Musculoskeletal Ultrasound

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Chapter 8
Foot and Ankle
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INTRODUCTION

The superficial location of most soft tissue and surface bone lesions of the ankle and foot makes this area readily accessible to high frequency (>10 MHz) ultrasound. The high resolution and clinical efficacy of ultrasound ensure that it is a comparable, and in many respects better, imaging modality than magnetic resonance imaging (MRI) when assessing most soft tissues of the foot and ankle. Imaging anatomy and pathology are best approached by arbitrarily dividing the ankle into anterior, posterior, medial, and lateral regions, whereas the foot is best approached by separately considering the hindfoot, midfoot, and forefoot.

PATIENT POSITION

For the anterior, medial, and lateral ankle and dorsal foot, the patient should be supine with the knee semiflexed. The knee is rotated medially and the ankle slightly inverted for the lateral ankle, and the knee rotated laterally with the ankle slightly everted for the medial ankle. For the posterior ankle and sole of the foot, the patient is best examined prone with the foot hanging freely or in a dorsiflexed position over the edge of the examination couch.

ULTRASOUND APPEARANCES OF LIGAMENTS, TENDONS, AND NERVES

The ultrasound appearances of ankle and foot ligaments, tendons, and nerves are similar to those in other parts of the body. In general, ligaments are seen as thin, regular, hyperechoic fibers aligned in parallel with a well-defined border and attached to bone at both ends. Ankle ligaments are best examined with the transducer aligned with the long axis of the ligament and the ligament stretched. Tendons have a thicker, tightly packed linear fibrillar echotexture when scanned longitudinally and have a speckled echogenic dot appearance on transverse scanning. Most tendons and nerves around the ankle joint are best examined in a transverse plane. Extended longitudinal views of the curved ankle tendons are difficult to obtain. Ankle tendons are prevented from “bowstringing” by overlying thin fibrous retinacula, which also serve as fulcra for the pulley action of the tendons. All ankle and foot ligaments and tendons are anisotropic and appear hypoechoic if the transducer is not perpendicular to their fibers. Anisotropy can be used to identify a tendon or ligament by distinguishing it from adjacent tissues. For example, tilting the transducer to make the anterior talofibular ligament (ATFL) appear hypoechoic accentuates the conspicuity of the ligament against the adjacent echogenic fat. Conversely, tendon or ligament echogenicity should always be interpreted in the light of anisotropy. Nerves are also tubular echogenic fascicular structures. They have a slightly coarser fibrillar echotexture than tendons and are found in predictable locations, typically in neurovascular bundles. If doubt exists, nerves can be differentiated from tendons by tracing their course proximally or distally, while scanning in short axis.

Tip:

In general, most ankle and foot tendons or ligaments are best assessed with ultrasound except for those on the plantar aspect of the foot, which are best assessed with MR imaging

RELEVANT ULTRASOUND ANATOMY

Anterior Ankle

Anterior Ligaments

The anterior inferior tibiofibular ligament is a strong multifascicular fan-shaped ligament that lies between the anterior aspect of the distal tibia (Tillaux-Chaput tubercle) and the adjacent fibula (Fig. 8.1). It runs slightly obliquely upward and medially from the anteromedial aspect of the distal fibula at the upper margin of the ankle joint and is widest at the tibial insertion (Fig. 8.1). Moving the transducer proximally in a transverse plane from the ankle joint shows the anterior tibiofibular ligament (Fig. 8.2). Ligament conspicuity can be improved by slight angulation of the transducer or by rotating the tibial end of the transducer slightly more cranially.

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The ligament is <2-mm wide at its mid-portion and provides major support to the distal tibiofibular syndesmosis.[1] The distal interosseous membrane is seen as a thin, hyperechoic (almost isoechoic to bone) line between the tibia and the fibula[2] proximal to the anterior tibiofibular ligament.

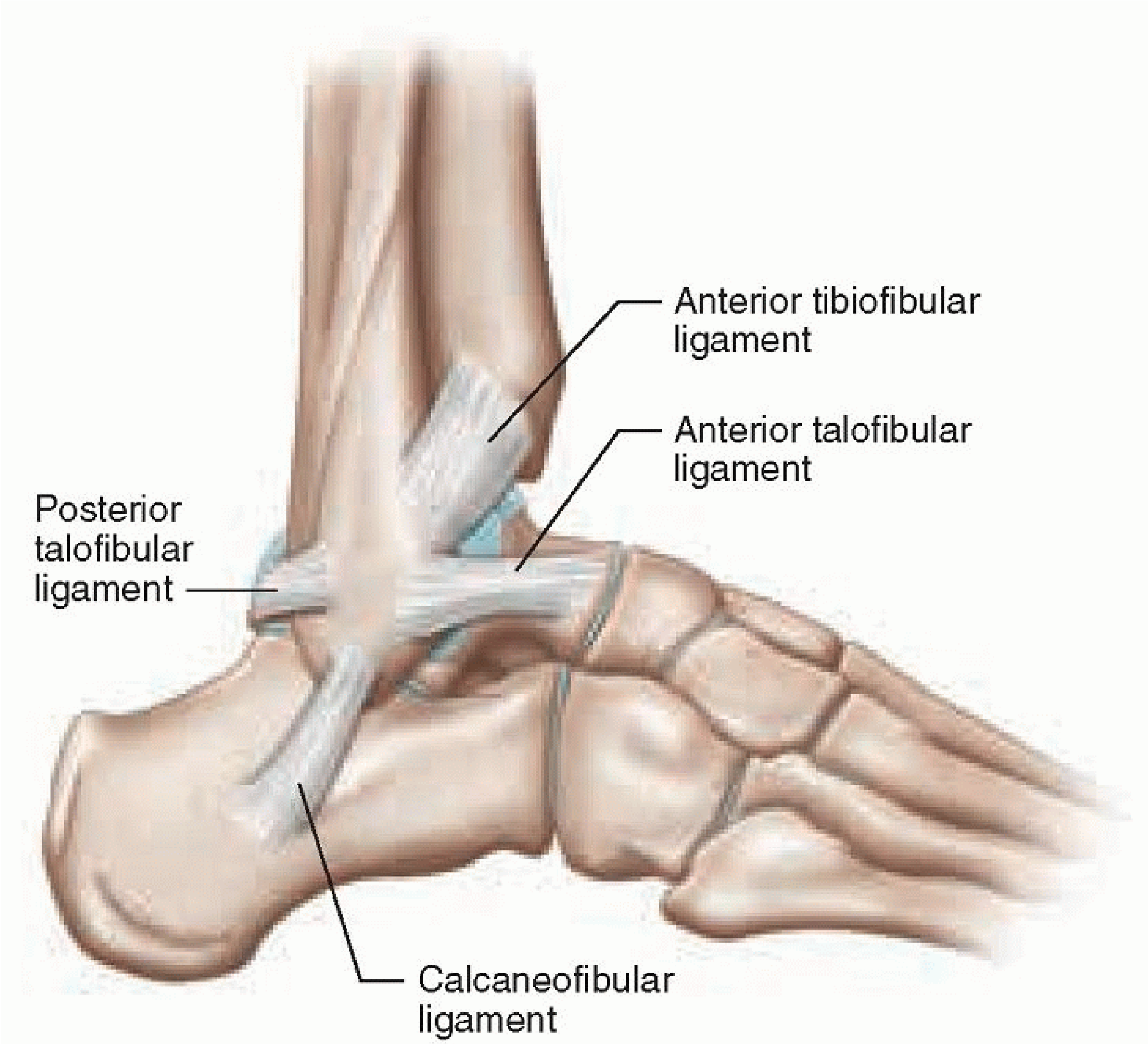

Figure 8.1. Schematic diagram of the ankle showing lateral and anterior ankle ligaments.

Tip:

The anterior inferior tibiofibular ligament is best seen by scanning transversely between the tibia and the fibula just proximal to the ankle joint.

Anterior Tendons

The anterior ankle tendons comprise the tibialis anterior, extensor hallucis longus (EHL), and extensor digitorum longus (EDL) from medial to lateral (Fig. 8.3). They can be memorized by "Tom Has a Date Perhaps" from medial to lateral (Tom = Tibialis anterior, Has = extensor Hallucis longus, A = Anterior tibial artery/vein and deep peroneal nerve, Date = extensor Digitorum longus, and Perhaps = Peroneus tertius). They are covered by retinaculum. The superior extensor retinaculum extends from the lateral malleolus to the medial malleolus and is continuous with the flexor retinaculum medially and superior peroneal retinaculum laterally. The inferior extensor retinaculum is Y-shaped. The stem of the Y lies on the lateral aspect of the retinaculum and divides into superomedial and inferomedial limbs, which attach to the medial malleolus and navicular-cuneiform area, respectively.

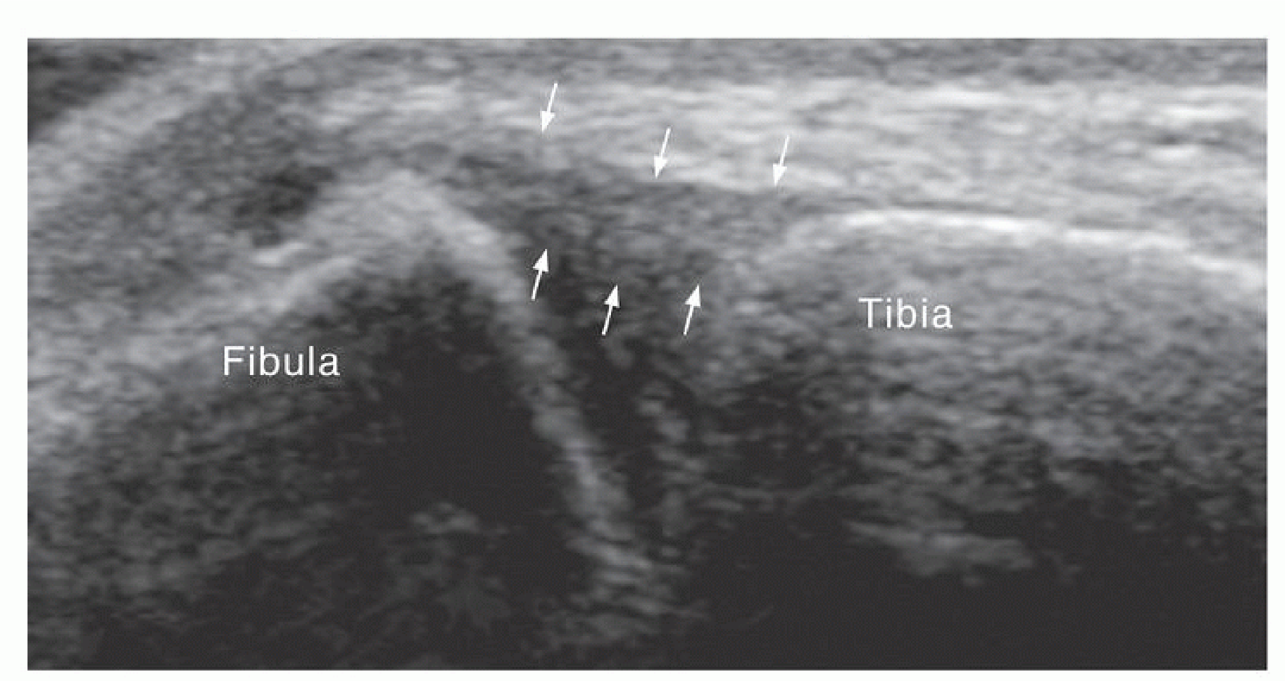

Figure 8.2. Transverse ultrasound showing normal anterior inferior tibiofibular ligament (arrows) with a wider attachment to the tibia than the fibula.

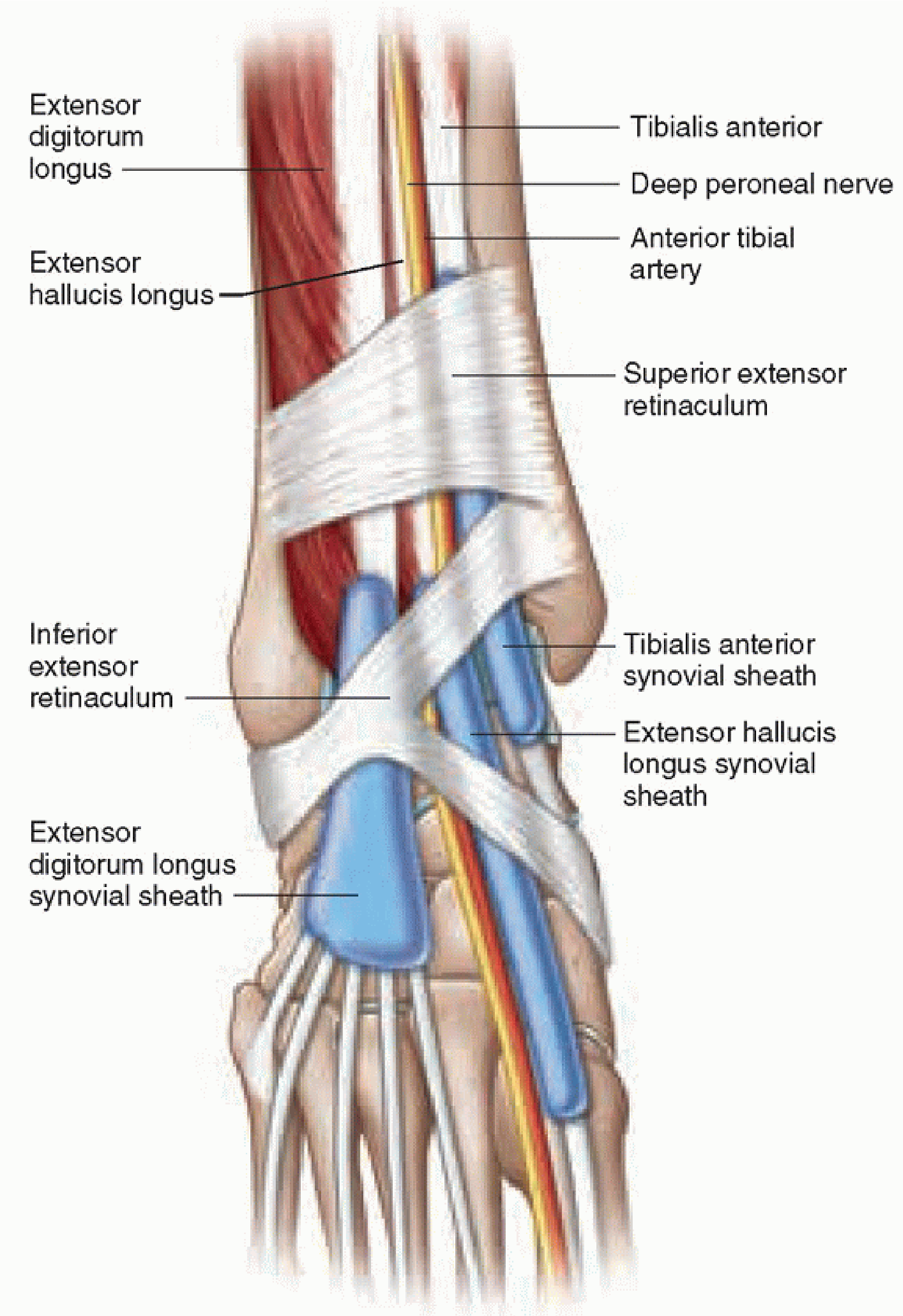

Figure 8.3. Schematic diagram of the anterior aspect of the ankle showing tendons running deep to the superior and inferior extensor retinacula. Note how the dorsalis pedis neurovascular bundle runs between the FDL and the flexor pollicis longus tendons. The deep branch of the peroneal nerve lies alongside the FHL tendon. FDL, flexor digitorum longus; FHL, flexor hallucis longus.

The tibialis anterior tendon is the strongest, largest, and most superficial of the anterior tendons. It begins at the junction of the lower and middle-thirds of the tibia. At the level of the ankle joint, it lies medial to the midline

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and then runs toward the medial border of the foot, where it attaches to the superomedial aspect of the medial cuneiform and the first metatarsal[3],[4] (Fig. 8.3). The tendon is ovoid proximally and becomes flatter distally, measuring <5 mm in width at 3 cm from its insertion.[5]

The EHL tendon lies just lateral to the tibialis anterior tendon and inserts dorsally on the base of the distal phalanx of the hallux. The anterior tibial artery, anterior tibial vein, and deep peroneal nerve lie just lateral to EHL. In the distal leg, the deep peroneal nerve lies on the medial side of the anterior tibial artery. At the ankle, the nerve crosses over the artery to lie on its lateral side (Fig. 8.3). This arrangement is important to note during aspiration of an ankle joint effusion using an anterior approach.

The EDL tendon is the most lateral of the anterior tendons, and branches at the anterior ankle joint line into separate tendons to the second to fifth toes (Fig. 8.3). The peroneus tertius tendon crosses the ankle joint lateral to the EDL tendon to insert into the dorsal surface of the fifth metatarsal base. It is absent in about 10% of individuals.

Tip:

All extensor tendons of the ankle have their own individual tendon sheath at the level of the inferior retinaculum.

The deep and superficial peroneal nerves arise from the common peroneal nerve at the level of the fibular neck and run distally deep to the peroneus longus muscle. At the ankle, the deep peroneal nerve runs deep to the extensor retinaculum initially medial to and then lateral to the anterior tibial artery. Just distal to the retinaculum it divides into a medial sensory branch, which supplies the first digital interspace, and a lateral motor branch, which supplies the extensor digitorum brevis muscle. It can become trapped between the extensor digitorum brevis muscle and the head of the talus.[6] The superficial peroneal nerve separates from the deep peroneal nerve 10 to 15 cm above the ankle. It pierces the investing fascia about 9 cm proximal to the lateral malleolus, where it is prone to injury,[7] then runs anterolateral to the lateral malleolus, superficial to the superior and inferior extensor retinacula, before dividing into medial and intermediate dorsal cutaneous nerves. It provides sensory innervation to the skin on the dorsum of the foot and most of the toes.

Tip:

The deep peroneal nerve lies initially medial to and then lateral to the anterior tibial artery at the ankle joint.

Anterior Ankle Joint

The anterior ankle joint is best assessed in the longitudinal plane with the ankle in slight plantar flexion. The anterior joint capsule is attached along the distal margin of the tibia and fibula approximately 10 mm from the ankle joint and extends to the neck of the talus.[8] High-resolution imaging is needed to distinguish the capsule from the triangular-shaped fat pad that lies slightly deeper and divides the anterior recess of the ankle into smaller superior and larger inferior recesses (Fig. 8.4). A small amount of joint fluid is normal and best appreciated in the medial and lateral recesses. Larger quantities of fluid distend the anterior and posterior recesses. Increased fluid in the anterior recess displaces the fat pad anteriorly. A thin (approximately 2 mm), hypoechoic layer of articular cartilage covers the anterior aspect of the talar dome when the ankle is plantar flexed and should not be confused with joint fluid. Articular cartilage in the central and posterior ankle joint cannot be seen with ultrasound.

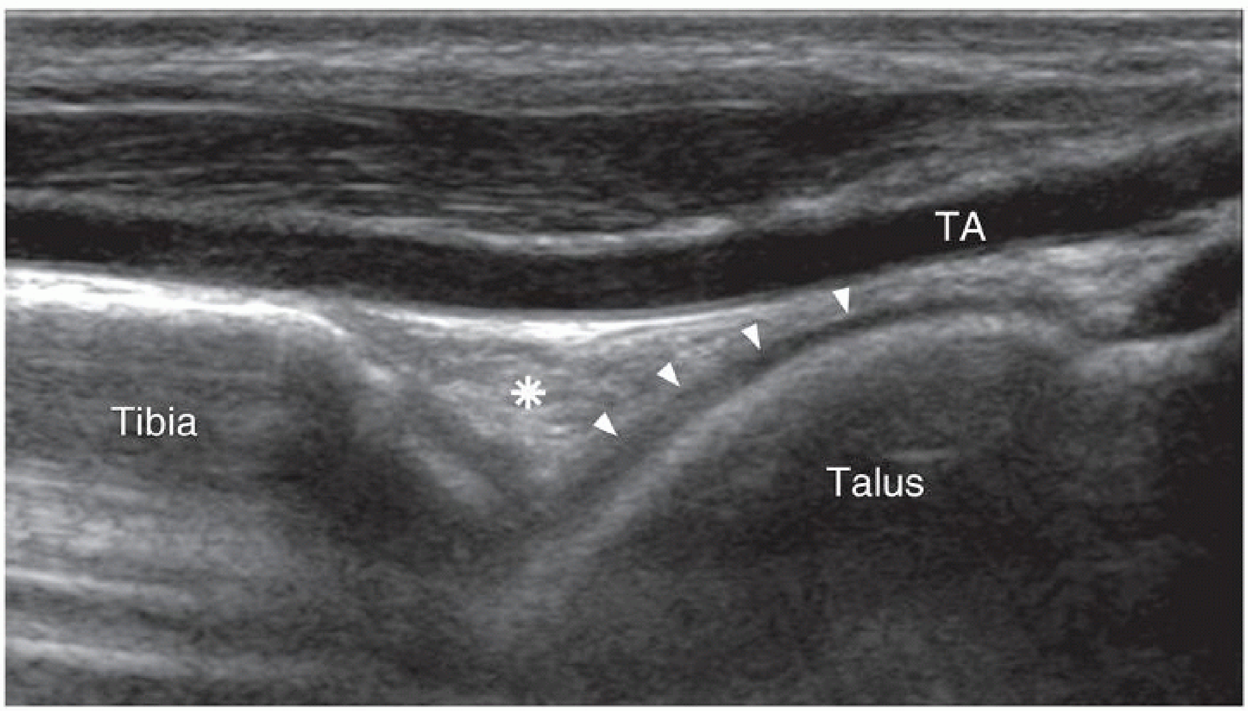

Figure 8.4. Longitudinal ultrasound of normal anterior aspect of ankle joint at the level of tibialis anterior artery (TA). Note the distal tibia and hypoechoic articular cartilage (arrowheads) on anterior aspect of the talar dome. Note the subcapsular triangular-shaped anterior fat pad (asterisk) segregating the anterior recess into smaller superior and larger inferior recesses.

The anterolateral recess or gutter is bounded laterally by the fibula, medially by the talus and tibia, anteriorly by the tibiotalar joint capsule, the anterior talofibular and tibiofibular ligaments, anteroinferiorly by the calcaneofibular (CFL) ligament, and superiorly by the tibial plafond and distal tibiofibular syndesmosis.[9] The anterolateral and anteromedial recesses should be routinely assessed on ultrasound of the ankle joint as fluid, crystal aggregates, synovial proliferation, and other pathologies may be visible, although not apparent in the anterior recess.

Lateral Ankle

Lateral Ligaments

The lateral ankle ligaments, or lateral collateral ligaments, comprise the anterior talofibular, CFL, and posterior talofibular ligaments (Fig. 8.1).

Anterior Talofibular Ligament (ATFL)

The ATFL is a condensation of the ankle capsule similar to the glenohumeral ligaments in the shoulder.[10] It extends
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from the anterosuperior aspect of the distal fibula at the level of the malleolar fossa to the lateral aspect of the neck of talus. It is best visualized with the ankle in a relaxed position (about 20 degrees plantar flexed, i.e., with the sole of the foot flat on the examination couch) and the ligament almost parallel to the ankle joint. The transducer is placed just anterior to the lateral malleolus and oriented horizontally. The distal end of the transducer is then turned slightly caudally.[11] The normal thickness of the ATFL is 2 mm, and it is about 20 mm in length.[12] The ligament is best seen when straight and taut (Fig. 8.5) by plantar flexing the ankle joint.

Tip:

The ATFL is best seen by scanning almost transversely when the foot is slightly plantar flexed just distal to the ankle joint.

Calcaneofibular Ligament

The CFL ligament extends posteroinferiorly from the inferior tip of the fibula to insert on the lateral aspect of the calcaneal body. It lies deep, almost at right angles to the peroneal tendons (Fig. 8.1), and has a curved alignment akin to a hammock when relaxed. The normal ligament is about 2 mm wide and 2 mm thick and can be seen deep to the peroneal tendons when the tendons are scanned transversely[12] (Fig. 8.6). The ligament is best seen with the ankle in dorsiflexion as this stretches the ligament and reduces its curvature. The transducer is positioned with its proximal end at the inferior tip of the lateral malleolus and its distal end aligned posteriorly and slightly toward the heel.[12] The ligament is hyperechoic in the distal two-thirds only. The curved proximal portion of the ligament usually appears hypoechoic due to anisotropy.

Tip:

The CFL ligament is best seen deep to the inframalleolar peroneal tendons when these tendons are scanned in a transverse plane.

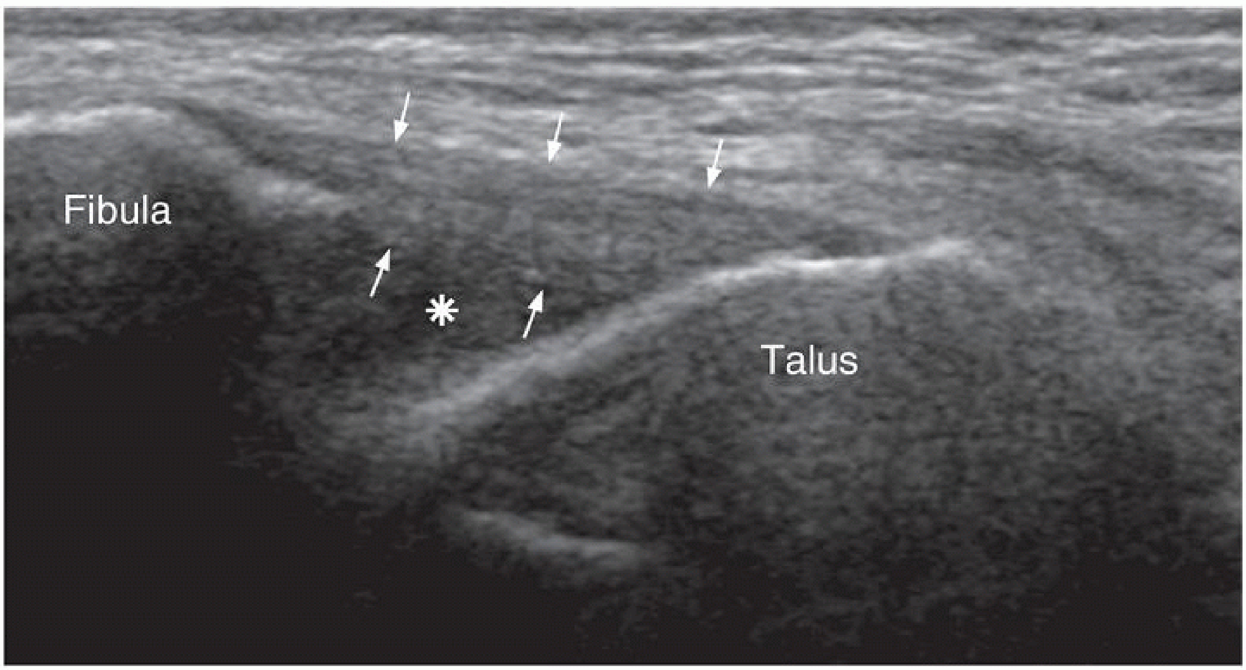

Figure 8.5. Transverse ultrasound showing normal ATFL (arrows). There is a small amount of fluid in the anterolateral ankle recess (asterisk). ATFL, anterior talofibular ligament.

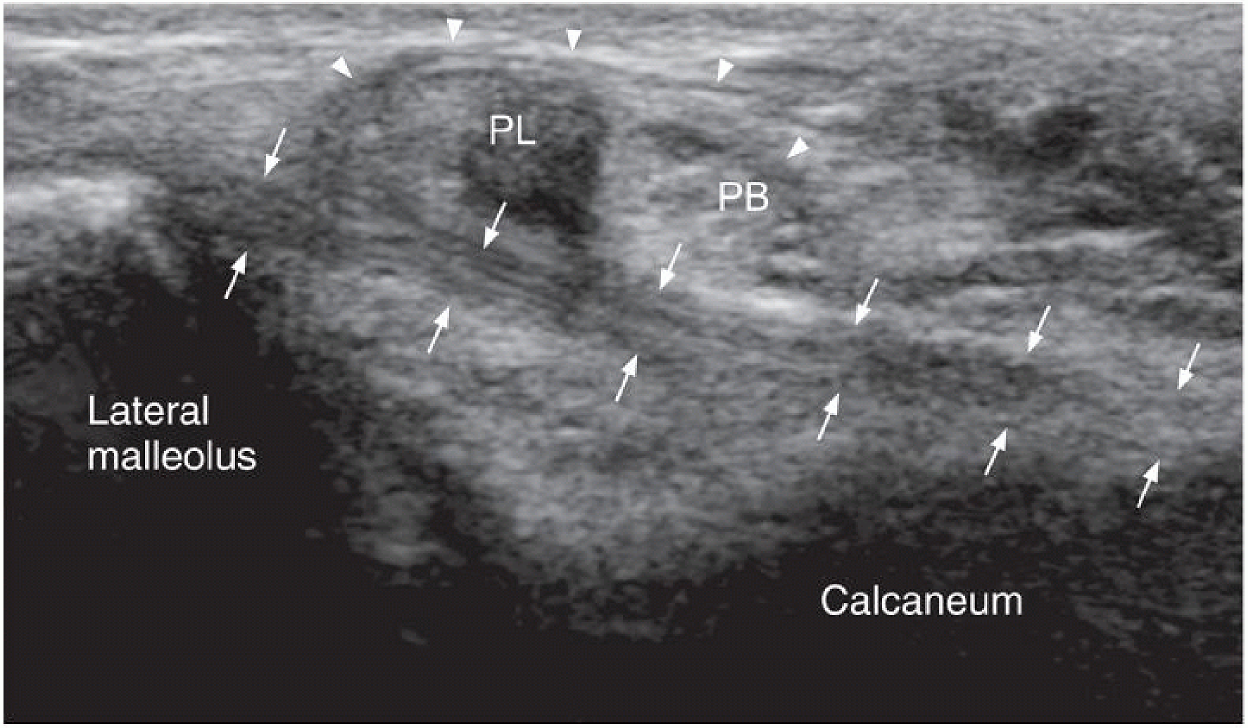

Figure 8.6. Transverse oblique ultrasound showing the CFL (arrows) extending between the fibular tip and the calcaneum. The ligament lies deep to the peroneus longus (PL) and brevis (PB) tendons. Note also the inferior extensor retinaculum (arrowheads) overlying the peroneal tendons at this location. CFL, calcaneofibular ligament.

Posterior Talofibular Ligament

This ligament is partially obscured by the fibula and is difficult to see in its entirety on ultrasound. It is orientated in a similar plane to the ATFL and extends from the distal aspect of the malleolar fossa and to the lateral tubercle of the posterior process of the talus. It is best seen with the patient prone.

Lateral Tendons

The tendons posterior to the lateral malleolus are the peroneus brevis and peroneus longus tendons (Fig. 8.7). The peroneus longus tendon starts about 4 cm above the level of the ankle joint and peroneus brevis more distally. Both tendons run posterior and then inferior to the lateral malleolus where they share a common supramalleolar and retromalleolar synovial sheath. They lie within the fibro-osseous retromalleolar groove of the fibula covered by the superior peroneal retinaculum. The groove is lined by fibrocartilage and varies in width and depth. The superior peroneal retinaculum is about 1mm thick, 1- to 2 cm wide; extends from the posterolateral edge of the distal fibula to the lateral wall of the calcaneum, although variations of this insertion exist[13]; and is the primary restraint to peroneal tendon subluxation. The peroneus longus tendon lies posterolateral to the brevis tendon in the retromalleolar groove and takes the longer route around the inferior aspect of the malleolus (remembered by the peroneus longus takes the longer route round the malleolus or peroneus brevis lies closer to the bone). The peroneus longus is marginally (10% to 15%) larger in cross-sectional area than the peroneus brevis tendon.[10]

Tip:

The peroneus longus tendon is slightly larger than the peroneus brevis tendon in cross-sectional area.

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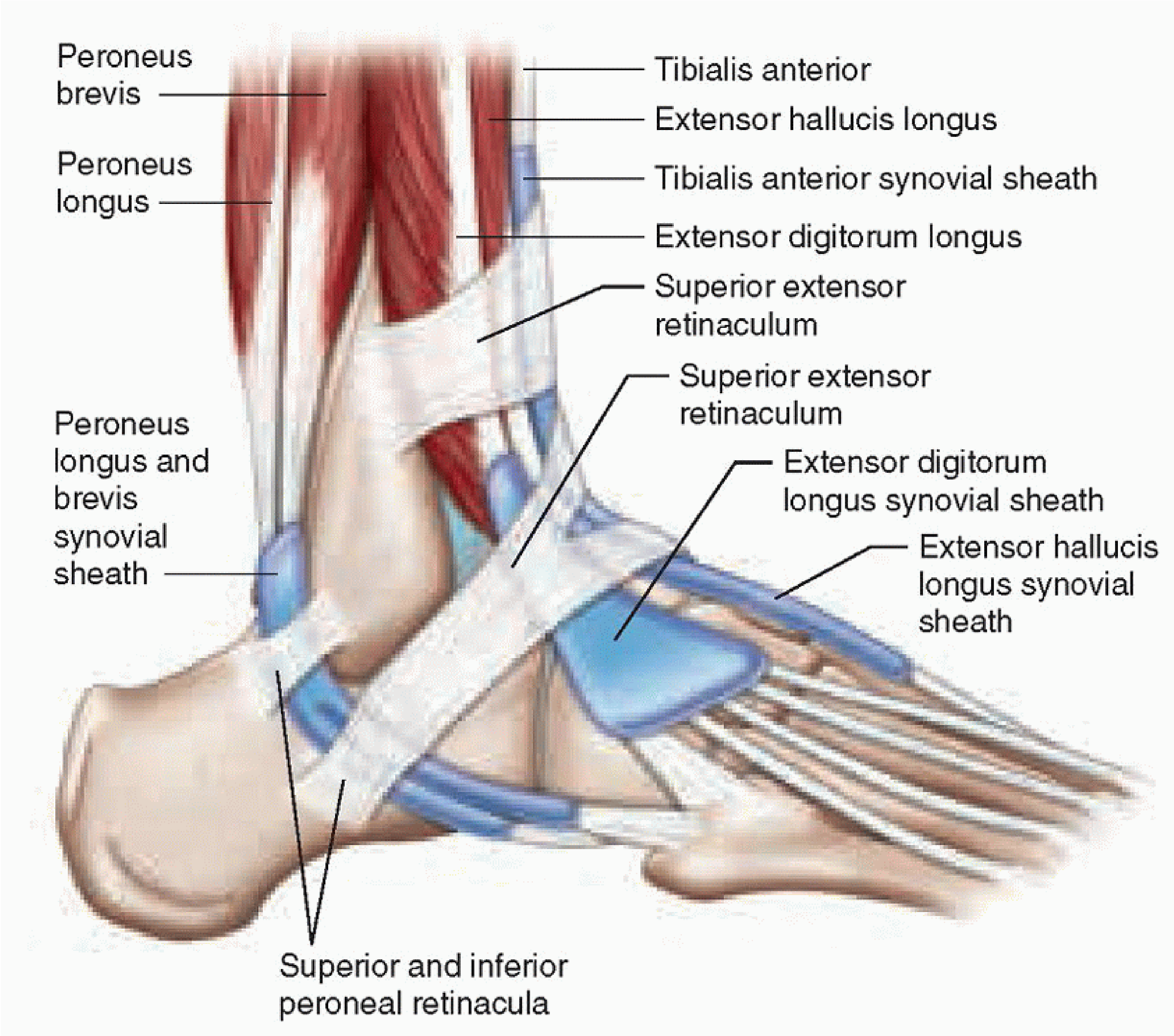

Figure 8.7. Schematic diagram of the tendons and retinacula on the anterior and lateral aspects of the ankle joint.

The common peroneal tendon sheath divides just proximal to the peroneal tubercle of the calcaneus, and the tendons run anteriorly in their respective tendon sheaths. Peroneus brevis runs dorsal and peroneus longus plantar to the peroneal tubercle, deep to the inferior peroneal retinaculum. The tubercle varies in size from a clearly palpable and distinct tubercle to a barely discernible one. As peroneus longus moves around the peroneal tubercle, it may become irritated when the tubercle is hypertrophied. Small amounts of fluid in the peroneal tendon sheaths are normal, particularly in the dependent inframalleolar region. The peroneus brevis tendon extends horizontally from the peroneal tubercle to insert on the base of the fifth metatarsal.

Peroneus longus passes into the sole of the foot running in a groove on the cuboid (the cuboid tunnel) before turning sharply and running obliquely across the midfoot to insert onto the undersurface of the medial cuneiform and the base of the first metatarsal. The os peroneum is a sesamoid bone located in the peroneus longus tendon just proximal to the cuboid tunnel in up to 30% of adults, bilateral in 60% of cases, bipartite in about 10%,[14] and seen as a small (5 to 15 mm) hyperechoic body with posterior acoustic shadowing. The primary function of the peroneus longus tendon is plantar flexion of the medial forefoot. Along with the peroneus brevis, it also everts and stabilizes the ankle joint.

Tip:

The os peroneum is located within the peroneus longus tendon and is sometimes associated with focal tendinosis of the peroneus longus and fracture.

An accessory peroneus quartus muscle, present in about 10% of subjects, lies medial and posterior to the peroneus brevis and longus. Its tendon runs in the common peroneal tendon sheath and usually inserts into the retrotrochlear eminence of the calcaneus, which is often hypertrophied, just posterior to the peroneal tubercle.[15] Other insertions include the peroneus longus and brevis tendons, the base of the fifth metatarsal, and the cuboid.[16] Occasionally, the muscle belly inserts directly into the retrotrochlear eminence.

The sural nerve runs initially between the Achilles tendon and the peroneal tendons accompanied by the small saphenous vein, then inferior to the peroneal tendons. It bifurcates into terminal lateral and medial branches at the base of the fifth metatarsal and provides sensory supply to the lateral ankle, foot, and the small toe. It anastomoses with branches of the superficial peroneal nerve.

Medial Ankle

Medial Ligament

Deltoid Ligament

The deltoid ligament is a strong, fan-shaped ligament extending from the medial malleolus to insert on the posterior talus, sustentaculum tali of the calcaneum, anterior talus, and navicular (Fig. 8.8A). It has superficial and deep components that are separable on ultrasound. The superficial fibers extend to the navicular bone, spring ligament, and calcaneum, and are known as the tibionavicular, tibiospring, and tibiocalcaneal fibers, respectively. The stronger and shorter deep fibers extend

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to the talus and are known as the anterior and posterior tibiotalar fibers. While the superficial and deep fibers are identifiable as separate structures, the individual components of the superficial and deep layers are seen as a continuum. In general, the superficial fibers tend to be located more anteriorly, though considerable overlap does occur. The deltoid ligament is best visualized in a longitudinal plane with the ankle slightly dorsiflexed and the hindfoot everted. The proximal end of the probe is placed on the medial malleolus, while the distal end of the probe is moved in a fan-shaped manner to show all components (Fig. 8.9). The anterior and mid-fibers are not as consistently well visualized as the posterior fibers which run posteriorly and inferiorly to the talus.[17] The deltoid ligament is normally 4- to 5 mm thick in its mid-portion.[18] On transverse scanning, the tibialis posterior tendon crosses the superficial component of the deltoid ligament.

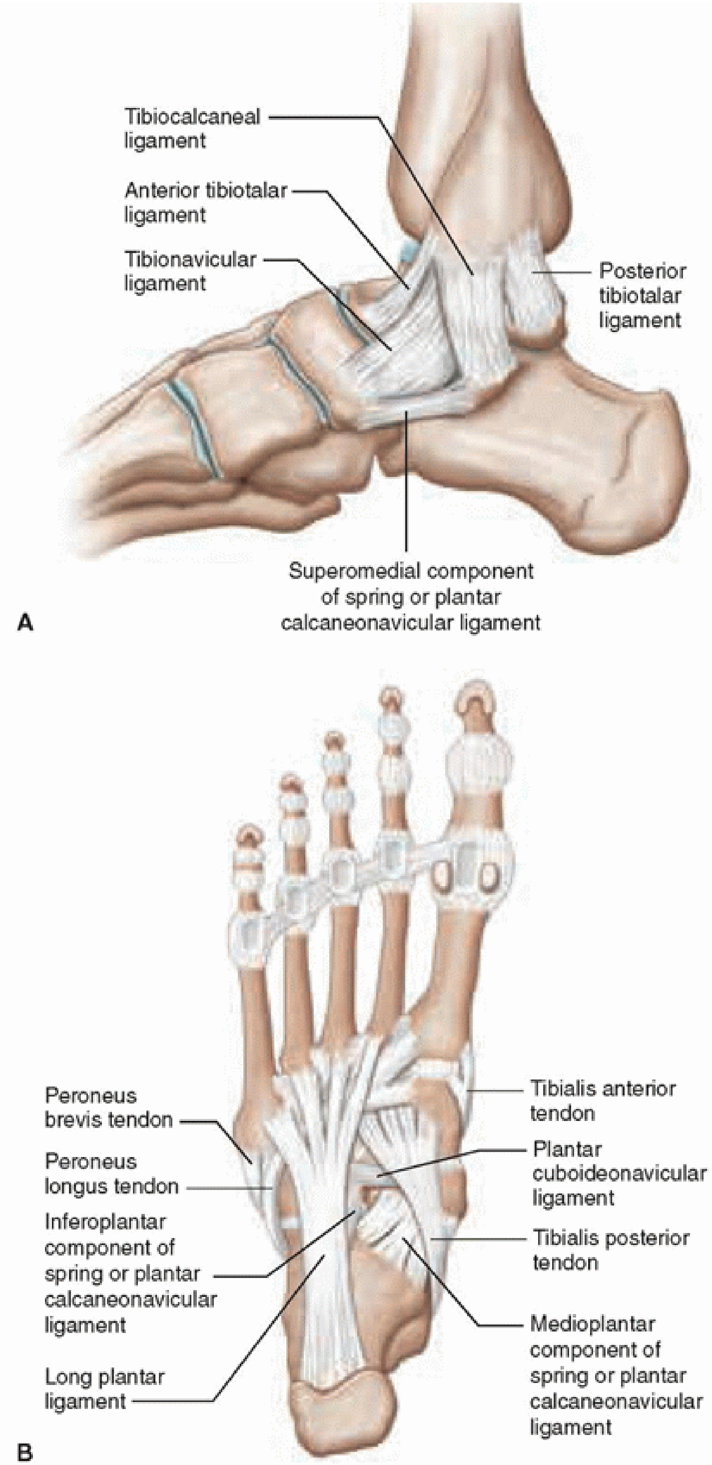

Figure 8.8. A: Schematic diagram of the medial aspect of ankle showing components of deltoid ligament. The superomedial component of the calcaneonavicular component is also shown. B: Schematic diagram of the plantar aspect of foot showing tendon insertions, and plantar components of spring ligament (inferoplantar and medioplantar components).

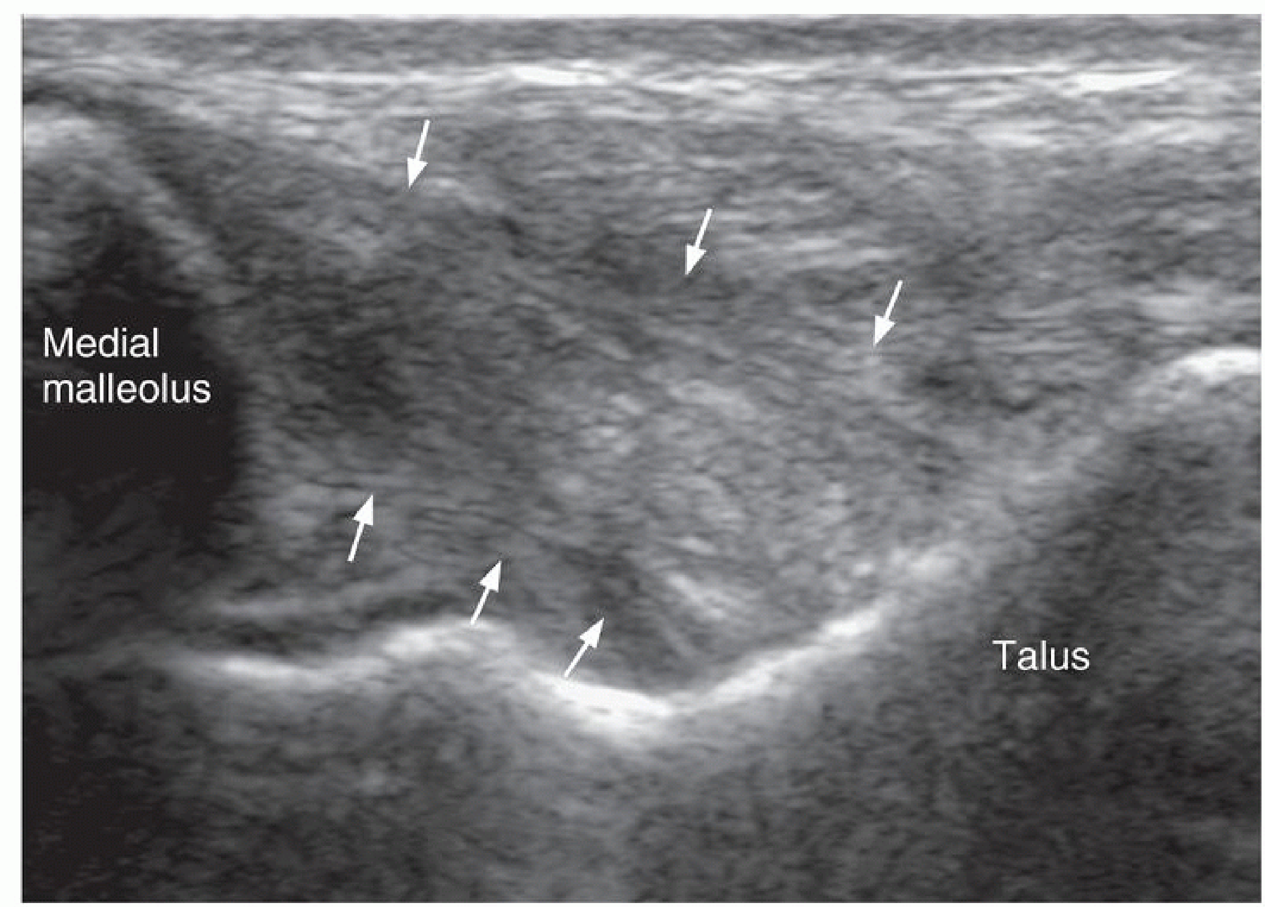

Figure 8.9. Longitudinal ultrasound showing normal fan-shaped deltoid ligament. The five different components of the deltoid ligament (arrows) are not readily distinguishable on ultrasound. The posterior fibers of the ligament extending between the medial malleolus and the talus are shown.

Tip:

The deltoid ligament is composed of superficial and deep fibers. The separate components of the fibers are inseparable on ultrasound.

Spring Ligament Complex

The spring (plantar calcaneonavicular) ligament complex is located between the calcaneus and navicular on the medial and inferoplantar aspects of the foot. It helps to support the head of the talus and, with the tibialis posterior tendon, forms a crucial support for the medial longitudinal arch[19] (Figs. 8.8A, B). It has superomedial, oblique medioplantar, and inferoplantar components (also known respectively as the medial, intermediate, and lateral components). The superomedial fibers are functionally the most important, and extend from the sustentaculum tali, over the surface of talar head, to

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insert onto the superomedial aspect of the navicular. There is a “gliding layer” of fibrocartilage between these fibers and the overlying posterior tibial tendon, though this gliding layer is not seen as a discrete separate structure on ultrasound imaging. There are also some connecting collagenous fibers between the posterior tibial tendon and the superomedial fibers of the spring ligament. The superomedial fibers are seen as an echogenic band just deep to the posterior tibial tendon,[20] best demonstrated by placing the probe with one end over the sustentaculum tali and the other over the talar head and medial aspect of the navicular bone.[20] The superomedial component of the spring ligament can also be seen deep to the posterior tibial tendon just proximal to the tendon insertion when the tendon is scanned transversely. Proximally, the normal thickness of the superomedial component of the spring ligament is 4 mm overlying the talar head.[21] The medioplantar and inferoplantar components of the spring ligament are functionally less important and not consistently seen on ultrasound (Fig. 8.8B).

Medial Tendons

The tendons that run posterior to the medial malleolus are from anterior to posterior (or medial to lateral), the posterior tibial (tibialis posterior), flexor digitorum longus (FDL), and flexor hallucis longus (FHL) tendons. The neurovascular bundle, comprising the posterior tibial vessels and tibial nerve, lies between the FDL and FHL tendons (Fig. 8.10). The arrangement can be memorized by “Tom, Dick, And Harry” from anterior to posterior (Tom = Tibialis posterior, Dick = FDL, And = posterior tibial Artery, concomitant veins and tibial nerve, and Harry = FHL). In the retromalleolar region, they are covered by the flexor retinaculum to form a fibro-osseous tarsal tunnel.[22] The retinaculum is a thin (<1 mm) echogenic band extending from the medial malleolus to the posterosuperior aspect of the calcaneus. The proximal tunnel is covered by a deep aponeurosis of the leg and has an osseous floor formed by the tibia and the talus. The more distal tunnel is covered by the flexor retinaculum and the fascial covering of the abductor hallucis muscle and has an osseous floor formed by the calcaneus, sustentaculum tali, and inferomedial navicular.

The tibial nerve trifurcates about 1.5 cm proximal to the tip of the medial malleolus.[23] Its first branch is the sensory medial calcaneal nerve, which arises proximal to the flexor retinaculum in 40% of subjects. When it does so, sensory deficit due to compression of the tibial nerve in the tarsal tunnel tends to spare the heel.[24] The tibial nerve then divides into the medial and lateral plantar nerves, deep to the flexor retinaculum and separated by the interfascicular septum. The medial plantar nerve runs with the posterior tibial tendon along the medial aspect of the foot to innervate the small muscles (abductor hallucis, flexor hallucis brevis, flexor digitorum brevis, and the first lumbrical muscles). The lateral plantar nerve descends under the midfoot to innervate the small muscles on the lateral side of the foot (abductor digiti minimi, abductor hallucis, quadratus plantae, interosseous muscles, second to fifth lumbricals).

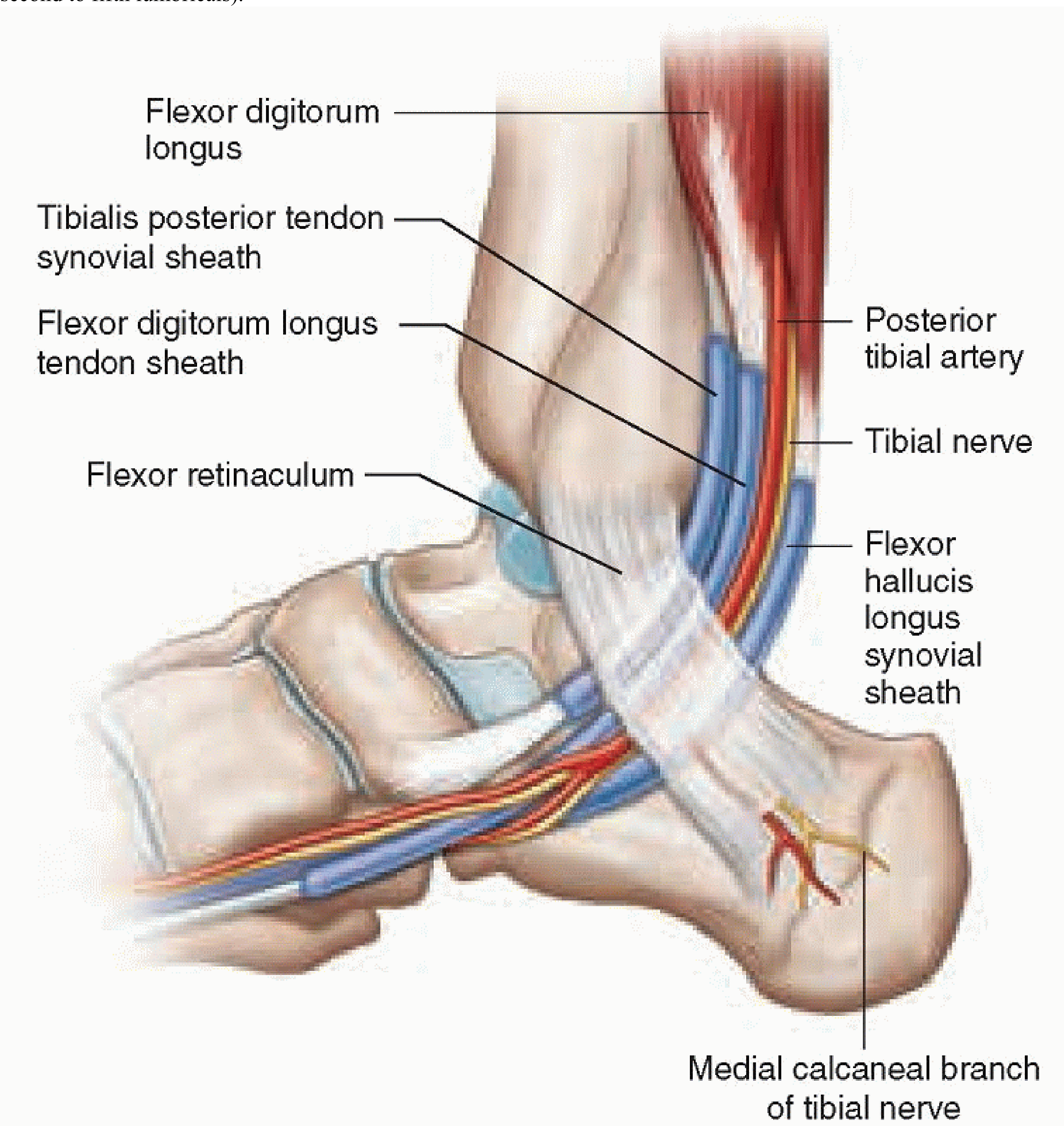

Figure 8.10. Schematic diagram showing “Tom, Dick, and Harry” arrangement of tendons on medial aspect of the ankle. The tibial nerve divides into the medial and lateral plantar nerves just distal to the medial retinaculum. The sensory medial calcaneal nerve often originates just proximal to or within the retinaculum.

The posterior tibial tendon is held against the posteromedial aspect of the medial malleolus by the flexor retinaculum. It courses around the inferior tip of the medial malleolus. The inframalleolar segment of the tendon runs over the deltoid ligament and the body of the calcaneum before inserting on the plantar aspects of the medial pole of the navicular, the cuneiforms, and the second to fourth metatarsal bases. Its tendon sheath extends from just distal to the musculotendinous junction to 1 to 2 cm proximal to the

navicular insertion. The tendon sheath is usually dry, but a trace of fluid (<2 mm in thickness and not circumferential) is normal. The posterior tibial tendon is best examined in short axis in the supra-, retro-, and inframalleolar regions. Distally, it is best examined in long axis as this allows easier evaluation of the insertion. The posterior tibial tendon normally fans out near its insertion, leading to thickening and hypoechogenicity that may mimic a tendon tear or tendinosis. It may insert into an elongated (cornuate) medial pole
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of the navicular or an accessory navicular. The normal cross-sectional area/mean width of the posterior tibial tendon are: retromalleolar <0.16 cm^2/<2 mm; inframalleolar <0.18 cm^2/<2.5 mm; and pre-insertional <0.27 cm^2/<3.5 mm. The retromalleolar and inframalleolar segments of the posterior tibial tendon are about twice the size of the adjacent FDL tendons. The largest variability in size of the posterior tibial tendon is the most distal portion where it flares out to insert onto the undersurface of the navicular medially, the three cuneiforms, and the medial metatarsal bases.[25]
Tip:
The retromalleolar and inframalleolar portions of the posterior tibial tendon are about twice the size of the EDL tendon.
The FDL tendon lies immediately posterolateral to the posterior tibial tendon behind the medial malleolus. It is important on transverse scanning to appreciate whether both tendons are present, as FDL can slip forward if tibialis posterior is ruptured and simulate a normal tibialis posterior tendon. In the inframalleolar region, FDL separates from the posterior tibial tendon and runs superficial to the sustentaculum tali. The normal caliber of the FDL is approximately half that of the tibialis posterior. Usually, there is no detectable fluid within the FDL tendon sheath on ultrasound.[26]
The FHL tendon is the most posterior and deepest ankle tendon in the medial ankle. The fleshy muscle belly extends to the level of the ankle joint. The tendon originates behind the ankle joint and lies in a fibro-osseous inframalleolar groove between the medial and lateral talar tubercle. The tendon is covered by the retinaculum and may become entrapped by reactive changes affecting the posterior talar process. Distally, the tendon courses inferior to the sustentaculum tali before inserting on the base of the distal phalanx of the hallux. Due to its deep location, it is less easily appreciated than the other medial ankle tendons on ultrasound. The retromalleolar portion is best seen on sagittal scanning medial to the Achilles tendon. Passive flexion and extension of the big toe during scanning help to confirm continuity of the FHL. In the hindfoot, FHL crosses the tendon of FDL to which it is connected by a thin fibrous slip. In 20% of normal individuals, the FHL tendon sheath communicates with the ankle joint, and an ankle effusion may result in considerable fluid distension of the FHL tendon sheath.
Tip:
Scanning in a sagittal plane medial to the Achilles tendon while flexing the big toe will help confirm continuity of FHL tendon.
Posterior Ankle
Posterior Ligaments
The posterior inferior tibiofibular and posterior intermalleolar ligaments are deep, not entirely accessible by ultrasound and better assessed by MRI. The posterior tibiofibular ligament is a short ligament between the posterior tibia and fibula. The posterior intermalleolar ligament is present in half of normal subjects and extends obliquely from the superior margin of the medial malleolus to the superior margin of the fibular malleolar fossa where it attaches just proximal to the posterior talofibular ligament.[27]
Posterior Tendon Groups
The Achilles tendon, the largest tendon in the human body, is a merger of the tendons of the triceps surae (i.e., gastrocnemius and soleus muscles) (Fig. 8.11). The gastrocnemius muscle ends in the mid-calf, soleus more distally. The Achilles tendon inserts via a broad attachment on the posterosuperior aspect of the calcaneus[28] (Fig. 8.12). There is a narrow band of fibrocartilage binding the tendon to the bone. The tendon is moderately echogenic with fine parallel echogenic lines on longitudinal scanning. It is generally oval-shaped with a flat or concave anterior border on transverse scanning and is covered by an incomplete tendon sheath or paratenon that envelops the dorsum and sides of the tendon. The paratenon is a thin echogenic band that outlines the moderately echogenic tendon. Anterior to the distal tendon is an echogenic fat pad known as
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Kager's fat. There are two bursae at the insertion. The retrocalcaneal bursa is a comma-shaped hypoechoic structure (2 to 3 mm in depth) between the tendon and the calcaneus. A subcutaneous bursa lies between the tendon and the skin. It is usually not distended or visible. Focal tendon calcification, fluid in the retrocalcaneal bursa, and bony contour abnormalities at the calcaneal insertion are seen in 2%, 35%, and 63% of asymptomatic subjects.[28] The normal Achilles tendon is 4 to 6 mm (mean 5.3 mm) wide and 5 to 6 cm long, although size is greater in taller and heavier subjects, and in men.[24,25] Achilles tendon measurements are best made on transverse scanning as measurements made on longitudinal scanning tend to be overestimations due to tendon obliquity. Vascularity on Doppler ultrasound is normally minimal or absent.[28] The middle third of the tendon is almost avascular,[28] while the proximal and distal portions are better vascularized from the soleus muscle belly and calcaneal periosteum, respectively. The Achilles tendon receives its sensory nerve supply from the nerves innervating the adjacent muscles and from cutaneous nerves, such as the sural nerve.[29]

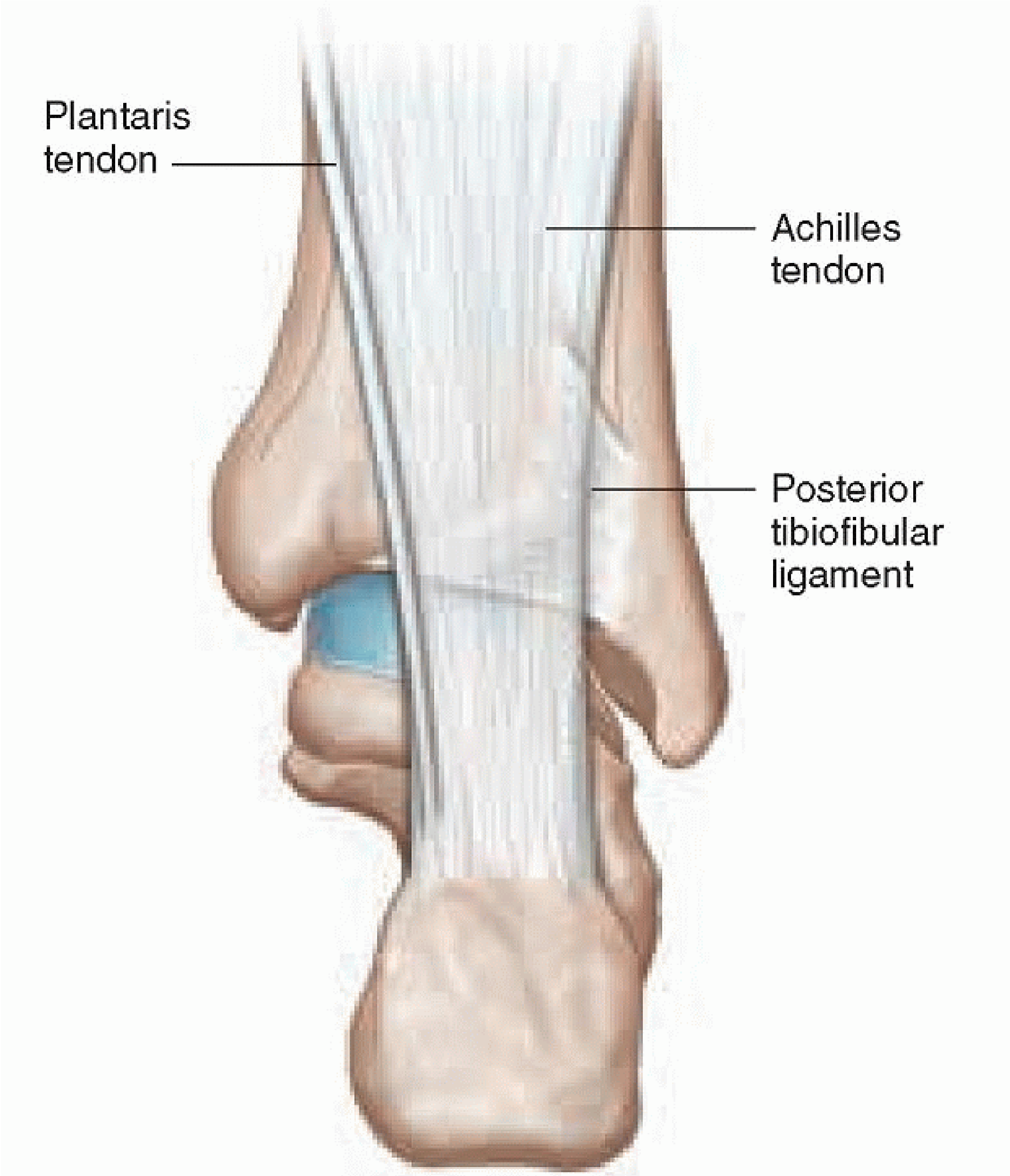

Figure 8.11. Schematic diagram of the posterior tendons showing the Achilles tendon and the plantaris tendon medially. The posterior tibiofibular ligament is also shown.

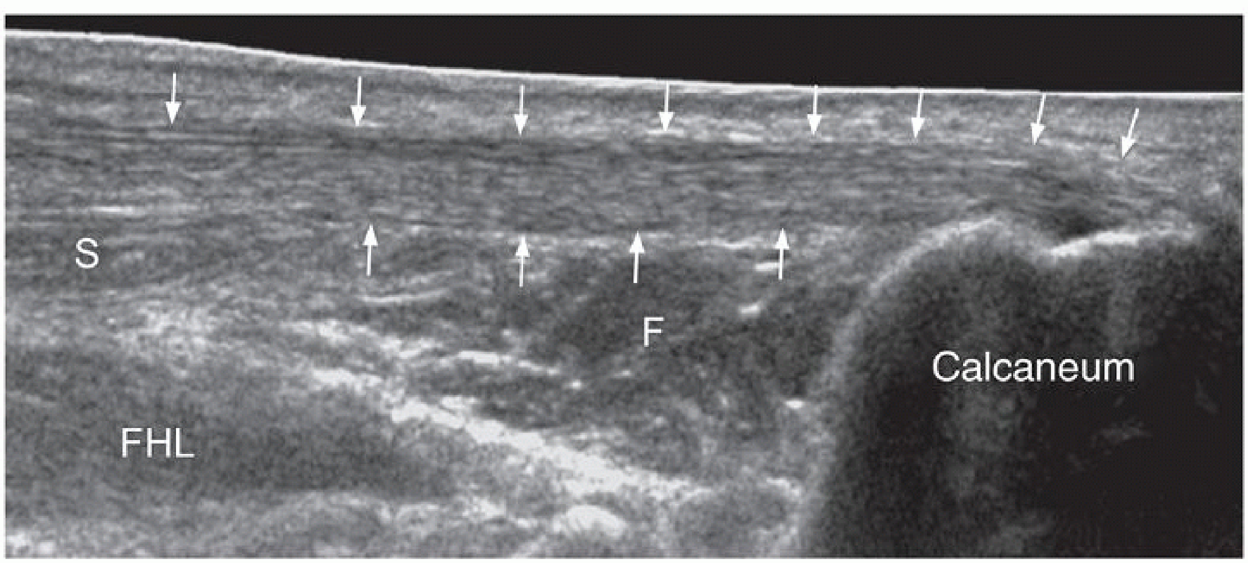

Figure 8.12. Longitudinal ultrasound showing normal Achilles tendon (arrows) attached to the posterosuperior aspect of the calcaneum. Note how the soleus muscle (S) joins the undersurface of the Achilles tendon. The gastrocnemius muscle joins the more superficial aspect of the tendon more proximally in the leg (not shown). Also shown are Kager fat pad (F) and the flexor hallucis longus (FHL) muscle.

Tip:

Measurements of the Achilles tendon are best made on transverse sections.

The plantaris muscle, which is present in about 90% of the population, has a short (7 to 10 cm) muscle belly arising from the lateral supracondylar region of the femur superficial to the origin of the lateral head of gastrocnemius.[30] The long slender plantaris tendon runs inferomedially, deep to the medial belly of gastrocnemius, to the medial side of the Achilles tendon, and either fuses with the Achilles tendon or inserts into the flexor retinaculum or the calcaneus. The myotendinous junction of plantaris is in the upper calf region.[31] The tendon is a thin ribbon-like structure between the medial head of gastrocnemius and the soleus muscle proximally, medial to the Achilles tendon distally (Fig. 8.13). It is best identified just proximal to the Achilles myotendinous junction, deep to the medial belly of gastrocnemius, as a small oval hyperechoic structure with an internal fibrillar appearance (Fig. 8.13). The whole course of the tendon may be traced, although it is more difficult to trace proximally than distally. Plantaris should be distinguished from an accessory soleus muscle and tendon. The accessory soleus muscle is a larger more fleshy structure, usually located at the anteromedial aspect of the Achilles tendon, and its tendon inserts into the medial side of the calcaneum.

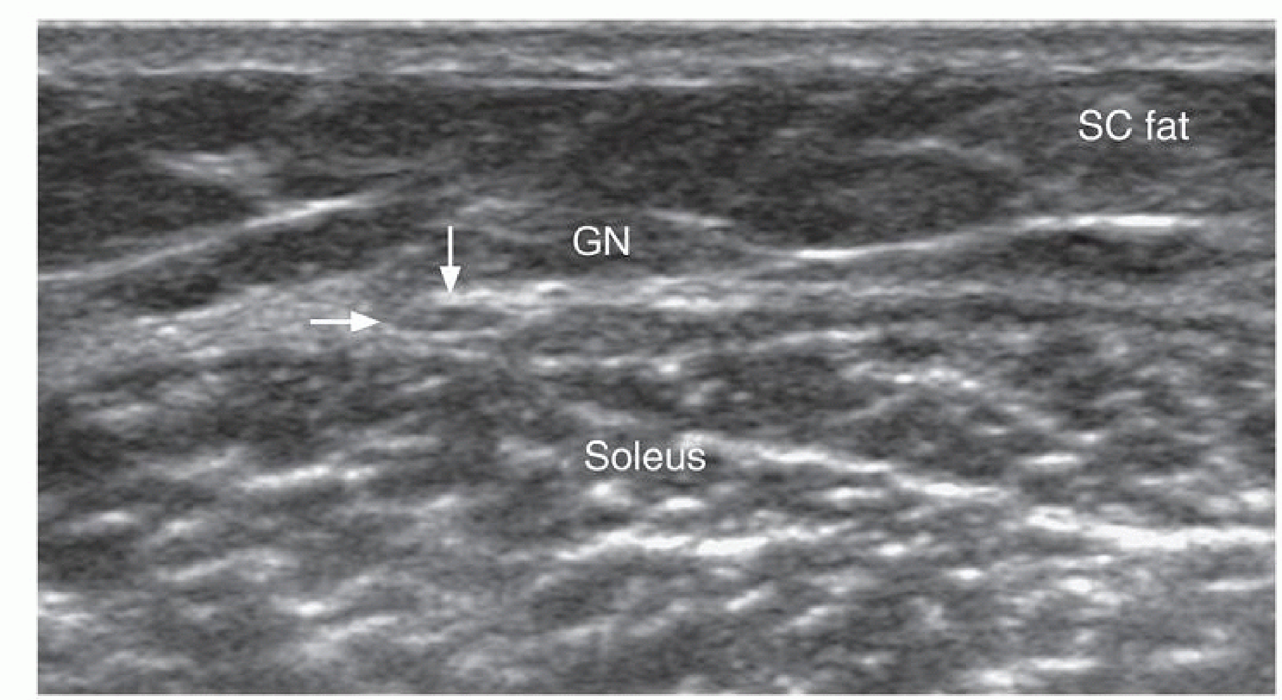

Figure 8.13. Transverse ultrasound of calf showing the ovoid-shaped plantaris tendon (arrows) between the medial belly of gastrocnemius muscle (GN) and the soleus muscle. SC fat, subcutaneous fat.

Posterior Ankle Joint

Fluid distension of the posterior recess of the ankle joint is best appreciated with the patient prone and can lead to unusual configurations such as an inverted “figure of 3” shape due to fluid bulging between the posterior tibiofibular and tibiotalar ligaments.

The posterior talocalcaneal articulation is continuous with the ankle joint in about 20% of normal subjects. The posterior talar process originates from a secondary ossification center that mineralizes between 7 and 14 years and fuses with the talar body within a year to form the lateral talar tubercle. If elongated, it is called a trigonal (Stieda) process. Failure of fusion occurs in approximately 25% of subjects, and the resulting ossicle, or os-trigonum, articulates via a synchondrosis with the talus. Ultrasound cannot usually distinguish between an os-trigonum and a Stieda process as acoustic shadowing precludes assessment of any synchondrosis.

Tip:

All the tendons crossing the ankle joint are enclosed in a tendon sheath except for the Achilles tendon (which has a paratenon) and the plantaris tendon. Only the Achilles paratenon can be seen in normal subjects on ultrasound. The synovial sheaths of the other ankle tendons cannot be seen in normal subjects.

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Foot

Plantar Foot

The plantar fascia is examined with the patient prone or supine. The plantar fascia is not strictly fascial but can be regarded as a common tendon aponeurosis for the superficial layer of the intrinsic plantar foot muscles. It is a thin structure that arises from the medial and lateral inferior calcaneal tubercles and attaches to the plantar plates of the metatarsophalangeal joints and the proximal phalangeal bases. It is a homogeneous echogenic band with internal linear interfaces on longitudinal scans, normally 2- to 3.6-mm thick near its calcaneal origin (Fig. 8.14), becoming thinner (1 to 2 mm) and more superficial distally. It comprises a thicker central cord and smaller medial and lateral cords that are not distinct entities but merge indistinguishably with each other. The plantar fascia is the main supporting structure for the longitudinal arches of the foot, connecting the posteroinferior calcaneus and the proximal phalanges.

The intertarsal joints, tarsometatarsal joints, metatarsophalangeal and interphalangeal joints, and their ligaments are assessed individually. The Lisfranc ligament extends obliquely between the lateral aspect of the medial cuneiform and the medial aspect of the second metatarsal base and is an important stabilizer of the tarsometatarsal joints, although it is not assessable by ultrasound. The normal joint space between the medial cuneiform and second metatarsal base is <1.0 mm.[32]

The sinus tarsi is an area bounded by the talus, calcaneum, talonavicular joint, and the posterior compartment of the subtalar joint. It contains the cervical and talocalcaneal interosseous ligaments, inferior extensor retinaculum, fat, and nerve endings of the deep peroneal nerve. The interosseous ligaments cannot be seen on ultrasound as they are deep, partially obscured by bone, and surrounded by adjacent echogenic sinus fat. The sinus tarsi region can be assessed by placing the probe anteroinferior to the tip of the lateral malleolus and angling toward the anterior subtalar joint.

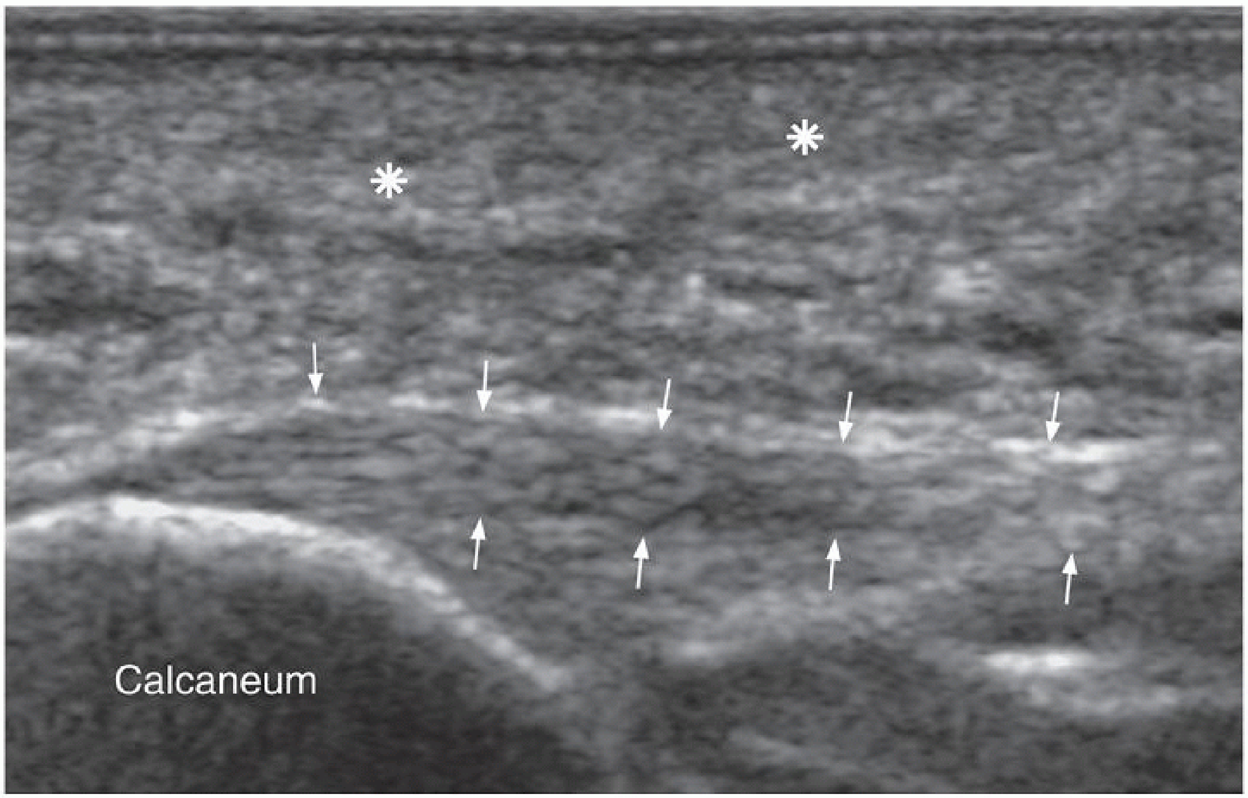

Figure 8.14. Longitudinal ultrasound showing normal plantar fascia (arrows) attached to the anteroinferior aspect of the calcaneum. As a general guide, the normal plantar fascia is <4-mm thick at the medial calcaneal tuberosity. Asterisks, heel fat pad.

Small plantar digital nerves and their accompanying vessels run between the metatarsal bones. The diameter of the normal plantar digital nerve is 1 to 2 mm at the level of the intermetatarsal heads, and the nerve can be seen on high-resolution (>10 MHz) ultrasound. The intermetatarsal bursa is normally present in each interspace, dorsal to the deep transverse intermetatarsal ligament and neurovascular bundle, and just proximal to the metatarsal head.[33] The bursa may reach ≤3 mm in asymptomatic subjects.[34]

The capsulo-ligamentous-sesamoid complex on the plantar aspect of the first metatarsophalangeal joint includes the joint capsule, collateral ligaments, fibrous plantar plate, and the flexor hallucis brevis, abductor hallucis, and adductor hallucis tendons. Flexor hallucis brevis originates on the cuboid and lateral cuneiform and splits beneath the first metatarsal into medial and lateral tendons that blend with the medial and lateral sesamoid bones just proximal to inserting into the proximal phalanx. The sesamoids are paired oval hyperechoic structures with posterior acoustic shadowing. The sesamoids are embedded in the medial and lateral tendon slips of the flexor hallucis brevis muscle and in the tendon of abductor hallucis muscle. Between the sesamoid bones runs the FHL tendon and deep to this, the thick interconnecting intersesamoid ligament. The sesamoids are bipartite in about 10% of subjects. In this situation, the summated width of the two bipartite ossicles is more than the width of the normal adjacent sesamoid allowing the distinction of bipartite sesamoid to be made.

The plantar plate is a triangular thick biconcave hyperechoic fibrocartilaginous structure on the plantar aspect of the metatarsophalangeal joints. It is firmly attached to the proximal phalanx and extends posteriorly to cover the articular cartilage on the plantar aspects of the metatarsal heads. The plantar plates are connected at the sides to the collateral ligaments of the metatarsophalangeal joints. The long and short toe flexors, invested in a synovial sheath, run just superficial to the plantar plates within a common fibrous sheath. In full-thickness tears of the plantar plate, the tendon sheath may communicate with the metatarsophalangeal joint.

Dorsal Foot

The tibialis anterior, EHL, and the four slips of the EDL tendons can be identified on the dorsal aspect of the midfoot (Fig. 8.3). The most medial tendon is the tibialis anterior tendon, which tapers distally to insert on the anteromedial aspect of the first cuneiform and the base of the first metatarsal bone. The distal portion of this tendon is more medial than dorsal and may normally show a mild longitudinal separation just prior to insertion. This
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should not be confused with a longitudinal split tear. The EHL tendon is thin and more easily appreciated by passive flexion and extension of the big toe. The four diverging slips of EDL insert onto the middle and distal phalanges of the toes. Peroneus tertius may be seen as an accessory fifth lateral slip of the EDL extending toward the base of the fifth metatarsal bone. The dorsal pedis artery and medial branch of the deep peroneal nerve both run between EHL and EDL distal to the ankle joint. The medial branch of the deep peroneal nerve lies just lateral to the dorsalis pedis artery.

The intertarsal, tarsometatarsal, metatarsophalangeal, and interphalangeal joints and corresponding ligaments are assessed individually. A small amount of fluid in the dorsal recess of the metatarsophalangeal or interphalangeal joints is normal. Extensor digitorum brevis and extensor hallucis brevis are the only muscles on the dorsal foot. They extend from the superolateral aspect of the calcaneus (just lateral to sinus tarsi) to the toes, the flat muscle belly lying deep to the EDL tendons.[34] The extensor hallucis brevis muscle is on the medial aspect of the extensor digitorum brevis muscle belly and may be indistinguishable from it. The extensor digitorum tendons insert into the lateral aspects of the EDL tendons.

Tip:

A small effusion in the dorsal recesses of the small joints of the toes is normal and should not be misinterpreted as synovitis.

ANKLE PATHOLOGY

Tendon Pathology

Anterior Tendons

The anterior tendons are the least prone of all the ankle tendons to tendinosis or tenosynovitis, and of these the tibialis anterior is the most commonly affected (Fig. 8.15). It is the strongest dorsiflexor of the ankle and also assists foot inversion. As it runs a relatively straight course within a fibrous tunnel, the mechanical demands on the tendon are low. Complete tears are rare but can result from acute injury or spontaneous rupture of a chronically degenerate tendon.[3] Acute rupture usually presents in younger patients and acute-on-chronic spontaneous rupture in elderly patients, predominantly males.[35,36] Predisposing factors include diabetes mellitus, inflammatory arthropathy, gout, and local steroid injections. The distal tendon is relatively avascular, and this, together with friction against the retinaculum, may explain why most tears occur distally. Ruptures occur at the medial cuneiform, beneath the superomedial limb of the inferior extensor retinaculum, or just distal to the superior extensor retinaculum. Ultrasound shows complete fiber discontinuity. The proximal end of the tendon usually retracts, appears moderately enlarged and hypoechoic, and is typically located deep to the superomedial limb of the inferior retinaculum. It may manifest clinically as a soft tissue mass anterior to the ankle joint. The distal end of the tendon tends not to swell or retract to the same degree and is not as well seen.

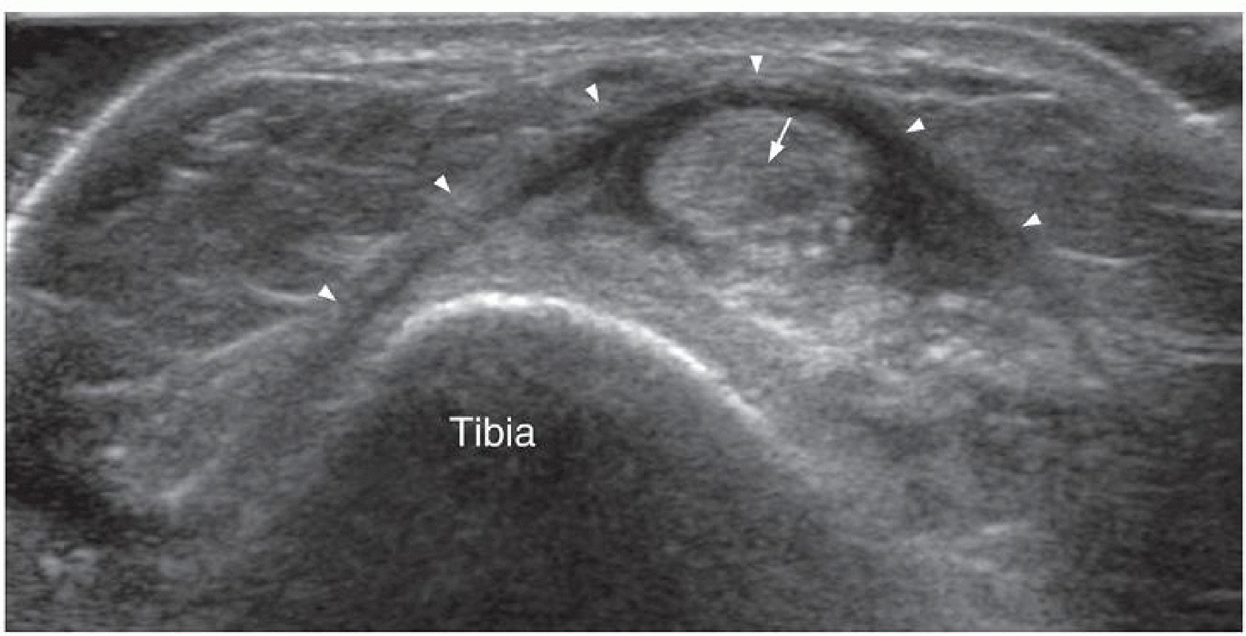

Figure 8.15. Transverse ultrasound of the ankle joint showing a severe tendinosis of the tibialis anterior tendon (arrow) with diffuse tendon thickening and a surrounding tendon sheath effusion and peritendinitis. Note the overlying thickened superior retinaculum (arrowheads).

The next most common disorder of the tibialis anterior tendon is tendinosis of the distal tendon, which results in a swollen, hypoechoic tendon.[5] Partial tears can occur on a background of tendinosis, usually located near the superomedial limb of the inferior retinaculum.[3,37] The tendon is focally swollen to a moderate degree and hypoechoic. Surface fraying or a discrete partial tear may be visible. Small partial tendon tears may be too small to detect, although swelling and hypoechogenicity are apparent.

Tenosynovitis, tears precipitated by large dorsal osteophytes, and cortical avulsion at the distal insertion are occasionally encountered in EHL and EDL.

Tip:

Tears of the tibialis anterior tendon may present as a mass anterior to the ankle joint.

Lateral Tendons

The peroneus longus and brevis tendons primarily evert the foot and stabilize the ankle. After the Achilles and posterior tibial tendons, disorders of the peroneal tendons are the next most common.

Tenosynovitis and Tendinosis

Tenosynovitis and tendinosis are the most frequent abnormalities. Tenosynovitis is primarily a disease of the tendon sheath with secondary involvement of the tendon, while tendinosis is primarily a disease of the tendon with secondary involvement of the tendon sheath. In most ankle tendons, one or other predominates, but in the peroneal tendons, a mixed picture with features of both

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tends to occur. Tenosynovitis produces distension of the tendon sheath with synovial proliferation ± fluid, peritendinous hyperemia, and a relatively unaffected tendon. Tendinosis manifests as tendon thickening, hypoechogenicity, intratendinous tears, and a variable degree of peritendinitis (Fig. 8.16). The more severe the tendinosis, the greater the likelihood of a tendon tear (Fig. 8.17). Tendons are prone to tendinosis at areas of greatest physical stress, and for the peroneal tendons this occurs at the tip of the lateral malleolus, around the peroneal tubercle and in the cuboid groove (for peroneus longus). Several anatomical variants predispose to tendinosis. A low-lying peroneus brevis muscle belly or an accessory peroneus quartus muscle can lead to stenosis within the fibro-osseous retromalleolar tunnel and stretching of the superior peroneal retinaculum. Hypertrophy of the peroneal tubercle, particularly if >5 mm in height, or a varus hindfoot alignment increase stress on the tendons.

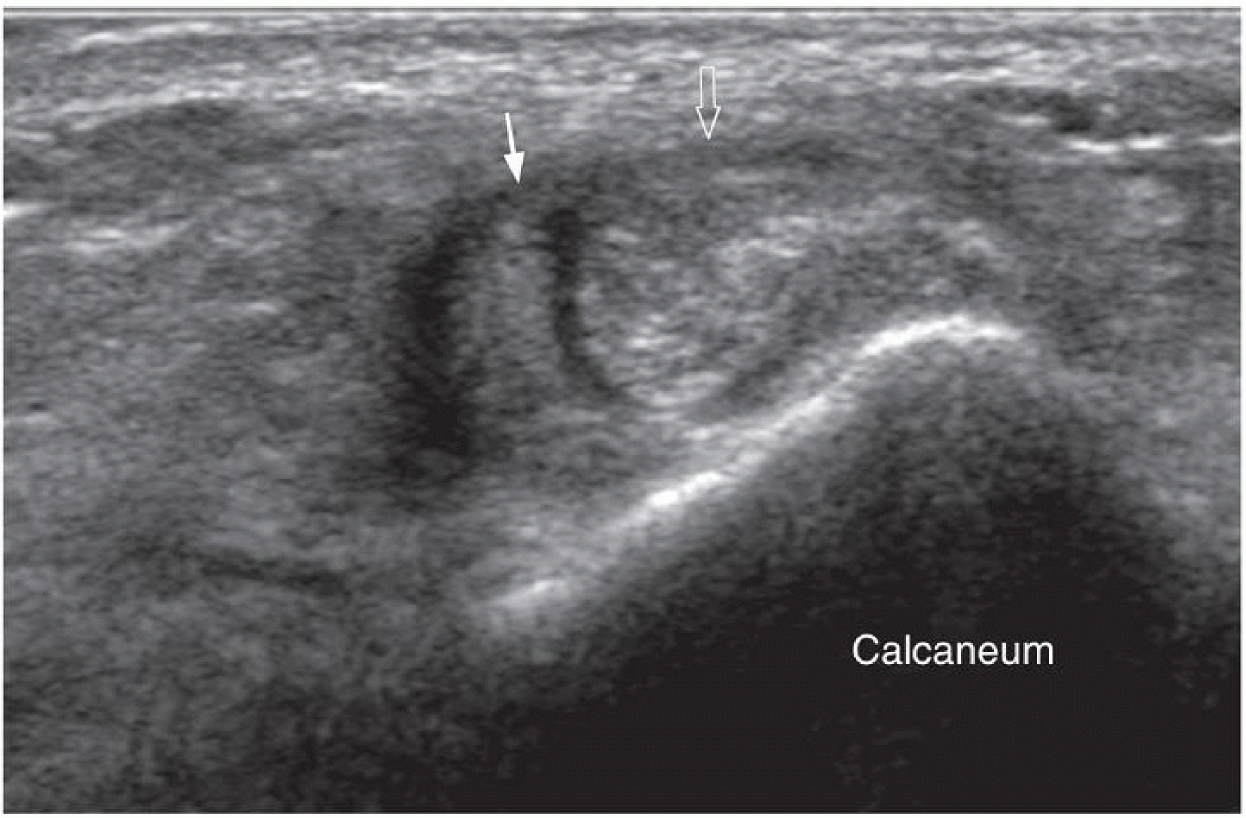

Figure 8.16. Transverse ultrasound lateral aspect of the hindfoot just distal to lateral malleolus. The peroneus longus (PL) tendon is moderately swollen, consistent with moderate-severity tendinosis (open arrow). Normally, the PL tendon is about one-third larger than the peroneus brevis tendon. The peroneus brevis tendon (closed arrow) is of normal size.

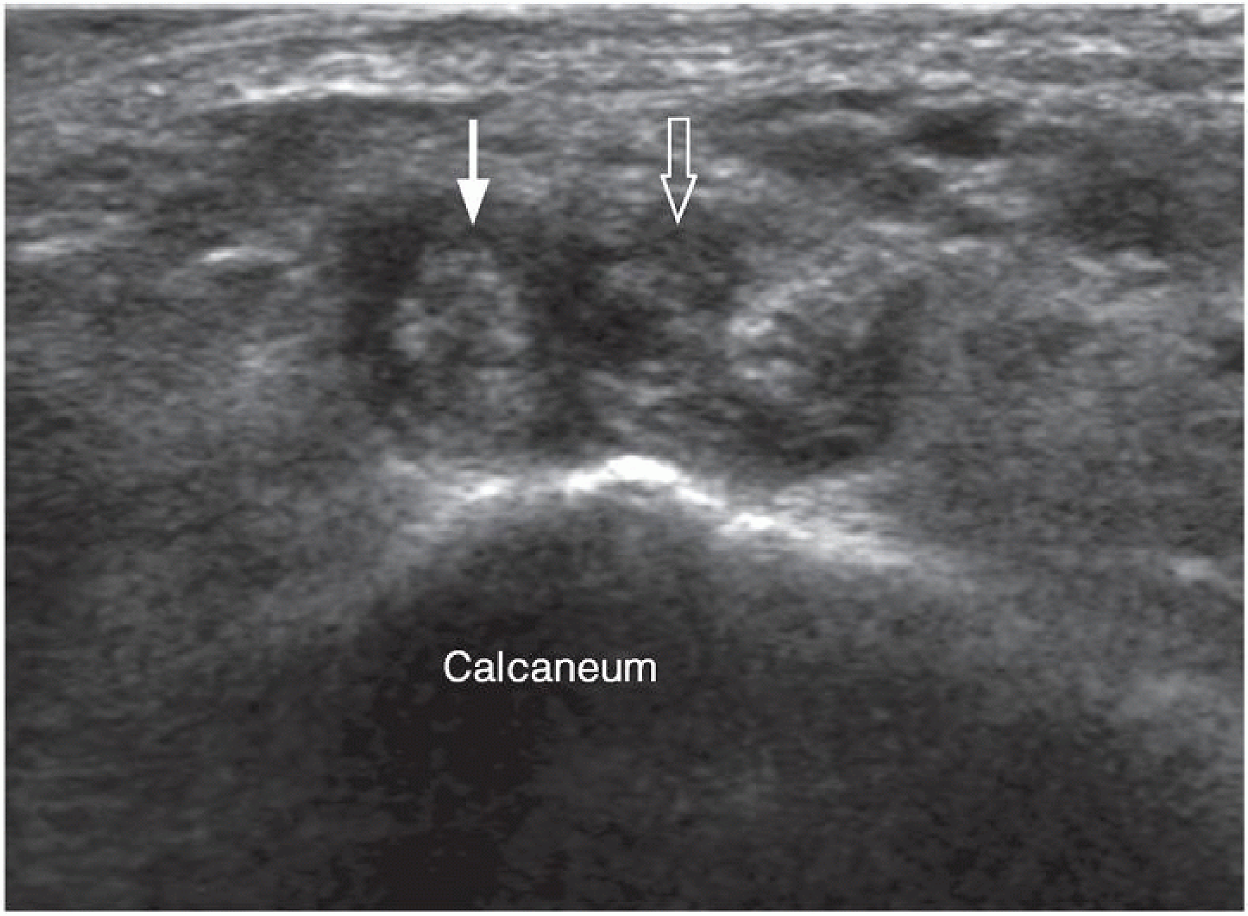

Figure 8.17. Transverse ultrasound lateral aspect of the hindfoot just distal to lateral malleolus. There is moderate to severe tendinosis of the peroneus longus (open arrow) and peroneus brevis tendons with tendon enlargement, heterogeneity, longitudinal tears (closed arrow), and irregular margin.

Peroneal Tendon Tear

Most peroneal tendon tears are longitudinal, intrasubstance partial-thickness tears, or split tears[38] (Fig. 8.17). Complete tears are uncommon and are transverse[39]. Tears of a healthy tendon may occur during acute ankle inversion. More commonly longitudinal tears, usually 2- to 5-cm long, occur in middle-aged patients on a background of tendinosis. Predisposing factors include tendon subluxation, a bony spur at the lateral malleolus, distal fibular or calcaneal fracture, or an os peroneum. Peroneus brevis tends to tear in the retromalleolar sulcus where it may be compressed by the overlying peroneus longus tendon. With more severe tears, peroneus brevis assumes a horseshoe shape on transverse scanning into which insinuates the peroneus longus tendon. Peroneus brevis may eventually split into two tendons, resulting in three apparent tendons being visible instead of the normal two in the retromalleolar region,[40,41] not to be confused with a concurrent peroneus quartus tendon, which usually inserts onto the retrotroclear eminence of the calcaneus. An effusion of the common peroneal tendon sheath is often present, and helps in visualization of the tendon tear. Dynamic examination with plantar or dorsiflexion may also accentuate any longitudinal tendon splits. Occasionally, multiple splits may occur, and the tendon looks like a horsetail at surgery.

Tip:

The peroneus brevis tendon is more prone to tear. Tears normally occur near the fibular tip and extend proximally and distally. Three rather than two tendons may be seen than peroneus longus.

Peroneus longus tends to tear near the peroneal tubercle and the cuboid groove. An os peroneum predisposes peroneus longus to tendinosis, partial splits, and complete tears, and the combination is known as os peroneum syndrome. An os peroneum is prone to fracture or stress reaction. A fracture is seen as a large gap in the ossicle with irregular margins usually associated with a full-thickness peroneus longus tendon tear.[13] The tendons and bone fragments retract ≥6 mm and have irregular margins, surrounding soft tissue swelling and tenderness on transducer pressure.[10,13] In an undisplaced os peroneum fracture or bipartite os peroneum, the gap is usually <2 mm. Stress reaction of the os peroneum and cuboid marrow edema in peroneal tendinosis are best seen on MRI.[1,10]

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Peroneal tendon tears tend to be underdiagnosed on ultrasound as the split fibers do not clearly separate (and even at surgery, tears may only be identified by probing the tendon). A high index of suspicion is needed. Partial tears are best visualized in short axis as thin hypoechoic splits in the tendon. A complete tear produces a distinct transverse gap with retraction of both ends of the tendon.

Occasionally, the torn margins remain quite closely apposed, and in this situation separation can be increased by ankle dorsiflexion and inversion.

Tip:

In general, ultrasound tends to underestimate the presence and extent of peroneal tendon tears.

Peroneal Tendon Subluxation

Subluxation of one or both peroneal tendons from the retromalleolar groove is uncommon[42,43] and results from a tear or attenuation of the superior peroneal retinaculum or, more typically, cortical avulsion or periosteal stripping of the retinaculum from its fibular attachment due to an acute inversion injury. Predisposing factors include a shallow fibular groove, congenital absence or laxity of the superior peroneal retinaculum, and crowding of the peroneal groove by a low-lying peroneus brevis muscle or an accessory peroneus quartus muscle. Ultrasound, particularly dynamic ultrasound with the foot in resisted eversion and dorsiflexion,[44] shows one or both peroneal tendons moving anteriorly and laterally over the lateral malleolus. The superior peroneal retinaculum may be thickened or torn. Sometimes, a small avulsed bony fragment (the "fleck sign") attached to the superior peroneal retinaculum can be identified. Recurrent subluxation usually leads to tendon degeneration and longitudinal splitting.[44]

In some subjects with a sensation of subluxation or a click, the retinaculum is intact and the tendons do not sublux from the retromalleolar groove, but their relationship in the groove changes. Either peroneus longus moves deep to the peroneus brevis or peroneus brevis splits longitudinally and subluxes around the peroneus longus tendon.[10] Intra-sheath subluxation shares some clinical features with true peroneal tendon subluxation such as retrofibular pain and a clicking sensation on ankle movement.[45]

Medial Tendons

Posterior Tibial Tendon Abnormality

The posterior tibial tendon plantar flexes and inverts the foot and, with the spring ligament and sinus tarsi ligaments, helps maintain the medial longitudinal plantar arch. Posterior tibial tendon dysfunction is a progressive spectrum characterized by degeneration of the tendon and acquired flat foot (pes planus). The tendon is prone to tendinosis rather than tenosynovitis. Tendinosis manifests by progressive tendon thickening, intrasubstance tears and hyperemia, and a variable degree of peritendinitis, usually involving the inframalleolar and distal tendon (Figs. 8.18A, B). Noting the degree of peritenditis is important since peritendinitis will respond to anti-inflammatory medication. The tendon should be examined in cross section from the supramalleolar region to the insertion. A cross-sectional area >20 mm^2 in the retro- and inframalleolar tendon indicates tendinosis. The tendon normally widens at its insertion, and cross-sectional measurements of the distal tendon are larger and more variable. Intratendinous vascularity on Doppler imaging

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is always abnormal.[46,47,48] Always examine an apparent longitudinal split with Doppler imaging as occasionally, a large vessel traversing the tendon may simulate a tear. Tendinosis (Fig. 8.19) is strongly associated with an accessory navicular bone (Fig. 8.20). Subclinical tendinosis is common, so seeing the same appearance on the opposite side is not always a reliable sign of normality.

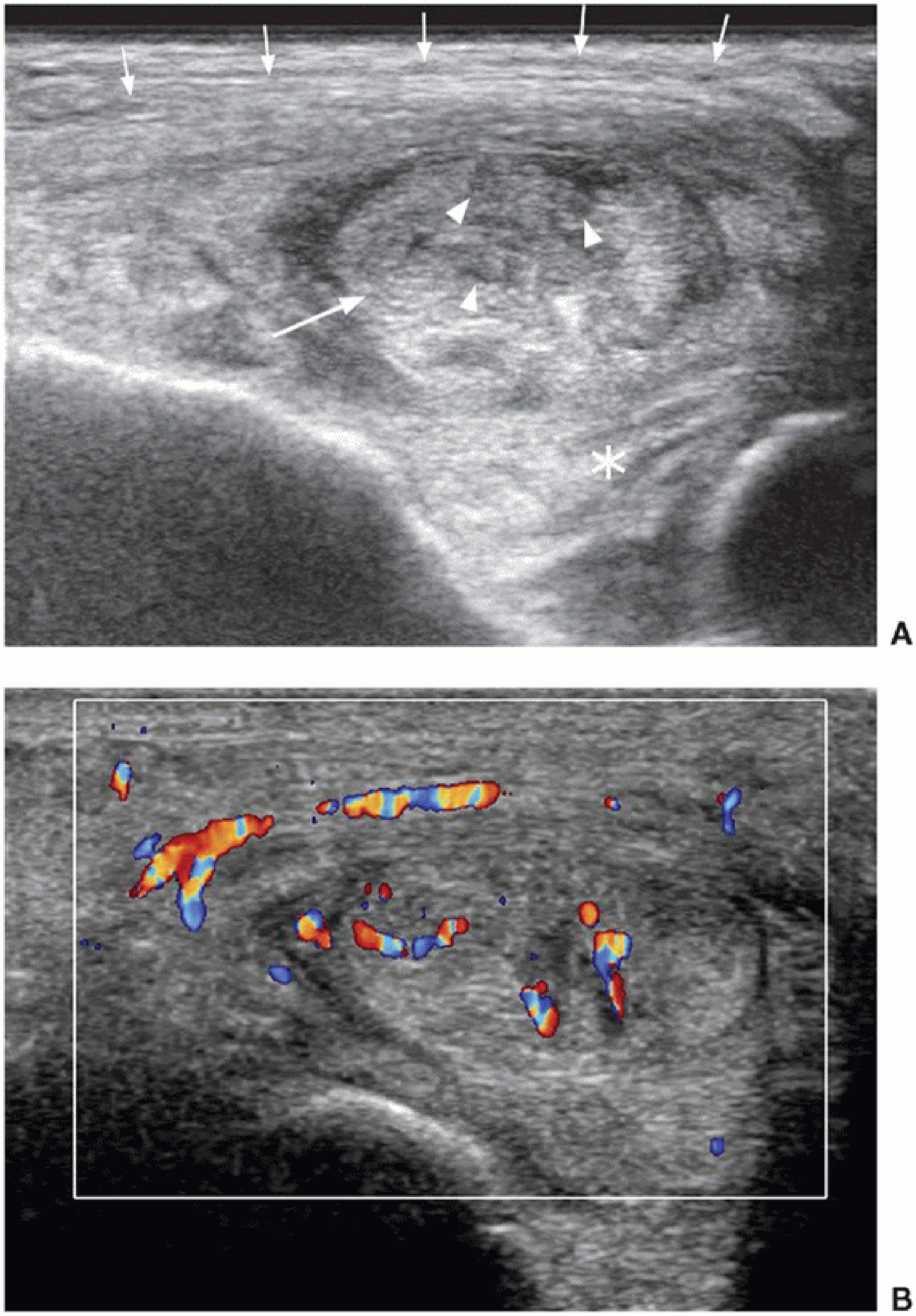

Figure 8.18. A: Transverse ultrasound of inframalleolar region showing moderate to severe tendinosis of posterior tibialis tendon (long arrow). The tendon is thickened, slightly hypoechoic with many small linear hypoechoic foci (arrowheads) representing small intrasubstance micro tears. The surface of the tendon is also irregular and frayed. There is moderate associated peritendinitis with surrounding soft tissue swelling, including the flexor retinaculum (arrows). Note the deltoid ligament (asterisk) deep to the tendon. B: Color Doppler imaging at a similar position to (A) showing mild peritendinous and intratendinous hyperemia.

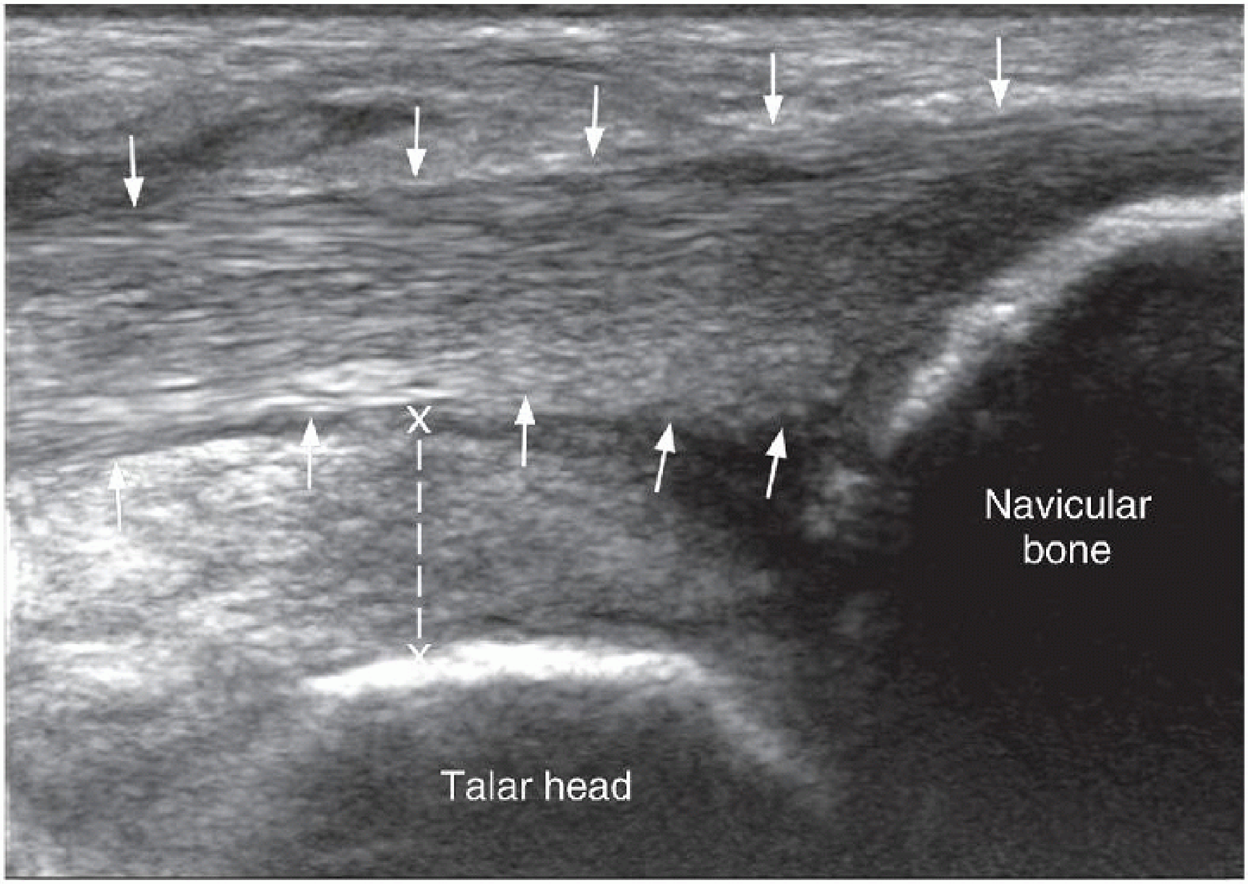

Figure 8.19. Longitudinal ultrasound medial aspect of midfoot showing mild insertional tendinosis of posterior tibialis tendon with thickening (arrows). There is also moderate thickening (dotted line) of the superomedial component calcaneonavicular (or spring) ligament deep to the posterior tibialis tendon. The ligament is 6.4-mm thick, while the normal ligament thickness should be <4.0 mm.

Complete rupture is uncommon, while short segment intrasubstance longitudinal tears are frequent.[4] Early diagnosis and treatment of posterior tibial dysfunction may prevent later disability and surgery.[46,47] An intact FDL tendon may move forward into the retromalleolar groove following posterior tibial rupture and mimic a normal posterior tibial tendon.

The vertical or superomedial component of the spring ligament complex becomes thickened with posterior tibial tendinosis/tears (in pes planus) (Fig. 8.19). The normal thickness of the superomedial portion of the spring ligament measured deep to the posterior tibialis tendon at the level of the talar neck is <4 mm. Superomedial component thickness of 4 to 5 mm is borderline, while thickness of >5 mm is generally abnormal.

Rarely, an accessory navicular may separate at the synchondrosis with the navicular, leading to retraction of the posterior tibial tendon.

Posterior Tibial Tendon Subluxation

Subluxation or dislocation of the posterior tibial tendon is due to forced dorsiflexion of the ankle with hindfoot supination and external rotation of the leg.[49] Patients may present late.[50] The flexor retinaculum tears or its medial malleolar attachment is avulsed, resulting in anteromedial dislocation of the tendon. Dislocation is readily appreciated on ultrasound, accentuated, if necessary, by ankle dorsiflexion and supination.[40,41]

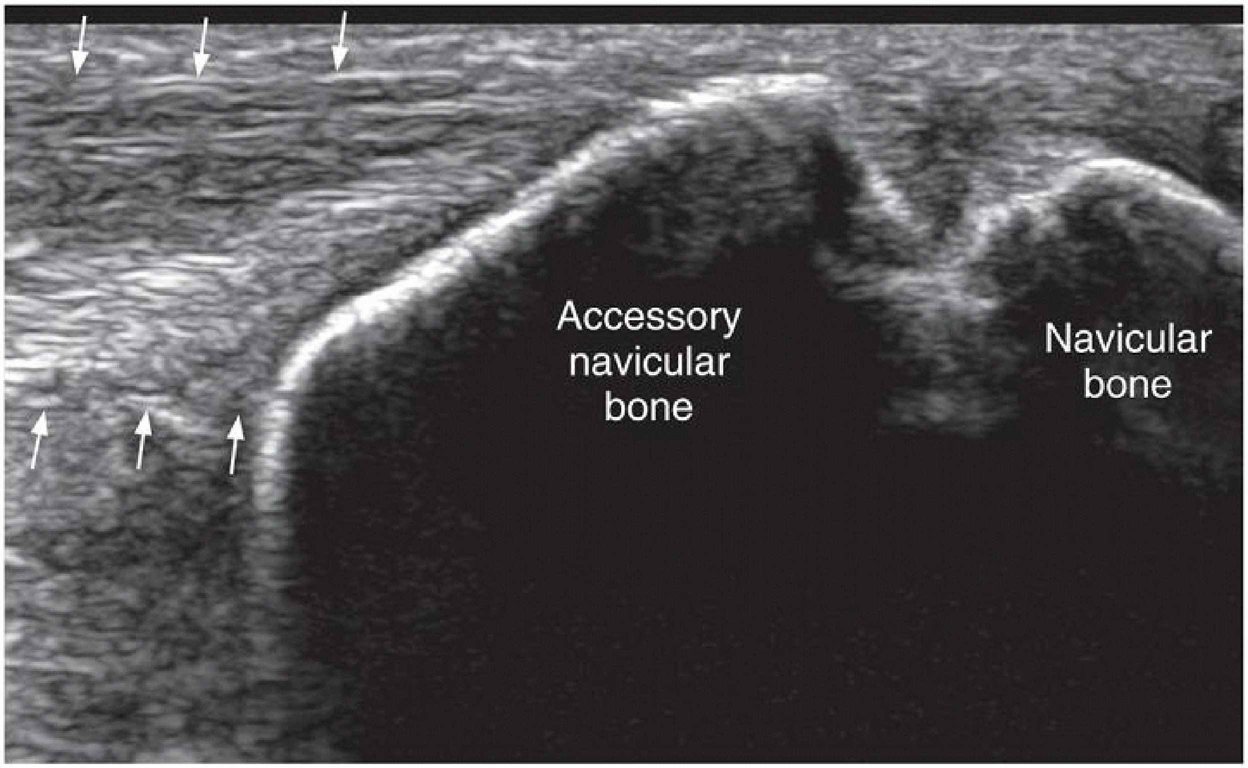

Figure 8.20. Longitudinal ultrasound medial aspect of the midfoot showing a quite large accessory navicular bone with attached posterior tibialis tendon (arrows) fibers. The junction with the main navicular bone is also shown.

Tip:

Subclinical tendinosis of the posterior tibial tendon, is common so you cannot necessarily rely on the contralateral side as a normal standard.

Posterior Tendons

Plantaris Tendon Pathology

The plantaris muscle is a weak flexor of the knee and ankle. Its tendon is thin and prone to acute tear, which is typically complete.[51] Isolated tears of the plantaris tendon are relatively uncommon. Tears usually occur in the mid-calf, and with tears of the medial head of gastrocnemius, constitute "tennis leg," which usually affects middle-aged "weekend warriors" who present with acute calf pain and swelling. More distal plantaris tendon tears are usually associated with Achilles tendon tears.

Plantaris tears present with calf pain and may mimic deep vein thrombosis, ruptured Baker cyst, Achilles tendon tear, or medial belly of gastrocnemius tear.[30,31,51,52] Hemorrhagic and serous fluid around the tear can be mistaken for a myofascial tear of gastrocnemius or a leaking Baker cyst as the fluid tracks between gastrocnemius and soleus, but these can be excluded by recognition of the normal half-arrowhead configuration of the distal medial belly of gastrocnemius and the absence of a Baker cyst, or a crisply defined inferior margin to the cyst if a Baker cyst is present.[51,52] The injured tendon is typically swollen and hypoechoic. The proximal end retracts, thickens, and has irregular margins. There is usually a thin hematoma in the gap and around the ends of the tendon.[52] In chronic rupture, the gap is filled by hypoechoic granulation tissue or more mature fibrous tissue.

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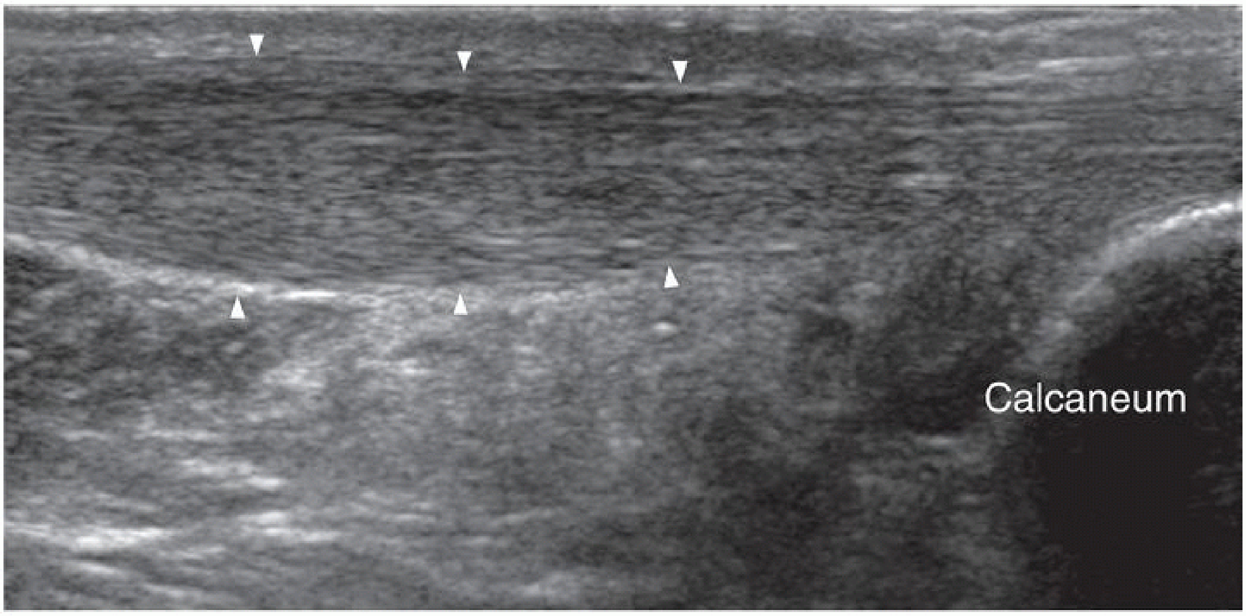

Figure 8.21. Longitudinal ultrasound showing moderate mid-portion Achilles tendinosis with moderate fusiform swelling and slight hypoechogenicity of the mid-third of the Achilles tendon (arrowheads). Color Doppler imaging (not shown) revealed mild intratendinous hyperemia. The distal one-third of the Achilles tendon is more normal in caliber and echo-texture.

Achilles Tendon Pathology

The Achilles tendon is the ankle tendon most prone to degeneration or tear. Pathology ranges from mild peritenonitis to a full-thickness tear.[53] Tendinosis, the most common Achilles tendon disorder, is characterized by pain and swelling, usually in mid-tendon (Fig. 8.21) or near the calcaneal insertion (insertional tendinosis) (Fig. 8.22). The incidence of Achilles tendinosis is rising as a result of greater participation in sports such as running, racquet sports, track and field, basketball, volleyball, and soccer.[54] Runners are about 10 times more likely than age-matched controls to develop Achilles tendinosis, and elite runners have a lifetime prevalence of up to 10%.[55] About one-third of subjects are nonathletes.[54] Histologically, tendinosis has features of a failed healing response with haphazard tenocyte proliferation, tenocyte degeneration, disruption of collagen fibers, and increase in proteoglycan matrix.[54] Tendon vascularity is typically increased. Vessels are arranged haphazardly and are generally perpendicular to the collagen fibers. Inflammatory lesions and granulation tissue are infrequent and, when present, are usually associated with partial tears. Vascular proliferation with an inflammatory infiltrate is common in the paratenon.[56] Tendinosis is characterized by progressive tendon thickening, hypoechogenicity with loss of the normal fibrillar pattern, and increase in tendon vascularity (Figs. 8.21 and 8.22). There may be a variable degree of paratenonitis manifested by swelling, edema, increased echogenicity, and hyperemia of the Achilles paratenon. Depending on the conspicuity of these features, the severity of Achilles tendinosis and paratenonitis can be graded as mild, moderate, or severe.

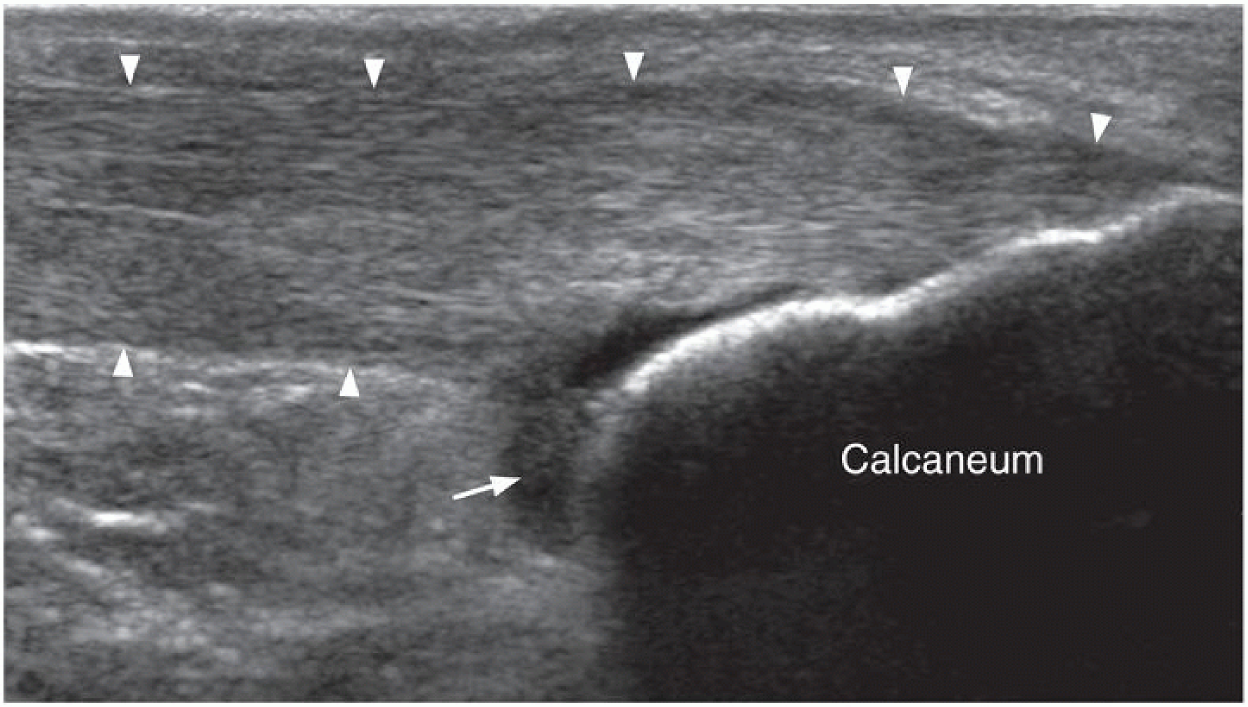

Figure 8.22. Longitudinal ultrasound of posterior aspect of ankle showing diffusely thickened distal Achilles tendon (arrowheads) consistent with moderate-severity tendinosis. There is a mildly distended retrocalcaneal bursa (arrow). In this patient, no cortical irregularity of the calcaneal insertional area is present.

The size of the Achilles tendon varies considerably,[28] and diagnosis should be based on an impression of overall tendon size and echotexture rather than relying on size measurements alone. Cross-sectional area can be compared with the opposite side or used to provide a baseline for longitudinal studies and is best measured at: (1) the soleus musculotendinous junction, (2) the mid-portion of the tendon, and (3) the calcaneal insertion. The normal cross-sectional areas are at level 1: 0.54 ± 0.11 cm^2; at level 2: 0.54 ± 0.12 cm^2;28 and at level 3: 0.72 ± 0.15 cm^2.

The severity of tendinosis and paratenonitis do not necessarily parallel each other; for example, there may be a severe degree of tendinosis but only a mild degree of paratenonitis, or there may be paratenonitis with no evidence of tendinosis (Figs. 23A, B). The distinction is clinically relevant as active paratenonitis responds to anti-inflammatory medication, whereas tendinosis does not.[57] Tendon calcification, fluid in the retrocalcaneal bursa, and bony contour abnormalities at the calcaneal insertional are seen in 2%, 35%, and 63%, respectively, of normal asymptomatic subjects, although they occur with greater frequency in subjects with insertional Achilles tendinosis[28] (Fig. 8.24). Tendinosis usually leads to diffuse or fusiform swelling of the tendon, although occasionally focal nodular expansion occurs due to localized accumulation of proteoglycans, partial tendon tears, or focal reparative fibrosis.[53] Partial tears are seen as focal discontinuity of the fibrillar echogenic fibers on a background of tendinosis. Small partial tears are an inherent part of any moderate to severe tendinosis, and ultrasound is not able to distinguish them from background tendinopathic changes. Tendon vascularity seems to be associated more with duration and chronicity of physical activity and severity of tendinosis than pain.[57]

Tip:

The degree of paratenonitis as well as the degree of Achilles tendinosis should be assessed independently.

Asymptomatic pre-existing tendinosis is often present in Achilles tendons that rupture.[58] Tears typically affect middle-aged men, often overweight and engaged in physical activity above their level of fitness.[59] The tear

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is usually 2 to 6 cm from the calcaneal insertion where the tendon is relatively avascular.[28] It is important to differentiate full-thickness from partial-thickness tears as full-thickness tears may benefit from surgical repair. Distinguishing mild-to-moderate partial tears from tendinosis is less important as both are treated conservatively. A complete tear is seen as discontinuity in the tendon, with distraction and posterior acoustic shadowing of the tear edges[60] (Fig. 8.25). The tear should be evaluated comprehensively by moving the transducer from medial to lateral and superior to inferior to avoid misdiagnosis of partial tear as complete tear. In acute tears, a mixture of blood products, debris, and herniated fat leads to variable echogenicity in the gap. An intact plantaris tendon may run across the medial aspect of the tear (Fig. 8.26). Dynamic ultrasound examination is valuable. Dorsiflexion of the foot may accentuate a tear by separating the tear edges and creating or widening the gap between them. Normally the tendon moves as one unit on plantar/dorsiflexion, but with a complete tear paradoxical movement may be seen: The distal segment of the

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tendon moves normally and inferiorly and the proximal tendon is retracted superiorly on dorsiflexion, whereas on plantar flexion, the two segments move toward each other. Plantar flexion can also be used to assess whether conservative management in an equinus cast is appropriate: The edges of the tendon should become apposed. Most surgeons will operate if the gap is ≥1 cm. Marking the site of the tear on the skin is helpful for surgical planning (Fig. 8.27). In chronic or missed Achilles tendon tears, isoechoic or hypoechoic granulation or fibrous tissue may fill or partially fill the gap (Fig. 8.28). Injuries at the insertion are normally avulsive, and the tendon remains attached to an avulsed bone fragment (Fig. 8.29).

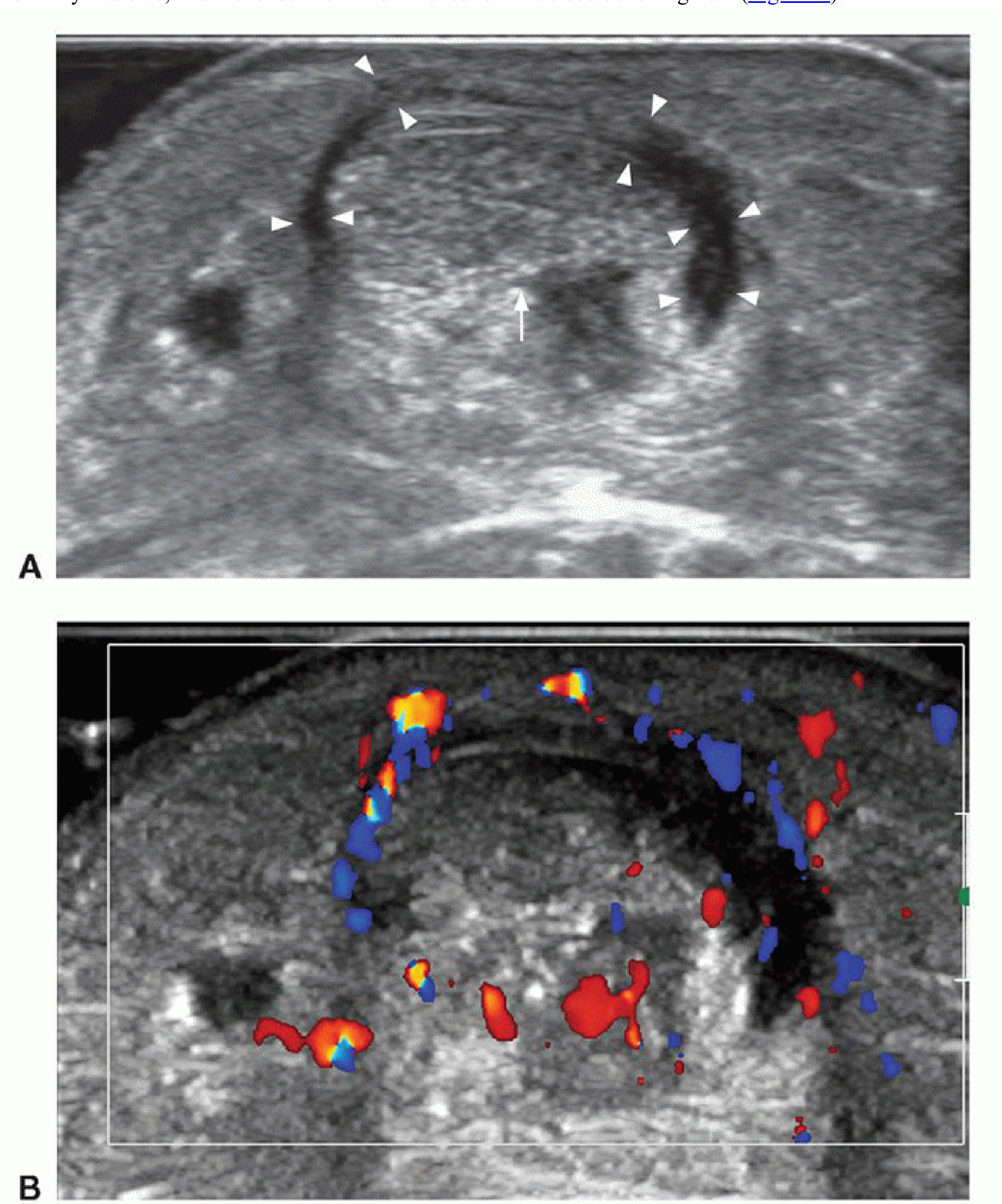

Figure 8.23. Transverse ultrasound of Achilles tendon (arrow) showing (A) a hypoechoic rim (arrowheads) around the sides and dorsal surface of the Achilles tendon due to a thickened paratenon. The Achilles tendon is normal. B: Color Doppler imaging shows a marked hyperemia around and, to a lesser degree, within this distended paratenon overall consistent with chronic paratenonitis.

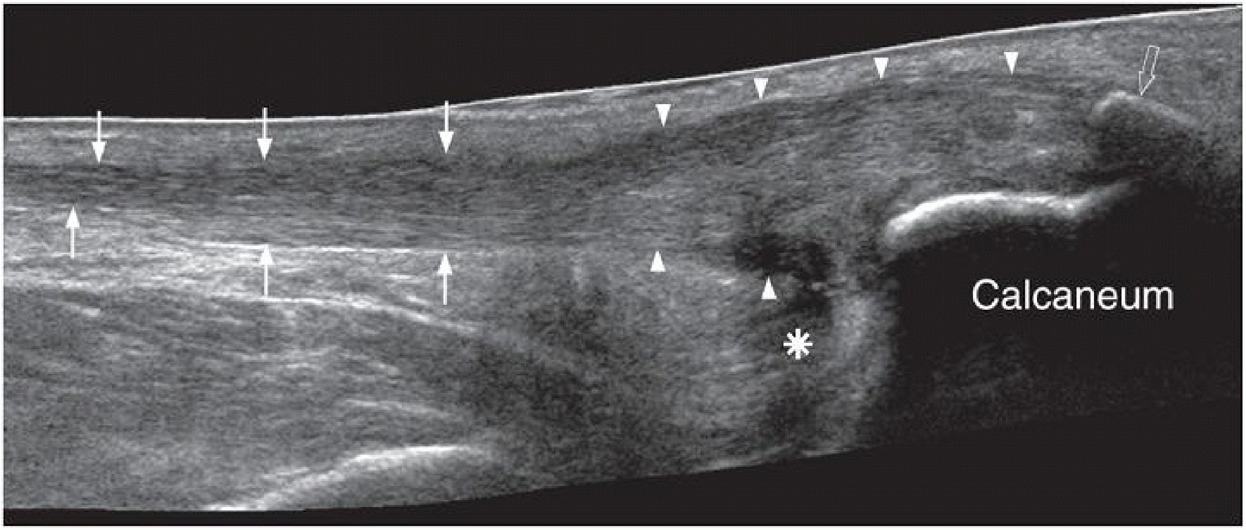

Figure 8.24. Longitudinal extended-field-of-view ultrasound showing severe insertional Achilles tendinosis. The distal one-third of the Achilles tendon (arrowheads) is thickened and hypoechoic with disruption of the normal fibrillar pattern. Color Doppler imaging (not shown) revealed mild peri- and intratendinous hyperemia. The tendon gradually tapers to a more normal caliber in the mid- to proximal one-third (arrows). Note mild distension of the retrocalcaneal bursa (asterisk) and a large posterior plantar calcaneal spur (open arrow).

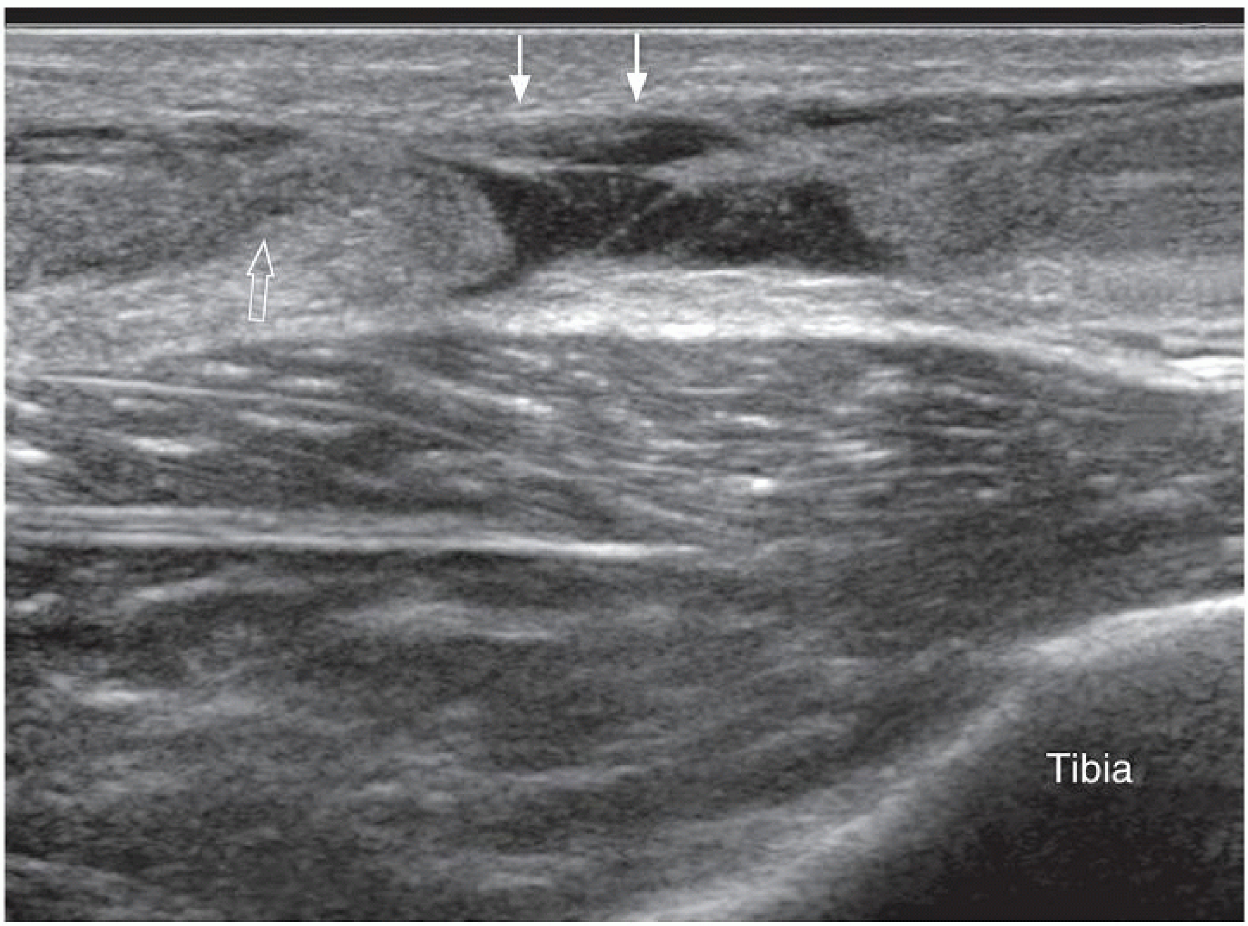

Figure 8.25. Longitudinal ultrasound showing complete Achilles tendon tear (arrows) just distal to the musculotendinous junction (open arrow). The tendon gap is filled with fluid.

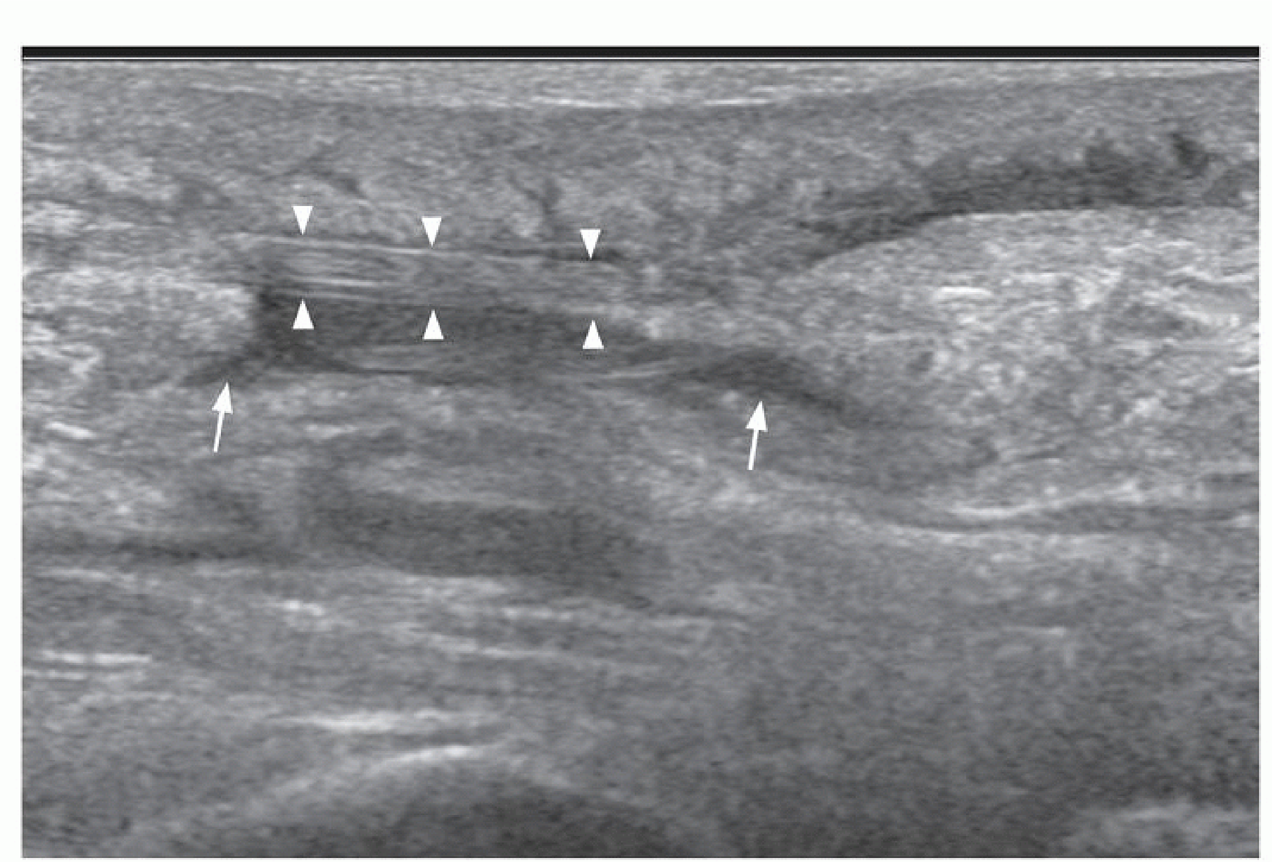

Figure 8.26. Longitudinal ultrasound of complete Achilles tendon tear, medial edge. The retracted ends of the tendon are shown (arrows) with the tendon gap filled with fluid. There is also an intact slightly swollen plantaris tendon (arrowheads) traversing the tendon gap. Do not confuse this with intact Achilles tendon fibers.

Figure 8.27. Clinical image showing how the edges of the Achilles tendon tear are marked on the skin. The rounded depressions (arrows) on the skin were made with the top of the marker pen prior to the ultrasound gel being removed for ink marking of the tendon rupture site.

Tip:

An intact plantaris tendon can mimic residual intact fibers of the Achilles tendon in complete Achilles tendon tear.

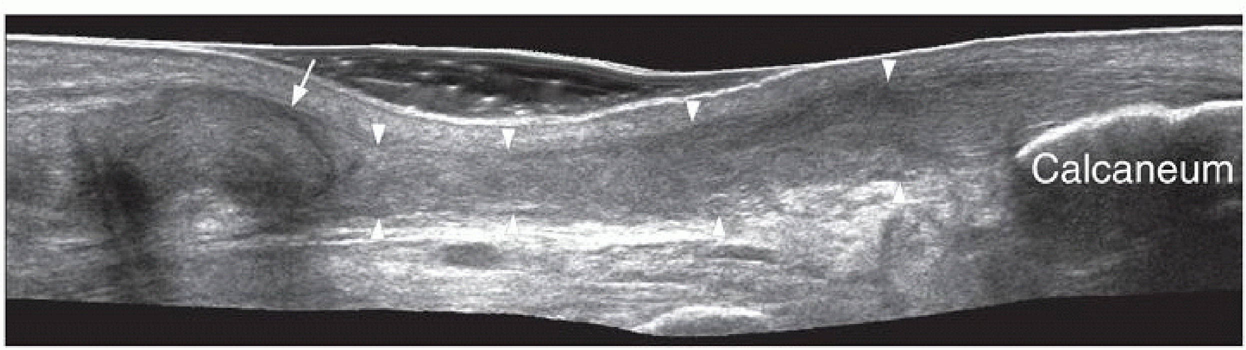

Figure 8.28. Longitudinal extended-field-of-view ultrasound of partial chronic Achilles tendon tear. The Achilles tendon (arrowheads) is elongated, although still continuous with a retracted tendon mass (arrow) close to the musculotendinous junction.

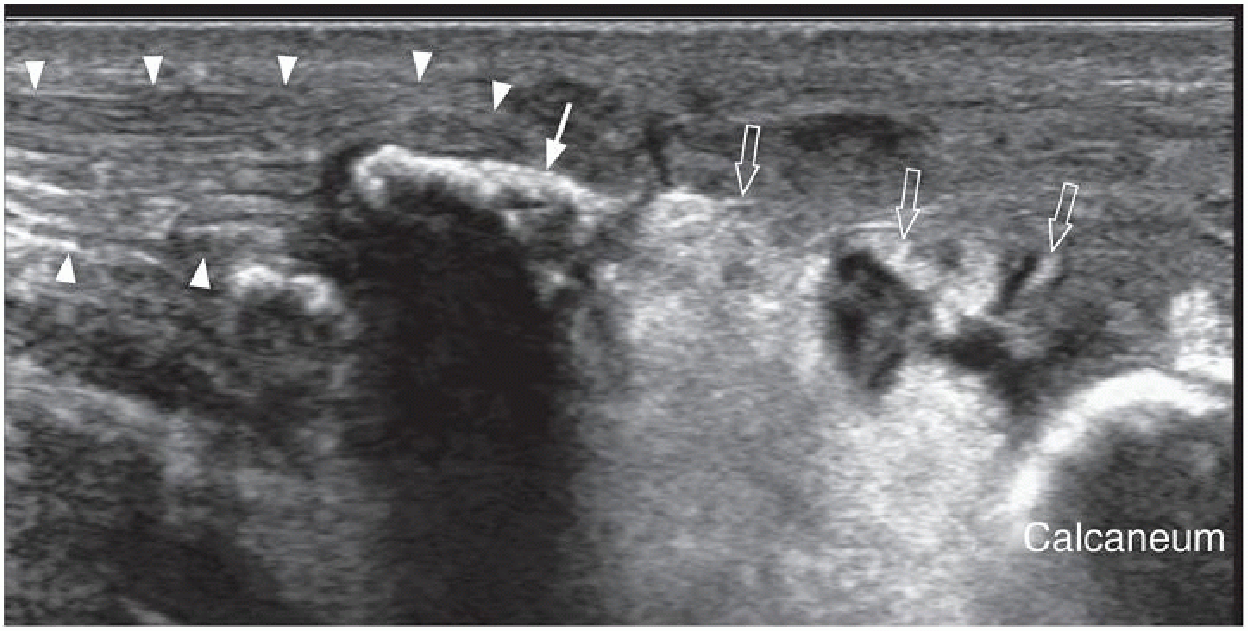

Figure 8.29. Longitudinal ultrasound showing Achilles fracture avulsion from calcaneal insertion. The Achilles tendon (arrowheads) is attached to a large fragment of bone (closed arrow) avulsed from the calcaneum. The intervening gap is filled with echogenic fat and hematoma (open arrows).

Ultrasound can assess Achilles tendon repairs. The postoperative tendon is normally moderately to severely diffusely swollen with an irregular contour (Fig. 8.30). The swelling increases postoperatively for the first year and decreases thereafter.[59] The site of repair varies from a hypoechoic to a mixed hypoechoic/hyperechoic appearance.[53] Large sutures such as a Krachow whip stitch can also be seen. The tendon ends should be closely apposed and reparative granulation tissue fills any gap. Occasionally, calcification occurs at the site of repair. In re-rupture, the repaired ends separate and sutures traverse the gap.[59]

In patients with familial hypercholesterolemia, the Achilles tendon can be diffusely swollen to a severe degree due to cholesterol deposition, and this can appear very similar to Achilles tendinosis[61],[62] (Fig. 8.31). Less frequently, focal deposition of cholesterol results in discrete intratendinous xanthomas (Fig. 8.31). Recognizing a family history of hypercholesterolemia, the cutaneous stigmata of hypercholesterolemia, or the involvement of other tendons, particularly the extensor tendons of the digits and, less frequently, the patellar tendons, is helpful.

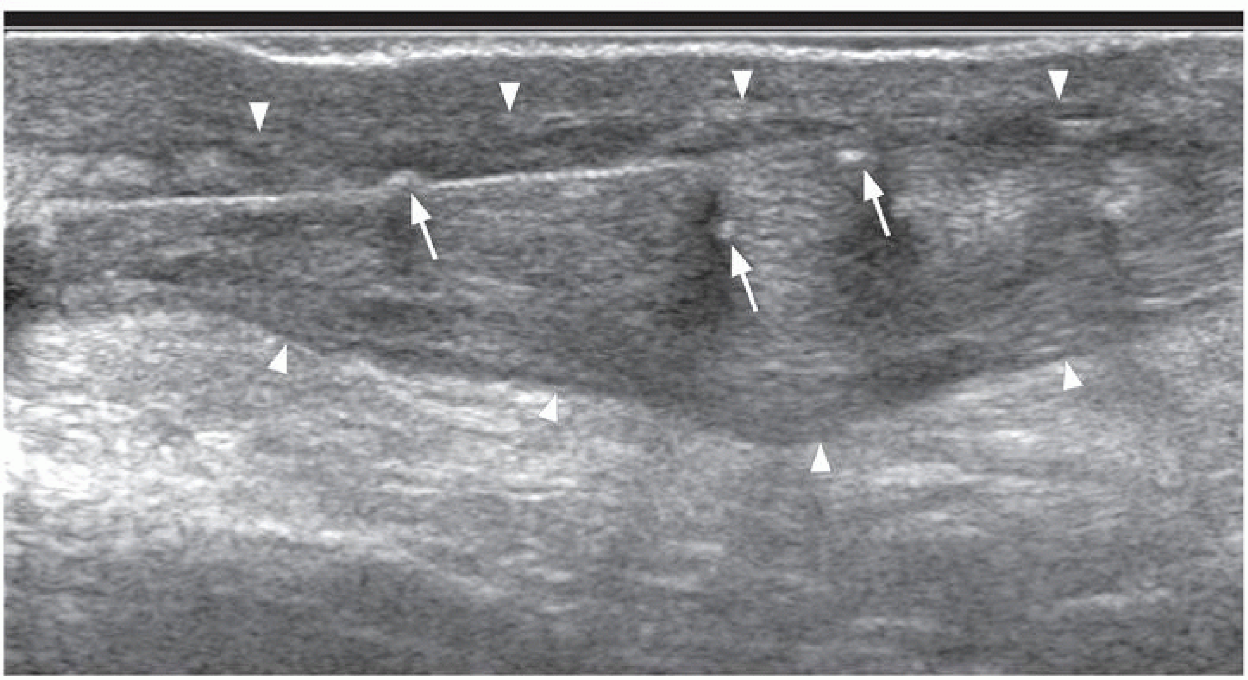

Figure 8.30. Postoperative appearances of Achilles tendon. The appearances vary according to the site and type of repair performed, although typically the tendon (arrowheads) remains thickened with clearly recognizable suture material (arrows). No re-tear is present.

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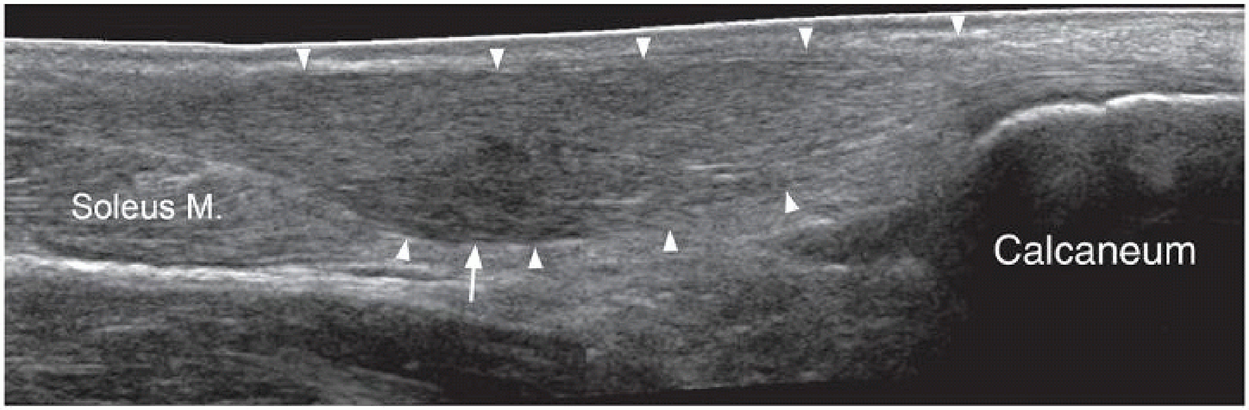

Figure 8.31. Longitudinal extended-field-of-view ultrasound showing diffusely thickened Achilles tendon (arrowheads) in patient with familial hypercholesterolemia. The tendon thickening is due to deposition of cholesterol-rich material between the collagen fibers. There is a more discrete hypoechoic area of cholesterol deposition on the deeper aspect of the mid-tendon, which could be termed a “xanthoma” (arrow).

Haglund syndrome

The Haglund syndrome is a constellation of bony and soft tissue abnormalities[63,64,65] characterized by a bony protuberance at the posterosuperior border of the calcaneus (Haglund deformity), Achilles tendinosis, and retrocalcaneal bursitis. The Achilles tendinosis and retrocalcaneal bursitis can be secondary to the bony deformity or occur together primarily. Ultrasound shows the bony prominence, abnormal thickening/heterogeneity of the tendon and distension, wall thickening, internal debris, or hyperemia of the bursa. Ultrasound-guided injection of local anesthetic and steroid into the bursa is both diagnostic and therapeutic. A short-axis approach and injection of 20 to 40 mg of methylprednisolone and 0.5 to 1 mL of 0.5% bupivacaine are appropriate. Pain relief is of variable duration. Recently, it has been proposed that the use of Haglund deformity or Haglund syndrome is inappropriate as the bony prominence is just as common in asymptomatic subjects,[66] and terminology such as Achilles tendinosis or tendinopathy, paratenonitis or retrocalcaneal bursitis is preferred.

Achilles tendon injection

Ultrasound-guided percutaneous interventions are used for Achilles tendinosis and at multiple other sites in the foot and ankle.[45,67,68,69,70,71,72,73,74,75,76,77,78,79] Ultrasound-guided treatment ensures accurate needle placement, avoids nerves and vessels,[45,67,68,69,70,71,72,73,74,75,76,77,78,79] and is employed when conservative methods have failed.[72] Treatments can be broadly divided into those designed to (1) obliterate tendon and paratenon neovascularity such as sclerotherapy, electrocoagulation, or high-volume saline injection,(2) stimulate a healing response such as dry needling, autologous blood injection, and hyperosmolar dextrose injection, or (3) control local inflammation, that is, anesthetic/steroid injection.

Neovascularity (and accompanying nociceptive fiber formation) is thought to be related to pain; hence methods such as sclerotherapy, electrocoagulation, and high-volume saline injection have been developed to reduce tendon and paratenon vascularity.[72] Sclerotherapy by ultrasound-guided injection of phenol or polidocanol into peritendinous (not tendon) vessels results in reduced vascularity. There is some evidence of clinical benefit,[73] although multiple treatments may be needed.

Electrocoagulation requires specialized equipment and is performed under local anesthesia. The electrocoagulation needle is placed against vessels entering the tendon under ultrasound guidance. Potential complications include infection, sural nerve damage, and tendon rupture. Brisement is high-volume injection (20 to 40 mL) of normal saline and local anesthetic between the Achilles tendon and the paratenon to strip the paratenon and break any adhesions.[72] A short-axis approach is best. High-volume injection of saline, local anesthetic, and steroid between the Achilles tendon and Kager fat pad[80] has been reported to improve pain scores, function, and symptoms.[74]

Dry needling by repeated fenestrations of the area of tendinosis precipitates intratendinous bleeding and is thought to stimulate healing and inflammation via deposition of blood-borne stem cells and inflammatory mediators. Autologous blood or platelet-rich plasma (PRP) injections contain various growth factors that are thought to promote healing. About 3 mL of PRP or autologous blood are injected into the area of tendinosis.

Prolotherapy (or regenerative injection therapy) involves ultrasound-guided injection of a small volume of irritant such as hyperosmolar dextrose in or around the tendon insertion to initiate a local inflammatory response.

Steroid injections remain the most frequently used percutaneous treatment of Achilles tendinosis, usually administered blindly, although ultrasound guidance can ensure accurate needle placement. Generally, peritendinous injections of 30 to 40 mg of methylprednisolone or triamcinolone mixed with lidocaine and/or bupivacaine are performed. Favored positions include deep to the paratenon adjacent to tendinosis or into the deep retrocalcaneal bursa. A short-axis approach is appropriate for both. Peritendinous injections often provide good short-term relief, but relapse, especially with vigorous rehabilitation, and soft tissue atrophy are common. Intratendinous injections are usually avoided because of the potential for tendon rupture, although good results have been reported for intratendinous injection of 40 mg of methylprednisolone.[72]

Ligament Pathology

The ultrasound appearances of ligament injury are dependent on the age and severity of injury. In acute partial tears, the ligament is swollen with an anechoic or hypoechoic zone due to edema or hematoma and disorganization of the normal internal echogenic fibrillar pattern. In complete tears, there is swelling and discontinuity of the ligament, sometimes with heterogenous

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hematoma filling the gap (Fig. 8.32). Avulsion of the bony insertion may be seen. There may be a hyeprechoic rim of quite intense edema surrounding any ligament tear with swelling of the adjacent soft tissues. As a tear heals, the gap fills in and becomes hyperechoic and heterogeneous, and the ligament appears thicker. Thickening continues for a long time after injury (Fig. 8.33). There is sometimes elongation of the ligament. Ligament continuity is usually eventually restored even after complete tears. Less frequently, the retracted torn ends undergo attrition and become reabsorbed, or the ligament heals in an attenuated fashion. Calcification or ossification rarely occur.[12] Stressing the ligament may be helpful when assessing acute or chronic ligament tears.

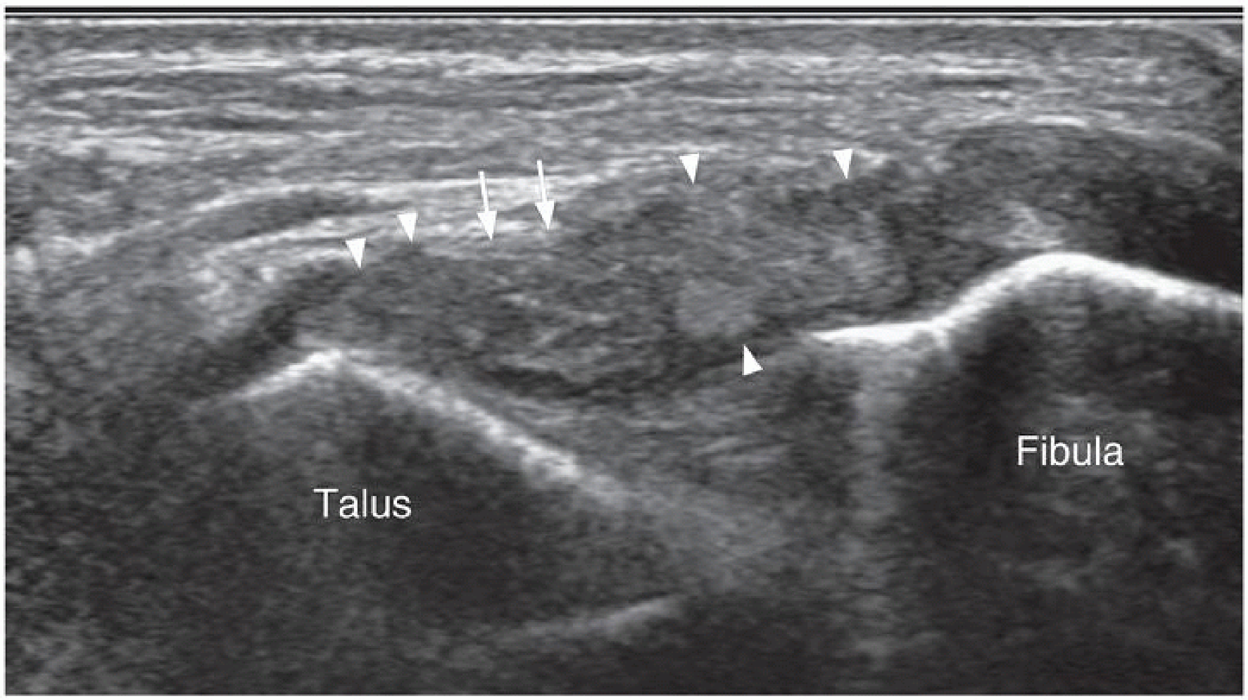

Figure 8.32. Longitudinal ultrasound lateral aspect of ankle showing complete tear of ATFL. The ligament (arrowheads) is very swollen with a complete tear (arrows) close to the talar attachment.

Lateral Collateral Ligament Injury

The lateral collateral ligaments are the most commonly injured ankle ligaments, the ATFL most frequently followed by the CFL, and rarely the posterior talofibular ligaments. They are injured during ankle inversion. Isolated ATFL injuries comprise up to 70% of ankle injuries. 1 Ultrasound is not usually indicated as the accuracy of clinical examination is high. Ligament tear can be divided into three types based on the severity of injury. A “sprain” is when the ligament is swollen indicative of ligament micro tear without visible ligament disruption. A partial-thickness tear is when a discrete tear presents in conjunction with some residual intact fibers. In full-thickness tears, the ligament substance is either completely torn or the ligament is completely avulsed from either its fibular or talar attachment, often with a thin slither of bone. In full-thickness tears, there is usually capsular rupture with leakage of synovial fluid into the soft tissues.[40],[41] Anteriorly drawing the inverted and slightly plantar-flexed foot forward over the edge of the examination bed (the anterior drawer test) while scanning longitudinally over the ATFL can help distinguish between partial and complete tear of the ATFL.

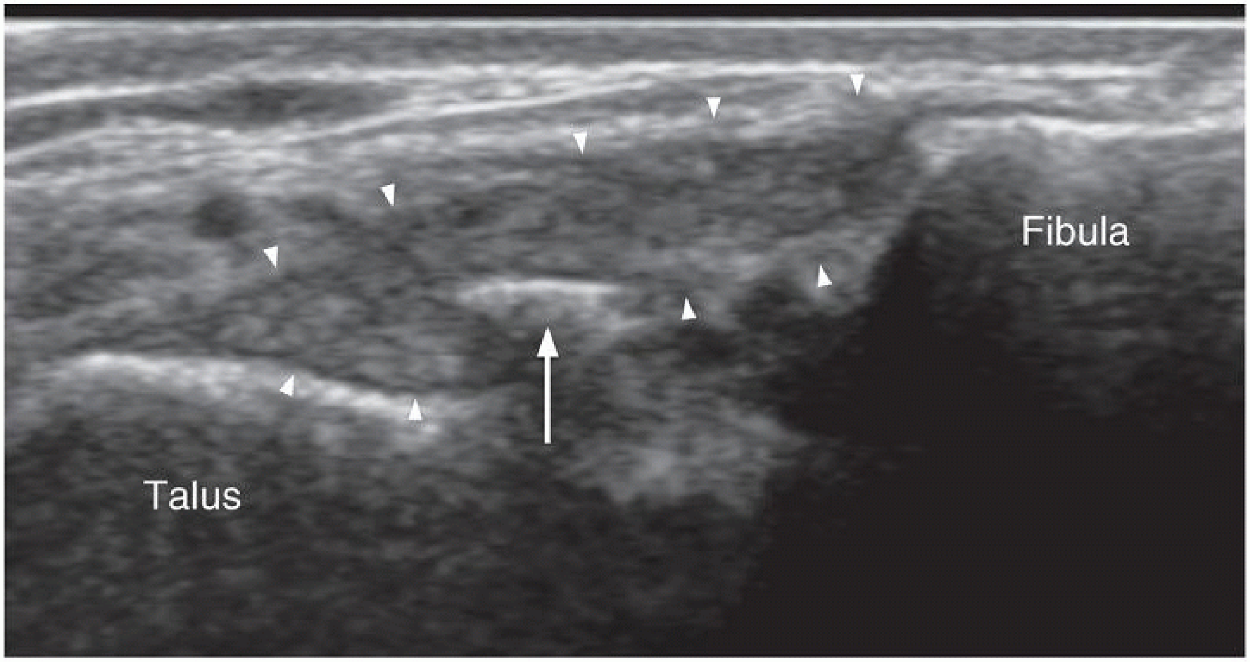

Figure 8.33. Longitudinal ultrasound anterolateral aspect of ankle. There is a healing tear of the ATFL with moderate ligament thickening (arrowheads) and a small avulsion fracture (arrow) most likely from the fibular tip. Overall, ligament continuity is maintained.

Tip:

Performing the anterior drawer test during ultrasound scanning can help differentiate partial and complete ATFL tears.

With healing of complete ATFL tears, ligament continuity can be spontaneously restored with the ligament being thickened, continuous, and functionally competent. This ligament thickening can remain for years. If ligament continuity is not restored, the free ligament will usually undergo progressive attrition and resorption.

The CFL nearly always tears in conjunction with an ATFL tear. In acute severe tears of the CFL, there is usually fluid distension of the common peroneal tendon sheath, and there may be free communication between the ankle joint and the peroneal tendon sheath.[40,41] During dorsiflexion, the normal CFL elevates the peroneal tendons toward the probe, while in complete CFL tears, the peroneal tendons remain close to the calcaneus (Fig. 8.34).

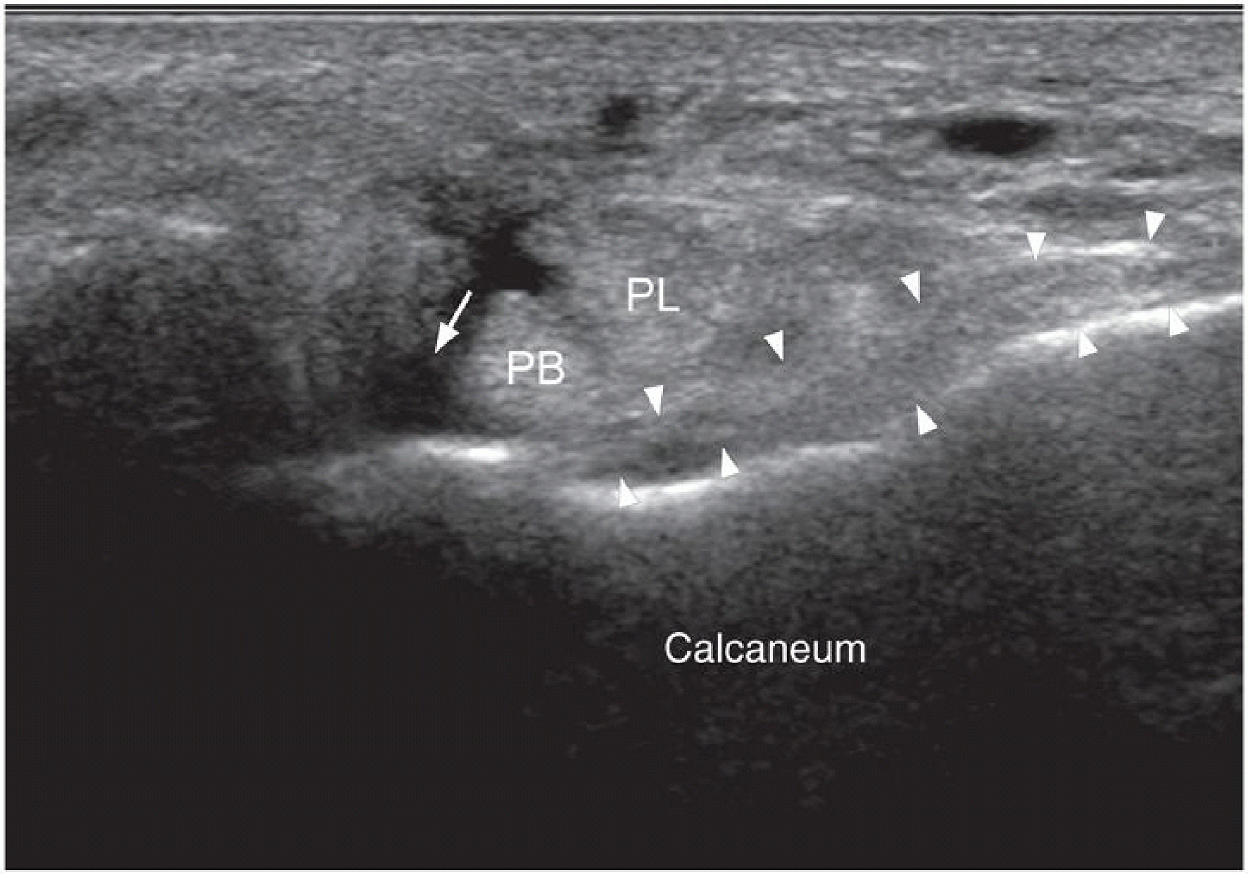

Figure 8.34. Longitudinal ultrasound of the lateral inframalleolar region. There is a complete tear (arrow) of the CFL from its fibular attachment. The torn ligament (arrowheads) has lost its normal "hammock effect" and is lying against the calcaneum deep to the peroneus longus (PL) and peroneus brevis (PB) tendons.

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Injury to the ATFL can also lead to synovial thickening on the deep aspect of the ligament, giving rise to anterolateral impingement syndrome.[9] This is discussed in the section on anterolateral impingement syndrome below.

Tip:

Isolated tears of the CFL ligament are very rare.

Syndesmotic Ligament Injury

The distal tibiofibular syndesmosis is strengthened by the anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, the interosseous ligament, and the transverse tibiofibular ligament. The anterior tibiofibular ligament is the most commonly injured syndesmotic ligament, known as "high ankle strain," and is often diagnosed late clinically. The injury leads to pain and tenderness at the anterolateral ankle, particularly on weight bearing.[1] Tears usually occur in the absence of any visible syndesmotic diastasis. Tears of the anterior inferior tibiofibular ligament usually occur as a result of an eversion injury, often in conjunction with deltoid ligament injury. Severe syndesmotic injury may involve the distal portion of the interosseous membrane between the tibia and the fibula. A torn distal interosseous membrane appears abnormally hypoechoic and poorly defined.[81] Usually the substance of the interosseous membrane is intact, although its periosteal attachment is stripped, mostly on the tibial side.

Medial Collateral Ligament (Deltoid Ligament)

The deltoid ligament is stronger than the lateral collateral ligament and less frequently torn.[1] Complete tears are rare and typically occur during severe eversion injury, often in association with lateral malleolar fracture and lateral displacement of the talus.[40,41] If there is rupture of the deltoid ligament, the posterior tibial tendon moves deeper, toward the articular and bony surface. In acute complete tears, the hematoma, joint effusion, and extension of joint fluid into the para-articular soft tissues give a similar appearance to severe acute lateral collateral ligament injuries.

Sinus Tarsi Ligament Injury

Tear of the sinus tarsi ligaments can manifest as a hypoechoic reparative mass on the lateral aspect of the ankle at the tarsal sinus level, although the ligaments themselves are not easily seen on ultrasound.[1] The sinus tarsi is best assessed on MRI, which shows replacement of the normal sinus tarsi fat on T1-weighted images and periligamentous edema on T2-weighted fat-suppressed images.

Spring Ligament Complex Injury

Isolated tears of the spring ligament are rare. Spring ligament insufficiency is commonly associated with posterior tibial tendon dysfunction and pes planus, which lead to repeated loading of the spring ligament. As a result of accelerated attrition, the ligament becomes lax or ruptures, resulting in acquired flat foot. Healing of partial ligament tears is characterized by thickening and hypoechogenicity.[19,21] Thickness >4 mm of the superomedial component of the spring ligament measured at its mid-portion deep to the posterior tibial tendon is considered abnormal.[20] The degree of posterior tibial tendinosis and thickening of the superomedial spring ligament do not necessarily parallel each other; for example, there may be only a mild degree of tendinosis but marked thickening of the ligament; hence each component should be individually assessed (Fig 8.19).

Tip:

As the degree of posterior tibial tendinosis, peritendinitis, and superomedial component of spring ligament thickening do not necessarily parallel each other, each component should be individually assessed and graded.

Ankle Impingement

Posterior Impingement

Posterior ankle impingement syndrome is caused by compression of the posterior process of the talus and adjacent soft tissue between the tibia and the calcaneum on repeated or forced ankle plantar flexion. It is also termed “os-trigonum syndrome,” “talar compression syndrome,” “posterior block,” or “posterior tibial talar impingement syndrome.”[82,83] It is characterized by posterior ankle pain in plantar flexion and commonly occurs in athletes, particularly ballet dancers, soccer players, downhill runners, and activities that involve regular forceful plantar flexion of the ankle.

The most common bony association is a large os-trigonum, less commonly a downward sloping posterior tibial margin (“posterior malleolus”) or prominent superior surface of the calcaneum. Soft tissue impingement involves the posterior recesses, posterior talofibular, posterior intermalleolar, and posterior tibiofibular ligaments that become inflamed, thickened, and fibrosed. Synovial thickening and swelling may be appreciated at the posterior recesses of the tibiotalar and posterior talocalcaneal articulations, superior and inferior to the posterior talar process, respectively. Ultrasound may reveal a prominent posterior talar process, nodular hypoechoic thickening of the posterior recesses, and an intact but thickened posterior talofibular ligament. The posterior intermalleolar and talofibular ligaments are deep and not easily accessed by ultrasound. The FHL tendon is particularly

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likely to be affected. Tenosynovitis or stenosing tenosynovitis may occur with increased fluid in the tendon sheath as well as more generalized adjacent soft tissue inflammation. In stenosing tenosynovitis, dynamic ultrasound may demonstrate tethering of the FHL tendon within the fibro-osseous tunnel behind the talus during flexion and extension of the great toe.[83] Ultrasound-guided injection of steroid and long-acting anesthetic provides a safe and effective treatment for posterior impingement symptoms.[76,84,85] A short-axis approach with the needle lateral and the transducer medial to the Achilles tendon avoids the tibial neurovascular bundle. A test injection of local anesthetic ensures that the needle is in the tendon sheath, and 20 to 40 mg of methylprednisolone is then injected.

Posteromedial Impingement

Posteromedial ankle impingement typically occurs 4 to 6 weeks after inversion injury and should be distinguished from tibialis posterior pathology, which has similar symptoms. The posteromedial tibiotalar capsule and posterior tibiotalar ligament become thickened and compressed between the talus and the medial malleolus.[27,83] Ultrasound shows thickening of the posteromedial capsule deep to the medial tendons that are displaced superficially.[86,87]

Anterior Impingement

Anterior impingement is a relatively common cause of anterior ankle pain due to impingement of hypertrophied soft tissue and anterior osteophytes of the distal tibia and talar neck. It is often associated with hypertrophic osteoarthritis and also commonly seen in soccer players. The osseous component forms a more significant part of the impinging mass, although the soft tissue component is also critical in producing the clinical syndrome. Capsular thickening, synovitis, and bony spurs are seen at the anterior margin of the ankle joint.

Anterolateral Impingement

Anterolateral impingement is caused by entrapment and inflammation of hypertrophied soft tissues within the anterolateral recess of the ankle and occurs most commonly in young athletes. Repeated inversion injuries and partial tears of the anterior talofibular and tibiotalar ligaments result in reparative hypertrophy of the anterolateral capsule and the ligaments without substantial instability. Chronic synovial-capsular thickening and inflammation leads to a hyalinized connective tissue mass termed a “meniscoid lesion”[9,27] that replaces fluid in the gutter. Marginal osteophytes can have a similar effect. Obliteration of the anterolateral gutter does not invariably imply that clinical impingement is present.[9]

Ultrasound shows thickened synovial tissue (>1 cm) of mixed echogenicity and nodular contour.[9,88] The mass bulges anteriorly, particularly with manual compression of the distal fibula against the tibia. Rarely soft tissue calcification and mild vascularity on Doppler imaging are seen.[9] The ATFL usually shows features of previous injury and is better seen if there is fluid in the anterolateral gutter. If ultrasound is negative or there is no improvement following injection therapy, MRI is recommended, particularly to assess for possible talar dome osteochondral injury.

Anteromedial Impingement

Anteromedial impingement is thought to be related to inversion injury, perhaps with a rotational component, leading to tearing of the anteromedial capsule, and is relatively uncommon.[27] Repeated microtrauma leads to synovitis and capsular thickening,[83] and there is usually a bony component. Findings include irregular anteromedial capsular thickening, synovitis, thickening of the anterior fibers of the deltoid ligament, and occasionally an anteromedial osteophyte.[83,89] If the ultrasound findings are not consistent with the presenting symptoms, MR is helpful in excluding a medial talar dome osteochondral lesion.

Tarsal Tunnel Syndrome

Tarsal tunnel syndrome is characterized by entrapment of the tibial nerve and/or its branches in the tarsal tunnel. Proximal entrapment occurs in the retromalleolar region, and distal entrapment in the inframalleolar region. Symptoms depend on the nerves affected and typically include pain or paresthesia along the medial aspect of the ankle radiating to the medial and/or lateral plantar aspects of the foot and toes. External compression from ill-fitting footwear or a tight plaster cast is the most common cause. Other causes include space-occupying lesions such as ganglia; talocalcaneal coalition; foot deformities such as heel varus or valgus; anomalous tendon and muscle such as an accessory FDL; schwannoma; or lipoma.[23,90,91] Ultrasound can trace the course of the tibial nerve and its branches and detect space-occupying lesions. A beak-shaped bony prominence protruding from the talus or calcaneus is due to talocalcaneal coalition. Local fusiform thickening of the tibial nerve, loss of the normal fascicular pattern, and size discrepancy between the medial and lateral branches suggest nerve compression within the tarsal tunnel.[78] Even small changes in nerve caliber can be detected by ultrasound.[22]

BONE AND JOINT DISORDERS

MRI or computed tomography (CT) is more sensitive in detecting radiographically occult fractures around the ankle and foot than ultrasound. However, ultrasound may show focal cortical disruption, callus, hematoma, or

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soft tissue swelling (Figs. 8.35A, B). Ultrasound is particularly useful in children where fractures may occur in unossified bone or in patients with osteoporosis who may have radiographically occult fractures.[92,93]

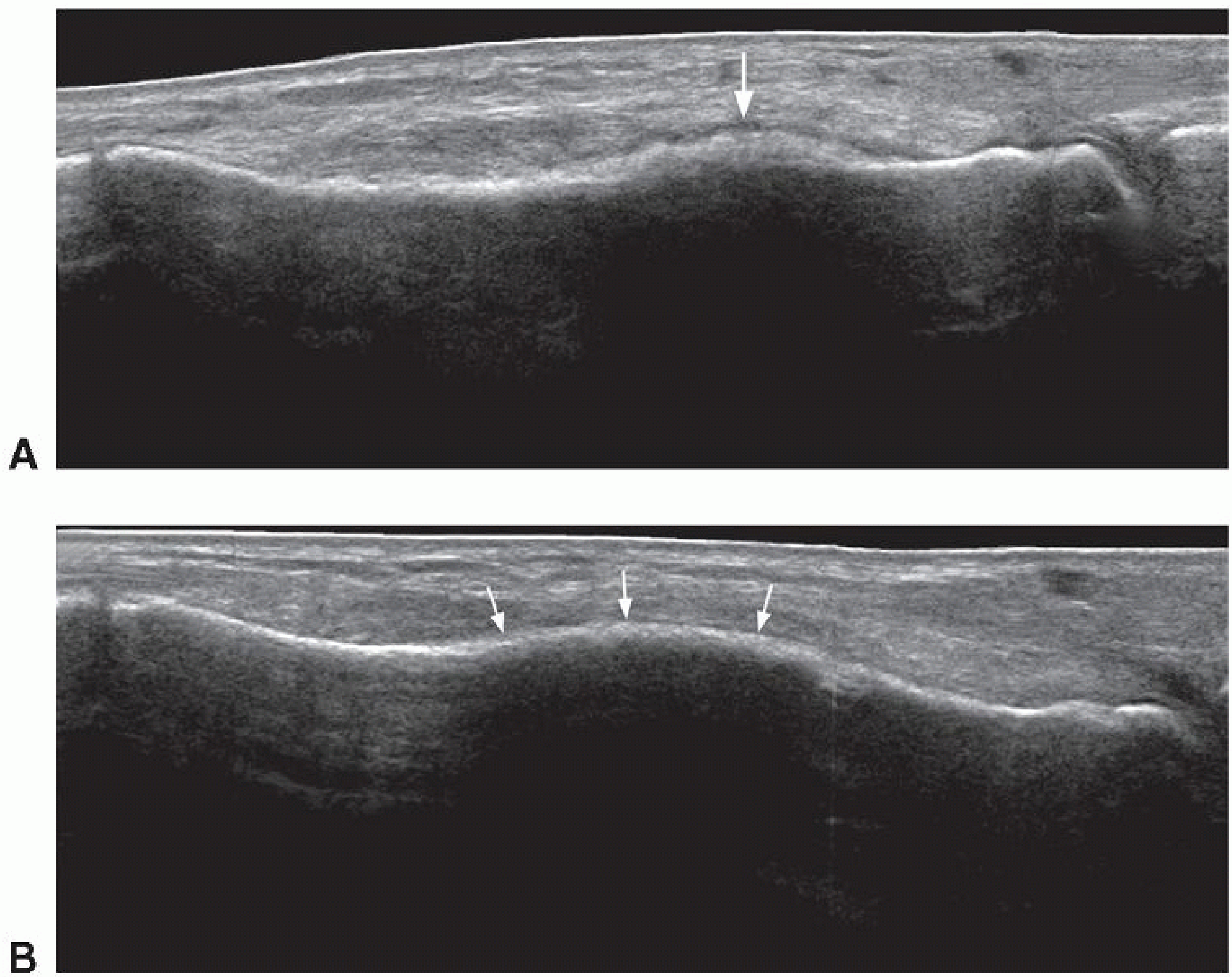

Figure 8.35. Longitudinal ultrasound of forefoot showing (A) an active fracture of the second metatarsal shaft with callus (arrow) and mild surrounding soft tissue swelling. B: On the contralateral foot, there is a healed fracture of the second metatarsal shaft with bone enlargement (arrows), no active callus, and no soft tissue swelling.

Because of their location, most osteochondral lesions of the talar dome are not visible on ultrasound. However, in patients with chronic ankle pain, a moderate or large ankle joint effusion without extra-articular ligament injury should raise the suspicion of an osteochondral lesion.[94]

Joint Disease

Ultrasound can detect as little as 2 mL of fluid in the ankle joint.[94] Plantar flexion of the ankle helps to show fluid in the anterior recess. A simple effusion is almost always anechoic (Fig. 8.36). Increased echogenicity suggests inflammation or hemarthrosis. Small echogenic foci with comet tail artifacts in joint fluid or attached to the synovium or cartilage suggest crystal arthropathy. It is not always possible to differentiate reactive synovitis, other inflammatory arthritis, infective arthritis, or hemarthrosis on ultrasound, although clinical correlation narrows the differential diagnosis (Figs. 8.37 and 8.38) and ultrasound usually distinguishes between joint fluid and synovitis. Chronic synovial proliferation is echogenic. Acute synovial proliferation is usually hypoechoic and can look similar to slightly echogenic joint fluid, but thickened folds of synovium and hyperemia on color or power Doppler distinguish acute synovitis from fluid.

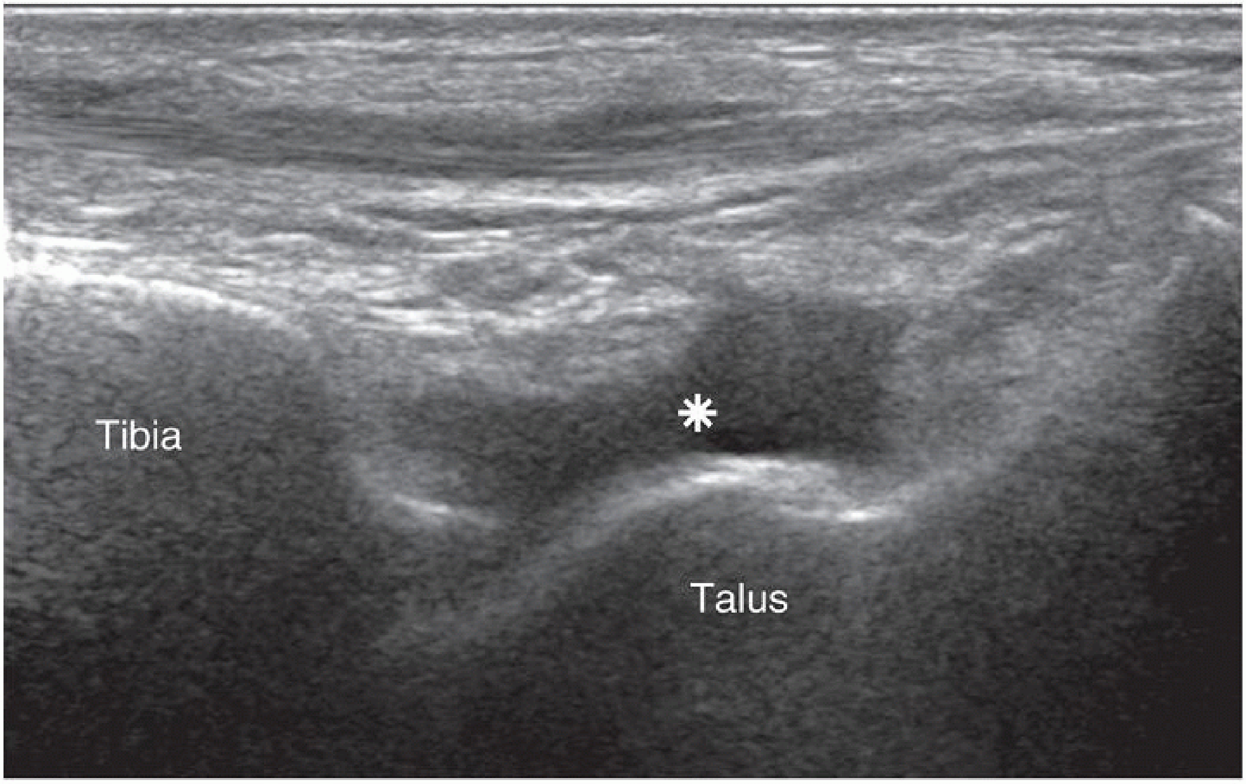

Figure 8.36. Longitudinal ultrasound anterior aspect of ankle. There is a moderate ankle joint effusion (asterisk) displacing the anterior fat pad.

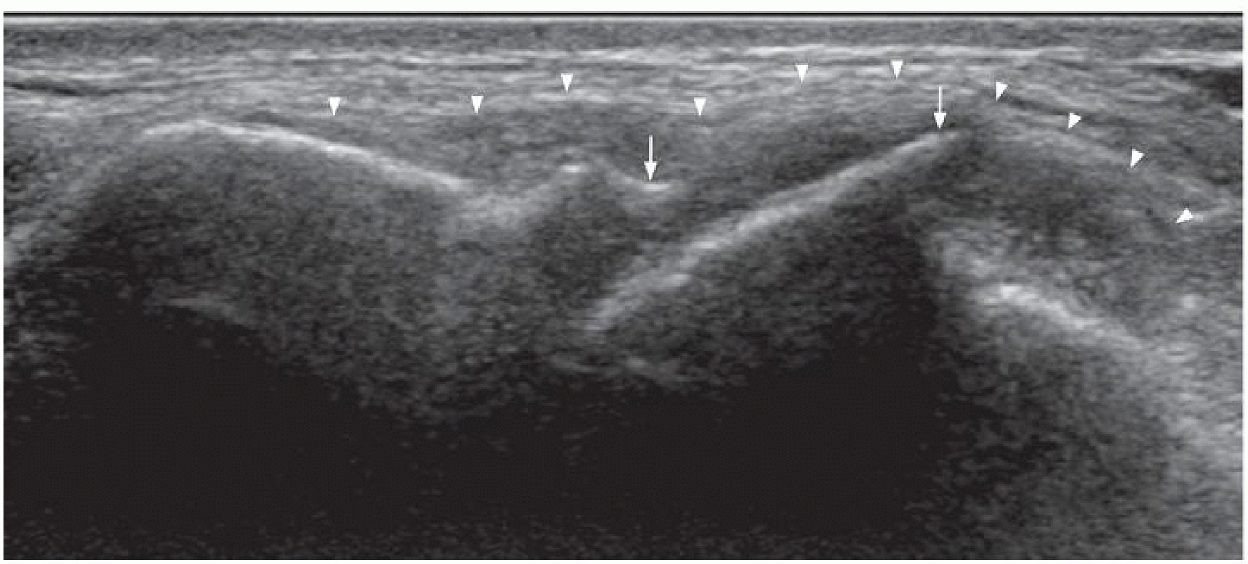

Figure 8.37. Longitudinal ultrasound anterolateral aspect of ankle showing quite severe osteoarthrosis with joint space narrowing, marginal osteophytosis (arrows), together with moderate capsular thickening (arrowheads).

Ultrasound-guided joint aspiration or synovial biopsy may be helpful. Ultrasound ensures accurate needle placement, avoids nerves and vessels, and helps to minimize the risk of cross contamination, for example, between joints and tendon sheaths.[45,67,68,69,70,71,72,73,74,75,76,77,78,79] A longitudinal anterior approach with the foot plantar flexed provides the best visualization of anterior tibiotalar joint. Remember that the deep peroneal nerve lies just lateral to the dorsalis pedis artery.

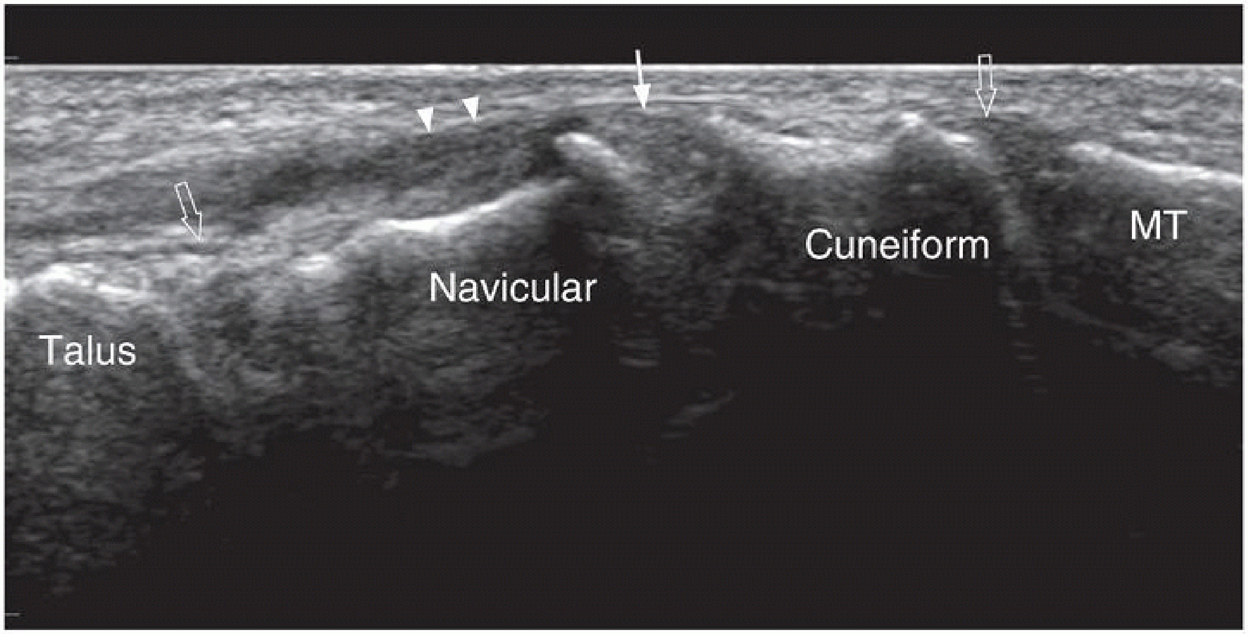

Figure 8.38. Longitudinal ultrasound dorsum of foot showing moderate osteoarthrosis of navicular:cuneiform articulation (arrow) with marginal osteophytosis and adjacent soft tissue thickening (arrowheads). MT, metatarsal. Open arrows point to talo-navicular and tarso-metatarsal joints.

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ANKLE/FOOT MASSES OR FOREIGN BODY

Ganglia

Ganglia are the most common soft tissue masses at the ankle and foot (Fig. 8.39). They contain gelatinous material of variable viscosity and have a fibrous capsule without a synovial lining.[95] Ganglia usually communicate with an adjacent joint or, less commonly, tendon sheath, although this communication is often not identified.[96] Ganglia around the ankle are generally more symptomatic and larger than at the wrist. They appear cystic with anechoic or hypoechoic contents and posterior acoustic enhancement. Septations and a lobulated border are more common than at the wrist.[97] Small comet tail artifacts may be seen, particularly in larger ganglia, due to aggregations of colloid.

Tip:

Most ganglia arise from adjacent joints. A track is usually present from the ganglion pointing toward or extending toward an adjacent joint rather than a clear continuity with the joint.

Other Ankle and Foot Masses

Other common ankle masses include lipomas, nerve sheath tumors, abscesses, and tophi.[96] Lipomas are usually subcutaneous, well defined, easily compressible, have variable internal echogenicity, and lack vascularity on power Doppler imaging.[98] Nerve sheath tumors are well defined, fusiform, and have low-to-mixed echogenicity, increased through-transmission, and moderate intrinsic vascularity on color Doppler[97] (Fig. 8.40). Continuity with and/or thickening of the entering/exiting nerve are confirmatory, although tumors arising from small peripheral nerves are often not associated with nerve thickening. The ultrasound appearances of gouty tophi depend on chronicity.[99] The early "soft tophus" appears as a hypoechoic mass with some internal echogenic foci, through-transmission, and surrounding edematous soft tissues. In the intermediate stage, more discrete and larger echogenic foci are visible within a hypoechoic tophus, and there is more acoustic shadowing. In the late stage akin to a "hard tophus," a dense echogenic mass with strong posterior acoustic shadowing is due to calcification.[99] Gouty arthropathy is more common in the foot than isolated tophi (Figs. 8.41A, B). Other common masses include epidermoid cyst, glomus tumor, and vascular leiomyoma (Fig. 8.42).

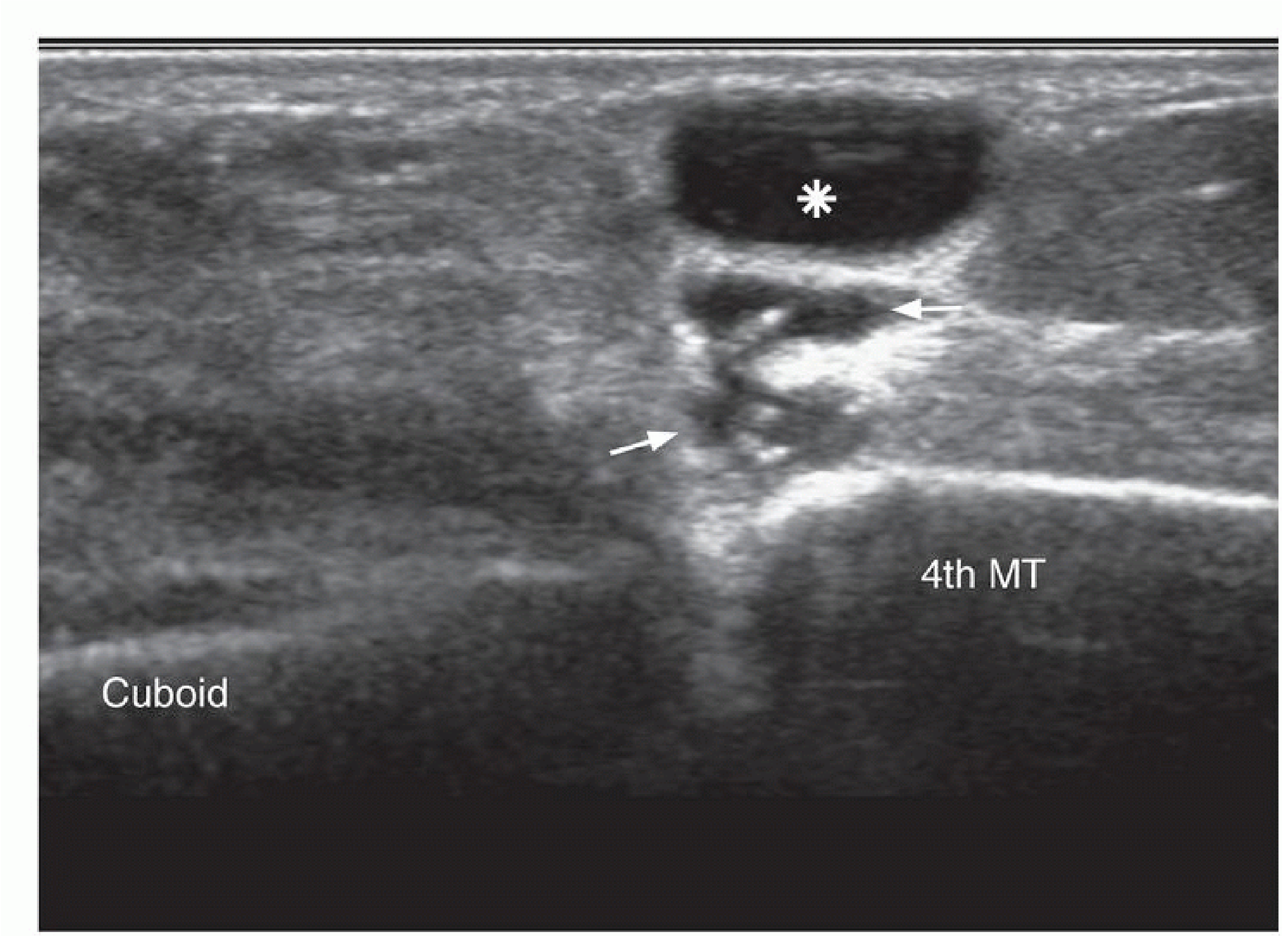

Figure 8.39. Longitudinal ultrasound of dorsum of foot showing medium-sized ganglion cyst (asterisk) with serpiginous track (arrows) leading to cuboid: 4th metatarsal (4th MT) articulation.

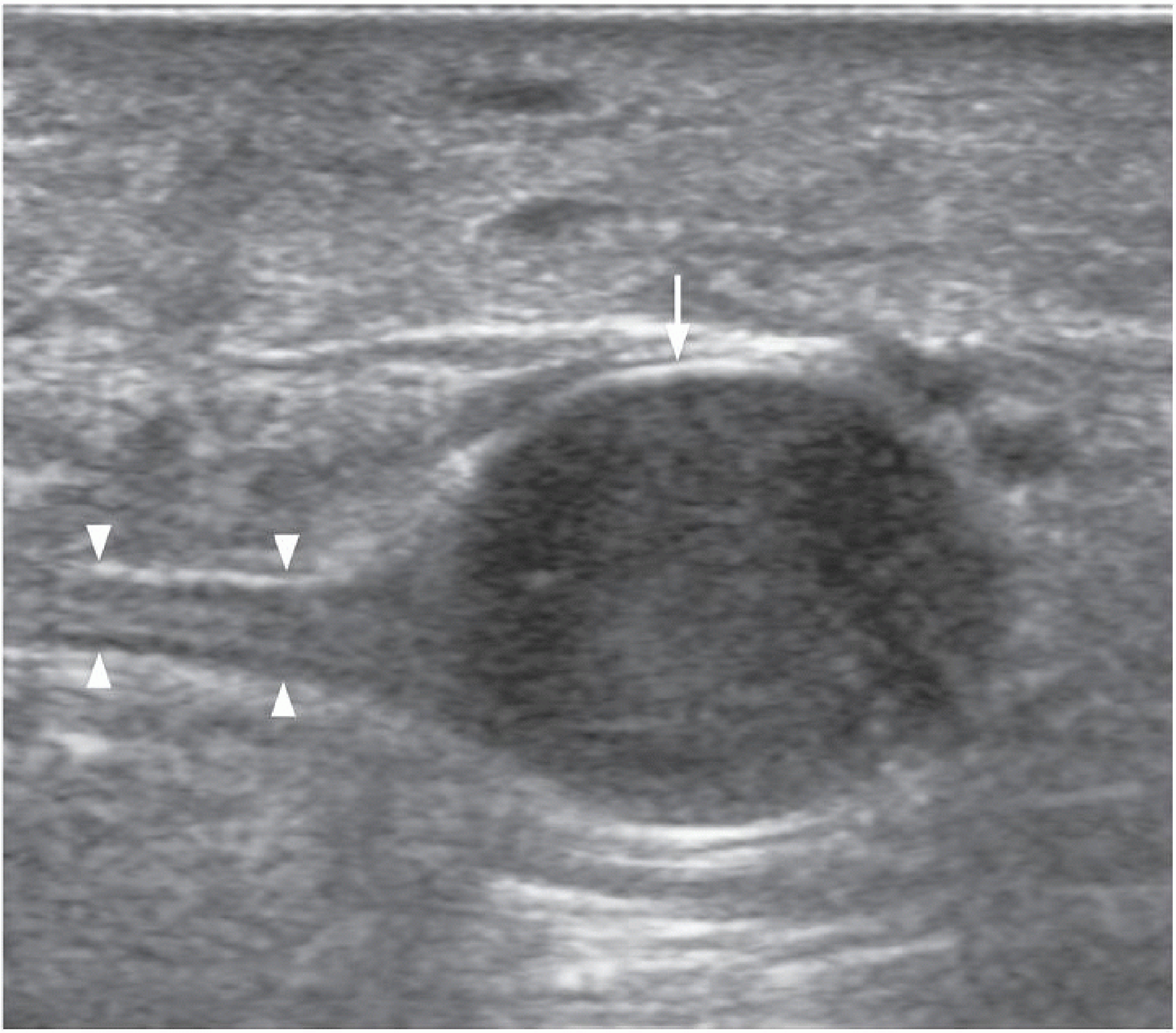

Figure 8.40. Longitudinal ultrasound of medial ankle showing a nerve sheath tumor (arrow) of the tibial nerve. The thickened entering nerve is shown (arrowheads). The exiting nerve is not visible in this image plane.

Foreign Body

Ultrasound detects radiolucent foreign bodies such as wood. Wood splinters tend to be narrow and elongated. They are echogenic, and the transducer may have to be rotated to show the full length of the splinter, which may have a halo.[100] A hyperechoic halo occurs acutely due to surrounding inflammatory tissue. A hypoechoic halo occurs later due to reactive granulation tissue and increases conspicuity of the foreign body. In the acute setting, if a foreign body is not visible on first inspection, it is useful to repeat the ultrasound about a week later when a halo may increase conspicuity. Ultrasound is also useful in localizing and guiding retrieval of foreign bodies.

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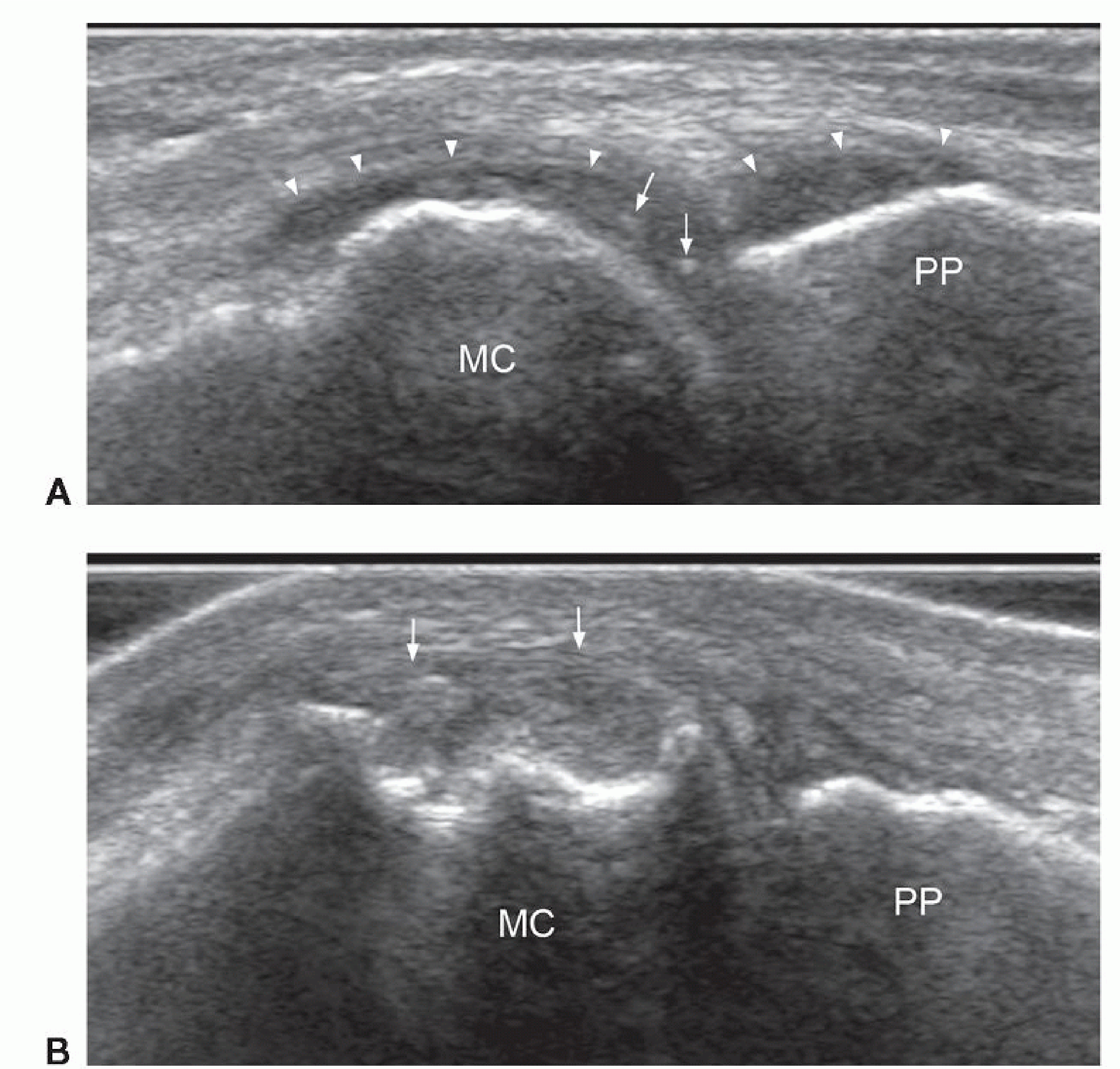

Figure 8.41. Longitudinal ultrasound of first metatarsophalangeal joint. A: Dorsal aspect of joint showing a small to moderate-sized effusion/synovial proliferation (arrowheads). There are several discrete echogenic foci within the joint consistent with crystal aggregates (arrows). B: Medial aspect of the joint. There is a wide deep erosion (arrows) with overhanging edges consistent with a gouty or crystal arthropathy. MC, metacarpal; PP, proximal phalanx.

FOOT PATHOLOGY

Tibialis Anterior and Extensor Tendon Abnormalities

Longitudinal splits of the distal tibialis anterior tendon have been noted in asymptomatic subjects.[101] Tendinosis of tibialis anterior tends to occur within 3 cm of the insertion on the medial cuneiform and first metatarsal. The extensor hallucis and extensor digitorum tendons are less prone to tendinosis or injury, and tears are usually secondary to direct trauma to the dorsum of the foot. All tendon pathology in the foot is readily seen with ultrasound (Fig. 8.43), except for the flexor tendons and peroneus longus tendon deep in the midfoot region when MRI should be employed.

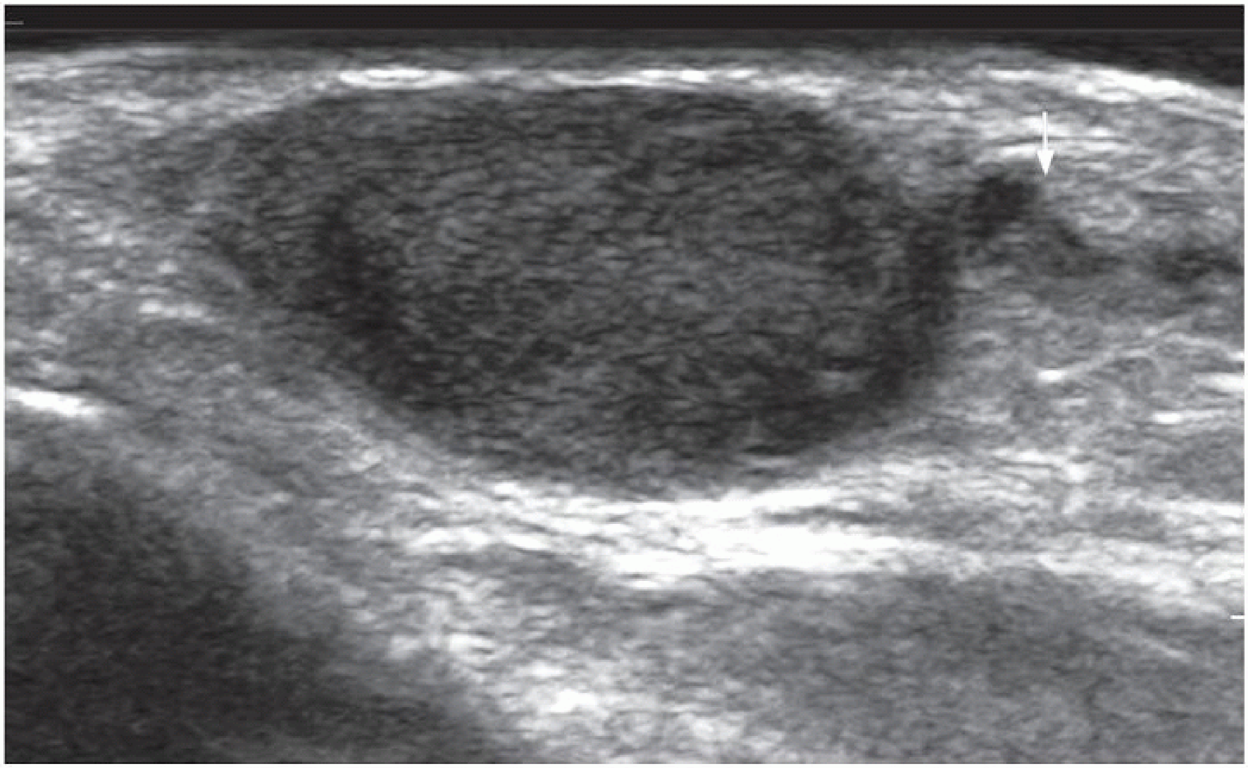

Figure 8.42. Longitudinal ultrasound dorsum of forefoot showing an ovoid hypoechoic mass in the subcutaneous tissues contiguous with a small artery distally (arrow). Histology revealed a vascular leiomyoma.

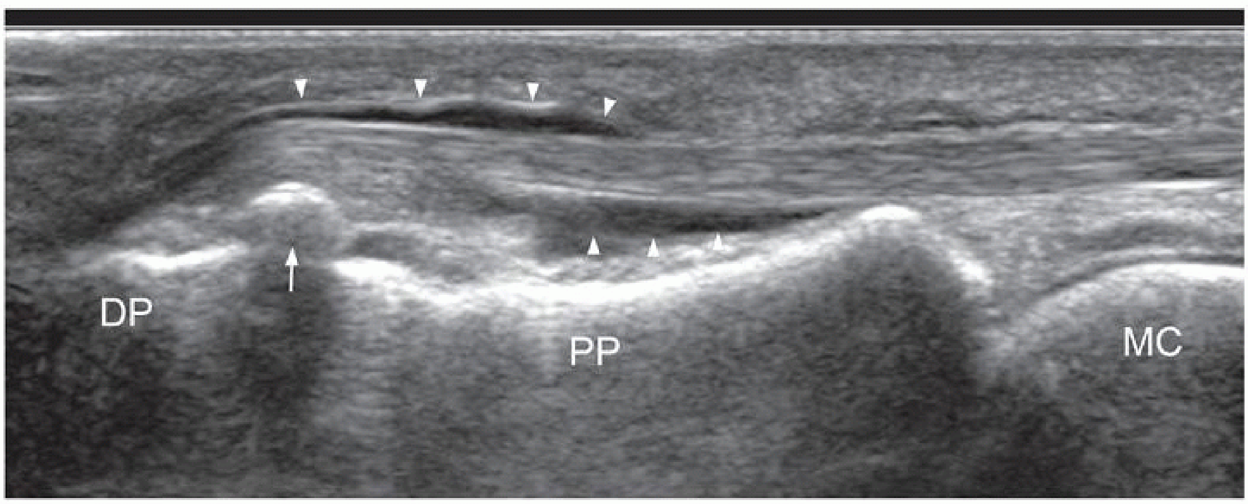

Figure 8.43. Longitudinal ultrasound of the big toe showing a moderate tenosynovitis (arrowheads) of the FHL tendon with a tendon sheath effusion surrounding a mildly swollen but otherwise normal tendon. Note the sesamoid bone (arrow) overlying the distal interphalangeal joint. MC, metatarsal; PP, proximal phalanx; DP, distal phalanx.

Plantar Hindfoot and Midfoot

Plantar Fasciitis

Plantar fasciitis is the most common cause of inferior heel pain and affects mainly middle-aged women and younger male runners.[102] It is related to overuse and repeated microtrauma, or occasionally associated with systemic enthesopathy, and involves the plantar fascia at its calcaneal insertion. Obesity is a risk factor.[103] Patients usually complain of heel pain, particularly on weight bearing, and there is deep tenderness at the medial calcaneal tuberosity. Thickening and hypoechogenicity of the fascia near its medial calcaneal insertion are the most consistent ultrasound findings[80,104] (Fig. 8.44A). Plantar fascia thickness <4 mm can be considered unequivocally normal, between 4 and 5 mm is borderline, and >5 mm unequivocally abnormal. A very thick plantar fascia has a slightly convex plantar contour (Fig. 8.44B). If symptoms are severe, there may be loss of definition of the fascia and perifascial edema close to the calcaneal insertion.[105] Calcaneal spurs may occur just deep to the calcaneal insertion. Intrafascial calcification is rare. Focal bony erosions and localized hyperemia at the attachment site may be seen with systemic enthesopathy.

Tip:

Plantar fascial thickness should be assessed at the medial leading edge of the calcaneum. Subclinical plantar fasciitis with plantar fascial thickening is not uncommonly present on the opposite, asymptomatic side.

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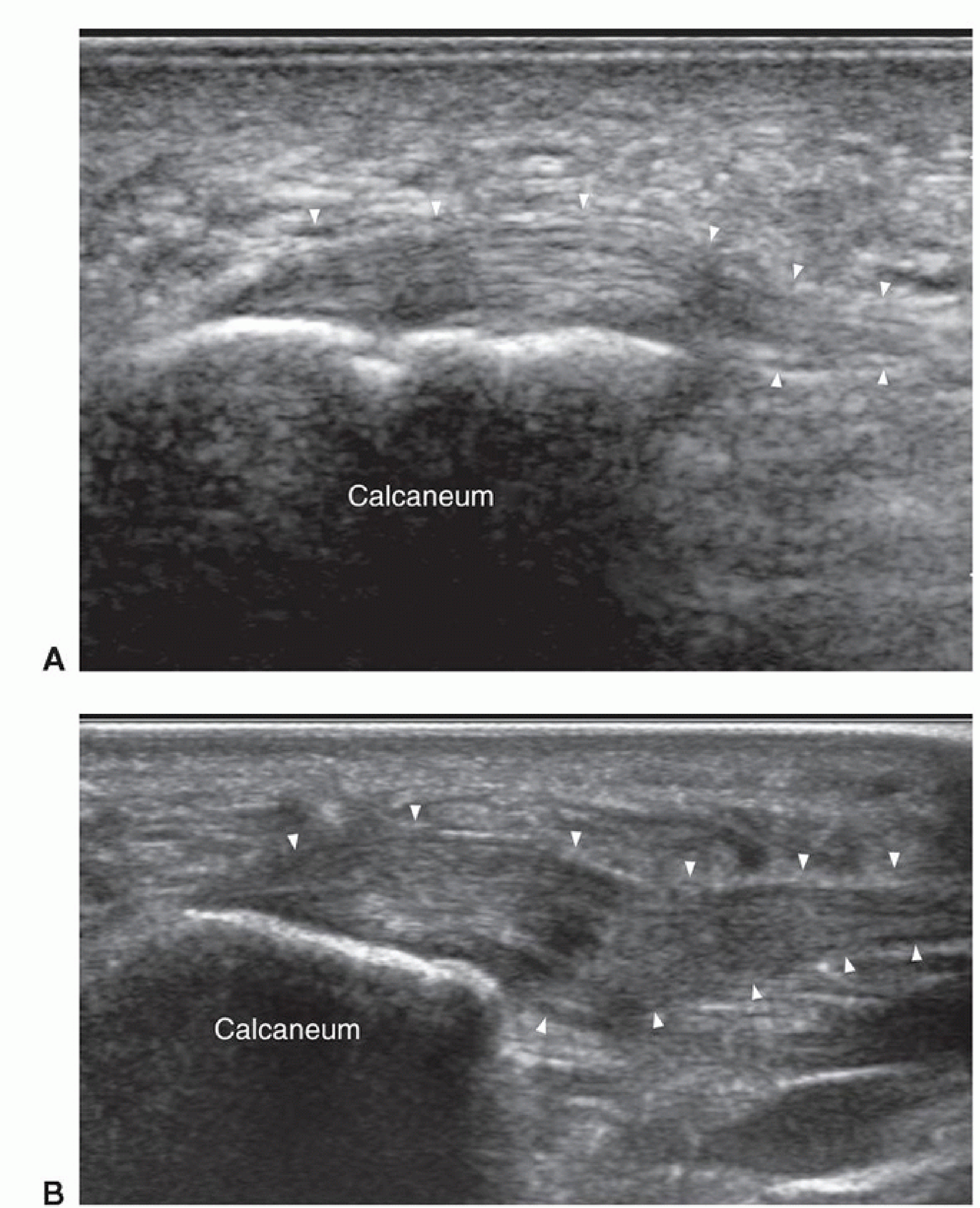

Figure 8.44. Longitudinal ultrasound of plantar heel. A: Mild (4.3 mm) thickening of the plantar fascia (arrowheads) at the leading edge of medial calcaneal tuberosity is present consistent with plantar fasciitis, although this degree of thickening can also be found in asymptomatic subjects. B: More marked (8.7 mm) thickening of the plantar fascia (arrowheads) is present, indicative of severe plantar fasciitis.

One treatment option is corticosteroid injection. Blind injections using bony landmarks have been associated with rupture of the fascia.[69] Ultrasound guidance minimizes direct intrafascial injection.[69,70,78] A longitudinal plantar approach is painful because of the thick skin on the sole of the foot. A medial, short-axis approach allows local anesthetic to be injected into the skin and is better tolerated. Dry needling may be performed initially and a steroid and local anesthetic mixture then injected deep and/or superficial but not into the fascia. Because of the risk of atrophy of the heel fat pad, short-acting steroids such as dexamethasone, or methylprednisolone of the long-acting steroids are preferred in a total dose of about 40 mg.

Plantar Fibromatosis

Plantar fibromatosis is a benign focal proliferation of fibrous tissue in the plantar fascia, and is most common between 30 and 50 years, affecting women almost twice as frequently as men.[106] Patients usually present with a slow-growing mass that is painful on weight bearing. Most lesions are solitary and unilateral, although about one-third are bilateral and one-quarter are multiple.[107] There is an increased prevalence in patients with Dupuytren contracture, penile fibromatosis (Peyronie disease), or those prone to keloid formation.[108] Ultrasound shows discrete fusiform or nodular thickening of the plantar fascia aligned along its long axis. Most swellings are avascular, well defined, hypoechoic without acoustic enhancement, <20 mm in length, and involve the central and medial portions of the fascia distal to the calcaneal insertion[106,107,108,109] (Fig. 8.45). They tend to bulge more from the superficial aspect of the fascia than deeply. Larger lesions are more rounded.[109] Plantar fibromatosis is readily differentiated from plantar fasciitis by morphology and position. Differentiation from a chronic partial tear is more difficult, although tears are less common. A visible tear, perifascial edema, and a history of preceding trauma favor the diagnosis of a partial tear. Aggressive plantar fibromatosis is a different disease and is associated with an enlarging locally invasive irregular fibrotic mass that can grow to a large size deep into the soft tissues of the midfoot and forefoot.

Tip:

Plantar fibromatosis is often multiple and bilateral. Both feet should be examined.

Forefoot

Metatarsal Stress Fracture and Freiberg Disease

Metatarsal stress fractures are among the most common fractures in the forefoot[110] and usually involve the second and third metatarsal shafts. Ultrasound shows cortical irregularity, periosteal thickening, and callus formation. Usually, a clear fracture line is not visible,

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but soft tissue swelling and inflammation and occasionally small fluid collections in the adjacent soft tissues are seen.[40,41]

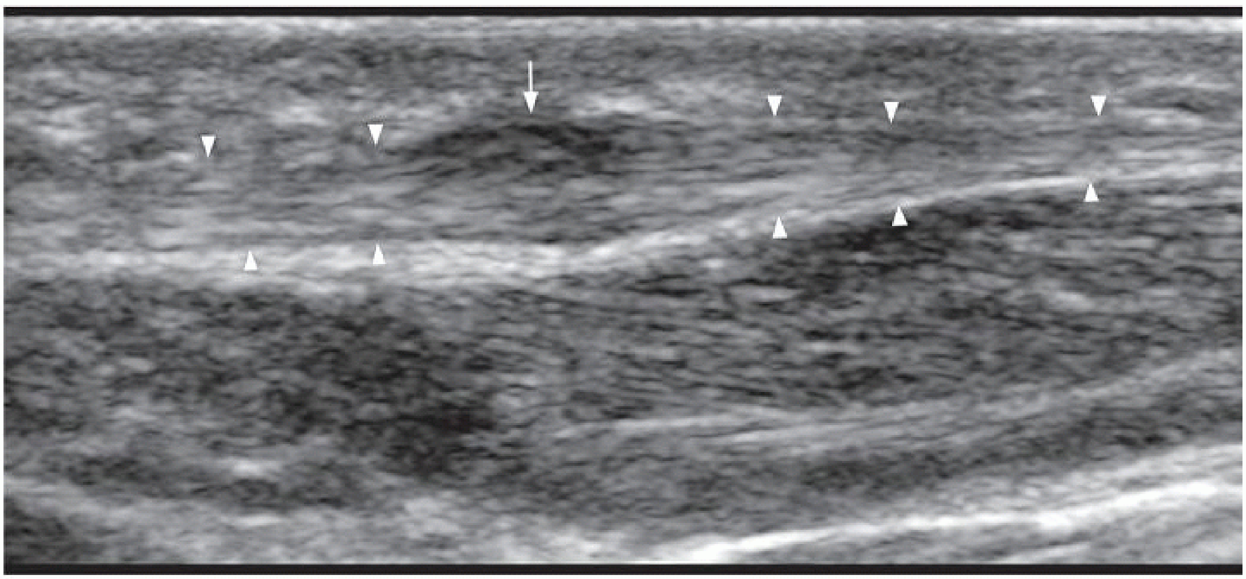

Figure 8.45. Longitudinal ultrasound of plantar aspect of foot showing medium-sized plantar fibroma (arrow) seen as fusiform, slightly hypoechoic swelling along the plantar fascia (arrowheads).

Freiberg disease is avascular necrosis of a metatarsal head. Ultrasound can identify the metatarsal head collapse and widening of the joint space, though this condition is usually diagnosed radiographically.

Plantar Plate Disruption (“Turf Toe”)

Turf toe is a hyperextension injury to the capsulo-ligamentous-sesamoid complex on the plantar aspect of the first metatarsophalangeal joint, involving first the capsule and then the plantar plate. The injury manifests as a hypoechoic, discontinuous swelling on the plantar aspect of the metatarsophalangeal joint. Usually the capsule or the plantar plate is injured close to the metatarsal head since this attachment is weaker than at the proximal phalanx.[40,41] Plantar plate injury is associated with synovitis, joint effusion, and periarticular soft tissue swelling. Dynamic scanning during flexion or extension of the toe accentuates visibility by widening the separation gap. Sesamoid fracture or displacement can be an associated feature.

Lisfranc Ligament Injury

Lisfranc injury comprises a fracture dislocation of the tarsometatarsal articulation and may result in premature osteoarthrosis and chronic foot pain.[32] Assessment of the Lisfranc ligament proper is not possible on ultrasound. However, assessment of the dorsal ligament between the medial cuneiform and the second metatarsal base and bony alignment can indirectly indicate a Lisfranc ligament injury. The normal dorsal ligament is 0.9- to 1.2-mm thick with a hyperechoic echotexture.[32] Non-visualization of this ligament and a distance between the medial cuneiform and the second metatarsal base of 2.5 mm or greater are indirect signs of a Lisfranc ligament tear.[32]

Morton Neuroma

Morton neuroma is a common cause of forefoot pain. It is a non-neoplastic fusiform enlargement of the plantar digital nerve, almost always between the second and third or third and fourth metatarsal heads. It is due to perineural fibrosis probably caused by chronic repetitive low-grade trauma[62] and is not a true neuroma. Women are particularly susceptible to Morton neuroma possibly due to tight-fitting high-heeled shoes. Patients present with pain or paresthesiae radiating from the midfoot to the toes, often exacerbated by tight shoes or walking. Tinel sign or Mulder sign (a palpable or audible click when squeezing the metatarsals heads) can be elicited clinically. Most Morton neuromas are round or oval, hypoechoic nodules. They sometimes contain an anechoic area due to an adjacent intermetatarsal bursa.[33] The neuroma is usually located more toward the plantar aspect of the intermetatarsal space at the level of the metatarsal heads or else midway between the metatarsal heads, typically on either side of the third metatarsal head. The intermetatarsal bursa may be difficult to distinguish from a neuroma on ultrasound, but its presence can be inferred if the mass shrinks when squeezed. Conspicuity of the neuroma can be improved by placing the transducer on the plantar aspect of the foot and applying finger pressure to the dorsal aspect of the intermetatarsal space. The neuroma is generally aligned parallel with the metatarsal shafts. Continuity with the plantar digital nerve is the most specific feature but is frequently not visible. Ultrasound-guided injections can be performed in cases refractory to the conservative treatment.[69,70,77] A dorsal approach is preferable as it is less painful than a plantar approach. Although the needle is placed in the mass, the injection sometimes shows swirling flow as the adjacent intermetatarsal bursa fills. Steroid injections, typically 40 mg of methylprednisolone along with bupivacaine, are the best first-line treatment options, often providing several months of pain relief.[111] After 3 months, if appreciable symptomatic relief from the first injection, a second steroid injection can be given. If the neuroma is large (>5 mm width) or does not respond to steroid injection, neuroablative therapy with alcohol injection may help to relieve symptoms.[112]

INFECTION

Ultrasound can assess soft tissue infection in the foot and distinguish between cellulitis, infective tenosynovitis, abscess, and joint infection (Fig. 8.46). MR should be used if osteomyelitis is suspected as ultrasound has low sensitivity for bone infection.

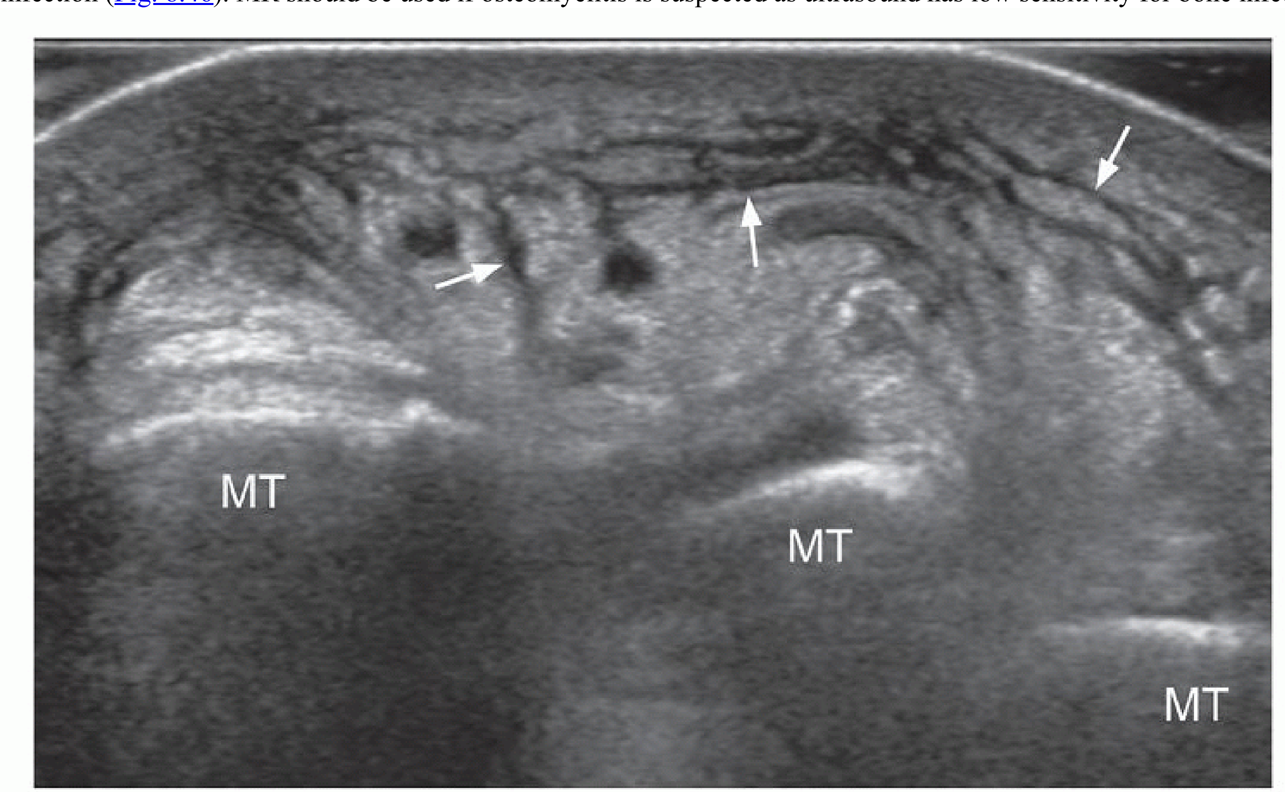

Figure 8.46. Transverse ultrasound of dorsum of forefoot. Severe edema of the subcutaneous fat is present with edematous thickening of the interlobular septa (arrows). Moderate hyperemia was present on color Doppler imaging. No discrete collection. Overall appearances are consistent with severe cellulitis. MT, metatarsal bones.

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