the clinical diagnosis of ALI is one of exclusion. Lesions producing similar symptoms have to be excluded before invasive treatment is instituted, because similar symptoms can be attributed to peroneal tendon tears or subluxations, sinus tarsi syndrome, stress fractures, loose bodies, osteochondral lesions, bony impingement, and degenerative joint disease.^{34,38} When the diagnosis is straightforward, no supplementary imaging examination is necessary. In complicated cases, CT arthrography, MRI, and MRA have been used to aid the diagnosis.³⁴⁻³⁹

The MR findings of an abnormal soft-tissue mass or fibrous band in the anterolateral ankle gutter, distinct from the anterior talofibular ligament, suggest the diagnosis of ALI.³⁴⁻³⁹ The mass has low signal on T1-weighted and low or intermediate signal on T2-weighted images. Care should be taken not to confuse the frayed margins of the torn ATF ligament with the meniscoid lesion.³⁴⁻³⁹

Controversies exist concerning the accuracy of the MRI in the diagnosis of ALI. Most authors believe that

the assessment of the anterolateral recess with conventional MRI is accurate only when a substantial joint effusion is present.³⁹ MRA has been proven to be an accurate technique for assessing the presence of soft-tissue scarring in the anterolateral recess of the ankle and elucidating its extent in patients with ALI before arthroscopy.³⁸ Another MRA finding of ALI is the absence of a recess of arthrographic fluid between the anterolateral soft tissues and the anterior surface of the fibula. This absence may be a result of the presence of adhesions and scar tissue that prevent fluid entering the normal recess between the fibula and joint capsule.³⁸ Anterolateral scarring and/or synovitis at MRA can be noted in patients without clinical features consistent with those of ALI (Fig 31). Nevertheless, the identification of abnormal soft tissue itself does not imply the presence of clinical ALI. Therefore, image confirmation of anterolateral soft-tissue abnormalities must be considered with the clinical findings.³⁸

Most patients with ALI respond to conservative therapy, including nonsteroidal anti-inflammatory drugs, rehabilitative physiotherapy, or local injection of steroids. If nonoperative treatment fails after 6 months, significant relief has been shown to be provided by arthroscopic debridement of hypertrophic synovial tissue in the lateral gutter.³⁴⁻³⁹

Anterior Impingement Syndrome

Anterior impingement is a relatively common cause of chronic pain in the ankle, especially in athletes subjected to repeated stress in ankle dorsiflexion, that is typical in soccer players.³⁴ This condition involves a beak-like prominence at the anterior rim of the tibial plafond, usually associated with a corresponding area over the apposed margin of talus proximal to the talar neck, well within the anterior ankle joint capsule. These osteophytes can impinge on each other, especially with ankle dorsiflexion, trapping soft tissues, and may actually limit motion.^{34,41} The cause and origin of anterior impingement are uncertain and many factors are probably involved. It has been suggested that forced dorsiflexion results in repeated microtraumas on the tibia and talus, leading to microfractures of trabecular bone or periosteal hemorrhage, which then heal with the formation of new bone. Another mechanism suggested in the etiology of these lesions is forced plantarflexion trauma, which causes capsular avulsion injury.^{34,41}

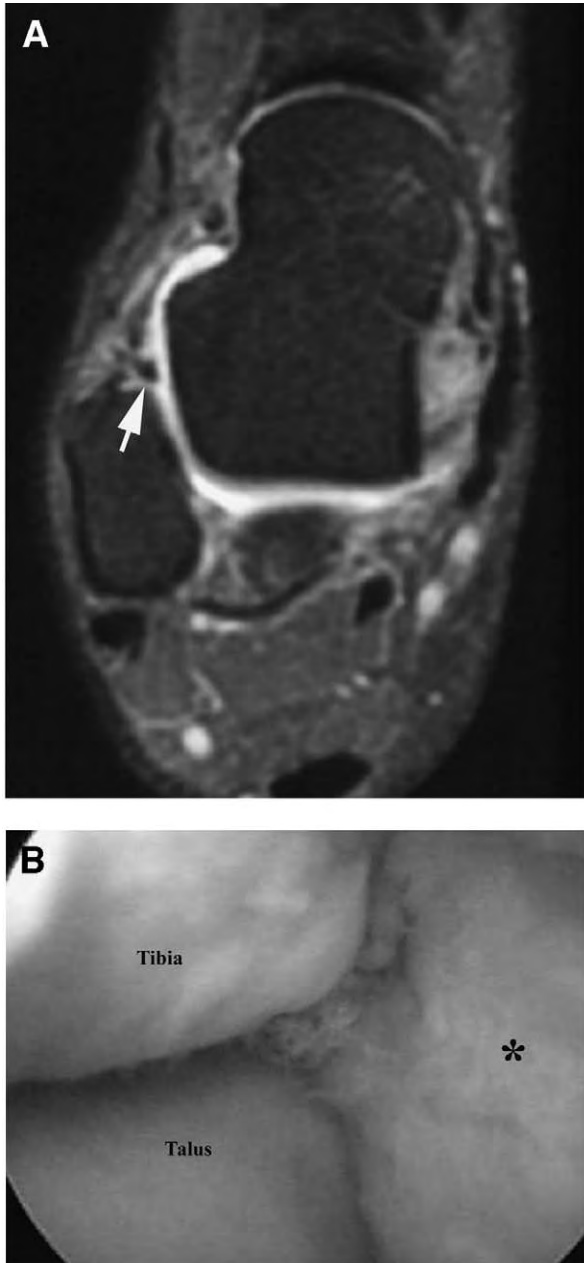


FIG 31. Anterolateral impingement. **A**, Axial T1-weighted spin-echo MRA shows focal nodular soft tissue thickening in the anterolateral gutter (arrow). **B**, Arthroscopic image demonstrates a thickened band of scarring and synovitis in the anterolateral gutter (meniscoid lesion) (asterisk).

Plain radiographs most often demonstrate anterior osteophytes, and lateral stress radiographs taken in maximum dorsiflexion may demonstrate physical impingement of the osteophytes. Scranton et al developed a classification for anterior ankle osteophytes, categorizing ankle spurs on the basis of the size of the spur and the presence of associated arthritis.⁴¹

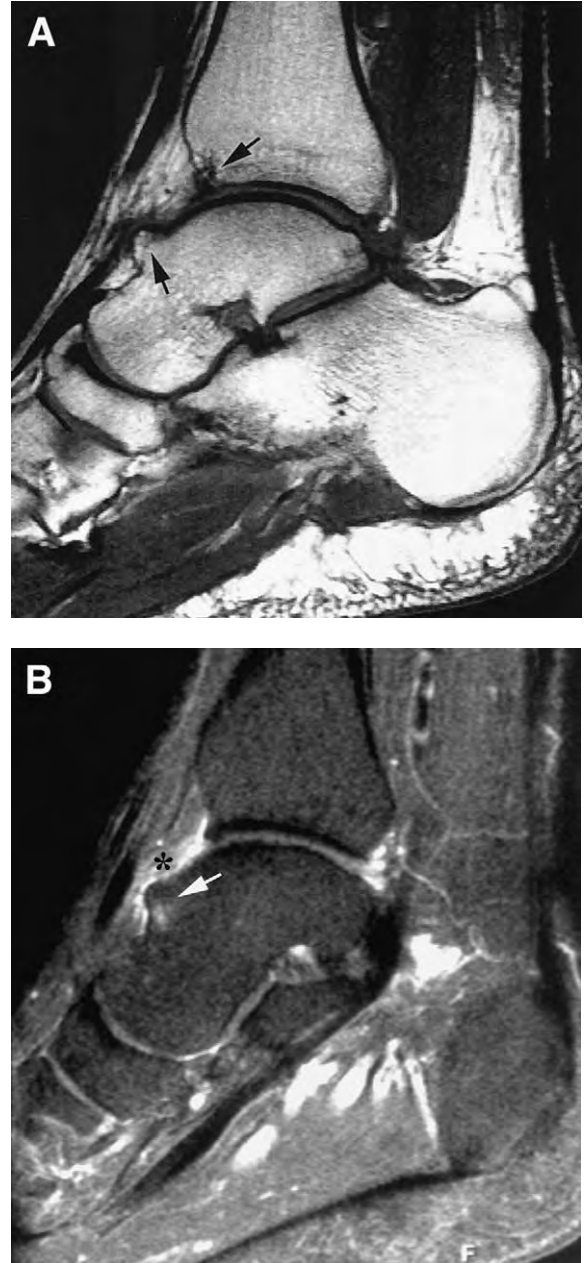


FIG 32. Anterior impingement. **A**, Sagittal T1-weighted spin-echo MRI shows anterior osteophytes in the tibia and talus (arrows). **B**, Sagittal fat-suppressed T1-weighted MRI after intravenous administration of gadolinium reveals bone marrow edema in the talar osteophyte (arrow) and synovial enhancement (asterisk).

MRI is useful in assessing the degree of cartilage damage and in detecting bone marrow edema and synovitis in the anterior capsular recess (Fig 32).³⁴ Conservative treatment consisting of heel lifts, rest, modification of activities, and physical therapy may be tried first. If there is persistent pain despite conservative treatment arthroscopic or open resection of the spurs may be considered.⁴²

Anteromedial Impingement Syndrome

Anteromedial impingement is a very uncommon cause of chronic ankle pain. It can be caused by a meniscoid lesion, represented by a mass of hyalinized connective tissue arising from a partially torn deep deltoid ligament or by a thickened anterior tibiotalar ligament.^{34,43-45} This thickened ligament or a meniscoid lesion, along with hypertrophic synovium, impinges on the anteromedial corner of the talus during dorsiflexion of the ankle. Anteromedial impingement is rarely an isolated condition but is most commonly associated with an inversion mechanism of injury with lateral as well as medial ligamentous injury.^{34,45}

Conventional MRI has not yet been proven useful in detecting the medial impingement syndromes. MRI can depict a partially torn deep deltoid ligament but is insensitive for other findings. MRA is the imaging method of choice, clearly defining the medial meniscoid lesion, the thickened anterior tibiotalar ligament, and chondral- or osteochondral-associated lesions.⁴⁵ If conservative treatment fails, debridement of the impinging lesion by arthroscopic methods yields good clinical results.

Posteromedial Impingement Syndrome

Posteromedial impingement is a very uncommon etiology of posteromedial ankle pain occurring after a severe ankle-inversion injury in which the deep posterior fibers of the medial deltoid ligament become crushed between the medial wall of the talus and the medial malleolus.⁴⁶ Initially, posteromedial symptoms do not predominate, compared with the symptoms of the lateral ligament disruption, and they usually resolve without specific treatment. However, inadequate healing of the contused deep posterior deltoid ligament fibers may lead to chronic inflammation and hypertrophic fibrosis and metaplasia. In such cases, this disorganized fibrotic scar tissue may impinge between the medial wall of the talus and the posterior margin of the medial malleolus.⁴⁶

MRI has been found to demonstrate the lesion, showing the thickened soft tissues and evidence of subchondral bruising of both the medial talus and medial malleolus. However, MRA is more sensitive and accurate than conventional MRI in the evaluation of this entity.⁴⁶

This posteromedial lesion usually resolves spontaneously or with nonoperative treatment. This lesion cannot generally be fully appreciated arthroscopically

via anterior portals in a stable ankle and requires a high index of suspicion and careful examination for the diagnosis to be made clinically. Arthroscopic surgery, via posterior portal, or limited open surgical excision of the lesion is successful in resolving the pain.⁴⁶

Posterior Ankle Impingement (PAI) Syndrome

PAI syndrome refers to a group of pathologic entities that result from repetitive or acute forced plantar flexion of the foot.⁴⁷⁻⁴⁹ Different names have been given to PAI syndrome, including os trigonum syndrome, talar compression syndrome, and posterior block of the ankle.⁴⁷⁻⁴⁹ The mechanisms of injury have been likened to a nut in a nutcracker because the posterior talus and surrounding soft tissues are compressed between the tibia and the calcaneus during plantar flexion of the foot. This syndrome has been extensively described in classical ballet dancers, but it also has been recognized in individuals who are active in sports, including soccer, basketball, running, and volleyball.⁴⁷ The anatomy of the posterior aspect of the ankle is a key factor in the occurrence of PAI syndrome. The more common causes are osseous in nature, such as the os trigonum (an accessory ossicle of the lateral tubercle that may persist unfused into adulthood in 7% of individuals), an elongated lateral tubercle termed a Stieda process, a downward sloping posterior lip of the tibia, the prominent posterior process of the calcaneus, and loose bodies.⁴⁷⁻⁴⁹ Soft tissue causes of impingement encompass synovitis of the flexor hallucis longus tendon sheath, the posterior synovial recess of the subtalar and tibiotalar joints, and the posterior intermalleolar ligament (IML).⁴⁷⁻⁵⁰ PAI syndrome may manifest as an inflammation of the posterior ankle soft tissues, as an osseous injury, or as a combination of both. The osseous injuries include fracture, fragmentation, and pseudoarthrosis of the os trigonum or lateral talar tubercle. As such, posterior ankle and subtalar synovitis as well as FHL tenosynovitis are soft-tissue changes associated with PAI syndrome.⁴⁷ The diagnosis of PAI syndrome is based primarily on the patient's clinical history and physical examination results and is supported by findings at radiography, scintigraphy, CT, and MRI.⁴⁷

MRI is useful in establishing the diagnosis of PAI syndrome, showing abnormal signal intensity in the lateral talar tubercle and/or os trigonum, consistent with bone marrow edema that is believed to be the

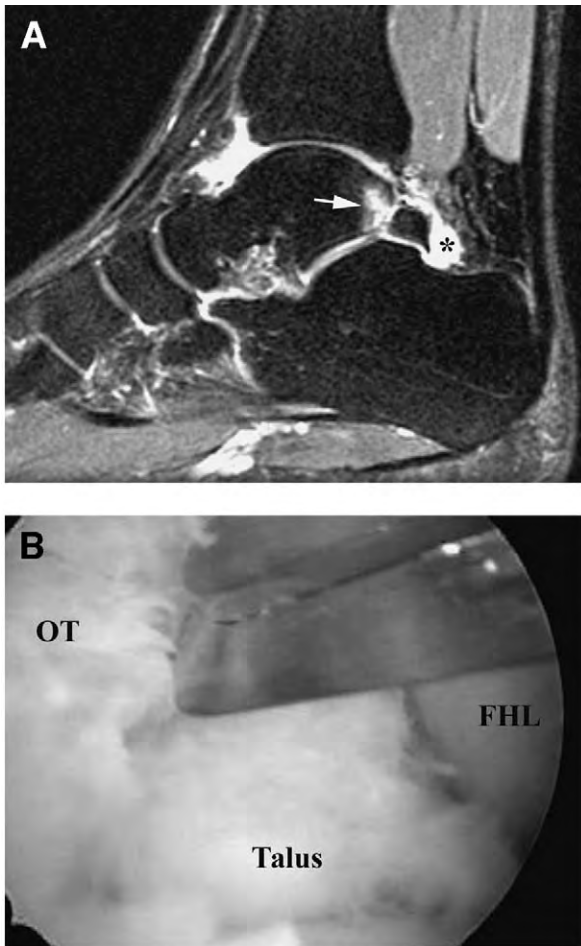


FIG 33. Posterior impingement. **A**, Sagittal fat-suppressed proton density image demonstrates abnormal high-signal intensity in the posterior aspect of the talus and in the os trigonum (arrow). Joint effusion in the posterior synovial recess of the tibiotalar and subtalar joints is noted (asterisk). **B**, Arthroscopic image showing the os trigonum and a normal flexor hallucis longus tendon.

result of bone impaction, and thus represents bone contusions or occult fractures (Fig 33).^{47,49}

MRI also depicts inflammatory changes in the posterior ankle soft tissues (the posterior synovial recess of the subtalar and tibiotalar joints and the FHL tendon sheath). The combined presence of marrow edema and posterior ankle synovitis may suggest the diagnosis of PAI.⁴⁷ Diagnosis of an abnormal IML on MRI requires a thickened IML, which can readily be separated from the surrounding posterior talofibular ligament and the transverse inferior tibiofibular ligament.^{50,51} It is equally important that MRI can specifically identify the wide range of pathology that may contribute to posterior ankle pain that might be clinically confused with PAI, including Achilles tendonitis/tear, retrocalcaneal bursitis, FHL tenosynovitis,

Haglund's deformity, ankle or subtalar arthritis, osteochondral lesion, tarsal tunnel syndrome, post-traumatic instability, sprain, or peroneal tendon subluxation.⁴⁷ The treatment of PAI syndrome is initially conservative. If conservative treatment fails, then surgical excision of the osseous fragments, with potential release of the FHL tendon, may be indicated.⁷

Plantar Fascial Injuries

The plantar fascia has been defined as the investing fibrous layer of the foot's plantar aspect, located subcutaneously and originating from the calcaneus to insert into the deep soft tissues of the forefoot, the proximal phalanges and the skin via superficial extension.

The plantar fascia comprises three variably developed components: central, lateral, and medial cords.⁵² The term *plantar fascia* usually refers to the large central component. It originates from the medial calcaneal tuberosity and extends anteriorly, adhering to the underlying flexor digitorum brevis muscle. At about the midsole, it splits into five bands, one for each toe, that insert on the proximal phalanges. The lateral and medial components of the plantar fascia act mainly as covering layers peripheral to the central component. The lateral component serves as the fascial covering of the abductor digiti minimi. It arises from the lateral margin of the medial calcaneal tuberosity and extends to the cuboid and base of the fifth metatarsal bone. The medial component is very thin and forms the investing fascia of the abductor hallucis muscle. The plantar fascia acts as a strong mechanical tie for the longitudinal arches by joining the three main weight-bearing points of the foot: the calcaneus, the first metatarsal head (including the two sesamoids), and the fifth metatarsal head.⁵²

On MRI, the normal fascia appears as a thin band with low-signal intensity with all pulse sequences; the thickness of the plantar fascia in normal individuals ranges between 2 to 4 mm.^{4,14,53-55} The plantar fascia is best examined by MRI in both sagittal and coronal planes.^{4,55} The coronal plane represents the true axial plane perpendicular to the plantar fascia. It allows simultaneous visualization of the three components of the plantar fascia and is optimal to assess the size, shape, and internal signal characteristics of these structures. The medial component appears as either a semilunar or fusiform-shaped sharply delineated band. Sagittal imaging allows optimal evaluation of the

plantar fascia along its length. On sagittal images, the normal fascia has a uniform thickness from its calcaneal origin through its proximal half, whereas more distally, progressively thins until it reaches its metatarsal insertions.^{4,55}

Plantar Fasciitis

Plantar fasciitis is a common overuse injury found in athletes engaged in sports that require running or jumping.³ Plantar fasciitis accounts for 7% to 9% of all running injuries. Repetitive microtrauma leads to microtears in the plantar fascia near its attachment at the inferior aspect of the calcaneus, and elicit a local inflammatory reaction.^{56,57} In cases of persistent overuse there will appear chronic changes with collagen necrosis, angiofibroblastic hyperplasia, and matrix calcification. Commonly cited predisposing factors of plantar fasciitis are excessive pronation, a flat or cavus foot, and a tight Achilles tendon. The type of training shoes worn and errors in training routines are predisposing factors in runners.

Pain on the undersurface of the heel on weight bearing is the principal complaint. The pain is often worse when weight is borne after a period of rest (eg, in the morning) and eases with walking. Localized tenderness without swelling is present over the antero-medial portion of the plantar surface of the calcaneus. Passive dorsiflexion of the toes often exacerbates the discomfort. Plantar fasciitis is essentially a clinical diagnosis. Radiological evaluation is reserved for ambiguous or clinically equivocal cases or for cases that are refractory to treatment. Radiographs, ultrasonography, and MRI have been used in the evaluation of plantar fascia injuries. Radiographs are mainly useful in evaluation of osseous structures to exclude unrecognized traumatic injuries. Also, radiographs may reveal a plantar calcaneal spur, although this entity may also be observed in asymptomatic adults.^{56,57} The etiologic significance of this spur remains controversial, but most authors believe that it is not the primary cause of pain.^{3,56,57}

Both ultrasonography and MRI have been recognized as useful tools in the assessment of the plantar fascia.^{4,5,14,53-55,58,59} MRI has an advantage over ultrasound imaging in that it allows an effective assessment of both soft-tissue and osseous structures. On MRI, plantar fasciitis is characterized by perifascial edema, fascial thickening, and increased signal intensity. Perifascial edema involves the adjacent fat pad and underlying soft tissues and has been described as

the most common MR finding in patients with plantar fasciitis (Fig 34).^{55,58}

Fascial thickening is often fusiform and typically involves the proximal portion and extends to the calcaneal insertion. Increased signal intensity of the plantar fascia reflects edematous and inflammatory changes as well as intrasubstance microtears. STIR sequences are often the most sensitive in the detection of both fascial and perifascial edema, which appear as poorly marginated areas of high-signal intensity (Fig 34).⁴ Limited marrow edema changes within the medial calcaneal tuberosity are a less common MRI finding.^{4,54,55} Successful outcome of plantar fasciitis is directly related to early diagnosis and treatment.⁵⁶ Therefore, in equivocal cases, especially in elite athletes, MRI can allow an early diagnosis of plantar fasciitis, and thus, improve the prognosis in these patients.

Treatment is conservative. Surgical treatment of plantar fasciitis is reserved to cases in which conservative treatment fails, especially in athletes engaged in high-level competition.^{3,56,57}

Plantar Fascial Rupture

Rupture of the plantar fascia is typically a sports-related injury. It occurs in athletes engaged in sports that require running and jumping, such as distance running, basketball, football, and tennis.⁶⁰⁻⁶² Most plantar fascial tears in athletes are acute.^{61,62} However, it can also be associated with local steroid injection in patients with plantar fasciitis.⁶³ Sudden plantar heel pain typically indicates a traumatic tear. The individual usually hears a clicking or snapping sound when the traumatic event occurs,⁶⁰ and a palpable, tender mass is detected at the site of injury. Clinical manifestations in patients with tears related to corticosteroid injection are more insidious.⁶³ Most cases involve the proximal portion of the plantar fascia near its calcaneal insertion, although more anterior tears have also been described.^{5,14,52} In a previous study, half of cases presented as ruptures located in the middle plantar fascia.⁵⁵

MRI is advocated as the modality of choice to confirm the diagnosis and to plan the treatment strategy in both clinical and radiological series.^{4,55,58,60,62} MRI findings of tears of the plantar fascia are similar to those seen in tendinous ruptures. Acute tears of the plantar fascia are characterized by partial or complete interruption of the normally low-signal intensity fascia by large areas of prominent increased signal-intensity

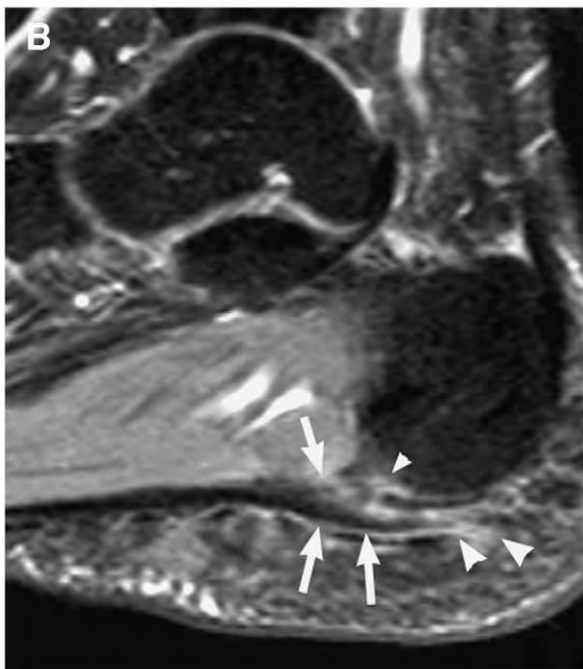
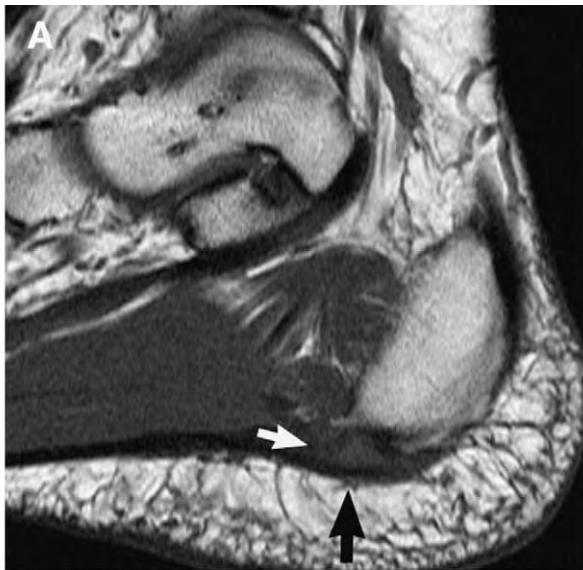


FIG 34. Plantar fasciitis. **A**, Sagittal T1-weighted MR shows marked thickening of the proximal plantar fascia (black arrow) with increased intrasubstance signal intensity (white arrow). Note also the perifascial edema, which has low signal intensity in **A** and high-signal intensity in **B** (arrowheads). **B**, Sagittal STIR MRI better demonstrates fascial thickening (arrows), intrasubstance increased signal as well as perifascial edema (arrowheads). Note also calcaneal bone marrow edema (small arrowhead).

on T2-weighted and STIR images, presumably representing edema and hemorrhage (Figs 35 and 36).^{4,5,14,60} Perifascial fluid accumulations are commonly seen on T2-weighted and STIR images (Fig 36).^{4,55} In addition, acute tears of the plantar fascia

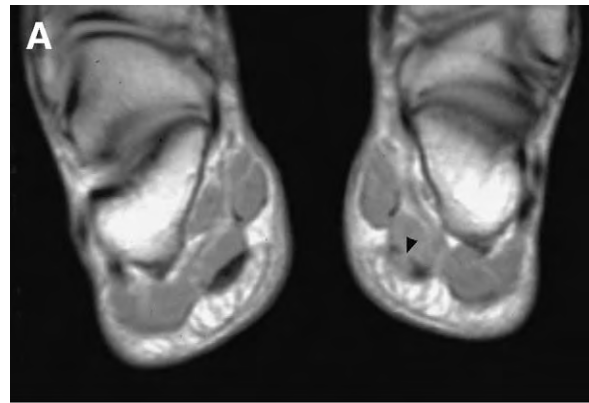


FIG 35. Partial plantar fascia rupture. **A**, Coronal proton-density-weighted MRI shows thickening of the central component of the left plantar fascia with abnormal intermediate signal intensity (arrowhead). **B**, Sagittal proton-density-weighted MRI demonstrates the fascial rupture at distance of the calcaneal insertion (arrows). Note fusiform fascial thickening and loss of its low-signal intensity.

commonly involve the underlying flexor digitorum brevis muscle.¹⁴ Acute and subacute muscle tears are characterized by high-signal intensity with a feathery appearance on both T1- and T2-weighted images representing muscle bleeding and edema (Fig 36). Less commonly, strains of other plantar muscles, such as abductor hallucis or quadratus plantae, are associated with plantar fascia rupture.¹⁴ Chronic tears of the plantar fascia are characterized by focal fascial thickening and intrafascial scar tissue, which show low-signal intensity on all pulse sequences.⁵⁵ Perifascial edema is usually absent in these chronic tears.⁵⁵

Conservative treatment consisting of rest, arch supports and orthotics, and physical therapy is sufficient in most cases.^{60,63}

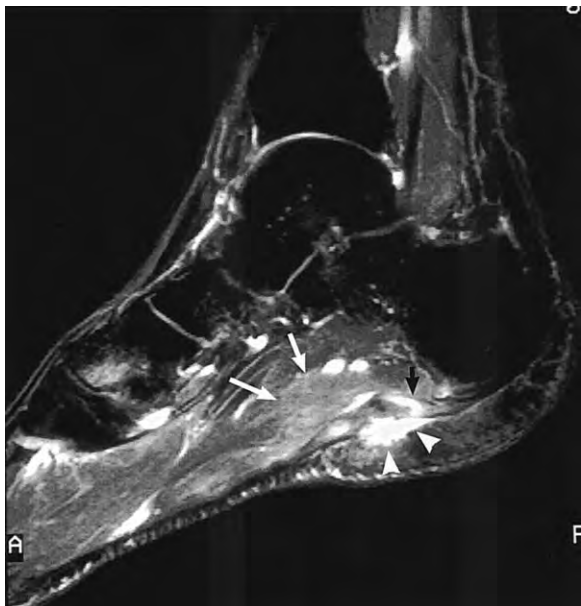


FIG 36. Plantar fascia rupture. Sagittal STIR MRI depicts the aponeurotic defect (black arrow), focal fusiform at the site of the rupture, and fluid collection superficial to the fascia (arrowheads). The underlying flexor digitorum brevis muscle shows changes in signal intensity representing edema and hemorrhage (white arrows).

Surgical treatment, including plantar fasciotomy and excision of scar tissue, is advocated in young active athletes.⁶²

REFERENCES

- Bencardino J, Rosenberg ZS, Delfault E. MR imaging in sports injuries of the foot and ankle. *MRI Clin North Am* 1999;7:131-49.
- Khan K, Brown J, Way S, et al. Overuse injuries in classical ballet. *Sports Med* 1995;19:341-57.
- Leach RE, DiLorio E, Hamey RA. Pathologic hindfoot conditions in the athlete. *Clin Orthop* 1983;177:116-21.
- Narvaez JA, Narvaez J, Ortega R, et al. Painful heel: MR imaging findings. *Radiographics* 2000;20:333-52.
- DiMarcangelo MT, Yu TC. Diagnostic imaging of heel pain and plantar fasciitis. *Clin Podiatr Med Surg* 1997;14:281-301.
- Rosenberg ZS, Beltran J, Bencardino J. MR imaging of the ankle and foot. *Radiographics* 2000;20:S153-79.
- Dunfee WR, Dalinka MK, Kneeland JB. Imaging of athletic injuries to the ankle and foot. *Radiol Clin North Am* 2001;40:289-312.
- Chandnani VP, Harper MT, Ficke JR. Chronic ankle instability: evaluation with MR arthrography, MR imaging, and stress radiography. *Radiology* 1994;192:189-94.
- Schweitzer ME, Caccese R, Karasick D, et al. Posterior tibial tendon tears: utility of secondary signs for MR imaging diagnosis. *Radiology* 1993;188:655-9.
- Schweitzer ME, Eid ME, Deely D, et al. Fluid in normal and abnormal ankle joints: amount and distribution as seen on MR images. *AJR Am J Roentgenol* 1994;162:111-4.
- Kvist M. Achilles tendon injuries in athletes. *Sports Med* 1994;18:173-291.
- Karjalainen P, Soila K, Aronen HJ, et al. MR imaging of overuse injuries of the Achilles tendon. *AJR Am J Roentgenol* 2000;175:251-60.
- Teitz C, Garret WR Jr, Miniaci A, Lee MH, Mann RA. Tendon problems in athletic individuals. *J Bone Joint Surg Am* 1997;79:138-52.
- Kier R. Magnetic resonance imaging of plantar fasciitis and other causes of heel pain. *MRI Clin North Am* 1994;2:97-107.
- Bottger BA, Schweitzer ME, El-Noueam KI, et al. MR imaging of the normal and abnormal retrocalcaneal bursa. *AJR Am J Roentgenol* 1998;170:1239-41.
- Chandnani VP, Bradley YC. Achilles tendon and miscellaneous tendon lesions. *MRI Clin North Am* 1994;2:89-96.
- Sammarco GJ. Peroneal tendon injuries. *Orthop Clin North Am* 1994;25:135-45.
- Tjin A, Ton ER, Schweitzer ME, Karasick D. MR imaging of peroneal tendon disorders. *AJR Am J Roentgenol* 1997;168:135-40.
- Bencardino J, Rosenberg ZS, Serrano LF. MR imaging features of diseases of the peroneal tendons. *MRI Clin North Am* 2001;9:493-505.
- Basset FH, Speer KP. Longitudinal rupture of the peroneal tendons. *Am J Sports Med* 2002;21:354-7.
- Rosenberg ZS, Beltran J, Cheung Y, et al. MR features of longitudinal tears of the peroneal brevis tendon. *AJR Am J Roentgenol* 1997;168:141-7.
- Schweitzer ME, Eid ME, Deely D, et al. Using MR imaging to differentiate peroneal splits from other peroneal disorders. *AJR Am J Roentgenol* 1997;168:129-33.
- Khoury NJ, El-Khoury GY, Saltzman CL, et al. Peroneus longus and brevis tendon tears: MR imaging evaluation. *Radiology* 1996;200:833-41.
- Major NM, Helms CA, Fritz RC, et al. The MR imaging appearance of longitudinal split tears of the peroneus brevis tendon. *Foot Ankle Int* 2002;21:514-9.
- Sammarco GJ. Peroneus longus tendon tears: acute and chronic. *Foot Ankle Int* 1995;16:245-53.
- Rademaker J, Rosenberg ZS, Delfaut EM, et al. Tear of the peroneus longus tendon: MR imaging features in nine patients. *Radiology* 2000;214:700-4.
- Oloff LM, Schullhofer SD. Flexor hallucis longus dysfunction. *J Foot Ankle Surg* 1998;37:101-109.
- Conti SF. Posterior tibial tendon problems in athletes. *Orthop Clin North Am* 1994;25:109-21.
- Porter DA, Baxter DE, Clanton TO, et al. Posterior tibial tendon tears in young competitive athletes: two case reports. *Foot Ankle Int* 1998;19:627-39.
- Schweitzer ME, Karasick D. MR imaging of disorders of the posterior tibialis tendon. *AJR Am J Roentgenol* 2000;175:627-35.
- Khoury NJ, El-Khoury GY, Saltzman CL, et al. MR imaging of posterior tibial tendon dysfunction. *AJR Am J Roentgenol* 1996;167:675-82.
- Miller TT, Staron RB, Feldman F, et al. The symptomatic accessory tarsal navicular bone: assessment with MR imaging. *Radiology* 1995;195:849-53.

33. Khoury NJ, El-Khoury GY, Saltzman CL, Brandser EA. Rupture of the anterior tibial tendon: diagnosis by MR imaging. *AJR Am J Roentgenol* 1996;167:351-4.
34. Umans H. Ankle impingement syndromes. *Semin Musc Radiol* 2002;6:133-40.
35. DeBerardino TM, Arciero RA, Taylor DC. Arthroscopic treatment of soft-tissue impingement of the ankle in athletes. *Arthroscopy* 1997;13:492-8.
36. Farooki S, Yao L, Sella EJ. Anterolateral impingement of the ankle: effectiveness of MR imaging. *Radiology* 1998;207:357-60.
37. Hauger O, Moinard M, Lasalarie JC, et al. Anterolateral compartment of the ankle in the lateral impingement syndrome: appearance on CT arthrography. *AJR Am J Roentgenol* 1999;173:685-90.
38. Robinson P, White LM, Salonen D, et al. Anterolateral ankle impingement: MR arthrographic assessment of the anterolateral recess. *Radiology* 2001;221:186-90.
39. Rubin DA, Tishkoff NW, Britton CA, et al. Anterolateral tissue impingement in the ankle: diagnosis using MR imaging. *AJR Am J Roentgenol* 1997;169:829-35.
40. Basset FH III, Gates HS III, Billys JB, et al. Talar impingement by anteroinferior tibiofibular ligament. A cause of chronic pain in the ankle after inversion sprain. *J Bone Joint Surg Am* 1990;72:55-9.
41. Scranton PE Jr, McDermott JE, Rogers JV. The relationship between chronic ankle instability and variations in mortise anatomy and impingement spurs. *Foot Ankle Int* 2002;21:657-64.
42. van Dijk CN, Tol JL, Verheyen CC. A prospective study of prognostic factors concerning the outcome of arthroscopic surgery for anterior ankle impingement. *Am J Sports Med* 1997;25:745.
43. Egol KA, Parisien JS. Impingement syndrome of the ankle caused by medial meniscoid lesion. *Arthroscopy* 1997;13:522-5.
44. Mosier-La Clair SM, Monroe MT, et al. Medial impingement syndrome of the anterior tibiotalar fascicle of the deltoid ligament of the talus. *Foot Ankle Int* 2000;21:385-91.
45. Robinson P, White LM, Salonen D, et al. Anteromedial impingement of the ankle: using MR arthrography to assess the anteromedial recess. *AJR Am J Roentgenol* 2002;178:601-4.
46. Paterson RS, Brown JN. The posteromedial impingement lesion of the ankle. A series of six cases. *Am J Sports Med* 2001;29:550-7.
47. Bureau NJ, Cardinal E, Hobden R, et al. Posterior ankle impingement syndrome: MR imaging findings in seven patients. *Radiology* 2000;215:497-503.
48. Hedrick MR, McCarroll JR. Posterior ankle impingement. *Foot Ankle Int* 1994;15:2-89.
49. Karasick D, Schweitzer ME. The os trigonum syndrome: imaging features. *AJR Am J Roentgenol* 1996;166:125-9.
50. Fiorella D, Helms CA, Nunley JA. The MR imaging features of the posterior intermalleolar ligaments in patients with posterior impingement syndrome of the ankle. *Skeletal Radiol* 1999;28:573-6.
51. Rosenberg ZS, Cheung Y, Beltran J, et al. Posterior intermalleolar ligament of the ankle: normal anatomy and MR imaging features. *AJR Am J Roentgenol* 1995;165:387-90.
52. Hedrick MR. The plantar aponeurosis. *Foot Ankle Int* 1996;17:646-9.
53. Berkowitz JF, Kier R, Rudicel S. Plantar fasciitis: MR imaging. *Radiology* 1991;179:665-7.
54. Roger B, Grenier P. MRI of plantar fasciitis. *Eur Radiol* 1997;7:1430-5.
55. Theodorou DJ, Theodorou SJ, Kakitsubata Y, et al. Plantar fasciitis and fascial rupture: MR imaging findings in 26 patients supplemented with anatomic data in cadavers. *Radiographics* 2000;20:S181-97.
56. Schepsis AA, Leach RE, Gorzyca J. Plantar Fasciitis. Etiology, treatment, surgical results, and review of the literature. *Clin Orthop* 1991;266:185-96.
57. Karr SD. Subcalcaneal heel pain. *Orthop Clin North Am* 1994;25:161-73.
58. Grasel RP, Schweitzer ME, Kovalovich AM, et al. MR imaging of plantar fasciitis: edema, tears, and occult marrow abnormalities correlated with outcome. *AJR Am J Roentgenol* 1999;173:699-701.
59. Theodorou DJ, Theodorou SJ, Farooki S, et al. Disorders of the plantar aponeurosis. *AJR Am J Roentgenol* 2001;176:97-104.
60. Rolf C, Gunther P, Ericsson J, et al. Plantar fascia rupture: diagnosis and treatment. *J Foot Ankle* 1997;36:112-4.
61. Leach R, Jones R, Silva T. Rupture of the plantar fascia in athletes. *J Bone Joint Surg Am* 1978;60:537-9.
62. Christel P, Rigal S, Poux D, et al. Surgical treatment of plantar fascia ruptures in athletes. *Rev Chir Orthop* 1993;79:218-25.
63. Sellman JR. Plantar fascia rupture associated with corticosteroid injection. *Foot Ankle Int* 1994;15:376-81.