Ankle: Update

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MR imaging has established itself as a very useful diagnostic tool for the evaluation of ankle disorders. In large part, this is due to its multiplanar capability, noninvasiveness, and its ability to accurately assess a wide spectrum of pathology.

TECHNIQUE

The ankle is best evaluated using a dedicated quadrature and/or phased array extremity coil. This permits high spatial resolution allowing assessment of fine anatomic detail such as ligaments and tendons. Including the opposite side for comparison is not worth the loss of signal-to-noise, and decreased spatial resolution necessitated by using a larger coil and larger field of view except in unusual cases. We use a 12-14 cm field of view with 3-4mm slice thickness.

Frequently utilized pulse sequences include: conventional T1-weighted spin-echo; T2-weighted fast spin-echo (FSE) with fat suppression; and FastSTIR. Our favorite protocol includes a T1 and FSE T2 (TE~40-60) with fat suppression in all three planes.

Pulse sequence protocol should be altered based on the clinical indication. Imaging is generally performed with the ankle in a neutral position which is a comfortable position for the patient (10-20 degrees plantar flexion) thus decreasing motion artifact. In this position, there are usually few problems with patient motion. Occasionally, altered positioning of the ankle joint can be helpful if a particular anatomic structure is to be evaluated. For example, the calcaneofibular ligament is best seen on axial images with the foot in 40-50 degrees of plantar flexion.

The presence of orthopedic hardware can significantly compromise image quality by producing artifacts secondary to magnetic susceptibility. These artifacts are reduced by utilizing conventional spin-echo and fast spin-echo sequences as opposed to gradient recalled echo sequences. Such ferromagnetic artifacts are further reduced by imaging on low field rather than high field strength MR systems.

In general, the axial images are best for assessing the ligaments and tendons. The sagittal images demonstrate the talar dome, sinus tarsi, and provide a second look at the tendons. The coronal FSE T2-weighted images best assess the ankle mortise and articular cartilage.

TENDONS

Normal tendons are uniformly low signal intensity on all pulse sequences. The spectrum of tendon pathology includes: tenosynovitis; peritendonitis; tendonitis; partial or complete tears; and subluxation or dislocation. Tenosynovitis represents inflammation of the synovial-lined tendon sheath which, on MR imaging, manifests fluid signal intensity in the tendon sheath associated with normal tendon morphology and signal intensity. Peritendonitis is pathophysiologically similar to tenosynovitis, but occurs in the loose connective tissue (the peritenon) surrounding those tendons which do not have a tendon sheath. In the ankle, this occurs only with respect to the Achilles tendon. Tendonitis is characterized by increased intratendonous signal on T2-weighted, T2*-weighted, and STIR images. The morphology of the tendon is also frequently abnormal in tendonitis with focal thickening or thinning frequently present depending on whether it is acute or chronic. On MR imaging, it may be impossible to differentiate tendonitis from a partial thickness tear. A clinical history of abrupt onset in the setting of trauma favors the diagnosis of a partial tear in such cases. The diagnosis of a complete tendon tear or rupture is highly suggested on MR imaging when a gap is present in the tendon. This frequently results in a "mop end" appearance of the torn ends of the tendon with fluid/hemorrhage signal intensity in the tendon gap. Such a completely torn tendon may retract significantly and may assume a wavy contour. The diagnosis of a tendon subluxation or dislocation is suggested on MR imaging when there is a change in the normal tendon position.

There are ten major tendons which cross the ankle joint. These can be divided into plantar flexors and extensors (dorsiflexors). Medially, the flexors include the tibialis posterior, flexor digitorum longus, and flexor hallicus longus. Laterally, the flexors include the peroneus longus and brevis. Posteriorly, the only flexor is the Achilles tendon. The extensors are located anteriorly and include the tibialis anterior, extensor hallicus longus, extensor digitorum longus, and peroneus tertius. These tendons are best seen in the axial plane on MR imaging. In approximate rank order of incidence, tendon pathology affects the ankle as follows: Achilles; tibialis posterior; flexor hallicus longus, peroneus longus and brevis; and tibialis anterior.

The Achilles tendon is derived from the gastrocnemius and solius muscles. It is the largest and strongest tendon in the body. Unlike most tendons, the Achilles tendon lacks a tendon sheath. Inflammation adjacent to the Achilles tendon, therefore, manifests as peritendonitis with/without associated retrocalcaneal bursitis. Ruptures of the Achilles tendon generally occur approximately 4-6 cm above the tendon insertion on the calcaneus where the blood supply is somewhat tenuous. This injury occurs most commonly in sedentary middle-aged men who have recently taken up strenuous exercise. The degree of injury in partial tears and the degree of separation in complete tears is well documented with sagittal MR imaging. In approximately 25% of cases an incorrect diagnosis is arrived at clinically. Conservative versus operative repair of torn Achilles tendons remains controversial. MR imaging does allow early identification of the injuries most likely to benefit from surgical therapy including those in which the tendon ends are grossly separated by hematoma. Following successful surgical repair, the Achilles tendon is usually significantly thickened. This may be impossible to differentiate from chronic tendonitis.

The tibialis posterior tendon is the principle invertor of the foot and is the major hindfoot stabilizer. This tendon maintains and supports the medial longitudinal arch of the foot. Chronic rupture of this tendon classically occurs in overweight middle-age females who present with an acute, painful flatfoot deformity which progressively worsens. The point of rupture is most commonly where the tendon passes beneath the medial malleolus. Other risk factors for spontaneous rupture of this tendon include rheumatoid arthritis, seronegative spondyloarthropathies, and steroid therapy. Traumatic rupture of this tendon can occur in young athletes. MR imaging accurately identifies and stages tears of the tibialis posterior tendon. Undiagnosed tibialis posterior tendon rupture results in progressive flatfoot deformity, and debilitating secondary osteoarthritis that may require subtalar joint arthrodesis. Early cases may be detected with MR imaging prior to complete rupture; these patients frequently benefit from synovectomy and debridement of the partially torn tendon. In chronic cases, the flexor digitorum tendon can be transferred to the navicular bone.

The flexor hallicus longus tendon is particularly susceptible to injury in people performing repetitive pushoff maneuvers. It is referred to as the "Achilles tendon of the foot" in ballet dancers reflecting the relatively high frequency of injury in this group of patients. While tenosynovitis and partial tears are relatively common, complete rupture of this tendon is unusual. It should be noted that in 20% of normal individuals, there is a communication between the ankle joint space and the tendon sheath of flexor hallicus longus. Tenosynovitis, therefore, should only be diagnosed on MR imaging when the amount of fluid in the tendon sheath is disproportionately greater than the amount in the joint space.

The peroneus longus and brevis tendons are the principle evertors and lateral dynamic stabilizers of the foot. These tendons pass behind the lateral malleolus with peroneus brevis anterior to peroneus longus. They are held in place by the superior and inferior peroneal retinacula. Disruption of the peroneal retinaculum can result in lateral dislocation/subluxation of the peroneal tendons. MR imaging best demonstrates this abnormal anterior subluxation of the tendons on axial images. A longitudinal split of the peroneus brevis tendon may occur in association with this injury due to "bowstringing" of the tendons. This injury is also associated with lateral collateral ligament sprains. Fluid in the peroneal tendon sheath is never normal and in the setting of a lateral ankle sprain suggests a possible tear of the underlying calcaneofibular ligament. Tears of the peroneus longus tendon also occur in the midfoot. This can be associated with marrow edema along the lateral calcaneal wall, and a hypertrophied peroneal tubercle.

The tibialis anterior tendon is the principle dorsiflexor of the foot. Repetitive or prolonged plantar flexion such as occurs in downhill running or hiking predisposes to injury. Complete ruptures are unusual, typically occurring in patients over the age of 50. These complete ruptures usually present as painless foot drop and, therefore, need to be differentiated from an L5 radiculopathy.

LIGAMENTS

The ankle joint is supported by an extensive complex of ligaments. These ligaments can be grouped medially into the medial collateral (deltoid) ligament, laterally into the lateral collateral ligament complex, and proximally into the syndesmotic ligament complex. Until recently, assessment of ligamentous injury relied entirely upon clinical examination, stress radiography, tenography, and CT arthrography. MR imaging has emerged as a very useful noninvasive method for evaluation of the ankle ligaments. On MRI, normal ligaments appear taut with uniform low signal intensity on all pulse sequences.

Medially, the triangular deltoid ligament fans out from the medial malleolus to the navicular, talus, and calcaneus. The medial collateral ligament is very strong and injury of this ligament is relatively uncommon. The mechanism of injury is usually forced eversion. Deltoid ligament tears may be associated with fibular fractures, tears of the syndesmotic ligament complex, or tears of the lateral collateral ligament complex.

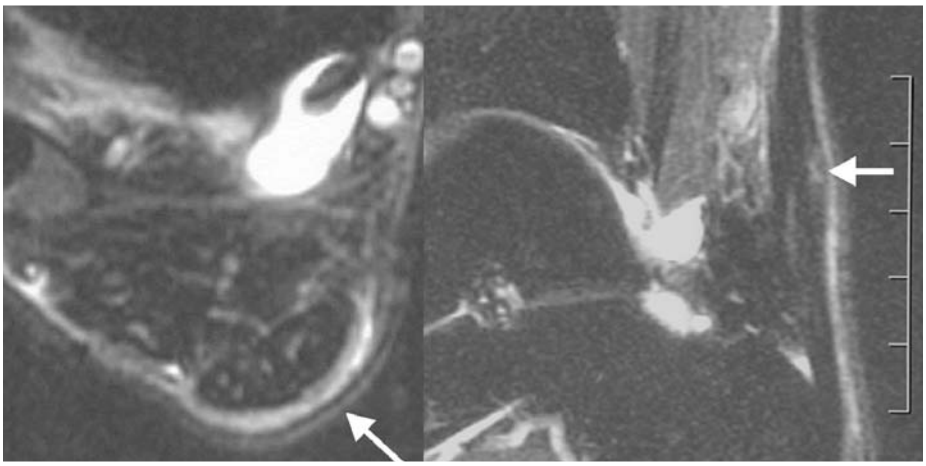
Injury of the lateral collateral ligament complex is the most frequent ligamentous injury of the ankle. In fact, this is the most common of all sports injuries and is commonly referred to as an ankle sprain. This ligamentous complex consists of three components: the anterior talofibular ligament, the calcaneofibular ligament, and the posterior talofibular ligament. It is important not to confuse the talofibular ligaments for the more proximal tibiofibular ligaments. On axial MR images, the talofibular ligaments are seen at the level of the malleolar fossa of the fibula. The tibiofibular ligaments are seen proximal to this level. Injury involving the lateral collateral ligament is most commonly due to an inversion mechanism. Of the three components of this ligamentous complex, the anterior talofibular ligament is the weakest and most frequently injured component. With more severe inversion, the calcaneofibular ligament may also be injured. Isolated tears of this ligament are very rare; disruption invariably is associated with anterior talofibular ligament rupture. Posterior talofibular ligament injury is the least common and invariably occurs only when both the anterior talofibular and calcaneofibular ligaments are also injured.

The syndesmotic ligament complex consists of the anterior and posterior tibiofibular ligaments. The most inferior portion of the posterior tibiofibular ligament is also referred to as the inferior tibiofibular ligament and normally extends inferiorly beyond the bony margin of the tibia as seen on sagittal MR images. This normal finding must not be mistaken for an intra-articular loose body or avulsion fracture fragment. Isolated tears of the syndesmotic ligament complex are uncommon. This injury is usually associated with fracture of the medial malleolus or rupture of the deltoid ligament. This particular injury complex is almost invariably associated with a fibular fracture proximal to the lateral malleolus.

On MR imaging, a torn ankle ligament may be seen as attenuated, wavy, lax, or discontinuous. In the acute setting, associated secondary findings include overlying soft tissue edema, evidence of capsular disruption with fluid evident in the anterior soft tissues (anterior talofibular ligament tear), or fluid within the peroneal tendon sheaths (calcaneofibular ligament tear).

SINUS TARSI SYNDROME

The sinus tarsi is an anatomic space bordered superiorly by the inferior aspect of the talus, and inferiorly by the superior aspect of the calcaneus just anterior to the posterior subtalar joint. It continues medially as a funnel-shaped space known as the tarsal canal just behind the sustentaculum tali and below the medial malleolus. The sinus tarsi normally contains fat, a neurovascular bundle, a small synovial bursa, and ligamentous structures. The ligaments of the sinus tarsi include the cervical ligament, talocalcaneal (interosseous) ligament, and the inferior extensor retinaculum. The sinus tarsi syndrome is characterized by pain in the



Left: *Axial posterior paratenonitis* ***Right:*** *Sagittal achilles partial tear* lateral aspect of the foot, overlying the sinus tarsi region, associated with the sensation of hindfoot instability. The pain is often relieved by injection of local anesthetic. Most commonly, this syndrome occurs as a sequelae of an inversion ankle injury associated with lateral collateral ligament complex injury. Trauma accounts for approximately 70% of the cases. Inflammatory conditions (rheumatoid arthritis, ankylosing spondylitis, gout) and foot deformities (pes cavus, pes planus) account for the remaining 30% of cases. The most reliable MR imaging finding associated with the sinus tarsi syndrome is replacement of the normal fat signal intensity within the sinus tarsi by edema/inflammatory tissue demonstrating low signal intensity on T1-weighted images and high signal intensity on T2-weighted or STIR images. Associated injuries of the lateral collateral ligament complex and tibialis posterior tendon should also be looked for on the MR images. Conservative treatment includes injection of local anesthetics and steroids. Patients not responding to conservative therapy are amenable to surgical treatment including complete excision of the inflammatory tissue filling the sinus tarsi.

TARSAL TUNNEL SYNDROME

The tarsal tunnel is a fibroosseous tunnel occupying the medial-posterior aspect of the ankle. It extends from the level of the medial malleolus to the level of the tarsonavicular. The floor consists of the medial surface of the talus, the sustentaculum tali, and the calcaneus. The flexor retinaculum forms the roof. Tarsal tunnel syndrome refers to an entrapment neuropathy of the posterior tibial nerve as it passes through this tunnel. This syndrome is characterized by pain or paresthesias along the plantar aspect of the foot and toes extending into the medial aspect of the heel. Symptoms tend to worsen with weight bearing. Etiologies include space-occupying lesions within the tarsal tunnel such as ganglion cysts, varicosities, lipomas, neurogenic tumors, and synovial hypertrophy due to rheumatoid arthritis. Alternatively, traction forces on the nerve due to post-traumatic scarring/fibrosis may be responsible. In many cases, the syndrome is idiopathic, possibly due to a taut flexor retinaculum. MR imaging demonstrates clearly the contents of the tarsal tunnel and is extremely useful to evaluate the presence and extent of lesions causing the tarsal tunnel syndrome. Treatment consists of surgical release of the flexor retinaculum and excision of any compressive lesion.

TARSAL COALITION

Tarsal coalition represents fibrous, cartilaginous, or osseous fusion of the tarsal bones which is either congenital or acquired. While congenital coalitions are present at birth, clinical presentation and diagnosis frequently do not occur until the second decade of life when ossification of the fibrous or cartilaginous connection occurs. In rank order of incidence, the types of tarsal coalition include: calcaneonavicular; talocalcaneal; talonavicular; and calcaneocuboid. Conventional radiographic signs may be subtle or absent prior to skeletal maturation. Osseous coalition is diagnosed equally well with either CT or MRI. Recent comparative studies suggest MR imaging is more accurate than CT in detecting and characterizing nonosseous coalitions.

AVASCULAR NECROSIS

The most common site of osteonecrosis in the ankle is the talar dome. AVN involving the talar dome may results in collapse of an important weight-bearing surface and premature degeneration of the joint. It is most commonly encountered as a complication of corticosteroid therapy. Avascular necrosis of the talus may also occur following a fracture of the talar neck which interrupts the blood supply to the proximal talus. As in the femoral head, MR imaging may demonstrate serpentine low signal intensity surrounding a fat signal intensity necrotic fragment. Associated marrow edema may also be seen as well as a “double line” sign which improves specificity. These findings are best seen in the coronal and sagittal planes.

Osteochondral Lesions

The terminology used to characterize osteochondral lesions of the talar dome has been a source of controversy and confusion. The terms osteochondral injury or osteochondral lesion are currently most accepted. These lesions have also been referred to as osteochondral fractures, transchondral fractures, or osteochondritis dissecans. These lesions are now considered to be posttraumatic in etiology. These lesions are most often seen in young adult males from 15-35 years. Lateral lesions are due to impaction of the talar dome against the lateral malleolus while medial lesions are due to impaction against the posteroinferior lip of the tibia.

MR imaging may detect radiographically occult disease as well as accurately stage the osteochondral injury. Stage I lesions represent subchondral bone contusions with intact overlying articular cartilage. Although the treatment is controversial, some advocate immobilization and nonweight-bearing of Stage I lesions to prevent progression to more advanced osteochondral lesions. Stage II lesions represent subchondral osseous injuries that extend and involve the articular surface cartilage. Stage III lesions demonstrate separation of the fragment from the subchondral bone without displacement. Treatment of Stage II and Stage III lesions depends in part on the stability of the fragment. Recent experience with MR imaging suggests that fragments are unstable when subchondral cystic lesions are seen or when fluid signal intensity is seen surrounding the base of the fragment. Stable fragments are immobilized and treated with nonweight-bearing. Large stable fragments may be treated surgically with drilling to facilitate revascularization or pinning. Advanced lesions with unstable fragments may be curettaged. Stage IV lesions are characterized by complete displacement of the fragment with an intraarticular loose body. Stage IV lesions are treated with surgical removal of the osteochondral loose body and debridement of the osteochondral injury site. It is important to note that the signal intensity of the osteochondral fragment has been reported to have no correlation with stability of the fragment.

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