Magnetic Resonance Imaging of the Tendons of the Ankle and Foot

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Abstract: Tendons of the ankle and foot are subject to considerable tensile forces with everyday activities such as walking and they are stressed further with recreational activities. Regular physical training is required to maintain tendon strength. However, chronic repetitive microtrauma can excede the tendon's capacity to regenerate, resulting in scar deposition and tendon degeneration. Pathologically, this is referred to as tendinosis. Clinically, it may initially be asymptomatic until an acute crisis supervenes or it may present with insidious pain and deformity as the tendon progressively fails. Tendon injury may also follow an acute traumatic episode. Abnormal stretching acutely strains or ruptures the tendon. Ruptures may be partial or complete. Such an injury invokes an inflammatory healing response and is usually acutely painful. Acute or chronic tendon injury also occurs. In addition to tendon injury, the paratenon and synovial-lined sheaths may become inflamed, resulting in paratendonitis or tenosynovitis. Certain biomechanical variations in anatomy, either developmental or acquired, predispose patients to tendon injury. Systemic illnesses such as the inflammatory arthropathies and local therapies such as steroid injections are predisposing conditions. The utility of magnetic resonance imaging (MRI) in diagnosing and assessing tendon injury in the ankle and foot will be reviewed. In particular, MRI features of the Achilles tendon, the peroneal tendons, the posterior tibial tendon, the flexor hallucis longus tendon, and the anterior tibial tendon will be discussed.

Introduction

Imaging modalities that can be used to evaluate the tendons of the ankle and foot include plain films, tenography, ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI). Of these tests, MRI is the most accurate and is noninvasive. Not only can it assess the specific tendon pathology, it can also evaluate predisposing conditions that may be amenable to therapy. Plain films and CT are useful in diagnosing acute avulsion fractures at tendon insertions, accessory ossicles, and tendon calcification that may occur with calcium deposition diseases or in chronic tendinopathies.

MRI Technique

To perform MRI of the ankle and foot, the patient's foot is immobilized within a regional radiofrequency coil such as a whole volume extremity coil and placed into the bore of the magnet. Generally, pulse sequences are selected to image the extremity in three planes (axial, sagittal, and coronal) with an anatomic sequence (T1 or proton density weighted) and a sequence sensitive to fluid and edema (T2 weighted). To improve visualization of fluid and edema, fat saturation techniques are frequently employed with the T2-weighted sequence on high field strength systems (≥ 1.0 Tesla). The short TI inversion recovery sequence (STIR) is very sensitive to fluid/edema and suppresses the signal from fat. It is often used in place of the T2-weighted sequence in the sagittal plane. The typical field of view is 12 cm to include the ankle mortise and the tarsometatarsal joints. A typical examination on a high field strength system lasts approximately 30 minutes. More recently, dedicated extremity and open MR systems have become commercially available. These systems have much lower field strength magnets (0.2–0.5 Tesla) and are much less expensive than the traditional high field strength systems. Their use, however, does entail some loss of image quality, which may affect diagnostic accuracy.

MRI of Normal Tendon Anatomy

Tendons connect muscle to bone. An individual tendon is a bundle of fibrils of collagen, elastin, and extracellular matrix arranged in parallel and is enclosed by loose connective tissue. Because they are composed of little free water, tendons are seen as low signal intensity areas on all MR pulse sequences. When a tendon is oriented at a 55 degree angle to the long axis of the bore of the magnet, it becomes mildly hyperintense on short TE sequences such as the T1 and proton density-weighted sequences because of proton-proton interactions. This effect, which is referred to as the "magic angle" phenomenon, is not seen on long TE sequences such as the T2-weighted sequence where the signal intensity of the tendon remains low [1]. This frequently occurs as tendons cross the ankle particularly the peroneal tendons and posterior tibial tendon. It is a pitfall in the MR imaging diagnosis of tendinosis [4]. With the exception of the Achilles tendon, which is enclosed only by connective tissue, the other tendons of the ankle and foot are enclosed within a synovial sheath. On MRI, this fluid-containing sheath is seen as a thin rim of intermediate signal intensity on T1 and proton density-weighted sequences and as a high
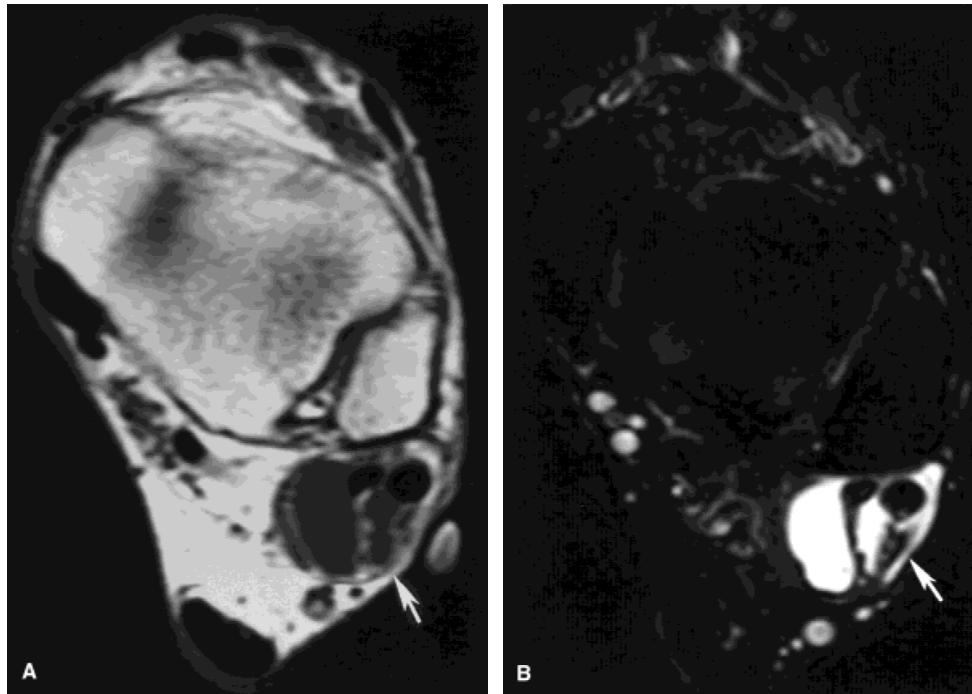
Fig. 1. Tenosynovitis of the combined sheath of the paired peroneal tendons. **A** (proton density-weighted axial sequence) and **B** (T2-weighted fat-suppressed axial sequence): An abnormally large amount of fluid surrounding the peroneus brevis and longus tendons is shown (arrow). Thickened synovium is present posteriorly, representing an inflammatory tenosynovitis.

signal intensity area on the T2-weighted sequence, which circumscribes the low signal intensity of the tendon. Normally, the amount of fluid is small [8].

MRI of Tendon Pathology

Pathologically, biomechanical factors lead to four general tendinopathies: (1) tenosynovitis or paratendonitis; (2) tendinosis, (3) partial tears; and (4) complete tears. Each of these tendinopathies has unique features on MRI. However, they are not mutually exclusive and combined pathologies frequently occur. Additional lesions of tendons and the peritendinous soft tissues such as gout tophi, soft tissue xanthomas, and pigmented villonodular synovitis will not be discussed.

Tenosynovitis

Tenosynovitis is inflammation of the tendon sheath surrounding the tendon. On MRI, a large amount of fluid is seen to distend the tendon sheath (Fig. 1). As previously mentioned, a small amount of fluid in the tendon sheath can be seen in normal subjects and caution should be exercised in diagnosing this condition. Occasionally, complicated tenosynovitis is observed, with lower signal intensity areas occurring amidst the high signal intensity fluid on the T2-weighted sequence. The low signal areas are believed to represent inflamed synovium or detritus. This should raise the possibility of an inflammatory arthropathy such as rheumatoid arthritis, a spondyloarthropathy, or infection. The Achilles tendon does not have a synovial sheath. It is sur-

rounded by a loose connective tissue called a paratenon, which may also become inflamed. Any amount of fluid circumscribing the Achilles tendon is pathologic and indicates paratendonitis (Fig. 2).

Tendinosis

Tendinosis occurs when chronic repetitive microtrauma to the tendon excedes its capacity to regenerate, resulting in
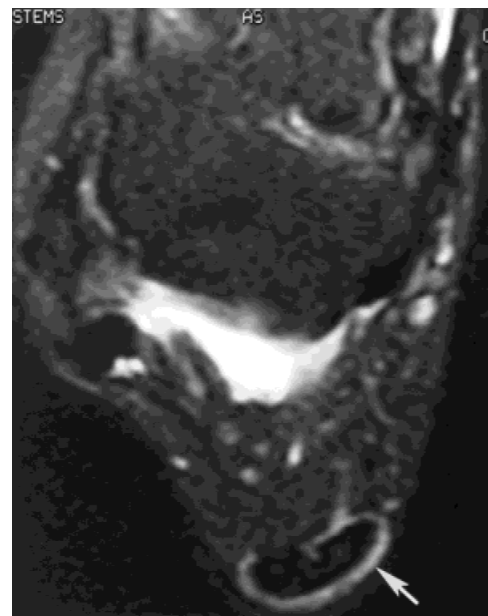
Fig. 2. Paratendonitis of the Achilles tendon. Axial T2-weighted fat-suppressed sequence demonstrates a thin rim of hyperintense fluid circumscribing the Achilles tendon (arrow).

scar deposition and tendon degeneration. Active individuals may develop tendinosis when an increase in their activities increases the trauma to their tendons. This is often attributed to poor training regimens. With advancing age, the tendon stiffens and loses its capacity to regenerate, predisposing to tendinosis without a change in activity levels. Underlying arthropathies such as rheumatoid arthritis and steroid injections into the tendon also increase the risk of tendinosis. With early degeneration, the composition of the tendon changes. Its signal intensity becomes intermediate on T1 or proton density-weighted sequences but remains normal in signal intensity or becomes only minimally brighter on the T2-weighted sequence (Fig. 3). Sometimes the degenerated tendon thickens. Some cases of chronic tendinosis of the Achilles tendon may present with just an enlarged tendon that remains normal in signal intensity on all sequences (Fig. 4).

The magic angle phenomenon can mimic tendinosis. This can be problematic, particularly around the ankle where the tendons make sharp turns around the malleoli. This artifact can be adjusted by repositioning the ankle within the bore of the magnet, which is rarely necessary. In most cases, the artifact can be distinguished from tendinosis by observing the signal intensity of an adjacent tendon that shares a similar orientation. For example, the paired peroneal tendons follow a similar course at the posterior aspect of the lateral malleolus. Therefore, the magic angle phenomenon in one tendon should be accompanied by magic angle in the other at that location (Fig. 5).

Tendon ruptures

These frequently occur following an acute traumatic episode. In almost all cases, a degenerated tendon with tendinosis ruptures [14]. Abnormal stretching of the degenerated tendon leads to partial or complete macrotears. On MRI, partial or full-thickness discontinuity of a degenerated tendon is seen. Within the gap between the torn fibers, the signal represents a combination of edema and hemorrhage. Edema is seen as an intermediate signal intensity area on T1 or proton density-weighted sequences and as a hyperintense area on the T2-weighted sequence. Figures 6A and 6B a partial-thickness and a full-thickness, respectively, tear of the Achilles tendon. With full-thickness tears, MRI can quantitate the degree of tendon retraction. There is good correlation between MRI and the surgical grading of tendon rupture [3]. One study reviewing the accuracy of MRI in assessing posterior tibial tendon ruptures found it to be superior to surgery in predicting surgical outcomes. This is because some tears are intrasubstance, deep, or otherwise beyond the surgical field of view [3].

MRI of Individual Tendons

The Achilles tendon is the largest tendon of the body, formed by the muscles of the medial and lateral heads of the gastrocnemius and the soleus muscle. The tendon of the small plantaris muscle often courses separately and inserts medially on the posterior tuberosity of the calcaneus. Achilles tendinopathy is particularly frequent in runners and in
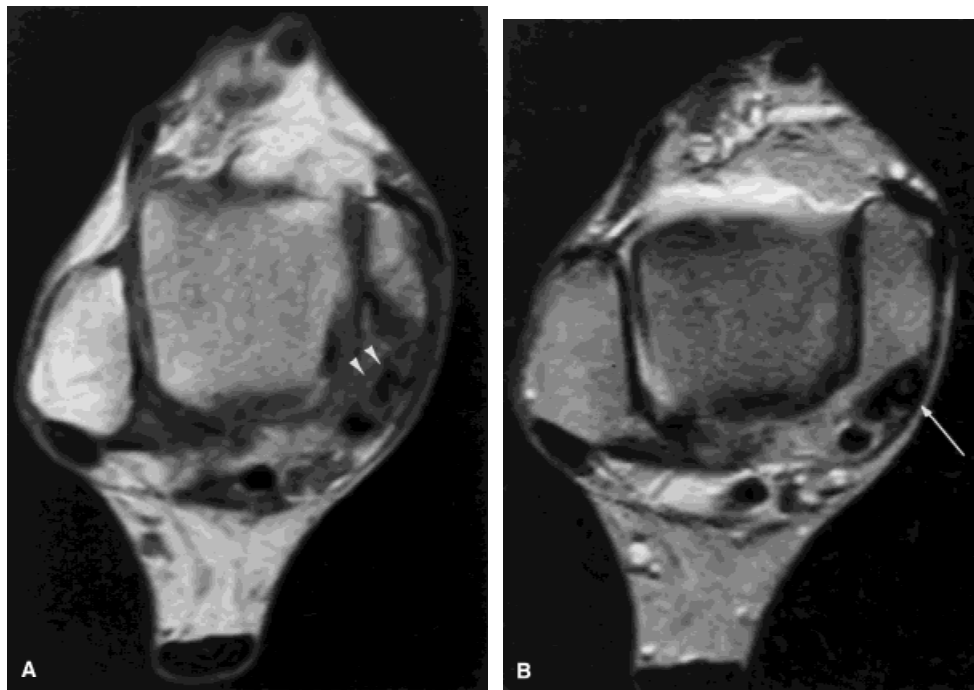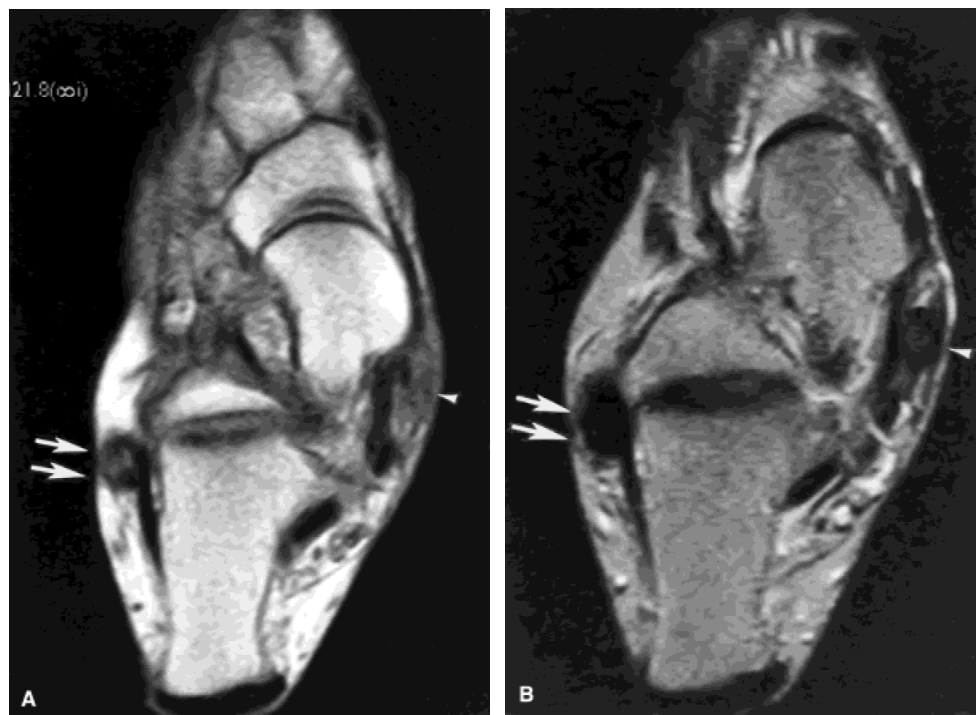

Fig. 3. Tendinosis of the posterior tibial tendon. A (proton density-weighted axial sequence) B (T2-weighted axial sequence) demonstrate abnormal signal intensity in the posterior tibial tendon as it courses around the medial malloelus. On the proton density-weighted sequence (**A**), the tendon is nearly completely replaced by hyperintense material (arrowheads), with only a small number of low signal intensity tendon fibers remaining. On the T2-weighted sequence (**B**), the tendon remains predominantly an area of low signal intensity, with only minimally increased signal intensity within its center (arrow).
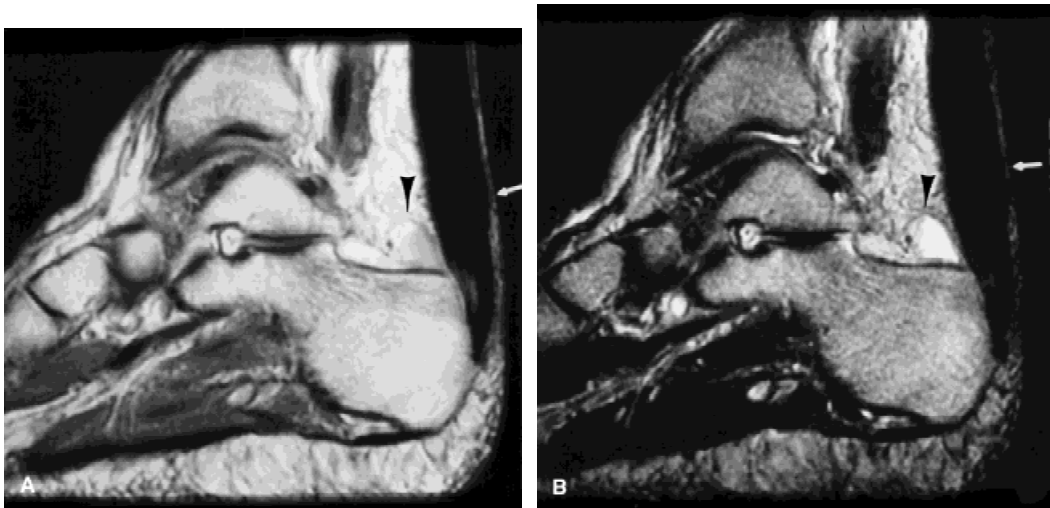
Fig. 4. Chronic tendinosis of the Achilles tendon. **A** (T1-weighted sagittal sequence) and **B** (T2-weighted sagittal sequence): Fusiform enlargement of the Achilles tendon proximal to its insertion site (arrow). The tendon remains an area of low signal intensity on all sequences. Retrocalcaneal bursitis is frequently present (arrowheads).

white collar office workers who pursue recreational activities intermittently. Paratendonitis of the Achilles tendon has already been described. Tendinosis and partial and complete tears are more frequently seen within the tendon 2–6 cm proximal to its insertion on the calcaneus [15]. In this location, the vascular supply to the tendon is most tenuous. The normal Achilles tendon is a relatively flat structure measuring less than 7 mm in thickness. Thickening of the Achilles tendon to 7 mm or more is an indicator of tendi-

nopathy (Fig. 4) [15]. A low insertion of the soleus muscle should not be mistakened for an abnormally thickened tendon [7]. Less frequently, Achilles tendinopathy occurs at its insertion site on the calcaneus. Haglund's syndrome is a triad of insertional Achilles tendinopathy, retrocalcaneal bursitis, and retro tendo-Achilles bursitis (Fig. 7). Haglund's syndrome may be associated with prominence of the posterior tuberosity of the calcaneus and with particular footwear such as hockey skates and women's shoes [9].

Fig. 5. Magic angle phenomenon in the peroneal tendons at the posterior subtalar joint. **A** (proton density-weighted axial sequence): Slightly increased signal intensity within the paired peroneal tendons as they course at a 55 degree angle relative to the long axis of the magnetic field (double arrows). **B** (T2-weighted axial sequence): Low signal intensity tendons (double arrows). This phenomenon is observed in both tendons in this particular location, which helps to categorize this occurrence as being due to the magic angle phenomenon rather than to tendinosis. There is tendinosis of the posterior tibial tendon (arrowhead).
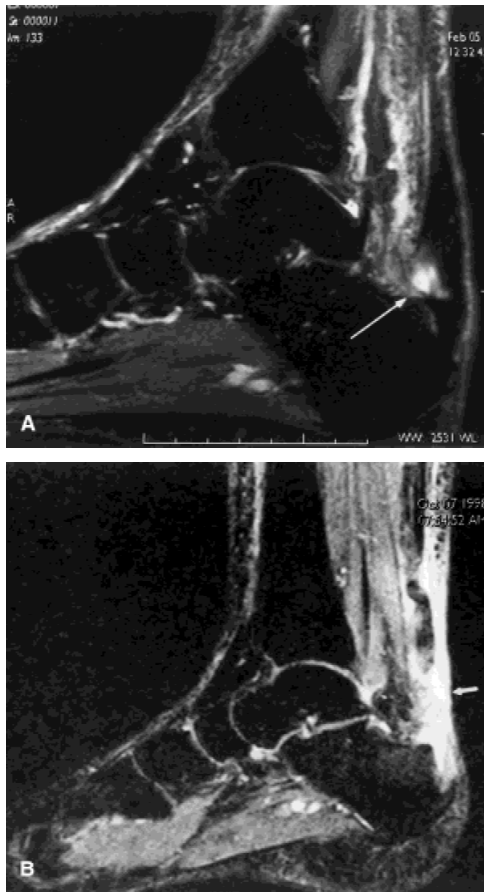
Fig. 6. Achilles tendon rupture. **A** (STIR sagittal sequence): Partial-thickness tear of a degenerated thickened Achilles tendon. Very hyperintense material at the anterior surface of the tendon represents the partial-thickness tear (arrow). The tendon remains continuous with intact low signal intensity fibers posteriorly. **B** (sagittal STIR sequence): Full-thickness tear of the Achilles tendon. Very hyperintense material fills the gap between the torn tendon end and the posterior tuberosity of the calcaneus (arrow).

Avulsion fractures of the posterior tuberosity of the calcaneus also occur.

The peroneus brevis and peroneus longus tendons course around the lateral aspect of the ankle. Each arises from separate muscles within the lateral compartment of the lower limb. Posterior to the lateral malleolus, they are contained in a common synovial sheath confined by the superior peroneal retinaculum. They course in the retromalleolar groove on the posterior aspect of the lateral malleolus. At the peroneal trochlea on the lateral aspect of the calcaneus, they divide into separate tendon sheaths [12]. The peroneus brevis courses superiorly and anteriorly to insert on the base of the fifth metatarsal. The os vesalianum is a rare sesamoid bone that may be contained within the tendon. The peroneus longus courses inferiorly in a groove under the cuboid toward its insertion on the base of the first metatarsal and the medial cuneiform. The os peroneum is a sesamoid bone that may be contained within the tendon at the lateral aspect of the cuboid. The peroneus brevis tendon is prone to tendinopathy as it courses in the retromalleolar groove where it is sandwiched between the peroneus longus tendon poste-
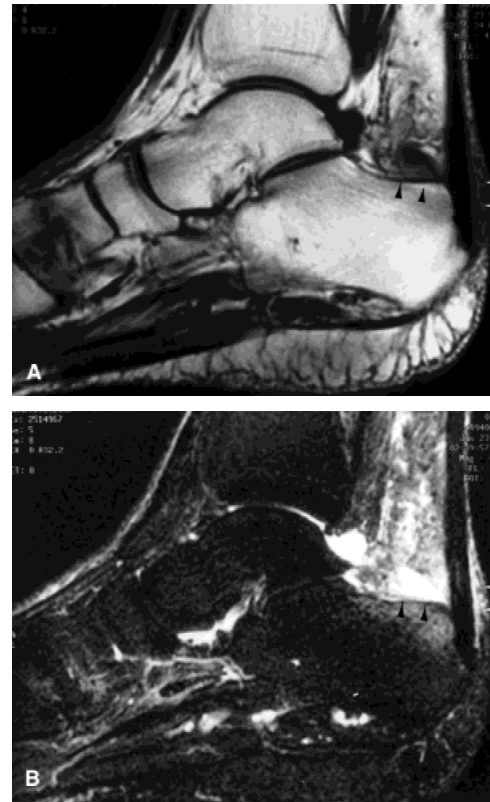
Fig. 7. Haglund's syndrome. **A** (T1-weighted sagittal sequence) and **B** (sagittal STIR sequence): Fluid in the retrocalcaneal bursa (black arrowheads), fluid in the retro tedo-Achilles bursa (white arrowheads), and tendinosis of the Achilles tendon at its insertion site. The Achilles tendon is thickened but remains normal in signal intensity on both of these sequences.

riorly and the bone anteriorly. The peroneus brevis tendon is also prone to partial tears, resulting in a "split tendon" (Fig. 8A). Proximal to the split, the tendon takes on a C-shaped appearance and partially envelops the peroneus longus tendon (Fig. 8B) [10,13]. A complete assessment of the peroneus brevis tendon also requires close inspection of the base of the fifth metatarsal where avulsion fractures occur.

The peroneus longus tendon is prone to tendinopathy as it courses around the lateral aspect of the cuboid. Acute ruptures of the peroneus longus tendon frequently occur through the os peroneum. When sufficiently ossified, this diagnosis can be made on the plain film when the sesamoid is fractured and/or retracted proximally [11]. Laterally dislocated or subluxing peroneal tendons are predisposed to tendinopathy at the retromalleolar groove. Both peroneal tendons should be confined by a tight superior peroneal retinaculum and should not extend laterally to the lateral cortex of the fibula. Subluxing tendons may require provocation to be demonstrated and, therefore, may be anatomically reduced at the time of MRI. Congenital laxity of the retinaculum and a congenitally convex retromalleolar groove may be causative. Lateral rim fractures of the fibula are avulsion fractures of the retinaculum that will result in dislocated or subluxing peroneal tendons. The peroneus quartus muscle is an accessory muscle present in as many as 10% of ankles at MRI (Fig. 9). It also occupies the superior
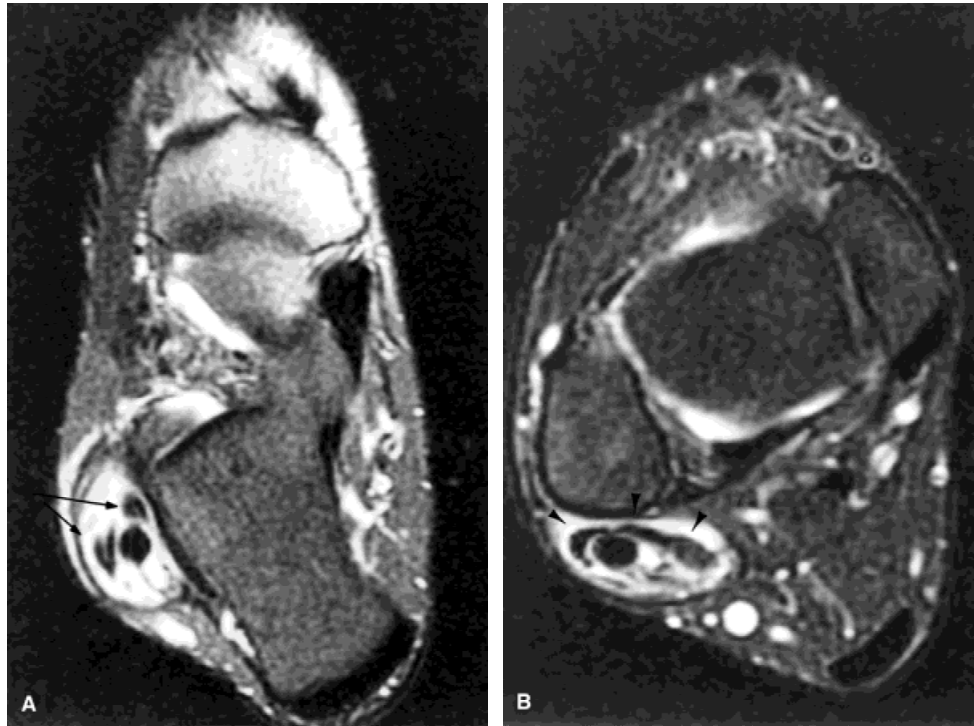
Fig. 8. Split peroneus brevis tendon. **A** (T2-weighted fat-suppressed axial sequence inferior to the lateral malleolus): Three low signal intensity tendons when there should only be two. The two anterior tendons (arrows) are the split components of the peroneus brevis tendon. The posterior tendon is the peroneus longus tendon. **B** (T2-weighted fat-suppressed axial sequence at the lateral malleolus): C-shaped appearance of the peroneus brevis tendon (arrowheads) partially circumscribing the peroneus longus tendon proximal to the actual split.

peroneal retinaculum with the peroneus brevis and longus tendons and can result in anatomic impingement of these tendons, contributing to tendinopathy [2].

The posterior tibial tendon courses around the medial aspect of the ankle. It is contained in a shallow groove at the posterior aspect of the medial malleolus by the flexor retinaculum. Also confined by this retinaculum are the flexor digitorum longus and flexor hallucis longus tendons. All
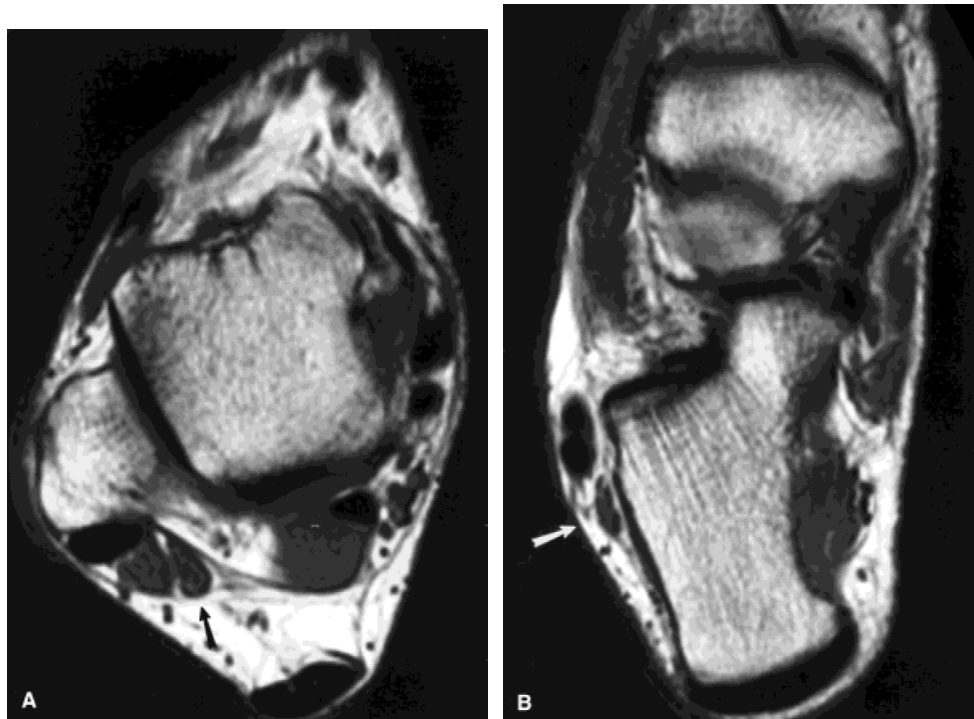

Fig. 9. Peroneus quartus muscle. Proton density-weighted axial sequences. At the lateral malleolus (**A**), there is an extra muscle (arrow) located posteriorly to the paired peroneal tendons confined by the superior peroneal retinaculum. Inferiorly (**B**), this accessory muscle takes an abbreviated course and inserts on the retrotrochlear eminence (arrow).
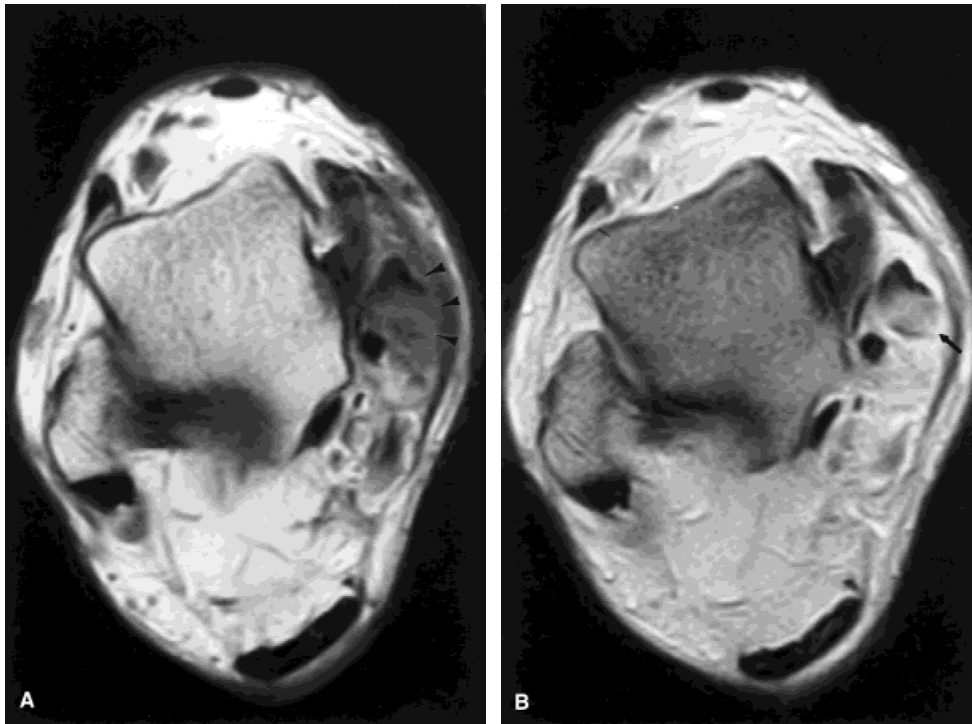
Fig. 10. Partial-thickness tear of the posterior tibial tendon at the medial malleolus. **A** (proton density-weighted axial sequence): Enlargement of the posterior tibial tendon, with hyperintense material partially replacing the tendon substance (arrowheads). **B** (T2-weighted axial sequence): Hyperintense material partially replaces the tendon substance. This material reaches the tendon surface (arrow) and represents the partial-thickness tendon tear. Nondisrupted low signal intensity tendon fibers are situated anteriorly. There is an associated tenosynovitis.

three muscles occupy the posterior compartment of the lower limb and are contained in their own individual tendon sheaths. The posterior tibial tendon has a broad insertion onto the navicular tuberosity and the cuneiforms and bases of the medial metatarsals. An os tibiale externum is a sesamoid bone that is occasionally seen within the tendon proximal to its insertion site. An accessory ossification center of the navicular may be present at the navicular insertion site.
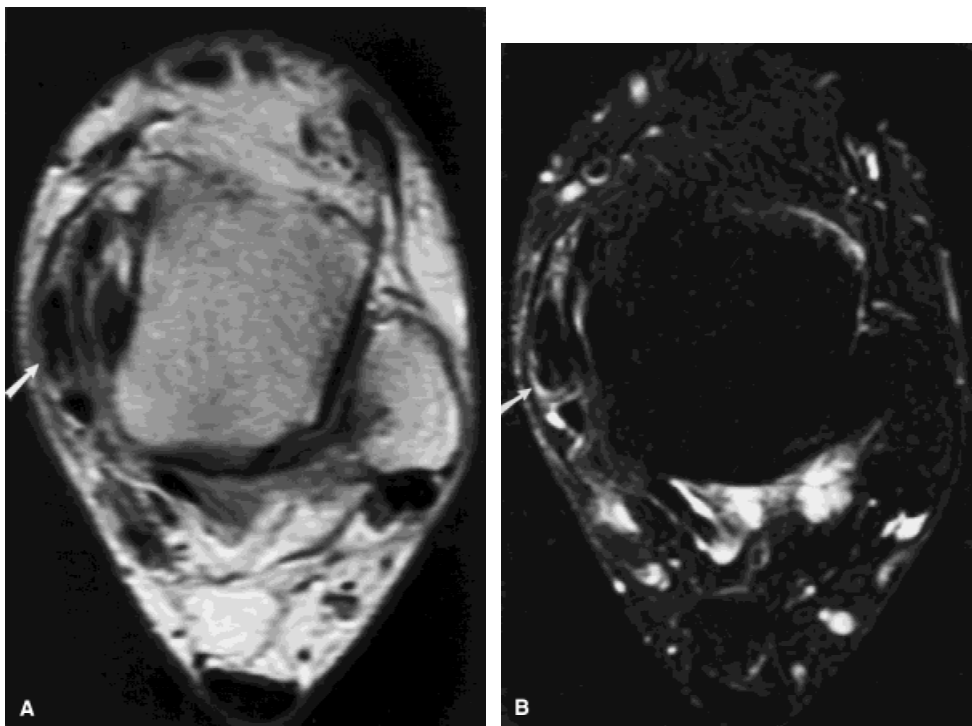
Fig. 11. Split posterior tibial tendon. **A** (proton density-weighted axial sequence) and **B** (T2-weighted fat-suppressed axial sequence): Linear area of increased signal intensity in the posterior tibial tendon (arrow).

The posterior tibial tendon is prone to tendinopathy as it courses around the medial malleolus. Patients are generally middle-aged females. Associated conditions include obesity, diabetes, and hypertension [6].

Chronic rupture of the posterior tibial tendon was surgically graded by Jahss [5]. Conti [3] described an MRI classification scheme. Both schemes categorize chronic rupture of the posterior tibial tendon into three major groups:

1. Type 1: The posterior tibial tendon is partially torn and focally enlarged. It contains multiple longitudinal splits that reach the tendon surface. Usually patients are symptomatic for less than one year (Fig. 10A,B).
2. Type 2: The posterior tibial tendon is partially torn and focally attenuated. Distal to the tear, the degenerated tendon is often enlarged over a short segment. The tendon lengthens and there is progressive hindfoot valgus. Patients are usually symptomatic between one and two years.
3. Type 3: The posterior tibial tendon is completely torn and no intact fibers bridge the torn ends. Peritendinous inflammatory changes may initially tether the torn ends to the tenosynovium, limiting tendon retraction. Peritalar subluxation and arthritis are progressive. Patients are usually symptomatic for more than two years.
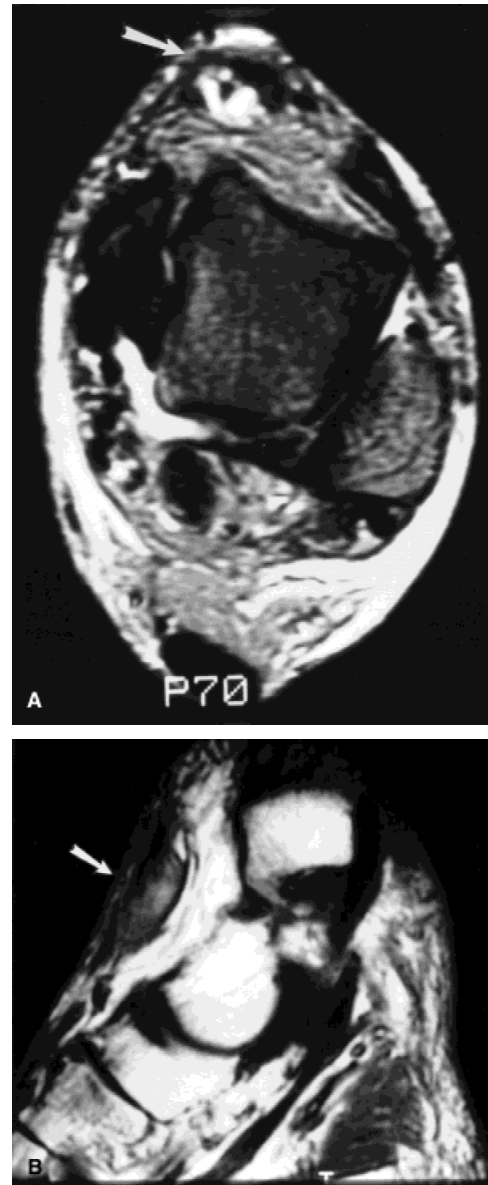
A split posterior tibial tendon analogous to the split peroneus brevis tendon occurs (Fig. 11). A dislocated or subluxing posterior tibial tendon has also been described [1].

Patients with an accessory ossification center for the navicular are predisposed to tendinopathy of the posterior tibial tendon at its insertion site. Caution must be made not to overdiagnose tendinosis in this area. In this location, the posterior tibial tendon frequently contains areas of intermediate signal intensity on the T1 and proton density-weighted sequences. An accessory ossification center for the navicular may be acutely painful. On MRI, there is edema at the subjacent bony surfaces and the synchondrosis may also separate (Fig. 12) [9].

The flexor hallucis longus tendon courses through a tunnel at the posterior aspect of the talus confined by the flexor retinaculum. It inserts on the base of the distal phalanx of the great toe. Its muscle also occupies the posterior compartment of the lower limb. The flexor hallucis longus tendon is prone to tenosynovitis and tendinopathy within the

Fig. 12. Stress reaction at the posterior tibial tendon insertion site on an accessory ossification center for the navicular. T2-weighted fat-suppressed axial sequence. The accessory ossification center is separated from the underlying navicular by a low signal intensity line representing the cortical margins of the synchondrosis (arrow). Abnormal hyperintense bone marrow edema is present in the subjacent marrow on either side of the synchondrosis. The low signal intensity posterior tibial tendon inserts on this accessory ossification center.

Fig. 13. Ruptured anterior tibial tendon. **A** (T2-weighted axial sequence) and **B** (T1-weighted sagittal sequence): Partial discontinuity of the anterior tibial tendon (arrow).

talar tunnel. This condition has been described in ballet dancers who perform point. A large os trigonum at the lateral aspect of the groove may contribute to impingement. Chronic nodular tenosynovitis may result in tethering of the tendon with triggering of the great toe [9].

The anterior tibial tendon courses over the anterior aspect of the ankle. Its muscle occupies the anterior compartment of the lower limb. Tendinopathies of this tendon have been described but are infrequent. Tendon rupture also occurs (Fig. 13).

In summary, MRI is the most accurate imaging modality for assessing pathologic conditions of the tendons of the ankle and foot. It accurately diagnoses tenosynovitis, para-tendonitis, tendinosis, and partial and complete tears. These disorders are variably symptomatic. They may be clinically occult or present with pain, disability, and progressive deformity. Tendon degeneration may be part of the process of aging. The imaging findings need to be correlated with the clinical scenario. When surgery is contemplated, MRI will qualify the degree of tendon derangement and associated anatomic conditions that might be causative. It will also identify normal tendons that may be utilized for surgical repairs.

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