MR Imaging of Ankle Anatomy and Review of Common Ankle Pathologies

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Learning objectives

The purpose of this pictoral essay is to:

- Familiarize the audience with normal ankle anatomy as it appears on MRI
- Aid in the interpretation and diagnosis of common ankle pathologies, including osseous, tendinous, and ligamentous injuries, as well as compressive neuropathies.

Background

Musculoskeletal disease of the ankle is very common, particularly in young active individuals. MR imaging has revolutionized the evaluation of the musculoskeletal system, and has become the modality of choice in the assessment of ankle pathology.

- MRI provides high resolution anatomic details and multi-planar imaging capabilities
- Particularly useful in the evaluation of soft tissue structures

Interpreting ankle MRI is complex secondary to the intricate anatomy, small structures, and range of pathologic processes. Knowledge of normal anatomic structures is crucial to accurately diagnosing pathologic conditions.

Findings and procedure details

BONES AND JOINTS (Fig 1)

The bony structures of the ankle include the tibial plafond, medial malleolus, lateral malleolus, and talus. The ankle joint consists of the tibiotalar, subtalar, and distal tibiofibular joints. The primary motions of the ankle are plantar and dorsiflexion, and the secondary motions include inversion, eversion, and rotation.

TENDONS (Fig 2)
ANTERIOR TENDONS (Fig 3)

Medial to lateral: tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius

Tibialis anterior (TA)

- The TA muscle arises from the proximal anterolateral tibia, and the tendon inserts into the medial and plantar aspects of the first metatarsal and cuneiform bones
- Actions: dorsiflex the foot at the ankle; invert the foot

The anterior aspect of the tendon is avascular, leaving it prone to spontaneous rupture

Extensor hallucis longus (EHL)

- The EHL muscle arises from the anterior surface of the middle 1/3 of the fibula and interosseous margin, and the tendon inserts into the base of the hallux P2 segment base
- Actions: dorsiflex and invert the foot at the ankle; extend the hallux

Extensor digitorum longus (EDL)

- The EDL muscle arises from the anterior aspect of the lateral tibial condyle and interosseous margin, and the tendons insert into the dorsal aspects of the 2nd through 5th P2 and P3 segments
- Actions: dorsiflex the foot at the ankle; extend toes 2-5

Peroneus tertius (PT)

- The PT muscles arise from the anterior surface of the distal 1/3 of the fibula, and the tendon inserts into the dorsal aspect of the 5th metatarsal base
- Actions: dorsiflex and evert the foot at the ankle

POSTERIOR (ACHILLES) TENDON (Fig 4)

- The tendons of the gastrocnemius and soleus muscles form the Achilles tendon, which inserts into the middle part of the posterior surface of the calcaneus
• Actions: Plantar flexes the foot; provides postural orientation for the visually impaired
• The Achilles tendon typically measures 15 cm in length, and 7mm in the AP dimension
• There is a hypovascular region 4 - 6 cm proximal to its insertion, where most injuries occur
• Does not have a tendon sheath

**MEDIAL TENDONS (Fig 5)**

Medial to lateral: tibialis posterior, flexor digitorum longus, flexor hallucis longus

Pass through the tarsal tunnel

**Tibialis posterior (TP)**

• The TP muscle arises from the interosseous membrane, posterolateral proximal tibia, and posteromedial proximal fibula
• The TP tendon courses posterior to the medial malleolus and inserts into the medial aspect of the navicular, into the 3 cuneiforms, cuboid, base of the 2\textsuperscript{nd} through 4\textsuperscript{th} metatarsal bases, and sustentaculum tali of the calcaneus
• Actions: inverts and plantar flexes the foot

**Flexor digitorum longus (FDL)**

• The FDL muscle arises from the posterior surface of the middle 1/3 of the tibia, medial to the TP muscle
• The FDL tendon passes lateral to the TP at the level of the medial malleolus, crosses over the FHL at the chiasma plantare, inserting into the plantar aspects of the 2\textsuperscript{nd} through 5\textsuperscript{th} P3 segment bases
• Actions: plantar flexes the foot; flexes the 2\textsuperscript{nd} through 5\textsuperscript{th} toes

**Flexor hallucis longus (FHL)**

• The FHL muscle originates from the distal 1/3 of the posterior surface of the fibula and the interosseous membrane
• The FHL tendon passes under the sustentaculum tali and between the hallux sesamoids, inserting into the plantar base of the hallux P2 segment
• Actions: plantar flexes the foot; flexes the hallux
• The FHL tendon sheath communicates with the ankle joint in 20% of the population
**LATERAL TENDONS (Fig 6)**

Peroneus longus (PL)

- The PL muscle originate from the head and proximal lateral 2/3 of the fibula, deep surface of the fascia, and intermuscular septa
- The PL tendon passes posterior to the distal fibula, posteroinferior to the peroneal tubercle of the calcaneus, crossing the plantar aspect of the foot to insert into the lateral plantar aspects of the medial cuneiform and first metatarsal
- Actions: plantar flexes and everts the foot at the ankle

Peroneus brevis (PB)

- The PB muscle arises from the distal 2/3 of the lateral fibula
- The PB tendon courses anteromedial to the PL tendon along the posterior aspect of the distal fibula, anterosuperior to the peroneal tubercle, and inserts into the lateral aspect of the 5th metatarsal base
- Actions: plantar flexes and everts the foot at the ankle

**LIGAMENTS (Fig 7)**

**MEDIAL COLLATERAL LIGAMENT (Fig 8)**

Also known as the deltoid ligament. Stabilize against valgus and pronation forces, and rotational forces on the talus.

Divided into the superficial and deep components

- Superficial: tibionavicular, tibiocalcaneal, tibiospring, and posterior superficial tibiotalar ligaments; have variable attachments across two joints
- Deep: anterior tibiotalar and posterior deep tibiotalar ligaments; intraarticular and covered by synovium; have talar attachments across one joint
- These components are variably present
- The most commonly present components include the tibiocalcaneal, tibiospring, and posterior deep tibiotalar ligaments

The superficial components arise from the anterior colliculus of the medial malleolus and the deep components arise from the posterior colliculus and intercollicular groove.
Tibionavicular ligament (TN): courses medial to the TP and FDL tendons, and inserts on the navicular tuberosity; only visible in 55% of patients

Tibocalcaneal ligament (TC): also courses medial to the TP and FDL tendons to insert on the sustentaculum tali of the calcaneus

Tibiospring ligament (TS): only component without an osseous attachment; joins the superomedial oblique band of the spring ligament proper

Anterior tibiotalar ligament (ATTL): inhomogeneous band that inserts onto the talar neck

Posterior deep tibiotalar ligament (PDTL): thick, inhomogeneous band with a broad insertion on the medial aspect of the talus

LATERAL COLLATERAL LIGAMENT COMPLEX (Fig 9)

Consists of three ligaments: anterior talofibular ligament (ATFL), posterior talofibular ligament (PTFL), and calcaneofibular ligament (CFL)

The ATFL arises from the anterior margin of the distal lateral malleolus and inserts onto the lateral surface of the talus.

- Restrains anterior talar motion
- Weakest and most frequently injured ligament

The PTFL has a broad-based origin from the fibular fossa and also has a broad-based attachment to the posterior aspect of the talus.

- Restrains posterior talar motion

The CFL originates from the apex of the lateral malleolus and inserts onto the lateral surface of the calcaneus.

- Courses deep to the PL and PB tendons

SYNDESMOTIC LIGAMENTS (Fig 10)

Consists of 4 ligaments: anterior inferior tibiofibular ligament, posterior inferior tibiofibular ligament, interosseous ligament, and posterior intermalleolar (transverse) ligament
The anterior tibiofibular and posterior tibiofibular ligaments join the tibia and fibula just proximal to the tibiotalar joint.

- Major stabilizer of the distal tibiofibular joint

The interosseous ligament consists of a thickened portion of the distal aspect of the interosseous membrane.

The transverse ligament connects the lateral malleolus to the posterior articular surface of the tibia.

**SINUS TARSI (Fig 11)**

Cavity located along the lateral aspect of the ankle, distal and slightly anterior to the lateral malleolus. The borders include the inferior aspect of the talar neck and anterosuperior aspect of the calcaneus. The sinus tarsi opens medially posterior to the sustentaculum tali, and separates the anterior and posterior subtalar joints. It contains multiple structures, including blood vessels, nerves, fat, interosseous talocalcaneal ligament, and cervical ligament.

**COMMON ANKLE PATHOLOGIES**

**OSSEOUS PATHOLOGIC CONDITIONS**

Common painful osseous abnormalities include contusions, fractures, osteochondral defects, osteonecrosis, and tarsal coalition

**OSSEOUS CONTUSION (Fig 12)**

Contusions are microfractures of the trabecular bone. They are manifested by decreased T1 and increased T2 signal intensity within the marrow. Contusions typically resolve in 8 - 12 weeks.

**ACUTE FRACTURE (Fig 13)**
Acute fractures manifest as a linear low T1/T2 signal intensity surrounded by marrow edema.

The talus, with its limited surface of penetrable bone, small nutrient blood vessels, and lack of collateral circulation is at an increased risk for developing avascular necrosis following fracture.

- Particularly true when fractures involve the talar neck

**OSTEochondral INjury (Fig 14)**

Most commonly located in the posteromedial or anterolateral talar dome. A crescentic low T1/T2 fracture line is identified on MR imaging. Unstable lesions have fluid (high T2 weighted signal) between the fracture fragment and underlying bone. Surrounding edema is noted in acute injuries. Surgery is reserved for unstable lesions or stable lesions that fail conservative management.

**TARSAL COALITION (Fig 15)**

Most commonly occurs at the calcaneonaviccular and middle facet of the subtalar joints. Coalition can be osseous, cartilaginous, or fibrous. The articulation is typically broadened, with surrounding marrow and possibly soft tissue edema identified. Treatment is typically conservative; surgical resection with fat or muscle interposition or triple arthrodesis are reserved for those who fail conservative management.

**OSTEONECROSIS (Fig 16)**

Bone marrow infarct can be secondary to trauma or an underlying systemic disease. Osteonecrosis of the talus is usually due to fracture with associated vascular compromise. MR imaging demonstrates central signal that follows fat on all sequences, and a serpiginous margin of high T2 weighted signal intensity with an outer margin of low T2 weighted signal intensity ("double line sign"). No treatment is necessary in uncomplicated osteonecrosis.

**TENDON PATHOLOGIC CONDITIONS**

Include ruptures, tendinitis/peritendinitis, tenosynovitis, entrapment, and dislocation
TENOSYNOVITIS (Fig 17)

Tenosynovitis is inflammation of the tendon sheath. It develops secondary to inflammation or mechanical irritation. Appears as excessive fluid accumulation, synovial proliferation, or scarring within the tendon sheath on MRI. May result in partial tendon rupture.

TENDINITIS (Fig 18)

Tendinitis results from inflammation within the tendon substance. It may be secondary to an acute or overuse injury. MR imaging demonstrates focal or diffuse tendon enlargement. Tendinitis can appear low in signal intensity on both T1 and T2 sequences, or have focal areas of increased T2 weighted signal intensity. These areas of high signal intensity complicate the differentiation between tendinitis and partial rupture.

TENDON RUPTURE (Figs 19 - 21)

Can be acute or chronic

Acute rupture is secondary to acute force applied to the tendon.

- Most common in the Achilles tendon in middle-aged men

Chronic rupture is due to degenerative tendinopathy without antecedent trauma.

- Most commonly involves the PT tendon

Increased T2 weighted signal intensity is visualized within the substance of the tendon in partial ruptures, and the tendon fibers are discontinuous in complete ruptures.

LIGAMENT PATHOLOGIC CONDITIONS (Figs 22 - 24)

Ankle ligament injuries are the most common injuries in patients participating in sports and recreational activities. On MR, ligamentous injury is signaled by discontinuity, detachment, thickening, thinning, irregularity, and abnormal signal characteristics.

The most common mechanism of injury to the ankle is inversion of the foot (85% of ankle injuries), which injures the lateral ligaments.
• The ATFL is the weakest ligament and, thus, the most frequently torn

An eversion injury damages the deltoid ligaments, and hyperdorsiflexion trauma causes injury to the syndesmotic ligaments.

• Syndesmotic and deltoid ligament injuries can have significant implications as 1mm of lateral translation of the talus results in 42% decrease in the tibiotalar contact area and 42% increase in tibiotalar contact pressure

Medial ligament injuries are typically associated with lateral ligament injuries.

Ankle ligamentous injuries are rarely treated surgically.

• The exceptions include high level athletes and in the case of displaced lateral malleolar fractures combined with tears of the deep medial collateral ligament - surgical repair is necessary to restore normal anatomy

SINUS Tarsi Syndrome (Fig 25)

Sinus tarsi syndrome is a clinical syndrome consisting of pain and tenderness along the lateral aspect of the hindfoot, which increases with weight-bearing, inversion, and eversion maneuvers. It is commonly associated with inversion injuries and tears of the lateral collateral ligaments, but can also be seen in patients with rheumatologic disorders or abnormal mechanics (i.e. pes planus). MR imaging demonstrates replacement of the fat with fluid or scar tissue, and the ligaments may or may not be intact. Treatment is conservative.

OS Trigonum Syndrome (Fig 26)

Results from posterior impingement of the ankle secondary to an os trigonum. It can be seen in athletes requiring repetitive forceful flexion (ballerinas, soccer players). MR imaging demonstrates excessive fluid in the synchondrosis, associated marrow edema, and surrounding soft tissue edema; isolated FHL tenosynovitis may also be seen. Treatment is conservative with surgery reserved for those who fail conservative management.

COMPRESSIONNEUROPATHY (Figs 27 - 28)
Direct compression upon a nerve causes tingling, numbness, pain, and muscle weakness.

Causes include repetitive external compressive force (extrinsic) or compression from the patients own soft tissues (intrinsic).

- Extrinsic causes include ankle/foot deformities, accessory ossicles, altered biomechanics
- Intrinsic causes include tumors, cysts, varicose veins, accessory/hypertrophied muscles, excess fatty tissue, synovial hypertrophy, and scar tissue

MR imaging demonstrates muscle edema in the acute setting, and fatty muscular atrophy when the process is chronic. Treatment depends on the cause, and ranges from conservative to resection of mass occupying lesions.

Images for this section:

Fig. 1: (a) Coronal T1 weighted image of the ankle demonstrates the tibiotalar joint (blue arrow) and distal tibiofibular joint (red arrow). The osseous structures include...
the tibia (T), tibial plafond (Tib Plaf), medial malleolus (MM), lateral malleolus (LM), talus (Tal), superior faces (SF), lateral malleolar facet (LMF), medial malleolar facet (MMF), calcaneus (C), peroneal tubercle (PT), and sustentaculum tali (ST). (b) Sagittal T2 weighted image again demonstrates the tibiotalar joint (blue arrow), as well as the subtalar joint (yellow arrow). The talus to include the head (TH), neck (TN), and body (TB) are noted, as well as the calcaneus (C).

**Fig. 2:** (a,b) Axial and coronal T1. The tendons of the ankle are divided into anterior (blue), posterior (green), medial (yellow), and lateral (red) compartments. The anterior compartment contains the tibialis anterior tendon, extensor hallucis longus tendon, extensor digitorum longus tendon, and peroneus tertius tendon. The posterior compartment contains the Achilles tendon. The medial compartment contains the tibialis posterior tendon, flexor digitorum longus tendon, and flexor hallucis longus tendon. The lateral compartment contains the peroneus longus and peroneus brevis tendons. Normal tendons are uniformly low in signal intensity throughout their course.
**Fig. 3:** Anterior tendons. (a,b) Axial and coronal T1. From medial to lateral - TA (blue arrows), EHL (yellow arrows), EDL (red arrows), and PT (orange arrows). The asterisk (a) represents the extensor retinaculum.
**Fig. 4:** Achilles tendon. (a) Axial T1. The Achilles tendon (red arrow) is concave anteriorly and convex along the posterior margin. (b) Sagittal PD FS. The normal Achilles tendon inserts onto the posterior surface of the calcaneus (blue arrow).

**Fig. 5:** Medial tendons. (a) Axial T1. From lateral to medial - FHL (blue arrow), FDH (red arrow), TP (red arrow). The neurovascular bundle (purple ellipse) is located between the FHL and FDL. (b) Sagittal PD FS. The PT (yellow arrow) and FHL (red arrow) pass posterior to the medial malleolus (MM), with the FHL coursing posterolateral to the PT in the region of the tarsal tunnel. (c) Coronal T1. Superomedial to inferolateral - PT (yellow arrow), FDL (red arrow), FHL (blue arrow). The FHL passes under the sustentaculum tali (ST) of the calcaneus. (d) Sagittal PD FS. The FHL (blue arrow) is again visualized passing under the sustentaculum tali. (e) Sagittal PD FS. The PT (yellow arrow) is identified inserting onto the navicular (N). (f) Axial T2 FS. The FHL (blue arrow) is visualized crossing deep to the FDL (red arrow) within the plantar midfoot, an intersection known as Henry’s knot.
Fig. 6: Lateral tendons. (a) Axial T1. The PB tendon (blue arrow) and PL tendon (red arrow) course posterior to the lateral malleolus (LM), with the PB tendon anteromedial to the PL tendon. The superior peroneal retinaculum overlies the tendons (orange arrow). (b) Coronal T1. The PB tendon (red arrow) is passes lateral to the PL tendon (blue arrow). (c) Sagittal PD FS. The PB tendon inserts onto the base of the 5th metatarsal (5th met). The PL tendon (red arrow) is partially visualized posterior to the PB tendon. (d,e) Axial and coronal T1. The PB tendon (blue arrow) passes anterosuperior and the PL tendon (blue arrow) courses posteroinferior to the peroneal tubercle of the calcaneus (PeT). (f) Sagittal PD FS. The PL tendon (red arrow) crosses the plantar aspect of the midfoot.
Fig. 7: (a-d) Ankle ligaments. Coronal and axial T1 weighted MR images. The ankle is bound by the deltoid ligament medially (red), the lateral collateral ligamentous complex laterally (blue), and the syndesmotic ligaments (yellow). Normal ligaments are predominately low in signal intensity, but may contain longitudinal streaks of intermediate signal intensity.
Fig. 8: Deltoid ligament. (a) Coronal T2 FS. The tibionavicular ligament (blue arrow) is seen coursing along the medial aspect of the talus. (b) Coronal T2 FS. The tibiospring ligament (red arrow) arises from the tip of the medial malleolus (MM), and joins the spring ligament complex distally. The flexor retinaculum (yellow arrow) overlies the deltoid ligament. (c) Coronal T2 FS. The tibiocalcaneal ligament (green arrow) connects the medial malleolus to the sustentaculum tali. The PT (purple arrow) and FDL (pink arrow) course medial to the ligament. (d) Coronal T2 FS. Connecting the medial malleolus to the talar neck is the anterior tibiotalar ligament (orange arrow). (e) Coronal T2 FS. The posterior tibiotalar ligament (white arrow) attaches the medial malleolus to the medial talar body. (f) Axial T2 FS. The anterior (orange arrow) and posterior (white arrow) tibiotalar ligaments demonstrate normal inhomogeneous signal intensity.
**Fig. 9:** Lateral ligaments. (a) Axial T1. The ATFL (blue arrow) extends from the distal lateral malleolus to the lateral talus. (b) Axial T2. The PTFL (orange arrow) is a broad band between the posterior tip of the LM and the lateral talus. (c) Coronal T2. The PTFL (orange arrow) is again seen extending from the distal LM to the talus. The posterior tibiofibular ligament (yellow arrow) lies superior to the PTFL, and attaches the LM to the tibial plafond (Tib Plaf). The normal inhomogeneous appearance of these ligaments is secondary to intervening fat. (d) Coronal T1. The CN ligament (red arrow) arises from the tip of the LM, and courses medial to the peroneal tendons (purple ellipse) to attach to the lateral calcaneus.
**Fig. 10:** Syndesmotic ligaments. (a,b) Axial and coronal T1. The anterior tibiofibular ligament extends from the distal fibular to the distal tibia, just proximal to the tibiotalar joint. (c,d) Axial and coronal T1. The posterior tibiofibular ligament (yellow arrows) is an inhomogeneous band that joins the posterior distal tibia and fibula. (e,f) Axial and coronal T1. The interosseous ligament (red ellipse) is the distal continuation of the interosseous membrane, uniting the distal tibia and fibula.

![Fig. 10](image)

**Fig. 11:** Sinus tarsi. (a) Coronal T1. The normal fat is visualized within the sinus tarsi (yellow ellipse). (b) Sagittal PD FS. The fat within the tarsal sinus suppresses normally (blue arrow). (c) Axial T1. The talocalcaneal interosseous ligament (red arrow) courses within the sinus tarsi, and functions to maintain apposition of the talus and calcaneus.

![Fig. 11](image)

**Fig. 12:** Osseous contusions. (a) Sagittal PD FS. Increased T2 weighted signal intensity is identified within the talus (blue arrow) and cuboid (red arrow). (b) Coronal T2. Again
identified is increased T2 weighted signal intensity within the talus, consistent with contusion.

**Fig. 13:** Fracture. (a) Coronal T1. A linear T1 weighted hypointense focus (blue arrow) is identified in the lateral malleolus. (b) Coronal T2. Linear T2 weighted hypointense signal is again evident in the lateral malleolus (yellow arrow), with surrounding marrow edema present (red arrow).
Fig. 14: (a) Coronal T1. A subcentimeter osteochondral fracture of the medial talar dome is present (red ellipse). (b) Coronal T2. A rim of T2 weighted hypointense signal is visualized along the margin of the osteochondral defect (yellow arrow). No intervening fluid is present to suggest an unstable injury.
**Fig. 15**: Talocalcaneal coalition. (a) Sagittal PD FS. The middle facet of the subtalar joint is broadened, with osseous (blue arrow) and fibrocartilagenous (yellow arrows) bridging identified. Associated mild edema is also noted in the talus and calcaneus (green arrows). Axial T2 FS. Talocalcaneal coalition of the middle facet is again evident (orange arrow). Increased T2 weighted signal intensity within the adjacent talus and calcaneus (orange arrows) is compatible with edema.
Fig. 16: Osteonecrosis in a 13 year old with leukemia. (a) Coronal T1. Serpiginous low T1 weighted signal intensity (blue arrow) surrounds normal fatty marrow (red arrow). (b) Sagittal T2 FS. The double line sign is visualized, as evidenced by a serpiginous inner margin of increased T2 weighted signal intensity (purple arrow) and an outer margin of decreased T2 weighted signal intensity (yellow arrow). Edema within the talus (green arrow) is concerning for early osteonecrosis.
Fig. 17: Tenosynovitis. (a) Axial T2 FS. Increased T2 weighted signal intensity is visualized about the FHL (blue arrow) greater than the FDL (red arrow) and PT (yellow arrow) tendons. (b) Sagittal T2 FS. Increased fluid is again noted within the FHL tendon sheath (blue arrow). (c) Sagittal T2 FS. A trace amount of increased fluid is noted within the PT tendon sheath.

Fig. 18: Tendinitis. (a) Axial T1. The PL tendon is mildly thickened (blue ellipse). (b) Sagittal PD FS. Again noted is focal, mild thickening of the PL tendon (red arrow).
Fig. 19: Axial T2 FS image demonstrates a split tear of the PB tendon (yellow ellipse).

Fig. 20: High grade partial rupture of the PT. (a) Sagittal PD FS. Increased signal intensity is visualized within the PT tendon distal to the MM (blue ellipse), compatible with partial rupture. (b) Coronal T2 FS. Intermediate T2 weighted signal intensity is visualized within the PT tendon (yellow arrow). The tendon just proximal to this partial rupture is mildly thickened (red arrow).

Fig. 21: Achilles tendon pathology. (a) Sagittal PD FS. A near complete tear of the Achilles tendon is visualized (blue ellipse). The tendon proximal and distal to the rupture demonstrates intermediate signal intensity (yellow arrows), consistent with superimposed severe tendinopathy. (b) Sagittal PD FS. Thickening of the Achilles tendon (orange
arrow) is noted, with minimal intrasubstance intermediate signal intensity, compatible with tendinitis. (c) Sagittal PD FS. Minimal thickening of the Achilles tendon is evident, with increased peritendinous signal intensity present, consistent with peritendinitis. Minimal retrocalcaneal bursitis is also identified (purple ellipse).

Fig. 22: Deltoid ligament sprain. (a,b) Coronal and axial T2 FS. Increased T2 weighted signal intensity is visualized within the posterior deep fibers of the deltoid ligament (yellow arrows), compatible with grade 2 sprain.
**Fig. 23:** Coronal T2 FS. The CN ligament is thickened and intermediate in signal intensity (red arrow). The ligament is discontinuous distally (blue arrow). (b) Axial T2 FS. The CN ligament has been avulsed from it’s lateral calcaneal attachment (blue arrow).

**Fig. 24:** Multi-ligamentous injuries. (a) Axial T2 FS. Intermediate T2 weighted signal intensity within and irregularity of the anterior tibiofibular ligament (blue arrow) is compatible with a partial tear. (b) Axial T2 FS. Complete disruption of the ATFL is evident (red arrow). The PTFL is intact, but appears mildly thickened with increased T2
weighted signal intensity (yellow arrow), consistent with partial tear. (c) Coronal T2 FS. Intermediate T2 weighted signal intensity is visualized within the deep posterior deltoid ligament (orange arrow), compatible with a partial tear. Opposing contusions are seen within the medial malleolus and medial talar dome (green arrows).

**Fig. 25:** Sinus tarsi syndrome. (a) Coronal T1. The normal T1 weighted hyperintense fat signal within the tarsal sinus is replaced by low signal intensity. (b) Coronal T2 FS. Increased T2 weighted signal intensity is visualized in the tarsal sinus, which does not suppress, consistent with edema (yellow ellipse). (c) Sagittal PD FS. Again, the fat within the tarsal sinus does not suppress, and remains high in signal intensity (red arrow).
Fig. 26: Os trigonum syndrome. Sagittal PD FS. A prominent os trigonum is evident (blue arrow). Significant surrounding soft tissue edema is present (yellow arrows). Increased signal intensity is also visualized within the accessory os and posterior talus (orange arrows), consistent with marrow edema.
Fig. 27: Tarsal tunnel syndrome. (a) Axial T1. There is marked enlargement of the tibial nerve (blue ellipse). (b) Axial T1. Post-surgical changes of prior flexor retinaculum release are visualized (red arrow), as evidenced by thickening and discontinuity of the retinaculum and overlying scarring of the subcutaneous tissues. (c) Axial T2 FS. Enlargement of and intermediated T2 weighted signal intensity within the abductor
hallucis muscle (yellow arrow) are evident. (d) Sagittal PD FS. Enlargement of and edema within the flexor digitorum brevis muscle (purple arrow) are also identified. This constellation of findings is consistent with tarsal tunnel syndrome and compression of the plantar branch of the tibial nerve.

**Fig. 28:** Baxter Neuropathy. (a,b) Sagittal and axial T1. Isolated fatty atrophy of the abductor digiti minimi muscle, consistent with chronic compression of the inferior calcaneal nerve.
Conclusion

In the past two decades, there has been an upsurge in the use of MR imaging for evaluating pathologic conditions of the ankle. MRI remains the modality of choice for evaluating tendons, ligaments, and other soft tissue structures, as well as some osseous abnormalities, as it is invaluable and unparalleled in the detection of many processes not visualized on other imaging modalities. Knowledge of the complex anatomic structures that comprise the ankle is crucial in proper assessment and diagnosis of these pathologic processes.

Personal information

References


