RadioGraphics

US and MR Imaging of the Extensor Compartment of the Ankle[1]

Joshua M. Ng, MD • Zehava Sadka Rosenberg, MD • Jenny T. Bencardino, MD • Zoraida Restrepo-Velez, MD • Gina A. Ciavarra, MD • Ronald Steven Adler, MD, PhD

SA-CME

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LEARNING OBJECTIVES FOR TEST 5

After completing this journal-based SA-CME activity, participants will be able to:

- Describe the normal anatomy of the ankle extensor compartment, including the anterior tibial, extensor hallucis longus, and extensor digitorum longus tendons; the extensor retinacular mechanism; and the anterior tarsal tunnel.
- Identify US and MR imaging features of common pathologic conditions of the ankle extensor compartment, including tenosynovitis, tendinosis, tendon tear, and retinacular injury.
- Recognize common pitfalls in US and MR imaging of the ankle extensor compartment.

TEACHING POINTS

See last page

Injuries to the extensor compartment of the ankle are uncommon and often are overlooked or misinterpreted at clinical presentation. Ultrasonography (US) and magnetic resonance (MR) imaging play a critical role in the diagnosis and evaluation of these injuries. US is a dynamic, quick, cost-effective imaging method for assessing ankle extensor compartment injuries as an alternative or adjunct to MR imaging. MR imaging provides multiplanar cross-sectional delineation of regional anatomic structures and also can be used to assess the predisposing cause, estimate the extent of injury, and aid in preoperative planning for requisite surgical repair. The spectrum of pathologic conditions affecting the ankle extensor compartment ranges from tendinosis secondary to degenerative, inflammatory, or depositional disease to traumatic tendon or retinacular rupture and entrapment neuropathy. Major components of the ankle extensor compartment at risk for injury include the anterior tibial, extensor hallucis longus, and extensor digitorum longus tendons; the extensor retinacular mechanism; and the anterior tarsal tunnel. Familiarity with the normal anatomic appearance and pathologic features of the ankle extensor compartment at US and MR imaging as well as potential imaging pitfalls is critical for accurate injury evaluation.

Introduction

Injuries to the extensor compartment of the ankle often are overlooked yet require prompt diagnosis and treatment to preserve function and produce a desirable outcome (1–3). Any structure within the ankle extensor compartment is subject to injury, with the most frequent injuries involving the tendons, most commonly the anterior tibial (AT) tendon followed by the extensor hallucis longus (EHL) and extensor digitorum longus (EDL) tendons.

Abbreviations: AT = anterior tibial, DPN = deep peroneal nerve, EDL = extensor digitorum longus, EHL = extensor hallucis longus, TE = echo time

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[1]From the Department of Radiology, New York University Langone Medical Center, Hospital for Joint Diseases, 301 E 17th St, New York, NY 10003 (J.M.N., Z.S.R., J.T.B., G.A.C., R.S.A.); and CediMed, Medellin, Colombia (Z.R.V.). Recipient of a Cum Laude award for an education exhibit at the 2011 RSNA Annual Meeting. Received August 22, 2012; revision requested September 20; final revision received June 17, 2013; accepted June 24. For this journal-based SA-CME activity, the authors, editor, and reviewers have no financial relationships to disclose. **Address correspondence to** J.M.N. (e-mail: *Joshua.ng2@nyumc.org*).

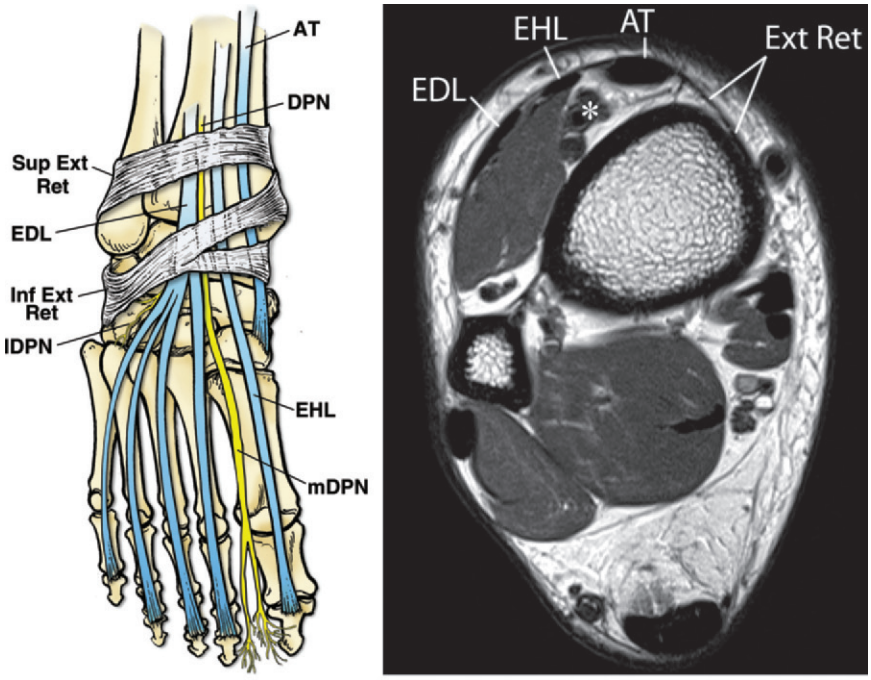


Figure 1. Normal anatomy of the ankle extensor compartment. **(a)** Frontal drawing of the foot and ankle shows the AT, EHL, and EDL tendons. The deep peroneal nerve (*DPN*) and its medial *(mDPN)* and lateral *(lDPN)* branches are shown descending deep to the transversely oriented superior extensor retinaculum *(Sup Ext Ret)* and Y-shaped inferior extensor retinaculum *(Inf Ext Ret)*. **(b)** Axial T1-weighted MR image shows the normal relationships of the AT, EHL, and EDL tendons as well as the superior extensor retinaculum *(Ext Ret)* and the DPN (∗) within the neurovascular bundle.

Injury to the ankle extensor compartment is relatively uncommon, in part because the straight course of the extensor tendons compared with that of other tendons in the ankle protects them from biomechanical stress (4). Injuries to the ankle extensor compartment often are overlooked because of a low index of clinical suspicion and relatively benign findings at physical examination. Radiologists may neglect to assess the ankle extensor compartment because of the rarity of injuries to this area and the prevalence of the "magic angle" phenomenon, which artifactually elevates the magnetic resonance (MR) imaging signal of the extensor tendons where they curve around the ankle. The frequent occurrence of this signal artifact may result in false-positive findings and erroneous dismissal of real findings of disease (5).

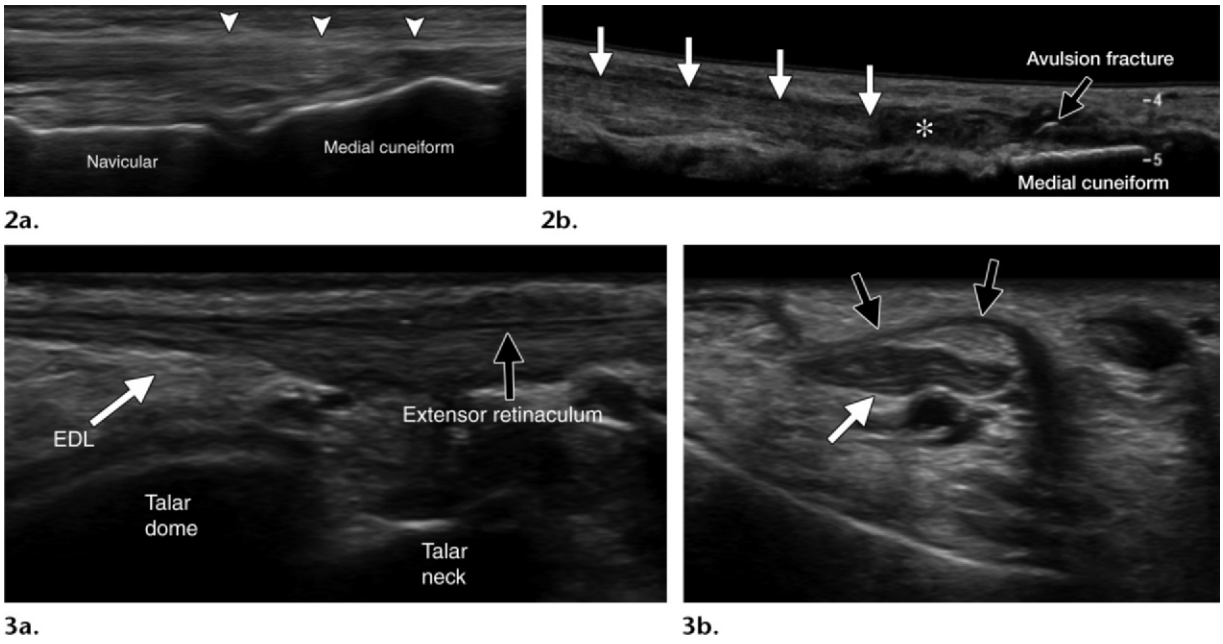
Ultrasonography (US) is a noninvasive, operator-dependent imaging modality useful for diagnosis of injuries to the ankle extensor compartment (6,7). The superficial location of the ankle extensor compartment allows easy depiction at US. US is an easily accessible, cost-effective method for dynamic evaluation of the ankle extensor structures and can be performed at the patient's bedside.

MR imaging is a noninvasive, operator-independent modality that allows cross-sectional anatomic evaluation of the ankle extensor compartment. MR imaging has become a mainstay in the diagnosis and workup of ankle extensor compartment injuries (8,9) that allows radiologists to gauge the extent of injury, identify culprit cofactors of disease predisposition, and provide valuable preoperative information in cases that require surgical repair (10,11).

This article discusses the normal anatomy and pathologic conditions of the ankle extensor compartment. US and MR imaging features as well as potential imaging pitfalls are presented. Components of the ankle extensor compartment include, from medial to lateral, the AT, EHL, and EDL tendons; the superior and inferior extensor retinacula; and the anterior tarsal tunnel (Fig 1). Primary focus is given to the ankle extensor tendons because tendinous injury accounts for the vast majority of diseases of the ankle extensor compartment.

US General Concepts

Normal tendons appear at US as uniformly echogenic bands with an internal fibrillar echotexture (Figs 2, 3). The surrounding paratenon appears as an echogenic line demarcating the tendon margin (7). High-frequency US provides higher-resolution imaging at the cost of decreased penetration and is recommended for imaging the superficially located ankle extensor compartment. The transducer should be held in true parallel or perpendicular alignment to the tendon during



2a. 2b.

3a. 3b.

Figures 2, 3. **(2)** Longitudinal US images of normal and torn AT tendons. **(a)** The fibrillar structure of a normal hyperechoic AT tendon (arrowheads) is shown near the medial cuneiform insertion site. **(b)** A torn AT tendon manifests with a tendon gap (*), thickening and retraction of the proximal tendon stump (white arrows), and a distal, fleck-like, hyperechoic focus (black arrow), findings compatible with avulsion fracture of the medial cuneiform. **(3)** Normal US appearance of the EDL tendon and its relationship to the extensor retinaculum. Longitudinal **(a)** and axial **(b)** images show the normal hyperechoic EDL tendon (white arrow) at the level of the talus. The extensor retinaculum (black arrows) appears as a thin curvilinear band of hypoechoic tissue superficial to the EDL tendon.

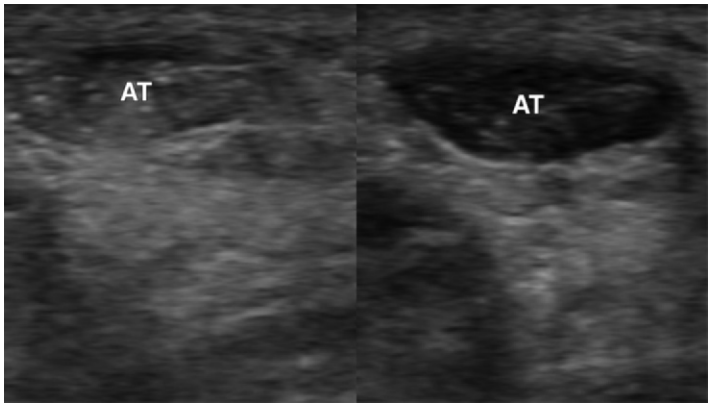


Figure 4. Axial dual-mode US images of the AT tendon with the transducer held in a true perpendicular position relative to the tendon fibers (left) and at an oblique angle relative to the tendon fibers (right). Note the artifactual hypoechogenicity of the tendon fibers when imaged at an oblique angle, a US artifact known as anisotropy that can be used to distinguish a tendon from an adjacent nonanisotropic structure.

longitudinal or transverse imaging, respectively, to avoid anisotropy and to ensure that the ultrasound beam is perpendicular to the tendon fibers. Anisotropy is a pitfall that occurs when the ultrasound beam is oriented oblique to the tendon fibers, resulting in an artifactually hypoechoic tendon that may mimic tendinopathy on US images (Fig 4) (12). However, anisotropy can be used advantageously at US to distinguish a tendon from an adjacent nonanisotropic structure such as a nerve (13).

US provides the imaging physician with the invaluable ability to confer with the patient at the time of study. US also allows the injection of diagnostic or therapeutic steroids and platelet-rich plasma into the site of pain. The injectate highlights tendon anatomy and disease by better outlining the tendon margins with fluid (Fig 5) and emphasizing linear clefts and tears within the tendon.

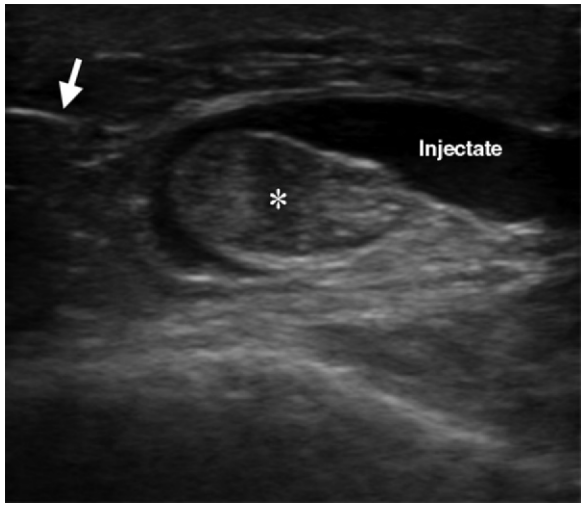
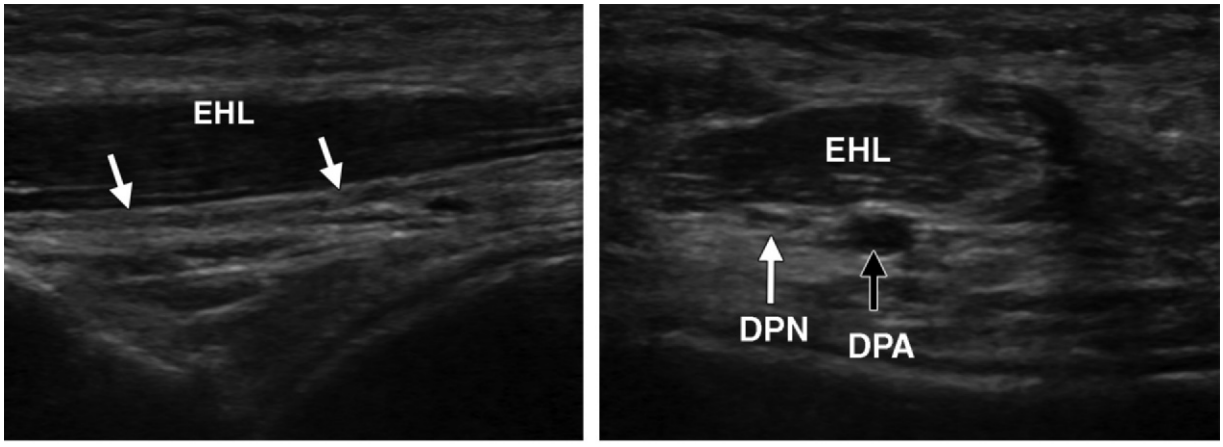


Figure 5. Axial US image obtained immediately after an injection into the AT tendon sheath. The injectate surrounds and highlights the anatomy of the tendon (*) within the sheath. The needle (arrow) is shown immediately after its withdrawal from the tendon sheath.



a. b.

Figure 6. Normal US appearance of the DPN. Longitudinal **(a)** and short-axis **(b)** US images show the DPN (white arrows) as a hypoechoic fasciculated structure running deep to the EHL tendon. Unlike its appearance at MR imaging, the DPN can be distinguished at US from the deep peroneal artery *(DPA)* (black arrow in **b**) by its fasciculated appearance and by the presence of color Doppler flow in the deep peroneal artery.

US Features of Tendon and Retinacular Injury

At US, tendinosis manifests as enlargement of the tendon diameter with increased hypoechoic space between echogenic fibrils (7). Dystrophic calcifications and cystic changes consistent with mucoid degeneration may be seen. Tenosynovitis, or inflammation of the tendon sheath, appears as anechoic fluid in the tendon sheath with or without concurrent tendinosis. A partial tendon tear manifests as an incomplete discontinuity or focal defect within the fibrillar architecture. A complete tendon tear appears as a discrete gap within the tendon that is filled with either a hematoma (in acute injury) or scar and granulation tissue (in chronic injury), with associated retraction of the proximal and distal tendon stumps (Fig 2) (7).

US demonstrates the retinaculum as a well-delineated, thin, hypoechoic band in close relation to the extensor compartment tendons (Fig 3). Injury to the extensor retinaculum manifests at US as a thickened, irregular, hypoechoic, or discontinuous retinaculum, with a highly vascular pattern seen at color Doppler US (14).

Unlike their appearance at MR imaging, the nerves of the anterior tarsal tunnel, including the DPN and its medial and lateral branches, are easily distinguished at US from their accompanying vessels. The nerves appear as ovoid fasciculated structures on transverse images and as multiple parallel hypoechoic linear areas separated by hyperechoic bands (Fig 6) (15). An injured nerve may demonstrate swelling and edema or entrapment neuropathy caused by direct compression by normal anatomic structures or pathologic lesions. US has the additional advantage of depict-

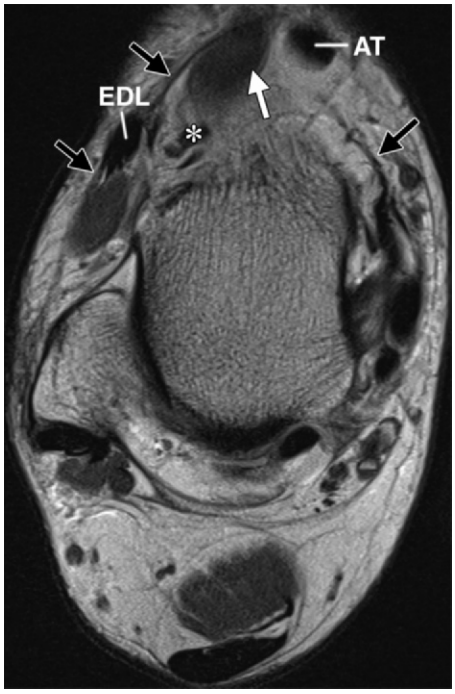


Figure 7. Magic angle effect. Axial T1-weighted MR image shows spuriously increased signal intensity of the EHL tendon (white arrow) at the point of greatest tendon obliquity (approximately 55°) relative to the primary magnetic field, a finding that may be mistaken for tendon disease when compared with the normal low signal intensity of the AT and EDL tendons. The normal inferior extensor retinaculum (black arrows) and DPN (*) within the neurovascular bundle are also shown.

ing dynamic nerve compression, a finding that may be missed on static MR images.

MR Imaging General Concepts

Normal tendons demonstrate low signal intensity on both T1-weighted (Fig 1) and fluid-sensitive MR images. The ankle extensor tendons are best assessed by using multiplanar MR imaging. Axial images offer visualization of the ankle extensor compartment tendons, but care should be taken to avoid misinterpreting the magic angle effect as tendon disease. Short-axis (oblique coronal) images of the midfoot and forefoot allow optimal assessment of the tendons distal to the ankle joint. Oblique sagittal images along the longitudinal course of the tendon, while most effective for evaluating tendons in their entirety, are inconvenient to obtain but can be indirectly obtained with three-dimensional isotropic imaging. Administration of a gadolinium-based contrast agent is uncommonly necessary to assess the ankle extensor compartment and may be used to assess entities beyond the scope of this article, such as soft-tissue masses and infectious processes.

The extensor retinacula manifest as thin, linear, low-signal-intensity structures on T1-weighted and fluid-sensitive MR images. Because of their transverse course across the ankle joint, they are best evaluated on axial images (Figs 1, 7). The superior extensor retinaculum is seen above the ankle joint at imaging and often is better visualized than the thinner and more distal inferior extensor retinaculum. Sagittal T1-weighted or proton-density–weighted MR images often depict the inferior extensor retinaculum and its roots as extending across the sinus tarsi fat. Visualization of the retinacula can be challenging because of their thin caliber and the magic angle effect.

The DPN traverses within the anterior tarsal tunnel, is longitudinally oriented, and is best depicted on axial images of the ankle and on short-axis images of the midfoot and forefoot (Figs 1, 7). The nerve and its medial and lateral branches exhibit intermediate signal intensity on T1-weighted and fluid-sensitive images and are best visualized on T1-weighted images, where perifascicular and perineural high-signal-intensity fat is most conspicuous (9). The nerve and its branches often are distorted by the magic angle effect and can be difficult to distinguish from adjacent accompanying vessels.

MR Imaging Features of Tendon and Retinacular Injury

Abnormal extensor tendons demonstrate a range of abnormalities at MR imaging depending on the nature and extent of injury. On cross-sectional images, a normal extensor tendon appears round or oval at the ankle joint and flattens out as the tendon travels distally. The AT tendon is the largest, most medial tendon, and its size approximates that of the posterior tibial tendon. The AT tendon normally is less than 5 mm thick within 3 cm of its distal insertion (10) and typically is located deeper than the other ankle extensor tendons.

Teaching Point

Unlike the flexor tendons of the ankle, the synovial sheaths of the ankle extensor tendons typically do not contain fluid. Tenosynovitis should be considered with a finding of even a small amount of fluid. Mechanical or inflammatory tenosynovitis is best seen on cross-sectional axial images of the ankle joint and on short-axis images of the midfoot and forefoot when disease is more distal.

Mild tendinosis usually appears as fusiform tendon thickening and increased signal intensity on T1-weighted or proton-density–weighted images. In advanced tendinosis, intrasubstance degeneration creates additional signal intensity abnormalities and longitudinal clefts visible on T2-weighted images. Partial tendon ruptures demonstrate a similar imaging appearance with more obvious morphologic changes of the tendon. However, advanced tendinosis and partial tears can be indistinguishable. Complete discontinuity of the tendon, with occasional thickening or fraying of the distracted ends, indicates a complete tear (9).

Teaching Point

Associated signs of tendinous injury include fluid in the soft tissues surrounding the tendon and occasional reactive bone marrow signal abnormalities at the osseous insertion sites.

MR imaging characteristics of extensor retinacular injury include retinacular thickening and heterogeneity on both T1- and fluid-sensitive images, retinacular enhancement, and surrounding edema (14,16). Discontinuity may be seen with complete rupture of the retinaculum. Remote retinacular injury is suggested by thickening without associated soft-tissue edema.

Magic Angle Phenomenon

The magic angle effect is a well-documented phenomenon characterized by increased intratendinous MR signal intensity when the tendon fibers are oriented approximately 55° relative to the primary magnetic field, B_0 (Fig 7). This artifactual signal abnormality is mostly seen on short echo time (TE) MR images, such as T1-weighted, proton-density–weighted, and gradient-echo images (5).

Teaching Point

The lack of concurrent tendon signal abnormality on long TE images and the absence of other signs of tendon abnormality prevent misinterpretation of the magic angle effect as tendinous disease.

The magic angle effect is the most frequently encountered pitfall when imaging the ankle extensor compartment and may be seen in all structures imaged with short TE sequences at the susceptible angle. Imaging the foot at an angle of 20° of plantar flexion helps minimize this artifact. Magic angle artifact occurs less commonly at MR imaging of the AT tendon (20% of examinations) (17) and is most pronounced at MR imaging of the EHL tendon.

Individual pitfalls inherent to the imaging of specific tendons and structures are discussed in the following sections.

AT Tendon

Anatomy

The AT muscle, the most medial extensor muscle of the ankle, originates from the proximal third of the lateral tibia, lateral tibial condyle, interosseous membrane, and deep fascia and intermuscular septum. The muscle becomes tendinous at the junction of the middle and distal thirds of the tibia. The proximal AT tendon is surrounded by a synovial sheath of variable length, while the last few insertional centimeters of the tendon are invested by a well-vascularized paratenon (18). During its course along the anteromedial ankle and midfoot, the AT tendon is retained anteriorly by three separate fibro-osseous tunnels formed by the superior extensor retinaculum and the oblique superomedial and oblique inferomedial bands of the inferior extensor retinaculum (Fig 1) (19). Retention against the anterior ankle and foot braces the AT tendon against several bony structures, including the talar head, navicular, and medial cuneiform (Fig 8).

AT tendon insertion sites vary (11,21), but most commonly the distal bifid tendon inserts via a dominant slip into the medial cuneiform and via a thinner slip into the first metatarsal base. Other insertion sites along the medial column of the foot have been documented in the orthopedic literature. Although injury to the AT tendon may not involve osseous insertion sites, evidence of anatomic variability could alter surgical planning and is valuable to the referring clinician (11).

The AT tendon is the largest of the extensor tendons, with a caliber approximately equal to that of the posterior tibial tendon. It is the strongest extensor tendon of the ankle and is responsible for approximately 80% of foot dorsiflexion, with the remainder performed by the EHL, EDL, and peroneus tertius tendons (4,11). The AT tendon also helps support the longitudinal arch and aids in foot supination and inversion.

Pathologic Conditions

The spectrum of injuries affecting the AT tendon includes tenosynovitis, tendinosis, and partial and complete rupture. Tendinosis of the AT tendon has received less attention than rupture, in part because of lack of recognition and delayed diagnosis. An increased prevalence of tendinosis is suspected and may be related to a higher rate of obesity in the general population.

MR imaging is instrumental to accurate diagnosis of AT tendon disease. Several salient features of AT tendinosis have been described (20). Tendinosis under the superior extensor retinaculum

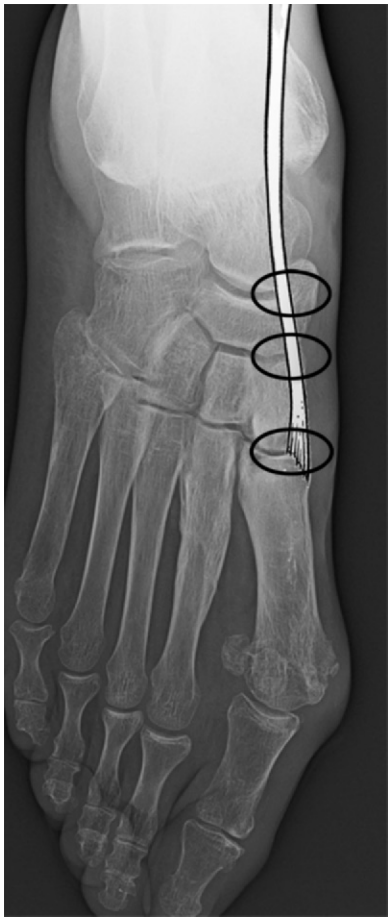


Figure 8. Anteroposterior radiograph shows the normal course of the AT tendon (black outline) over the dorsal aspects of the talar head and talonavicular, medial naviculocuneiform, and medial tarsometatarsal joints. Osteophytes in these regions (circles) have been shown to correlate with AT tendinopathy (3,20).

has been identified in young, athletic individuals, particularly uphill and downhill runners, and is related to overuse and poor conditioning. It also is frequently noted in women between the ages of 50 and 70 years, with a correlation to elevated body mass index or in association with inflammatory arthritis. Patients present with nocturnal or, less commonly, activity-related sharp burning pain at the medial midfoot, with occasional dorsomedial midfoot swelling. Point tenderness at the AT tendon insertion site and relative plantar flexion of the first metatarsal during active foot dorsiflexion support a diagnosis of tendinosis.

The proposed etiology of tendinosis includes an increased medial arch load in patients who are overweight or obese, midfoot arthrosis, and tight-fitting or high-heeled shoewear. Both AT tendon rupture and tendinosis correlate with imaging findings of dorsal osteophytes at the talonavicular, medial naviculocuneiform, and medial tarsometatarsal joints (10). Although this correlation could be coincidental given the prevalence of midfoot arthrosis, it also could reflect direct tendinous attrition from bone abrasion or increased biomechanical stress on the AT tendon as it functions against stiff arthritic joints (20).

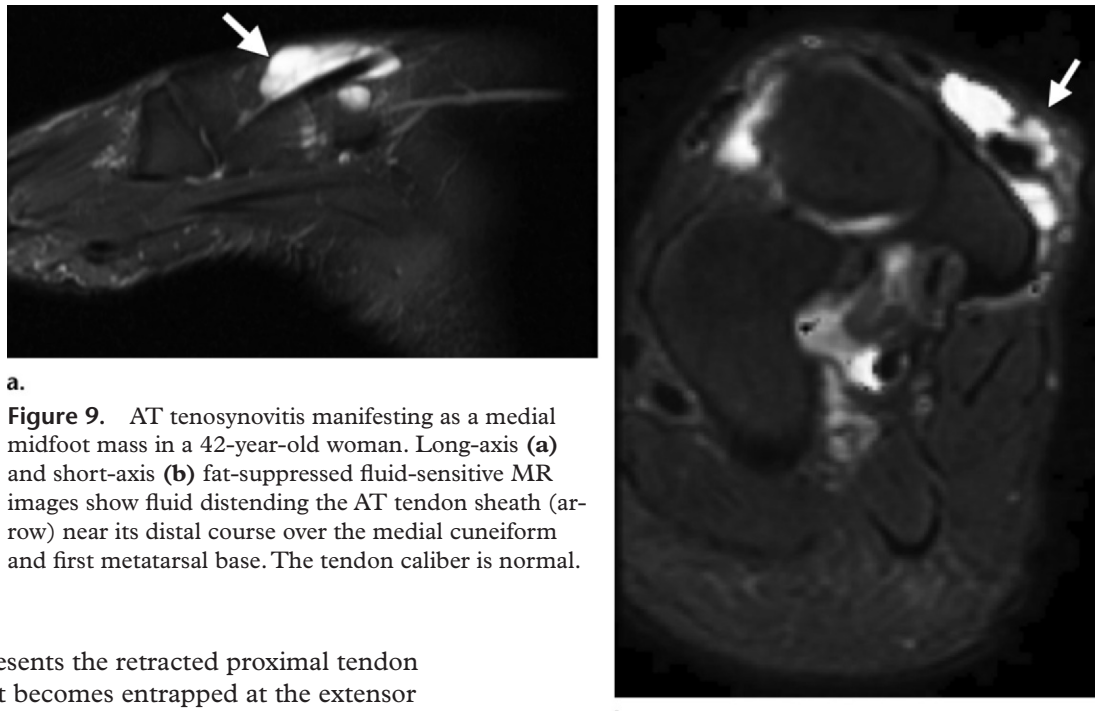
Teaching Point

Rupture of the AT tendon is rare and usually occurs 0.5–3.0 cm proximal to the tendon insertion site, where the tendon passes deep to the inferior extensor retinaculum (2,22,23). Rupture manifests as acute traumatic rupture or more commonly as spontaneous rupture in the setting of chronic tendon degeneration. Younger patients typically experience acute AT tendon rupture from laceration or penetrating injury by a sharp object such as a ski boot, hockey boot blade, or skate blade (24). Traumatic rupture occurs rarely with a closed fracture of the tibia or fibula (25). In contrast, spontaneous AT tendon rupture most commonly affects men aged 60–70 years, results from minor trauma in patients with comorbid medical conditions, and occurs mainly with underlying hypoxic or mucoid tendinous degeneration (26). Predisposing conditions include repetitive microtrauma, inflammatory arthritides, diabetes mellitus, infection, deposition disease such as gout, systemic lupus erythematosus, hyperparathyroidism, and local or systemic steroid therapy (1,27). Spontaneous rupture occasionally has been reported in persons involved in athletic activities such as fencing (11) or cross-country skiing (28).

The classic mechanism of AT tendon rupture is sudden eversion and plantar flexion of a dorsiflexed foot with buckling of the tendon against the sharp edge of the retinaculum (1,11), such as the motion that occurs when a person misses a stair step.

Patients with AT rupture often do not seek immediate medical attention, cannot recall a precipitating event, and present with mild symptoms and subtle clinical findings (2,11,22). These factors potentiate the likelihood of a delayed or missed clinical diagnosis. The typical presentation includes dropped-foot or slapping gait (mimicking DPN palsy or L5 neuropathy), weakness in dorsiflexion or inversion, pain over the anterior ankle and medial aspect of the foot, and swelling over the dorsum of the ankle. Some degree of dorsiflexion often is preserved even with complete AT tendon rupture because of compensatory recruitment by the other ankle extensor tendons.

Physical examination commonly reveals a mass (ie, pseudotumor) over the anterior aspect of the ankle, a palpable defect within the AT tendon, and weakness during dorsiflexion and inversion. A mass over the anterior aspect of the ankle most



a. b.

Figure 9. AT tenosynovitis manifesting as a medial midfoot mass in a 42-year-old woman. Long-axis **(a)** and short-axis **(b)** fat-suppressed fluid-sensitive MR images show fluid distending the AT tendon sheath (arrow) near its distal course over the medial cuneiform and first metatarsal base. The tendon caliber is normal.

likely represents the retracted proximal tendon stump as it becomes entrapped at the extensor retinaculum (24). Other causes, such as an organized hematoma or fibrous tissue surrounding the torn tendon stump or fibrosis of the synovial sheath and retinaculum, have been suggested (16).

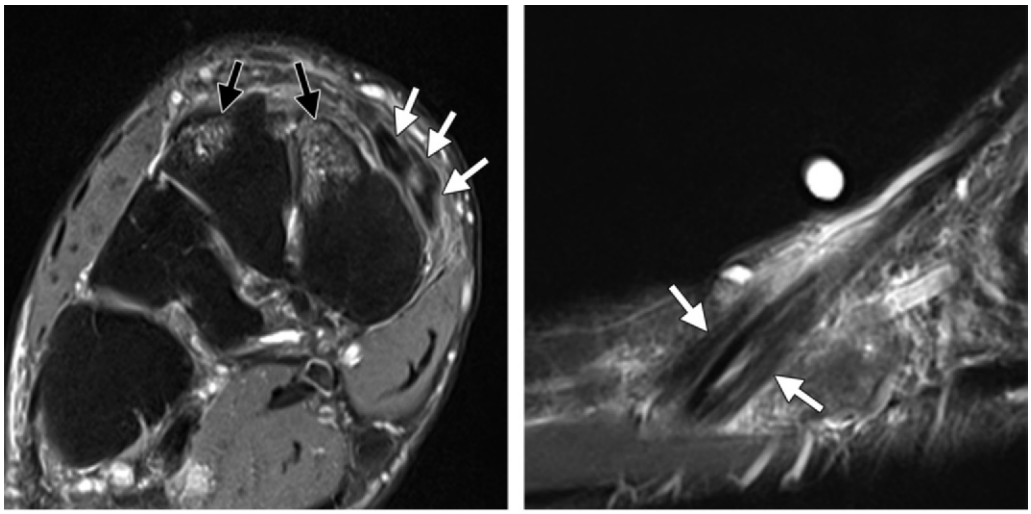
Tendon vascularity as a proposed cause of AT tendon rupture is controversial. Although an early study showed the AT tendon to have a homogeneous dual blood supply from branches of the AT artery and medial tarsal arteries (23), a later study identified a longitudinal hypovascular zone of approximately 4.5–6.7 cm within the anterior half of the AT tendon (29). The location of this avascular zone correlates with the portion of the tendon retained by the extensor retinacula and with the most frequent site of AT tendon rupture. The avascular zone, located where the AT tendon curves against the unyielding extensor retinaculum, may be vulnerable to repetitive microtrauma and subsequent hypoxic and mucoid degeneration.

Both surgical repair and conservative treatment have been proposed for AT tendon rupture. Most authors recommend surgical repair for a subacute rupture diagnosed within 3 months of injury in active patients of any age (1,2,11,30). For patients with chronic rupture, delayed diagnosis, and low functional demands, conservative treatment may be as effective as surgical repair (1,2,27,30). Partial tears and chronic tendinosis are more likely to warrant a trial of conservative management. Recalcitrant tendinosis may require débridement and tendon augmentation.

Imaging Features

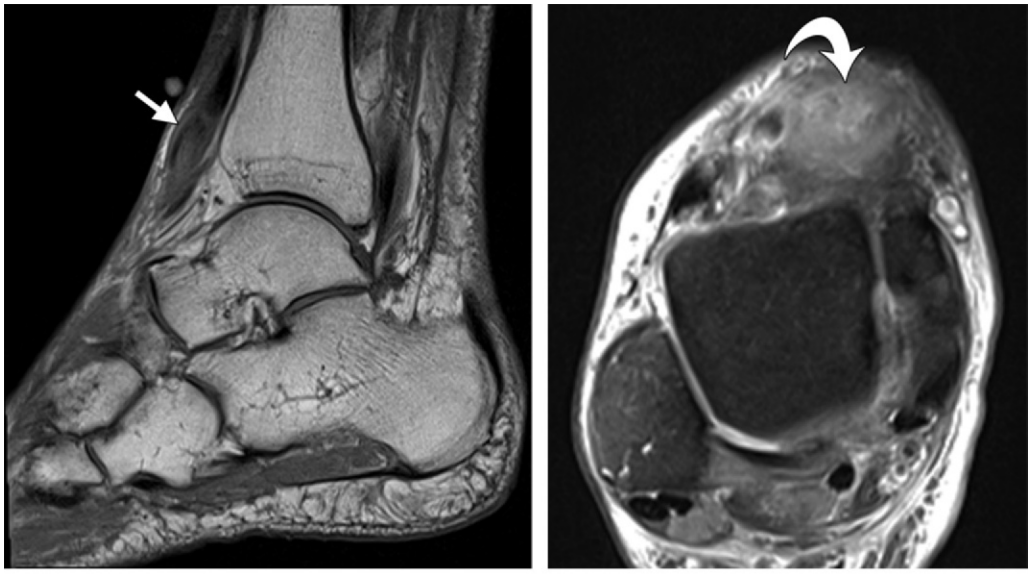
At US, tendinosis manifests as an enlarged tendon diameter with increased hypoechoic tissue within the tendon. Findings of peritendinous and intratendinous hypervascularity confirm tendinosis and may provide additional information about disease activity. Partial tears demonstrate incomplete fibrillar disruption. A full-thickness tear manifests as a tendinous gap filled with fluid, a hematoma, or scar tissue, depending on the age of the injury (Fig 2). Acute or chronic tears may show tendon irregularity (6).

At MR imaging, fusiform thickening and increased signal abnormality indicate AT tendon injury and, depending on the extent and severity of findings, reflect tendinosis or partial rupture. Accompanying tendon sheath distention correlates with a finding of concomitant tenosynovitis (Fig 9). Tendinosis and partial rupture of the AT tendon often are noted close to the insertion site, and the most medial sagittal and most distal axial MR images of the ankle should be used to assess the tendon. A distal longitudinal split tear should be distinguished from a normal bifid tendon. Although there is no tendon sheath at the insertion site, peritendinous edema aids in diagnosis of tendon disease (Fig 10). Advanced partial or complete tears are usually easy to diagnose and are best seen on sequential sagittal and axial MR



a. b.

Figure 10. Insertional AT tendinosis and partial tear in a 68-year-old man. Short-axis **(a)** and long-axis **(b)** fat-suppressed T2-weighted MR images show increased signal intensity of the AT tendon and longitudinal splitting (white arrows). Midfoot medial column osteoarthritis demonstrated by productive changes, marrow edema, and subchondral cysts (black arrows in **a**) may have predisposed toward the tear.

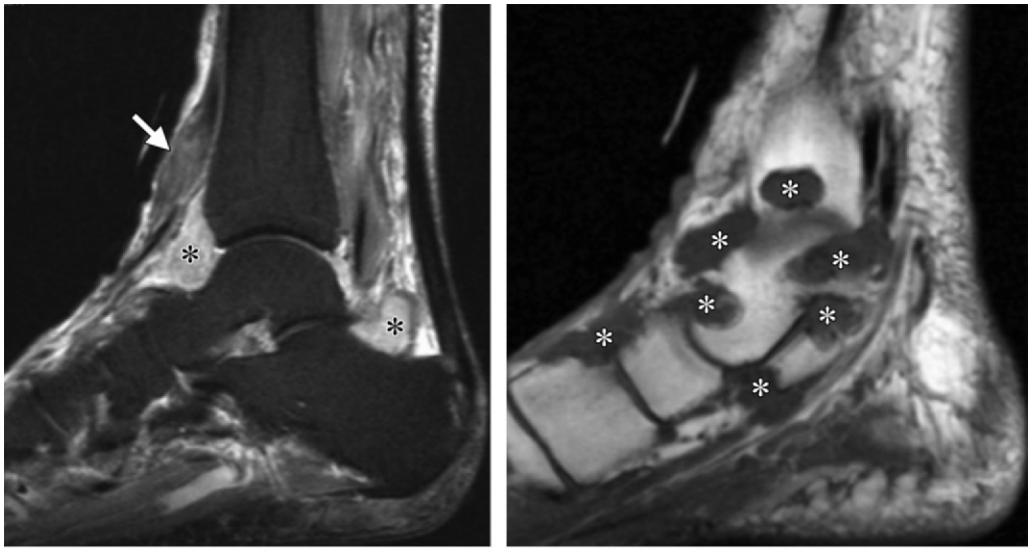


a. b.

Figure 11. Complete AT tendon tear in an 83-year-old man. **(a)** Sagittal T1-weighted MR image shows complete disruption of the AT tendon with thickening and retraction of the proximal tendon stump (arrow). **(b)** Axial fat-saturated T2-weighted MR image shows absence of the tendon (arrow) with surrounding edema.

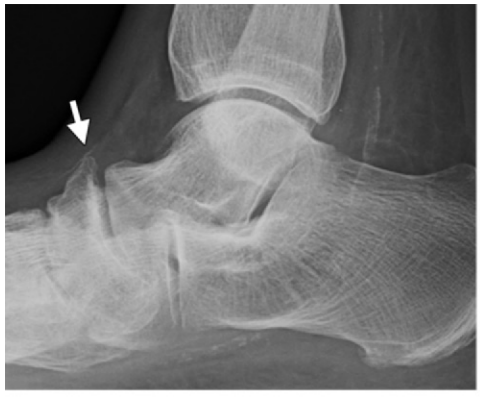
images of the ankle or on short-axis MR images of the midfoot. Not infrequently, the patient may report a dorsal soft-tissue mass, and the technologist can place a marker at the suspected injury site. A full tear manifests as complete disruption of the tendon fibers with retraction of the proximal tendon stump, typically to the level of the ankle joint (Fig 11), and a fluid-filled gap (9).

Comorbid conditions such as gout, inflammatory arthritides (Fig 12), and underlying osseous or postsurgical abnormalities (Figs 13, 14) should also be considered. Particular attention should be given to arthritis and impinging dorsomedial osteophytes along the course of the



a. b.

Figure 12. Sagittal T2-weighted MR images show suspected intratendinous gout producing a high-grade partial AT tendon tear. **(a)** Marked AT tendon thickening and heterogeneity (arrow) is seen with suspected intratendinous gout involvement of the ankle joint (*). **(b)** Multiple erosions and soft-tissue masses (*) are compatible with suspected gout arthropathy.



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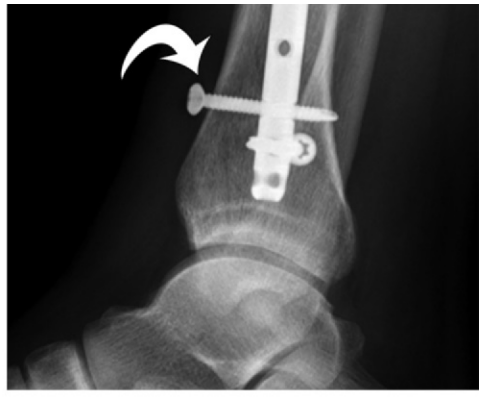
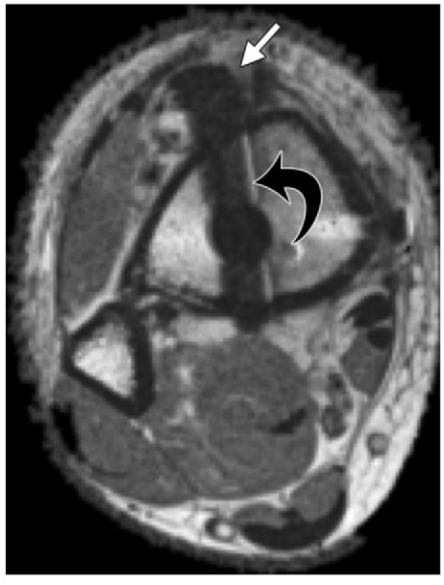


14a.



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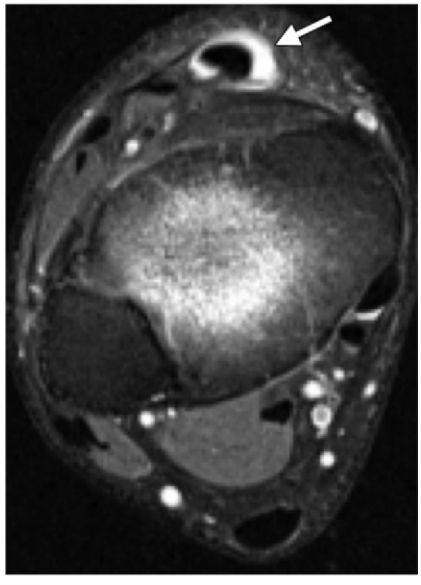

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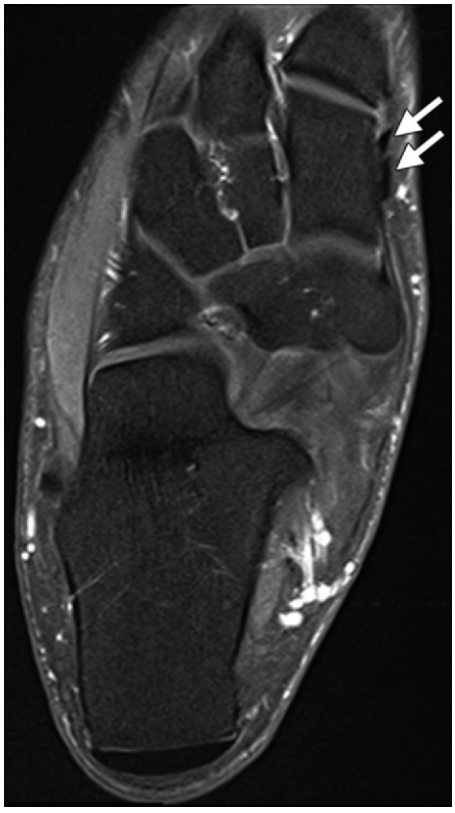


Figure 15. Normal splitting of the distal AT tendon in a 32-year-old woman. Axial fat-suppressed T2-weighted MR image shows the normal division of the distal AT tendon into two insertional slips (arrows).

tendon, which originate from the talar neck and talonavicular, navicular–medial cuneiform, and first tarsometatarsal joints.

Imaging Pitfalls

At US, the anisotropic effect, which occurs when the beam is not perpendicular to the tendon fibers, can result in an artifactually hypoechoic AT tendon. As previously mentioned, the most common MR imaging pitfall is the magic angle phenomenon, which is seen less frequently at imaging of the AT tendon (20% of examinations) than at imaging of the other ankle tendons. Apparent splitting of the AT tendon at its insertion into the medial border of the foot, a reflection of multiple AT tendon insertion slips (Fig 15), is another pitfall and may be mistaken for a distal longitudinal split tear. Correlation with clinical history, absence of other signal and morphologic changes in the tendon, and lack of peritendinous edema or enthesopathy will prevent misinterpretation of normal tendon anatomy.

EHL and EDL Tendons

Anatomy

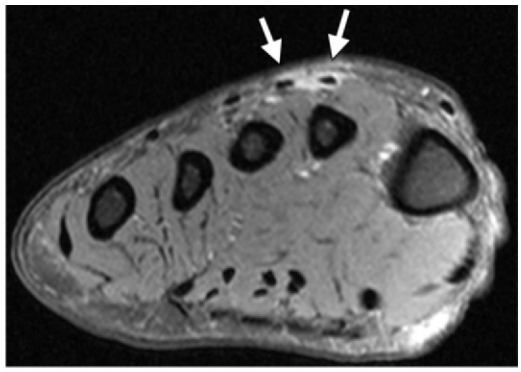
The EHL muscle originates from the middle half of the fibula and adjacent interosseous membrane and descends vertically between the AT and EDL muscles to become tendinous at the distal third of the tibia. The EHL tendon obliquely courses deep to the superior and inferior extensor retinacula before inserting medially onto the dorsal surface of the distal phalangeal base of the hallux (Fig 1) (19). The tendon inserts as an expansion of a thin, triangular, aponeurotic sheath that covers the dorsal surface of the distal phalangeal base. There is considerable anatomic variation of the EHL tendon, including one to three muscle bellies and tendons and a shared muscle slip with the EDL tendon (31). The principle function of the EHL is to extend the hallux, although the EHL tendon also contributes to overall foot dorsiflexion and inversion.

The EDL muscle arises from the lateral tibial condyle, proximal three-fourths of the anterior fibula, interosseous membrane, deep fascia, and intermuscular septa. Beyond the musculotendinous junction at the distal one-third of the leg, the EDL courses behind the superior extensor retinaculum in a sling formed by the medial root of the stem of the inferior extensor retinaculum. The EDL then exits the extensor retinacula, divides into four slips, and traverses the dorsum of the foot to insert onto the dorsal aspect of the middle and distal phalanges of the second through fifth digits in extensor expansion sheaths (Fig 1) (19). The EDL functions primarily in phalangeal extension but also contributes to foot dorsiflexion.

Pathologic Conditions and Imaging Features

Injuries to the EDL or EHL tendons are less common than those to the AT tendon and include tenosynovitis, tendinosis, and tendon tear or lac-

◀ **Figures 13, 14.** **(13)** Lateral radiograph of the foot shows an osteophyte (arrow) of the dorsal talonavicular joint that may predispose the AT tendon to tendinosis and tearing. **(14)** AT tenosynovitis and partial tear caused by screw fixation. Lateral radiograph **(a)** and axial T1-weighted **(b)** and fat-suppressed T2-weighted **(c)** MR images show a screw (curved arrow in **a** and **b**) extending beyond the cortex and abutting the AT tendon, with resultant distention of the tendon sheath (straight arrow in **b** and **c**). The tendon is slightly irregular in morphology but otherwise normal. (Case courtesy of George Nomikos, MD.)



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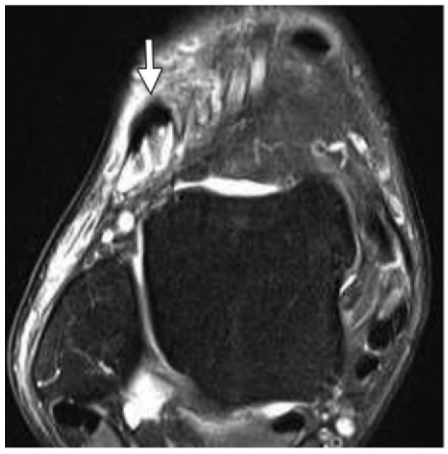

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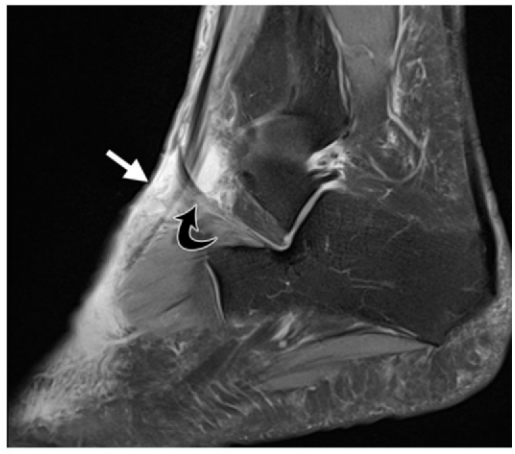


18.

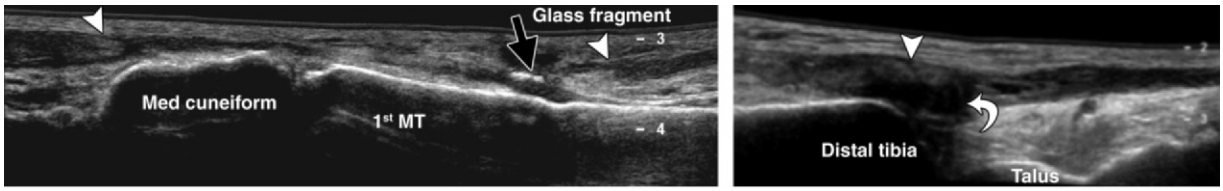
Figures 16–18. **(16)** Tenosynovitis of the distal second and third EDL tendon slips in a 63-year-old woman. Short-axis fat-saturated proton-density–weighted MR image at the level of the metatarsal shafts shows fluid distending the tendon sheaths of the second and third EDL divisions (arrows). Unlike in the flexor compartment of the ankle, fluid in the extensor tendon sheaths, even when minimal, is almost always pathologic. **(17)** EDL tenosynovitis in a 28-year-old man. Axial fat-suppressed T2-weighted MR image shows a distended EDL tendon sheath (arrow). This finding should not be confused with a ganglion extending from the sinus tarsi. (Courtesy of George Nomikos, MD.) **(18)** EDL tenosynovitis in a 53-year-old woman. Sagittal fat-saturated T2-weighted MR image shows fluid distending the EDL tendon sheath (white arrow). Note the extensor retinaculum crossing the EDL tendon anteriorly (black arrow).

Teaching Point

eration. Given their superficial location, the EDL and EHL tendons are especially vulnerable to penetrating trauma and laceration during industrial accidents (32). Because such injuries may appear shallow and produce little blood, they often are overlooked by the patient and clinician, and a high index of suspicion is necessary for accurate diagnosis.

Closed tendon ruptures, which are caused by active tendon contraction against resistance, are unusual and typically are associated with a predisposing cause such as mechanical overuse, inflammatory arthritides, infection, diabetes mellitus, crystal deposition disease, and midfoot arthropathy (33,34). Mechanical impingement by bone spurs or tight-fitting, high-laced boots can be predisposing factors. Sports-related injuries incurred during activities such as ultramarathon running (35) and tae kwon do (36) and iatrogenic injury after arthroscopic thermal ablation have also been described (37).

Both nonsurgical and surgical treatments of EHL and EDL tendon tears have been advocated (34). Lacerations to the EHL tendon distal to the extensor expansion compartment may not cause significant loss of function or tendon retraction and thus may be treated conservatively. Conversely, more proximal lacerations create significant tendon retraction and function loss and require surgical intervention, particularly in young patients (38). The surgical technique varies depending on the location of injury, partial or complete tear, and time from injury to treatment.



a. b.

Figure 19. Longitudinal US images of complete and partial EHL tears in two different patients. **(a)** Complete EHL laceration is seen with proximal and distal retracted EHL fibers (arrowheads) and a glass fragment (arrow). *1st MT* = first metatarsal, *Med* = medial. **(b)** High-grade partial tear of the EHL (arrowhead) is seen at the tibiotalar joint. Anechoic material (arrow) is visible deep to the tendon, reflecting fluid or debris in the tendon sheath.

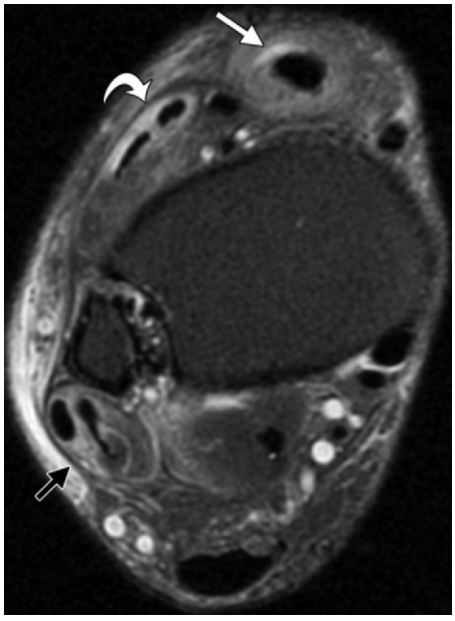


Figure 20. Reactive arthritis and tenosynovitis of multiple tendons in a 38-year-old man. Axial fat-suppressed T2-weighted MR image shows synovitis and sheath distention of the AT (straight white arrow), EDL (curved white arrow), and peroneal tendons (black arrow) with debris or synovial hypertrophy. Involvement of multiple tendons and debris within tendon sheaths usually reflect inflammatory arthritis. (Courtesy of Mini Pathria, MD, San Diego, Calif.)

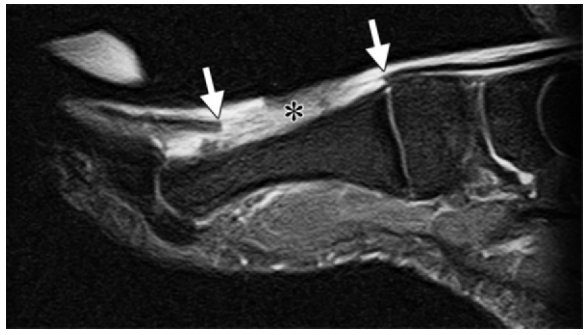


Figure 21. Complete EHL tear in a 41-year-old woman with a midfoot laceration injury. Sagittal MR image of the forefoot at the level of the first toe shows abrupt EHL tendon discontinuity (arrows) and a 4-cm tendon gap (*). Lacerations from sharp objects that fall on the dorsum of the foot are a frequent cause of extensor tendon tears.

At US and MR imaging, fluid is seen distending the tendon sheath with tenosynovitis of either mechanical or inflammatory origin (Figs 16–18). Any amount of fluid within the extensor tendon sheaths is indicative of disease. US is a quick and dynamic imaging method to verify tendon laceration in the setting of suspected penetrating injury to the EHL or EDL tendon (Fig 19). US also helps identify the site of injury relative to the retinaculum (Fig 3). MR imaging findings include fusiform tendon thickening, increased intrasubstance signal intensity, and frank fiber discontinuity, reflecting tendinosis, partial tear, and complete tear, respectively (Figs 20, 21). If there is complete rupture with tendon retraction, US may be advantageous over MR imaging to locate torn tendon edges (Figs 19, 21). Both modalities are useful for assessing intrasubstance tearing in more chronic injury.

Imaging Pitfalls

When evaluating the EHL and EDL tendons at US, the beam should be oriented perpendicular to the tendon fibers to avoid anisotropy artifact. At MR imaging, the EHL and EDL tendons are more susceptible than the AT tendon to magic angle artifact (Fig 7) (17). As with imaging of the AT tendon, increased signal intensity should be correlated with clinical symptoms, location relative to the course of the tendon, tendon appearance on long TE images such as T2-weighted images, and other evidence of disease at imaging. The magic angle effect is less prominent along the midfoot and forefoot sections of the tendons.

The descending EDL tendon appears initially flat on axial MR images but becomes bulbous, simulating tendinosis, just before it divides into its

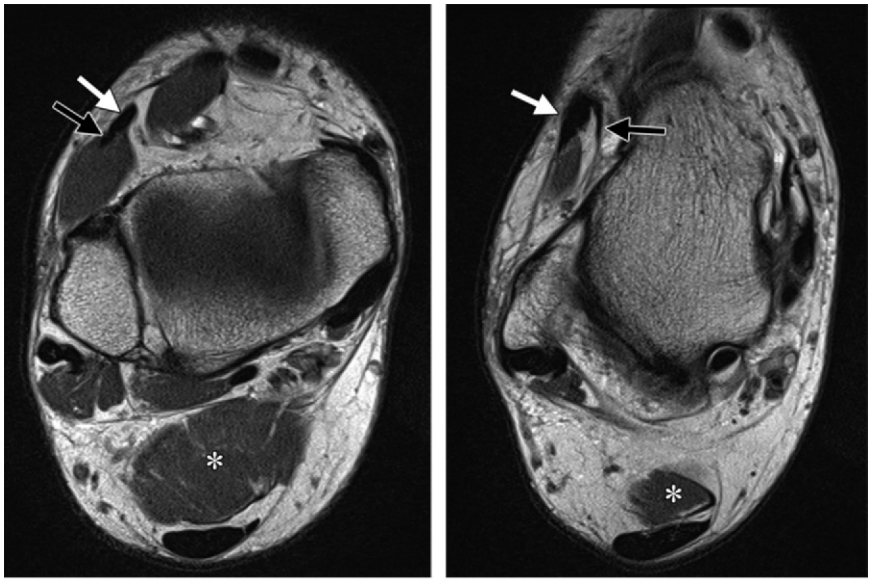


Figure 22. Pitfalls of MR imaging of the EDL tendon in a 46-year-old man. Axial images show the EDL tendon (white arrow) as flat at the level of the ankle joint **(a)** but becoming bulbous more distally **(b)** before it divides into its tendon slips. The tendon margins appear irregular laterally in **a** because of the adjacent peroneus tertius tendon (black arrow in **a**) and appear irregular medially in **b** because of the adjacent linear low-signal-intensity medial root of the inferior extensor retinaculum (black arrow in **b**). An accessory soleus muscle (*) is also shown.

respective slips. This characteristic appearance (Fig 22) and specific location, along with an absence of signal intensity abnormality, should prevent misinterpretation of the thickened tendon as tendinosis. Because of their close proximity, the peroneus tertius and EDL tendons sometimes are confused as a longitudinal split tear of the EDL tendon.

The medial root of the inferior extensor retinaculum abuts the periphery of the EDL tendon, resulting in irregularity of the medial and sometimes lateral tendon margins. This normal anatomy should not be misconstrued as a longitudinal split tear (Fig 22). A sinus tarsi ganglion insinuated in the roots of the extensor retinaculum can partially surround or displace the EDL tendon and mimic tenosynovitis. To assist in diagnosis, fluid should be traced within the sinus tarsi and along the roots of the extensor retinaculum (Fig 23).

Extensor Retinacular Mechanism

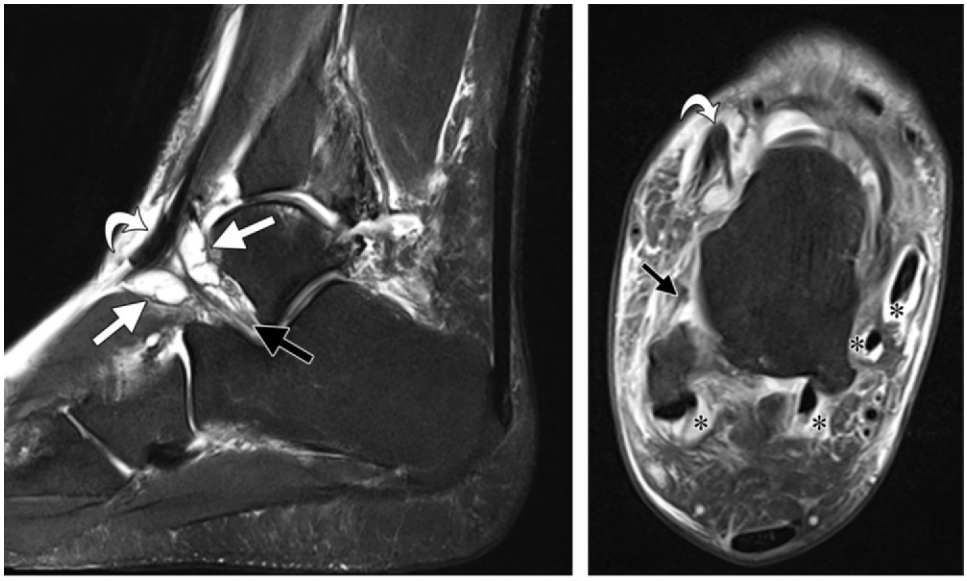
Anatomy

The extensor retinacula are reinforced thickenings of the aponeurosis of the lower leg. They consist of superior and inferior components that cross the AT, EHL, EDL, and peroneus tertius tendons. The superior extensor retinaculum is a transverse band superior to the ankle joint that constrains the extensor tendons against the distal leg. It often forms a separate loop for the AT tendon. The inferior extensor retinaculum is an X- or Y-shaped structure subdivided into roots and oblique superomedial, oblique inferomedial, and variable oblique superolateral limbs below the ankle joint. The stem, particularly the medial root, of the inferior retinaculum forms a constraining loop around the EDL and peroneus tertius tendons. The superomedial and inferomedial bands form slings around the AT and EHL tendons (Fig 1) (16,19).

The extensor retinacula restrict the extensor tendons to tight fibrous tunnels and prevent forward bowstringing. The retinacula also function as fibrous pulleys, allowing the tendons to change direction and providing low-friction gliding action as the tendons move along the long axis of the fibrous tunnels.

Pathologic Conditions and Imaging Features

Trauma, in particular forceful dorsiflexion of the foot, is the most common injury mechanism of the extensor retinacula (14,16). Retinacular injury occasionally is seen with bone avulsion.



a. b.

Figure 23. Sinus tarsi ganglion mimicking EDL tenosynovitis in a 43-year-old man. Sagittal **(a)** and axial **(b)** fat-suppressed T2-weighted MR images show a sinus tarsi ganglion (straight white arrows in **a**) insinuating between the retinacular roots (black arrow in **a**) and partially enveloping the EDL tendon (curved white arrow). A tear of the anterior talofibular ligament (black arrow in **b**) is seen with fluid distending the sheaths of the flexor and peroneal tendons (∗ in **b**), findings related to a recent inversion injury.

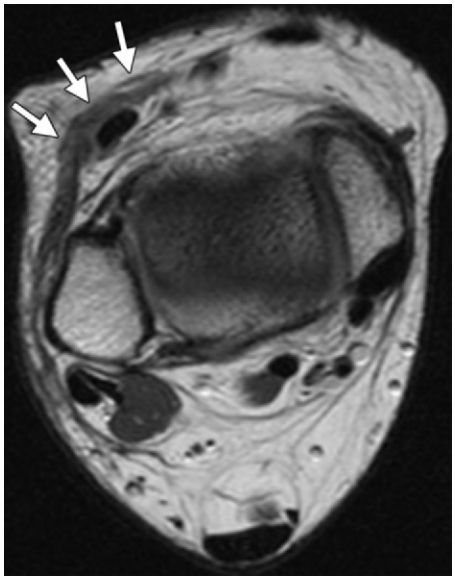
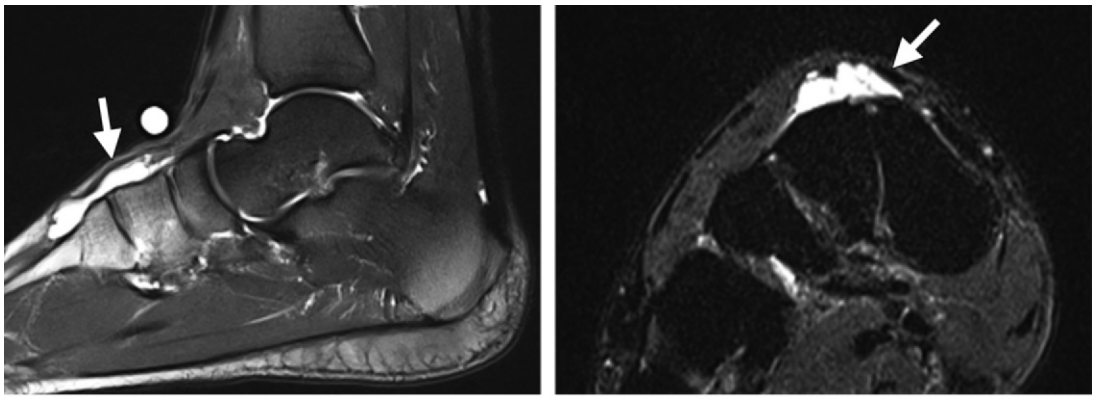


Figure 24. Retinacular injury after ankle inversion injury in a 29-year-old woman. Axial proton-density–weighted MR image of the distal aspect of the ankle shows marked thickening and heterogeneity of the inferior extensor retinaculum (arrows).

As previously mentioned, the extensor retinacula may play an important role in the pathogenesis of AT tendon injury, given the typical location of rupture 0.5–3 cm from the bony insertion of the AT tendon and the zone of anterior hypovascularity in the distal AT tendon as it courses under the retinaculum.

A retinacular tear manifests at US as a thickened, irregular, hypoechoic band. In acute or subacute injury, a hypervascular pattern and edema of the surrounding soft tissues may be seen at Doppler US. Dynamic imaging maneuvers can be used to distinguish partial from complete retinacular tears. MR imaging of retinacular injury shows poor definition, thickening, and, depending on injury severity, retinacular disruption (Fig 24). Fluid-sensitive MR imaging highlights edema in surrounding soft tissues (14). Small avulsion fragments and traction-related spurs or ossifications at the retinacular insertion sites may be seen at both US and MR imaging. Fluid may collect in the space confined by the extensor retinacula (16). In cases of AT



a. b.

Figure 25. Ganglion cyst displacing the EHL tendon in a 33-year-old woman with a 1-month history of foot pain. Sagittal **(a)** and short-axis **(b)** fat-suppressed T2-weighted MR images of the midfoot show a multilobulated mass of fluid signal intensity displacing the EHL dorsally (arrow). A ganglion at that site could also cause entrapment of the medial branch of the DPN.

tendon rupture, MR imaging is particularly useful to establish the integrity of the extensor retinaculum and its relationship to the AT tendon because the proximal torn end of the tendon often is entrapped at the inferior extensor retinaculum. Successful surgical repair of the AT tendon often depends on maintenance of the normal anatomic relationship (24) between the two structures.

Anterior Tarsal Tunnel and DPN

Anatomy

The anterior tarsal tunnel is a fibro-osseous tunnel between the inferior extensor retinaculum and the talus and navicular that constrains the DPN and its branches as they pass deep to the EHL and EDL tendons (Figs 6, 7) (39). The DPN typically divides into lateral and medial branches within the tarsal tunnel, although it occasionally bifurcates distally or not at all (40). The lateral motor branch supplies the extensor digitorum brevis muscle and less commonly the extensor hallucis brevis muscle, while the medial branch provides sensory and occasional motor innervation to the first interspace and adjacent sides of the great and second toes (19).

Pathologic Conditions and Imaging Features

Entrapment of the DPN as it passes beneath the inferior extensor retinaculum has been termed *anterior tarsal tunnel syndrome* and manifests clinically as weakness of the extensor digitorum brevis muscle and numbness between the first and second digits. Pain that worsens at rest may be localized to the dorsomedial aspect of the foot. The tarsal tunnel, a tight anatomic space enclosed by unyielding retinacula, is subject to repetitive mechanical microtrauma (such as that caused by wearing high boots or tightly laced or tight-fitting shoes) and iatrogenic injury. Compression of the DPN by the underlying talar head, spurs at the talonavicular joint, and ganglia also can occur (Fig 25) (39). Sports-related causes include tight-fitting footwear worn by dancers or skiers (41) and direct repetitive trauma to the dorsal aspect of the foot as seen in soccer players (42). Treatment typically involves an initial trial of conservative measures such as rest, anti-inflammatory medications, foot stabilization with an orthotic, or local corticosteroid injection (43). Failure of conservative therapy may necessitate surgical nerve release.

The DPN and anterior tarsal tunnel can be assessed at US and MR imaging (44). US is advantageous in tracing the small nerve and distinguishing it from accompanying vascular struc-

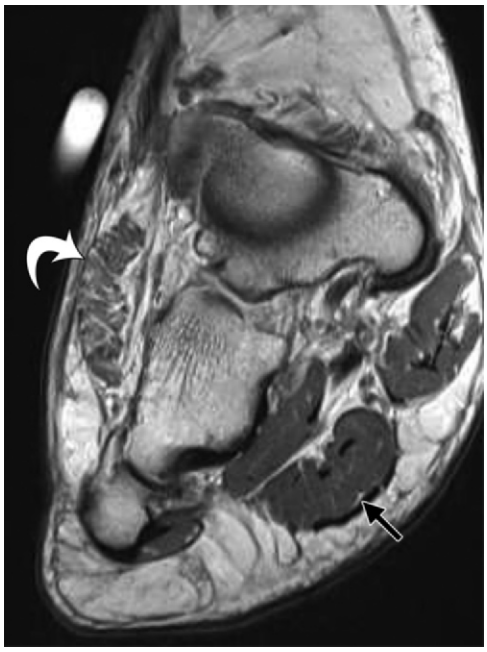


Figure 26. Indirect evidence of DPN injury. Short-axis T1-weighted MR image of the midfoot demonstrates denervation-related atrophy of the extensor digitorum brevis muscle (white arrow), which is innervated by the lateral branch of the DPN. Note the fatty replacement and decreased size of the extensor digitorum brevis muscle relative to the plantar muscles (black arrow). (Courtesy of George Nomikos, MD.)

tures. Conversely, the nerve can be difficult to depict at MR imaging because of the magic angle phenomenon and because its tiny size makes it difficult to distinguish from adjacent structures. Occasionally, however, MR images show direct signs of DPN neuropathy, such as increased signal intensity or contrast enhancement around the nerve. MR imaging also can reveal indirect signs of injury, such as denervation-related changes to the extensor digitorum brevis muscle (Fig 26). Both US and MR imaging can depict and potentially characterize masses in the anterior tarsal tunnel that impinge on the nerve.

Summary

Disorders of the ankle extensor compartment are rare but often underdiagnosed, leading to more challenging treatment and increased morbidity. Familiarity with their common US and MR imaging features and potential imaging pitfalls aids in accurate and timely diagnosis.

References

1. Markarian GG, Kelikian AS, Brage M, Trainor T, Dias L. Anterior tibialis tendon ruptures: an outcome analysis of operative versus nonoperative treatment. Foot Ankle Int 1998;19(12):792–802.
2. Ouzounian TJ, Anderson R. Anterior tibial tendon rupture. Foot Ankle Int 1995;16(7):406–410.
3. Lipscomb PR, Kelly PJ. Injuries of the extensor tendons in the distal part of the leg and in the ankle. J Bone Joint Surg Am 1955;37-A(6):1206–1213.
4. Scheller AD, Kasser JR, Quigley TB. Tendon injuries about the ankle. Orthop Clin North Am 1980; 11(4):801–811.
5. Erickson SJ, Cox IH, Hyde JS, Carrera GF, Strandt JA, Estkowski LD. Effect of tendon orientation on MR imaging signal intensity: a manifestation of the "magic angle" phenomenon. Radiology 1991;181 (2):389–392.
6. Bianchi S, Zwass A, Abdelwahab IF, Zoccola C. Evaluation of tibialis anterior tendon rupture by ultrasonography. J Clin Ultrasound 1994;22(9): 564–566.
7. Fessell DP, Vanderschueren GM, Jacobson JA, et al. US of the ankle: technique, anatomy, and diagnosis of pathologic conditions. RadioGraphics 1998; 18(2):325–340.
8. Cheung Y, Rosenberg ZS, Magee T, Chinitz L. Normal anatomy and pathologic conditions of ankle tendons: current imaging techniques. RadioGraphics 1992;12(3):429–444.
9. Rosenberg ZS, Beltran J, Bencardino JT. MR imaging of the ankle and foot. RadioGraphics 2000;20(Spec No):S153–S179.
10. Mengiardi B, Pfirrmann CW, Vienne P, et al. Anterior tibial tendon abnormalities: MR imaging findings. Radiology 2005;235(3):977–984.
11. Anagnostakos K, Bachelier F, Fürst OA, Kelm J. Rupture of the anterior tibial tendon: three clinical cases, anatomical study, and literature review. Foot Ankle Int 2006;27(5):330–339.
12. Khoury V, Guillin R, Dhanju J, Cardinal E. Ultrasound of ankle and foot: overuse and sports injuries. Semin Musculoskelet Radiol 2007;11(2):149-161.
13. Jamadar DA, Robertson BL, Jacobson JA, et al. Musculoskeletal sonography: important imaging pitfalls. AJR Am J Roentgenol 2010;194(1):216–225.
14. Demondion X, Canella C, Moraux A, Cohen M, Bry R, Cotten A. Retinacular disorders of the ankle and foot. Semin Musculoskelet Radiol 2010; 14(3):281–291.

15. Peer S, Kovacs P, Harpf C, Bodner G. High-resolution sonography of lower extremity peripheral nerves: anatomic correlation and spectrum of disease. J Ultrasound Med 2002;21(3):315–322.
16. Lee MH, Chung CB, Cho JH, et al. Tibialis anterior tendon and extensor retinaculum: imaging in cadavers and patients with tendon tear. AJR Am J Roentgenol 2006;187(2):W161–W168.
17. Mengiardi B, Pfirrmann CW, Schöttle PB, et al. Magic angle effect in MR imaging of ankle tendons: influence of foot positioning on prevalence and site in asymptomatic subjects and cadaveric tendons. Eur Radiol 2006;16(10):2197–2206.
18. Petersen W, Stein V, Bobka T. Structure of the human tibialis anterior tendon. J Anat 2000;197(Pt 4):617–625.
19. Sarrafian SK, Kelikian AS. Anatomy of the foot and ankle: descriptive, topographic, functional. 3rd ed. Philadelphia, Pa: Lippincott Williams & Wilkins, 2011; 120–162, 223–291.
20. Beischer AD, Beamond BM, Jowett AJ, O'Sullivan R. Distal tendinosis of the tibialis anterior tendon. Foot Ankle Int 2009;30(11):1053–1059.
21. Arthornthurasook A, Gaew Im K. Anterior tibial tendon insertion: an anatomical study. J Med Assoc Thai 1990;73(12):692–696.
22. Dooley BJ, Kudelka P, Menelaus MB. Subcutaneous rupture of the tendon of tibialis anterior. J Bone Joint Surg Br 1980;62-B(4):471–472.
23. Geppert MJ, Sobel M, Hannafin JA. Microvasculature of the tibialis anterior tendon. Foot Ankle 1993;14(5):261–264.
24. Sammarco V, Sammarco G. Injuries to the tibialis anterior, peroneal tendons, and long flexors of the toes. In: Porter DA, Schon LC, eds. Baxter's the foot and ankle in sport. 2nd ed. Philadelphia, Pa: Mosby Elsevier, 2007.
25. Ebrahimi FV, Tofighi M, Khatibi H. Closed tibial fracture associated with laceration of tibialis anterior tendon. J Foot Ankle Surg 2010;49(1):e19–e22.
26. Jozsa L, Kannus P. Spontaneous rupture of tendons. In: Human tendons: anatomy, physiology and pathology. Champaign, Ill: Human Kinetics Publishers, 1997; 254–325.
27. Trout BM, Hosey G, Wertheimer SJ. Rupture of the tibialis anterior tendon. J Foot Ankle Surg 2000;39(1):54–58.
28. Stuart MJ. Traumatic disruption of the anterior tibial tendon while cross-country skiing: a case report. Clin Orthop Relat Res 1992;(281):193–194.
29. Petersen W, Stein V, Tillmann B. Blood supply of the tibialis anterior tendon. Arch Orthop Trauma Surg 1999;119(7-8):371–375.
30. Gwynne-Jones D, Garneti N, Wyatt M. Closed tibialis anterior tendon rupture: a case series. Foot Ankle Int 2009;30(8):758–762.
31. Tezer M, Cicekcibasi AE. A variation of the extensor hallucis longus muscle (accessory extensor digiti secundus muscle). Anat Sci Int 2012;87(2):111–114.
32. Al-Qattan MM. Surgical treatment and results in 17 cases of open lacerations of the extensor hallucis longus tendon. J Plast Reconstr Aesthet Surg 2007;60(4):360–367.
33. Fadel GE, Alipour F. Rupture of the extensor hallucis longus tendon caused by talar neck osteophyte. Foot Ankle Surg 2008;14(2):100–102.
34. Scaduto AA, Cracchiolo A 3rd. Lacerations and ruptures of the flexor or extensor hallucis longus tendons. Foot Ankle Clin 2000;5(3):725–736x.
35. Kobayashi H, Sakurai M, Kobayashi T. Extensor digitorum longus tenosynovitis caused by talar head impingement in an ultramarathon runner: a case report. J Orthop Surg (Hong Kong) 2007;15(2):245–247.
36. Lee KT, Choi YS, Lee YK, Lee JP, Young KW, Park SY. Extensor hallucis longus tendon injury in taekwondo athletes. Phys Ther Sport 2009;10(3):101–104.
37. Tuncer S, Aksu N, Isiklar U. Delayed rupture of the extensor hallucis longus and extensor digitorum communis tendons after breaching the anterior capsule with a radiofrequency probe during ankle arthroscopy: a case report. J Foot Ankle Surg 2010;49(5):e1–e3.
38. Kass JC, Palumbo F, Mehl S, Camarinos N. Extensor hallucis longus tendon injury: an in-depth analysis and treatment protocol. J Foot Ankle Surg 1997;36(1):24–27; discussion 80.
39. Liu Z, Zhou J, Zhao L. Anterior tarsal tunnel syndrome. J Bone Joint Surg Br 1991;73(3):470–473.
40. Aktan Ikiz ZA, Ucerler H, Uygur M. Dimensions of the anterior tarsal tunnel and features of the deep peroneal nerve in relation to clinical application. Surg Radiol Anat 2007;29(7):527–530.
41. Lindenbaum BL. Ski boot compression syndrome. Clin Orthop Relat Res 1979;(140):109–110.
42. Kanbe K, Kubota H, Shirakura K, Hasegawa A, Udagawa E. Entrapment neuropathy of the deep peroneal nerve associated with the extensor hallucis brevis. J Foot Ankle Surg 1995;34(6):560–562.
43. Schon LC, Baxter DE. Neuropathies of the foot and ankle in athletes. Clin Sports Med 1990;9(2):489–509.
44. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. II. The knee, leg, ankle, and foot. RadioGraphics 2010;30(4):1001–1019.

US and MR Imaging of the Extensor Compartment of the Ankle

Joshua M. Ng, MD • Zehava Sadka Rosenberg, MD • Jenny T. Bencardino, MD • Zoraida Restrepo-Velez, MD • Gina A. Ciavarra, MD • Ronald Steven Adler, MD, PhD

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Page 2051
Unlike the flexor tendons of the ankle, the synovial sheaths of the ankle extensor tendons typically do not contain fluid. Tenosynovitis should be considered with a finding of even a small amount of fluid.

Page 2052
Associated signs of tendinous injury include fluid in the soft tissues surrounding the tendon and occasional reactive bone marrow signal abnormalities at the osseous insertion sites.

Page 2052
The lack of concurrent tendon signal abnormality on long TE images and the absence of other signs of tendon abnormality prevent misinterpretation of the magic angle effect as tendinous disease.

Page 2053
Rupture of the AT tendon is rare and usually occurs 0.5–3.0 cm proximal to the tendon insertion site, where the tendon passes deep to the inferior extensor retinaculum.

Pages 2058
Given their superficial location, the EDL and EHL tendons are especially vulnerable to penetrating trauma and laceration during industrial accidents. Because such injuries may appear shallow and produce little blood, they often are overlooked by the patient and clinician, and a high index of suspicion is necessary for accurate diagnosis.