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PICTORIAL ESSAY

Pathologies and Postoperative Features of Posterior Tibial Tendon Dysfunction: A Pictorial Essay

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INTRODUCTION

The posterior tibial tendon (PTT) is the largest tendon in the medial compartment of the ankle and the most important dynamic stabiliser of the longitudinal foot arch.[1] It is also the most common site of tendon abnormalities in the medial ankle, where dysfunction would result in a cascade of failures of other secondary supporting structures, eventually leading to collapse of the longitudinal arch and acquired pes planus deformity.[1] Knowledge of the imaging appearance of posterior tibial tendon dysfunction (PTTD) enables early diagnosis and treatment, hence preventing progression to fixed pes planus deformity.

ANATOMY AND PATHOPHYSIOLOGY

The PTT runs posterior to the medial malleolus and inserts mainly on the medial aspect of the navicular, with minor slips inserting into the cuneiforms and the first to fourth metatarsals.[1] As the tendon passes posterior to the axis of the ankle joint and medial to the subtalar joint, it acts as a plantar flexor and invertor of the foot, as well as the adductor of the forefoot at the midtarsal joint.[2-4]

PTTD is a spectrum of pathologies ranging from tenosynovitis, tendonitis, and partial tear to complete rupture.[5] It is most prevalent in middle- to old-aged females,[5] commonly resulting from chronic degeneration but may also be caused by acute trauma or by conditions with alterations of anatomy, mechanical forces, and tendon vascularity. Risk factors include pre-existing pes planus, obesity, hypertension, chronic steroid use, gout, and inflammatory arthropathies such as rheumatoid arthritis.[3,6] Ischaemia and mechanical stress are the main underlying pathophysiologies of most PTTD.[6] In particular, the PTT around the level of the medial malleolus is the most susceptible site due to a number of factors.[6] First, the mid tendon has relatively poor vascular supply; second, the synovial sheath ends at the midportion of the talus, distal to which the mesotendon

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is absent; third, the PTT curves around the medial malleolus, creating a focal point of mechanical strain.[6] The second most common location for tears is at the distal portion of the tendon.[6]

In PTTD, the tendon's normal antagonist, the peroneus brevis, acts as a deforming factor in foot eversion, leading to hindfoot valgus and forefoot varus deformities. Progression to arch collapse is associated with tension on the spring and deltoid ligaments and the talonavicular capsule. Hindfoot valgus also leads to an eversion force of the Achilles tendon on the calcaneus, leading to equinus deformity due to shortening of this tendon.[2,4,7-9]

CLASSIFICATION

Johnson and Strom's four-stage classification is a widely adopted clinical staging system for PTTD, which serves as a guide to management.[10] The clinical findings and corresponding magnetic resonance imaging (MRI) features of PTTD are described in the Table.[10-12]

PRIMARY IMAGING FINDINGS

Tenosynovitis

Tenosynovitis can be caused by mechanical trauma or inflammatory arthropathy such as seronegative spondyloarthropathies, rheumatoid arthritis, systemic lupus erythematosus, and gout.[3,6,13] Tenosynovitis manifests radiologically as an abnormal amount of fluid in the tendon sheath on both MRI and ultrasound (Figures 1 and 2). The tendon itself appears normal at this early stage with normal size and signal intensity on MRI.[5] On ultrasound, increased flow in the peritendon area and hypoechoic tissue around the tendon may be detected.[14]

Tendinosis

The next phase of the disease is tendinosis, with collagen degeneration, local necrosis, calcification, and hypocellularity.[6] On MRI, the normal PTT should appear black on all spin-echo images[5] and is about twice the size of the adjacent round flexor digitorum longus (FDL) and flexor hallucis longus tendons.[1] In posterior tibial tendinosis, the tendon appears thickened with normal or increased signal intensity on both T1- and proton density–weighted images (Figure 3); these intrasubstance signals represent severe degeneration or intrasubstance tears not reaching the tendon surface, thus there is a certain degree of overlap of the appearance of tendinosis and partial tear.[5] Contrast enhancement of the tendon is also a common finding in tendinosis.[14]

On sonography, the PTT normally shows homogeneous echogenic longitudinal fibres with no tendinous or peritendinous vascularity.[14] In tendinosis, the tendon is enlarged and appears inhomogeneous, with internal vascular flow sometimes detected on Doppler examination (Figure 4).[14]

Table. Clinical staging and corresponding magnetic resonance imaging findings for posterior tibial tendon dysfunction.[10-12]

Stage	PTT and foot pathology	MRI findings
I	• Inflamed PTT • Intact longitudinal foot arch	• PTT tenosynovitis, low-grade tendinosis
II	• Degenerated, elongated, and dysfunctional PTT • Reversible/flexible pes planus deformity • Subclassification on weight-bearing radiography: (a) minimal forefoot abduction and < 50% uncovering of talar head (b) significant forefoot abduction and > 50% uncovering of talar head (c) forefoot varus with concomitant stage 2a/b deformity	• PTT tendinosis ± low-grade partial tear ± tenosynovitis • Spring ligament abnormality • Hindfoot valgus
III	• Degenerated ± torn PTT and non-functional PTT • Subtalar joint osteoarthritis • Hindfoot valgus • Rigid/fixed pes planus deformity	• High-grade PTT tear ± tenosynovitis • Spring and tibiospring ligamentous abnormalities • Talar head uncoverage • Hindfoot valgus • Early talocalcaneal ± calcaneofibular impingement • Subtalar joint osteoarthritis
IV	Stage III findings with additional: • Irreversible osteoarthrosis of ankle and midfoot • Fixed hindfoot valgus	Above findings with additional: • Deltoid sprain • Tibiotalar and subtalar osteoarthritis • Talocalcaneal and calcaneofibular impingement

Abbreviations: MRI = magnetic resonance imaging; PTT = posterior tibial tendon.

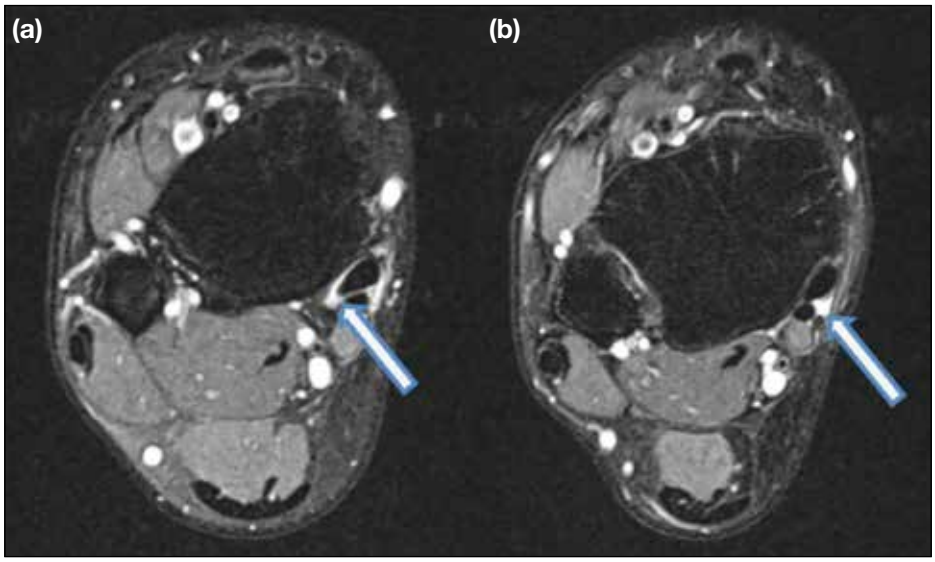

Figure 1. A 25-year-old man with known seronegative spondyloarthropathy complained of right ankle pain. (a, b) Axial fat-saturated proton density–weighted magnetic resonance images showing mild tenosynovial effusion along the posterior tibial tendon (arrows), suggestive of mild tenosynovitis. There was also Achilles tendinosis (not shown). His symptoms improved after starting on golimumab.

Partial and Complete Tears

In chronic tenosynovitis or tendinosis, the PTT is weakened, and tears of the tendon can occur. They can be classified into three types according to Rosenberg et al.[15] Type 1 is a partial tear with associated tendon hypertrophy. On MRI, the tendon is thickened, appearing rounded with loss of its normal oval shape and shows increased linear or heterogenous intrasubstance signal in all sequences, which represents internal tears (Figure 5).[5] A severe form of this type of injury is a longitudinal split of the PTT into two separate parts, which together with the adjacent FDL and flexor hallucis longus tendons, give rise to the appearance of four medial ankle tendons, known as the four-tendon sign (Figure 6a-c).[13] Type 2 is a more severe partial tear with reduced tendon calibre and further increase in internal signal.[5] The segment proximal and distal to the segment of thinned tendon is frequently hypertrophied, which may be due to the background chronic type 1 injury.[5,13] Type 3 injury is a complete tear characterised by a visible gap in the tendon within the tendon sheath (Figure 7).[5]

SECONDARY AND ASSOCIATED IMAGING FINDINGS

Accessory Navicular Bones

There are three types of accessory navicular bones. Type I is a 2- to 3-mm sesamoid bone in the PTT separated from the navicular bone; type II is connected to the navicular bone by a thin layer of cartilage (Figures 6d-e and 8a); type III is a prominent protuberance fused with the navicular tuberosity (Figure 5e).[3] Types II and III may be associated with a younger onset of midfoot pain and planovalgus foot,[3] which are associated with a more proximal insertion of the PTT, straightening the distal tendon curve.[6,16] As a result, there is more focal frictional wear and tear on the tendon at the medial malleolus.[6,16]

MRI features include bone marrow oedema in the accessory navicular bone and the adjacent tuberosity (Figures 6d-e and 8a), fluid in the synchondrosis, soft tissue swelling, adventitial bursa formation, and fracture of the synchondrosis from the chronic pulling of the PTT.[3,6]

Tibial Spur

Osteophytes may develop at the posteromedial aspect of the medial malleolus adjacent to the tendon, presumably due to reactive periostitis in chronic tears (Figure 5j-l).[5,15]

Spring Ligament Injury

As the PTT weakens, it fails to invert the hindfoot and lock the transverse tarsal joints, causing the force of the gastrocnemius-soleus muscle complex to act at the talonavicular joint rather than the metatarsal joints.[17] This leads to increased stress on the talar head and injury to the spring ligament.[17] On MRI, insufficiency of the spring ligament is characterised by thickening (> 6 mm) and increased signal heterogeneity on proton density–weighted images (Figures 5g-i and 6f).[18]

Sinus Tarsi Syndrome

Increased plantar flexion of the talus and hindfoot

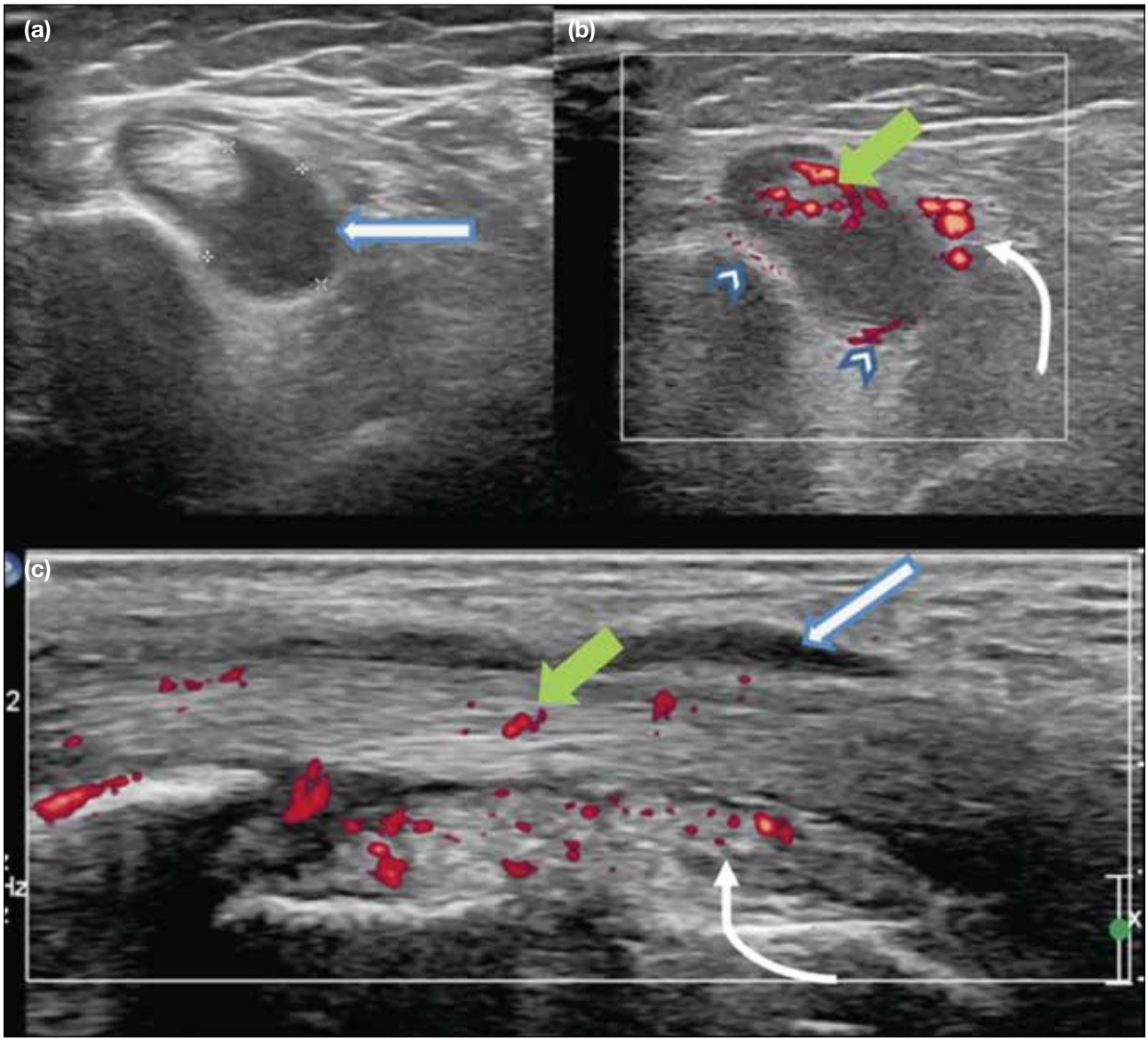

Figure 2. A 10-year-old girl with enthesitis-related arthritis (HLA-B27–positive) presented with right medial ankle pain. (a, b) Short-axis ultrasound (US) images of the posterior tibial tendon (PTT) showing effusion (blue and white arrow) around the tendon, which is thickened with a moderate increase in vascularity within the tendon (green arrow), in the tendon sheath (arrowheads), and adjacent soft tissue (curved arrow). (c) Long-axis US image of the PTT showing similar findings, with effusion (blue and white arrow) and increased vascularity in the tendon (green arrow) and the adjacent soft tissue (curved arrow). Findings are suggestive of tenosynovitis and tendinosis.

valgus due to PTTD also increases the stress on the interosseous talocalcaneal ligaments and cervical ligaments within the sinus tarsi, causing insufficiency of these ligaments over time.[3,18] On MRI, the normal fat signal around the ligament in the sinus tarsi is replaced by abnormal tissue that shows T1 hypointensity and T2 hyperintensity (Figure 8). When fibrotic change within the abnormal tissue predominates, the signal may show T1 and T2 hypointensity.[3,18] Other imaging features include partial or complete tears of the tarsal sinus ligaments.[3]

Plantar Fasciitis

Plantar fasciitis has been found to have a low association with advanced PTT injury, due to increased strain on the plantar fascia which is responsible for supporting the longitudinal arch.[3,18] MRI shows thickening (> 4 mm), irregularity or increased heterogenous signal within the fascia, associated with perifascial and bone marrow oedema.[3]

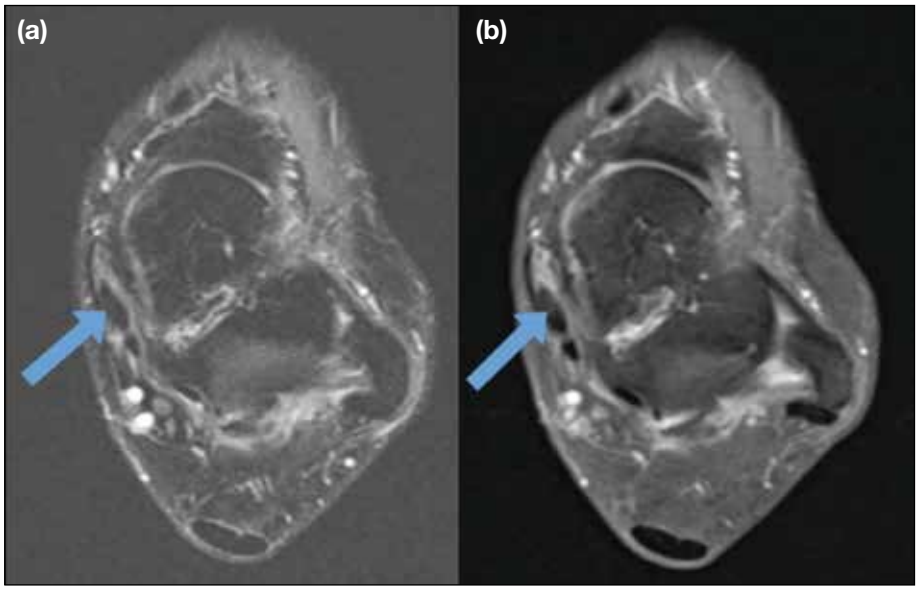

Figure 3. A 63-year-old woman presented with left foot and ankle pain with flexible flatfoot deformity. (a) Axial fat-saturated (FS) proton density–weighted and (b) axial post-contrast FS T1-weighted magnetic resonance images showing posterior tibial tendinosis as evidenced by mildly increased intratendinous signal (arrows).

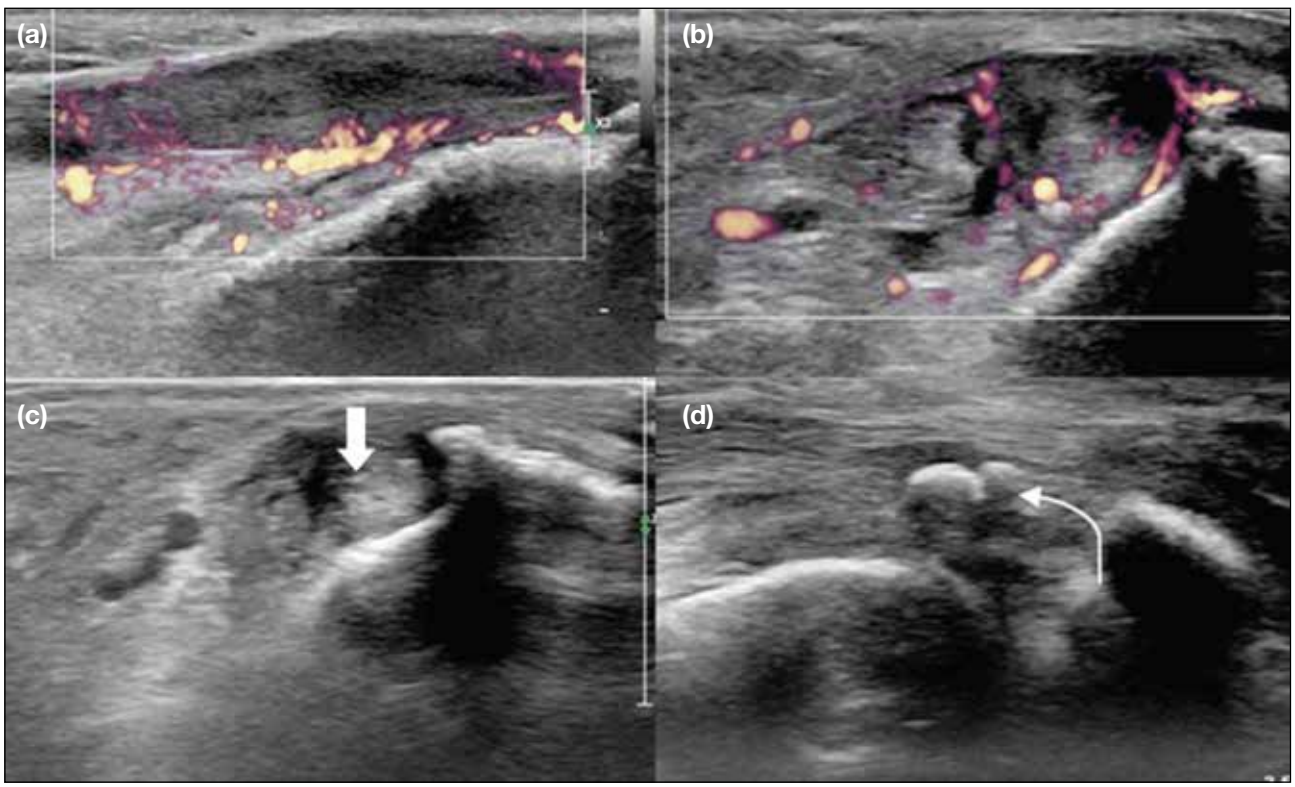

Figure 4. Ultrasound images of a 61-year-old woman presenting with bilateral progressive flexible flatfeet, more severe on the left. (a) Long-axis and (b) short-axis images of the left posterior tibial tendon (PTT) demonstrate enlargement and increased heterogeneity and vascularity of the tendon, suggestive of tendinosis. (c) Short-axis image of the left PTT at the supramalleolar level showing a partial tear (arrow). (d) Calcification/ossification (curved arrow) seen in the long-axis image of the PTT insertion.

Deltoid Ligament Complex Injury

The deltoid ligament complex consists of deep and superficial layers. The deep layer opposes ankle valgus and stabilises the tibiotalar articulation, while the superficial ligaments limit hindfoot eversion and inward displacement of the talar head to stabilise the

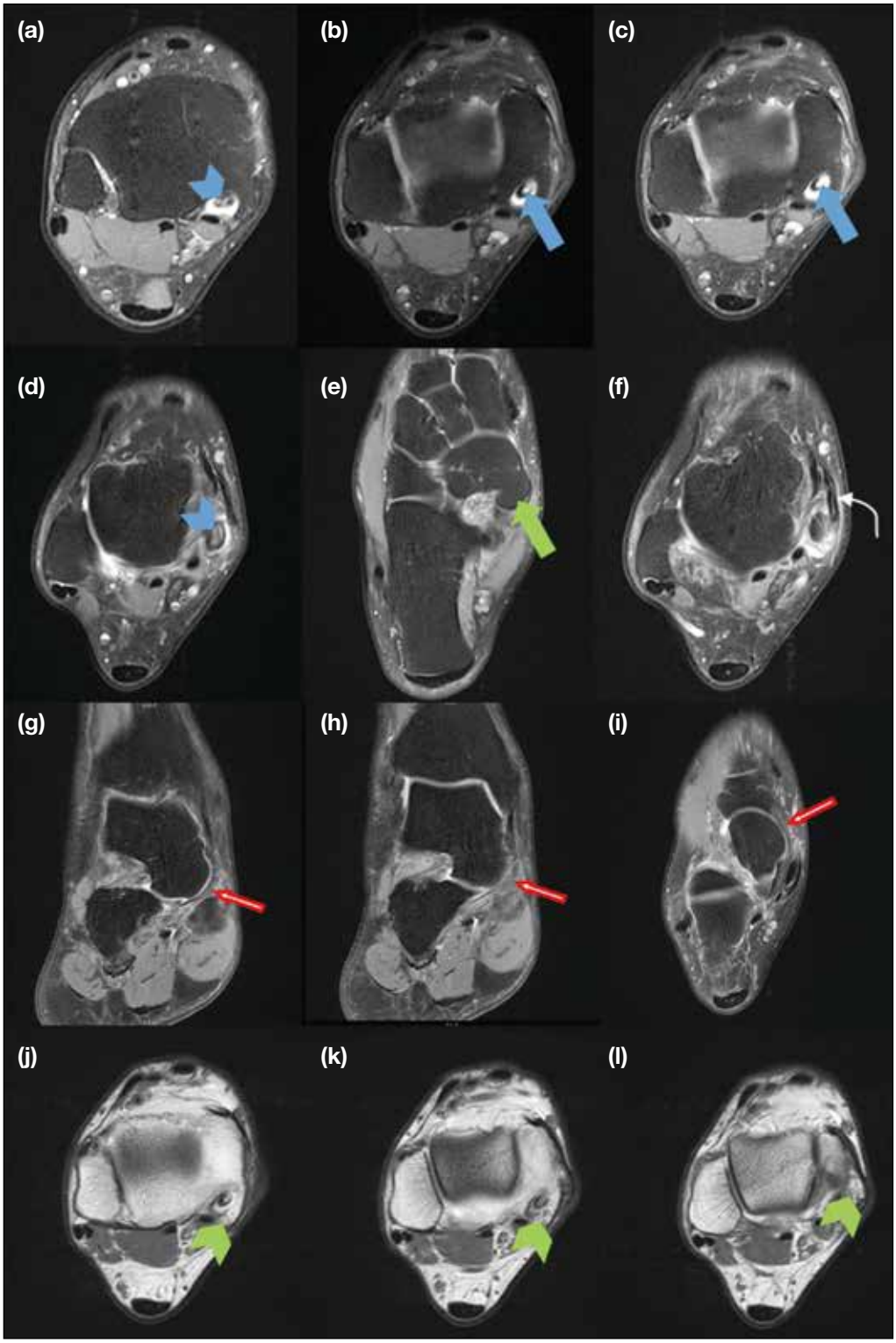

Figure 5. A 61-year-old man complained of right heel pain for approximately a year. Magnetic resonance imaging of his right ankle is suggestive of posterior tibial tendon dysfunction. (a-d) Axial fat-saturated (FS) proton density (PD)–weighted magnetic resonance imaging (MRI) of the posterior tibial tendon from proximal to distal show increased signal and abnormal thickening of the tendon at the level of distal tibia and talus (blue arrowheads in [a] and [d]), suggestive of tendinosis. Fluid signal within the tendon suggests partial tear (blue arrows in [b] and [c]). Small amount of fluid within the tendon sheath are in keeping with tenosynovitis. (e) A cornuate (type III) os naviculare is present (green arrow). (f) Axial FS PD-weighted MRI of the overlying flexor retinaculum showing thickening with increased signal (curved arrow) could be chronic injury or a partial tear. (g-i) Increased signal in and thickening of the spring ligament on the coronal and axial FS PD-weighted MRI (red arrows) is probably related to abnormal stress. (j-l) Axial PD-weighted MRI of the right tibia from proximal to distal shows a prominent spur at the posteromedial aspect of the distal tibia (green arrowheads).

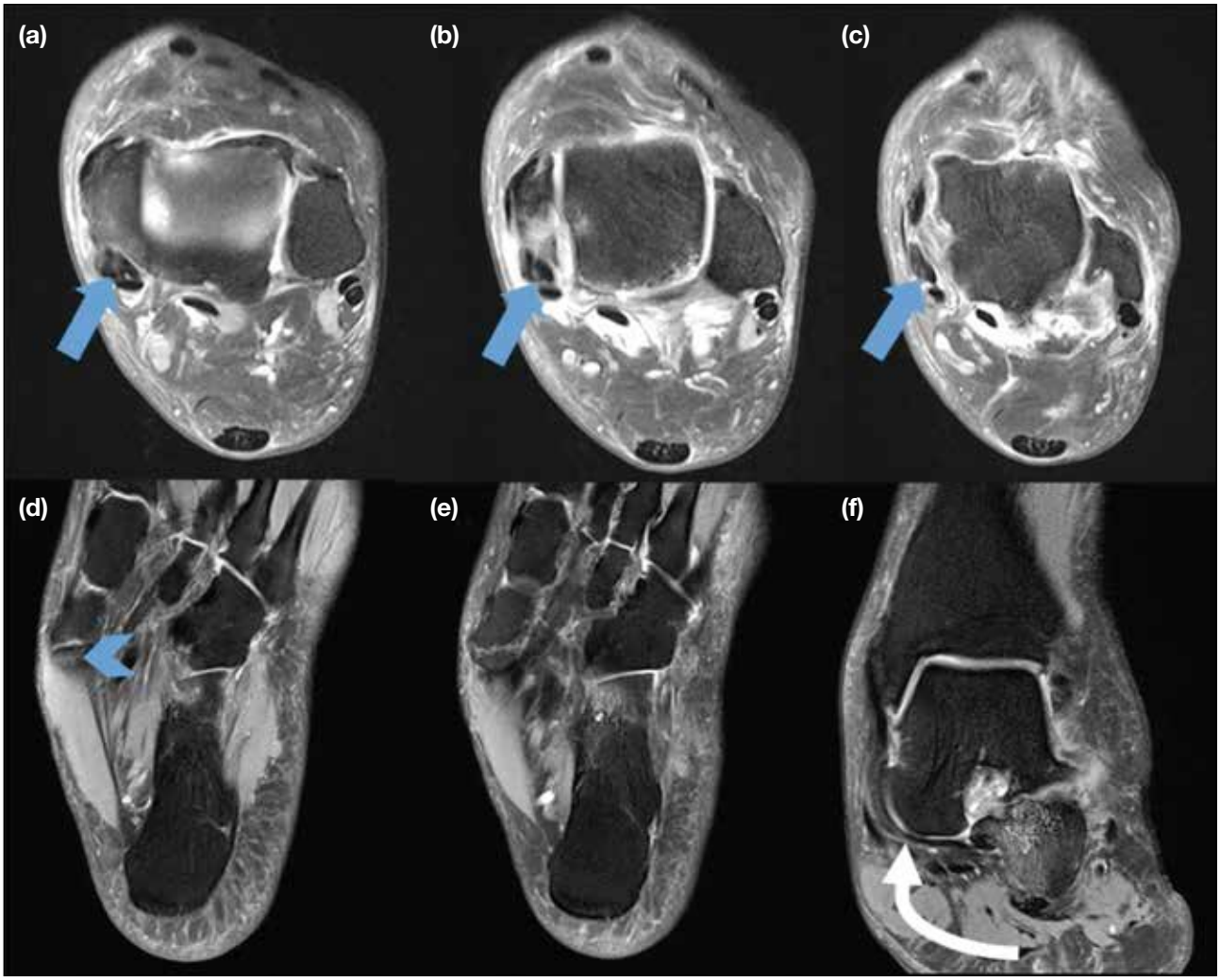

Figure 6. A 57-year-old woman presented with persistent left ankle swelling and pain over the medial aspect of the foot. Magnetic resonance imaging (MRI) of the left ankle revealed a split tear of the posterior tibial tendon (PTT). (a-c) Axial fat-saturated (FS) proton density (PD)–weighted MRI from proximal to distal reveal a grossly thickened PTT with a full thickness split tear, leading to the four-tendon sign (arrows). (d, e) A type II os naviculare with slight increase in marrow fluid signal (arrowhead in [d]) on the FS PD-weighted sequence could represent os naviculare syndrome. (f) Coronal FS PD-weighted MRI showing thinning of the superomedial calcaneonavicular ligament (curved arrow), a component of the spring ligament complex, suggestive of partial tear.

talonavicular joint.[3] Insufficiency of the deltoid ligament complex is seen in late stage of PTTD.[18] On MRI, low-grade sprain causes amorphous signal with loss of the normal striated appearance (Figure 7c and 7d), while high-grade injuries manifest as fluid-filled gaps or even discontinuity of the ligament.[3]

Bony Malalignment

Dysfunction of the PTT causes tension and eventual failure of the secondary supporting structures including the spring ligament, tarsal sinus ligaments, and deltoid ligaments. The biomechanics of the foot are altered, causing a cascade of foot deformities.

Pes Planus

Collapse of the medial longitudinal arch results in pes planus, which can be demonstrated by a reduction in the calcaneal inclination angle on lateral weight-bearing radiographs.[19] The calcaneal inclination angle is drawn between the plane of support and the calcaneal inclination axis (i.e., the line connecting the most inferior point of the calcaneal tuberosity with the most distal and inferior point of the calcaneus along the calcaneocuboid joint) [Figure 9].[19,20] The normal range is 20° to 30°.[19,20]

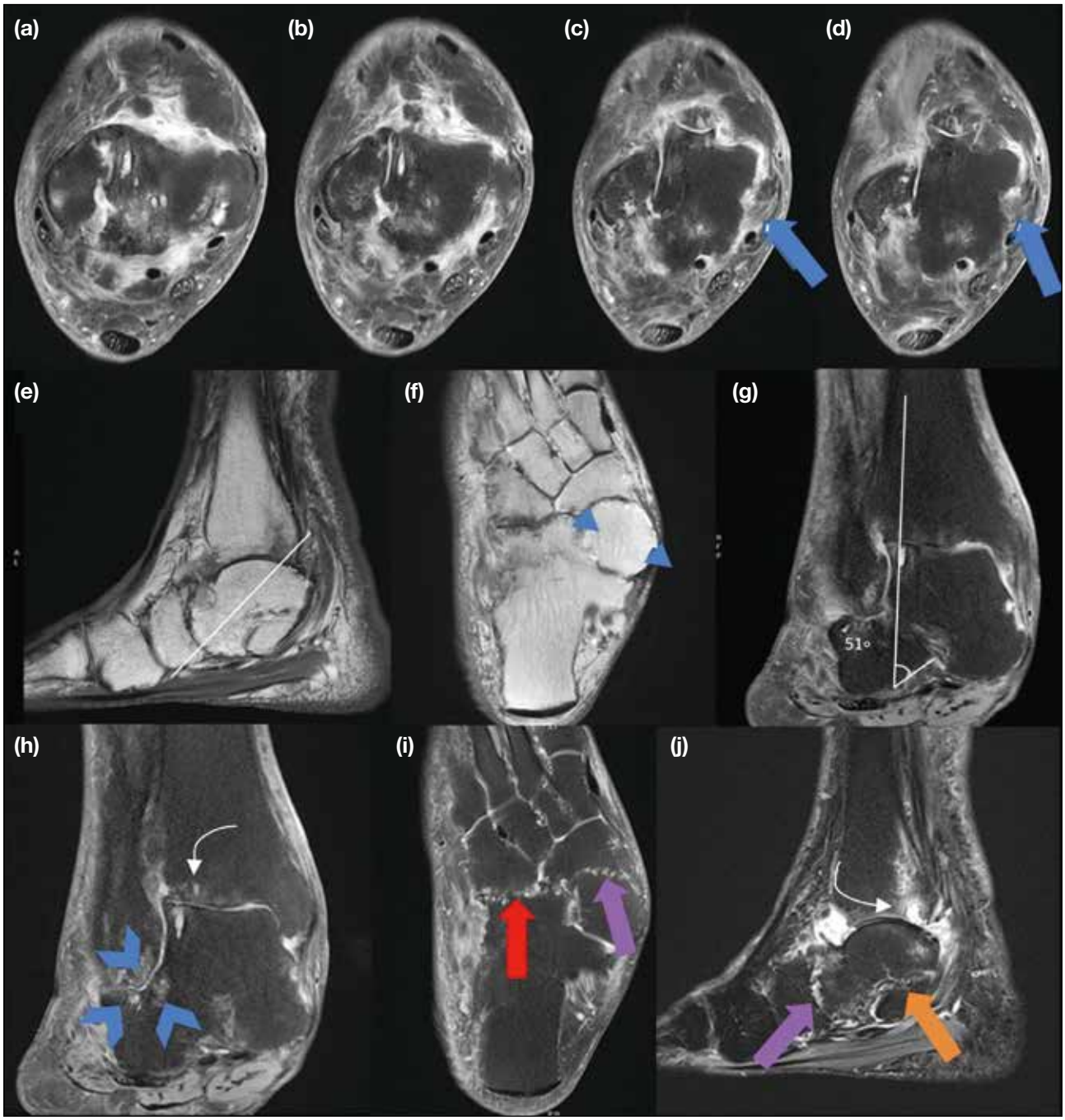

Figure 7. A 67-year-old woman with a history of rheumatoid arthritis reported longstanding bilateral ankle pain and stiffness, more severe in the medial aspect of the right ankle. She had bilateral fixed pes planus, as well as ankle, subtalar, and forefoot deformity. Magnetic resonance imaging (MRI) of her right ankle was performed. (a, b) Axial fat-saturated (FS) proton density (PD)–weighted MRI of the right ankle from proximal to distal show absence of the right posterior tibial tendon, suggestive of complete tear. (c, d) Increased signal in the deltoid ligament in the FS PD-weighted sequence (arrows) suggests partial tear. Sagittal T1-weighted MRI reveals plantar flexion of the talus and inferomedial migration of the talar head. The long axis of the talus, which is indicated by the white line in (e), is inferiorly tilted, instead of bisecting the navicular as in a normal foot. (f) On axial PD-weighted MRI, the navicular covers only about half of the talar head articular surface (the medial and lateral edges of which are marked by the blue triangles). (g) Coronal FS PD-weighted MRI demonstrates hindfoot valgus with an increased tibiocalcaneal angle (51°). (h) Coronal FS PD-weighted image of the right hindfoot shows talocalcaneal and subfibular impingement, bone marrow oedema, and subchondral cysts (arrowheads), signifying secondary osteoarthritis. There was also osteoarthritis of the tibiotalar joint (curved arrow). (i) Axial FS PD-weighted image illustrating osteoarthritis of the calcaneocuboid (red arrow) and talonavicular joints (purple arrow). (j) Osteoarthritis of the subtalar joint (orange arrow), tibiotalar joint (curved arrow), and talonavicular joint (purple arrow) are seen on sagittal FS T2-weighted image.

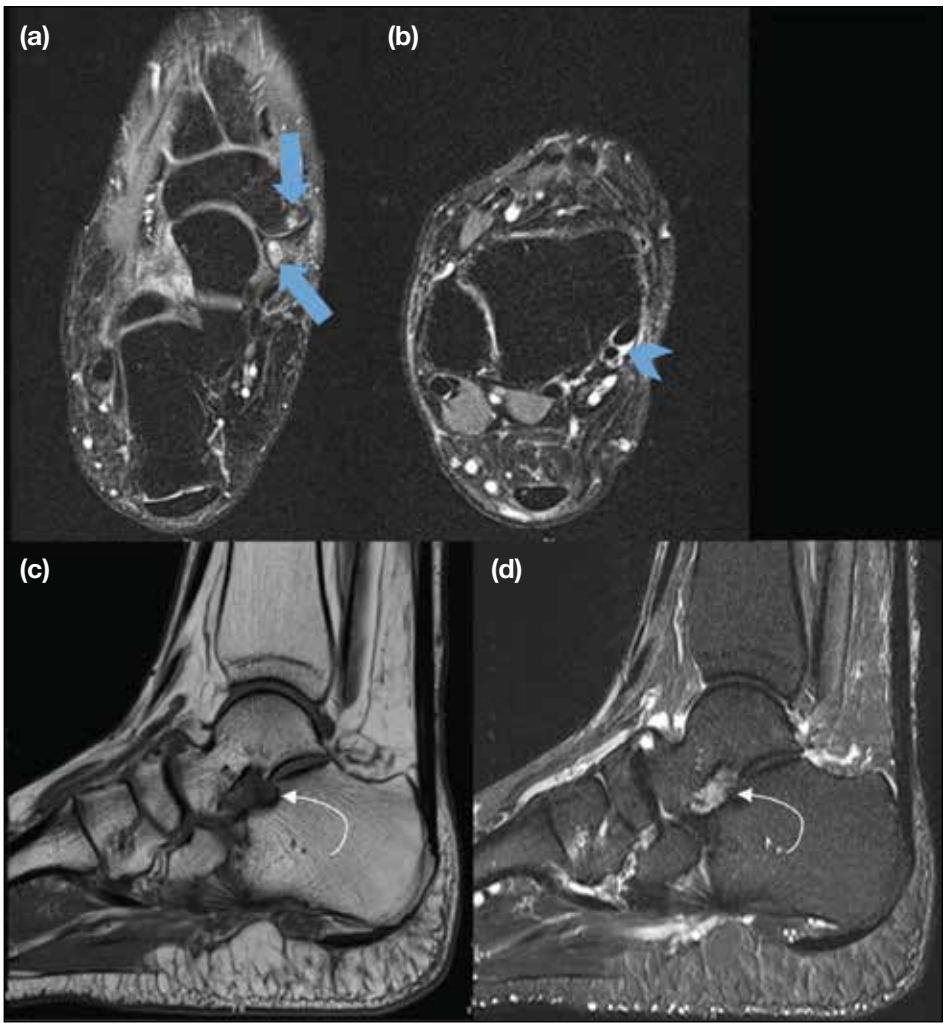

Figure 8. A 38-year-old woman presented with bilateral flatfoot deformity. Magnetic resonance imaging of her right ankle showing tenosynovitis of the posterior tibial tendon (PTT), os navicular syndrome, and sinus tarsi syndrome. (a) A type II os naviculare with bone marrow oedema (arrows) is evident on an axial fat-saturated (FS) proton density–weighted sequence. (b) Mildly increased tendon sheath fluid is seen along the PTT (arrowhead), suspicious for tenosynovitis. (c) Sagittal T1-weighted and (d) sagittal FS T2-weighted images showing abnormal T1 hypointense, T2 hyperintense soft tissue signal in the sinus tarsi (curved arrows), respectively, suggestive of sinus tarsi syndrome.

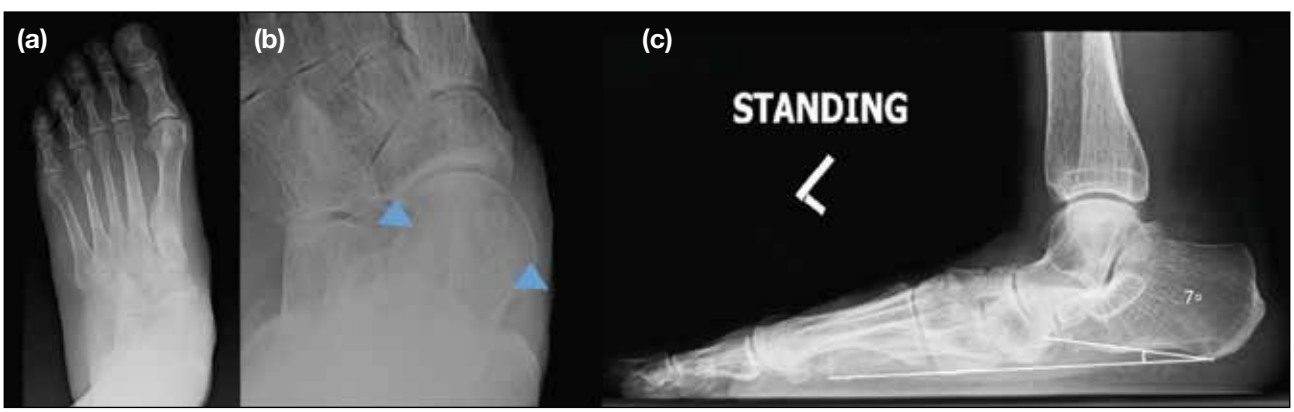

Figure 9. Radiographs of the left foot of the same patient with flatfoot deformity in Figure 4. (a) Standing dorsoplantar (DP) radiograph of the left foot and (b) cropped DP view focusing on the talonavicular joint demonstrate reduced talonavicular coverage. The navicular covers only about half of the talar head articular surface (the medial and lateral edges of which are marked by the blue triangles in [b]). (c) Lateral weight-bearing radiograph of the left foot showing marked reduction of the calcaneal inclination angle (7°), suggestive of pes planus.

Talonavicular Fault

As pes planus progresses, there is excessive plantar flexion of the talus, resulting in talonavicular fault which can be demonstrated on weight-bearing lateral radiographs of the foot or sagittal MRI.[6,19] On sagittal images including the base of the first metatarsal, a long axis drawn along the talus should normally divide the navicular into equal superior and inferior halves. If the line is inferiorly positioned, it suggests talonavicular fault (Figure 7e).[6]

Talar Head Uncoverage

The unopposed pull of the peroneus brevis abducts the forefoot and causes lateral subluxation of the talonavicular joint and uncoverage of the talus.[6,16,19] In a normal foot, most (75%-100%) of the talar head is covered by the navicular; in flatfoot deformity, the talar head becomes more uncovered (Figures 7f and 9b).[21]

Hindfoot Valgus

The degree of hindfoot valgus can be measured on radiographs or on the most posterior coronal image that includes the tibia and calcaneus on MRI, by measuring the angle between the long axis of the tibia and a line along the medial calcaneal wall (Figure 7g), with the normal range measuring 0° to 6°.[22]

Lateral Hindfoot Impingement

As hindfoot valgus progresses, lateral hindfoot impingement, including talocalcaneal impingement and/or subfibular impingement, may occur (Figure 7h).[18] On MRI, talocalcaneal impingement causes bone marrow oedema, cysts, and sclerosis at the site where the lateral talar process impinges on the lateral cortex of the calcaneus.[18] Common findings in subfibular impingement include low signal on T1-weighted imaging and predominantly low signal on T2-weighted imaging from soft tissue entrapment between the distal fibula and calcaneus, direct osseous contact between calcaneus and fibula, and distal fibular oedema.[18,22]

Secondary Osteoarthritis

In late-stage disease, chronic uneven stress and bony malalignment result in secondary osteoarthritis of the subtalar, talonavicular, calcaneocuboid, and tibiotalar joints.[10]

MANAGEMENT

Conservative treatment is the first-line treatment for PTTD and is indicated before operative treatment is considered. It involves treatment of the acute inflammation in the early stages, physiotherapy and accommodation of the deformity in late stages, including appropriate footwear (e.g., flat lace-up shoes) and orthosis.[7,23,24]

Operative treatment is indicated if conservative treatment fails, typically in Stages II to IV diseases and less frequently in Stage I disease, with reported good to excellent outcome in > 80% of the patients at 5 years' follow-up. A wide variety of surgical options have been reported; in general, they consist of different combinations of soft tissue procedures addressing the PTTD and osseous procedures addressing the malalignment. A few commonly performed procedures are discussed below.

In early PTTD (Stages I and II diseases), surgical release, simple tenosynovectomy and tendon debridement are indicated in cases of mild tendon inflammation, which have been reported to be helpful in pain relief.[25] However, combined procedures including soft tissue reconstructions (e.g., tendon transfer and side-to-side anastomosis) and bony procedures are often performed to address the PTTD and osseous deformities.[23]

In more advanced Stage II disease, tendon transfer is often the choice of treatment (Figures 10 and 11).[3] The FDL tendon is the most commonly transferred tendon, which is transferred to replicate the function of the PTT by augmenting hindfoot inversion strength and reducing adduction across the tarsal joint.[23] A calcaneal osteotomy is often performed at the same time to correct hindfoot valgus and medialise the pulling force of the Achilles tendon.[3,23,25]

Patients with an accessory navicular bone causing PTTD can benefit from the Kidner procedure, in which the accessory navicular bone is excised and the PTT is rerouted to a more plantar position at the undersurface of the remaining navicular (Figure 12).[26] Modifications of the Kidner procedure have been reported, which involve advancement of the PTT insertion using suture anchors, biotenodesis screws, or osseous tunnels to reattach the PTT.[23] The Kidner procedure and its modifications remain the current standard of care, with reported success rates of up to 96%.[27,28]

In the late stages of PTTD (Stages III and IV), surgical management often involves different forms of arthrodesis, with or without concomitant soft tissue reconstruction and osteotomy procedures.[3,23,24]

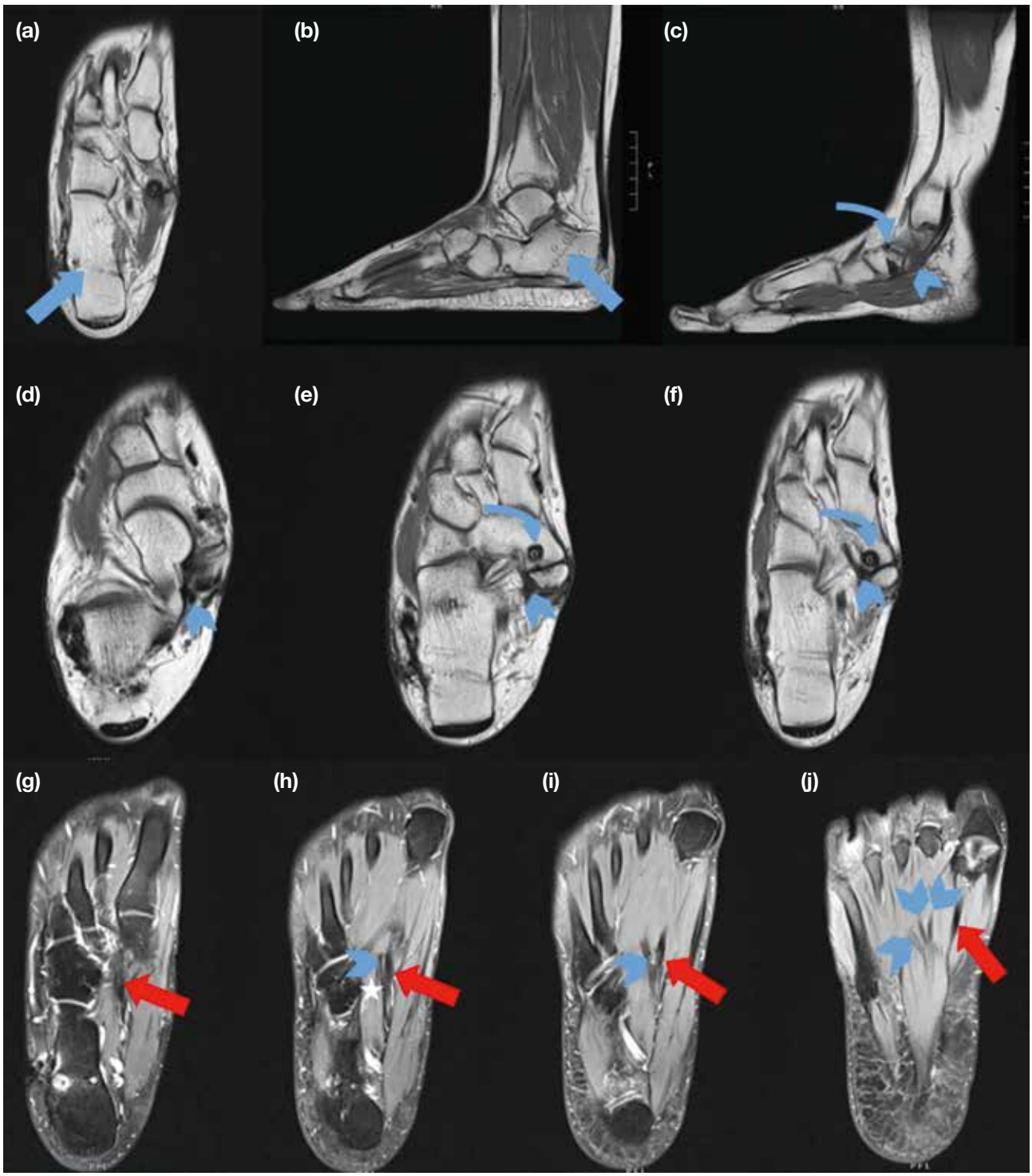

Figure 10. Postoperative magnetic resonance imaging (MRI) of the right ankle of a 26-year-old woman who underwent flexor digitorum longus (FDL) tendon transfer and medialising calcaneal osteotomy for posterior tibial tendon dysfunction after failing conservative treatment. (a, b) Axial and sagittal proton density (PD)–weighted MRI of the right ankle showing evidence of medialising calcaneal osteotomy with satisfactory bony union (arrows). (c) Sagittal and (d-f) axial PD-weighted MRI of the right ankle from proximal to distal showing the distal FDL tendon (arrowheads) transferred into a bony tunnel in the navicular and fixed with a screw (curved arrows in [c], [e], and [f]). (g-j) Axial fat-saturated PD-weighted MRI of the right ankle from proximal to distal demonstrating the FDL tendon (arrowheads in [h] to [j]) attachment to flexor hallucis longus tendon (red arrows) at the level of the Knot of Henry (star in [h]).

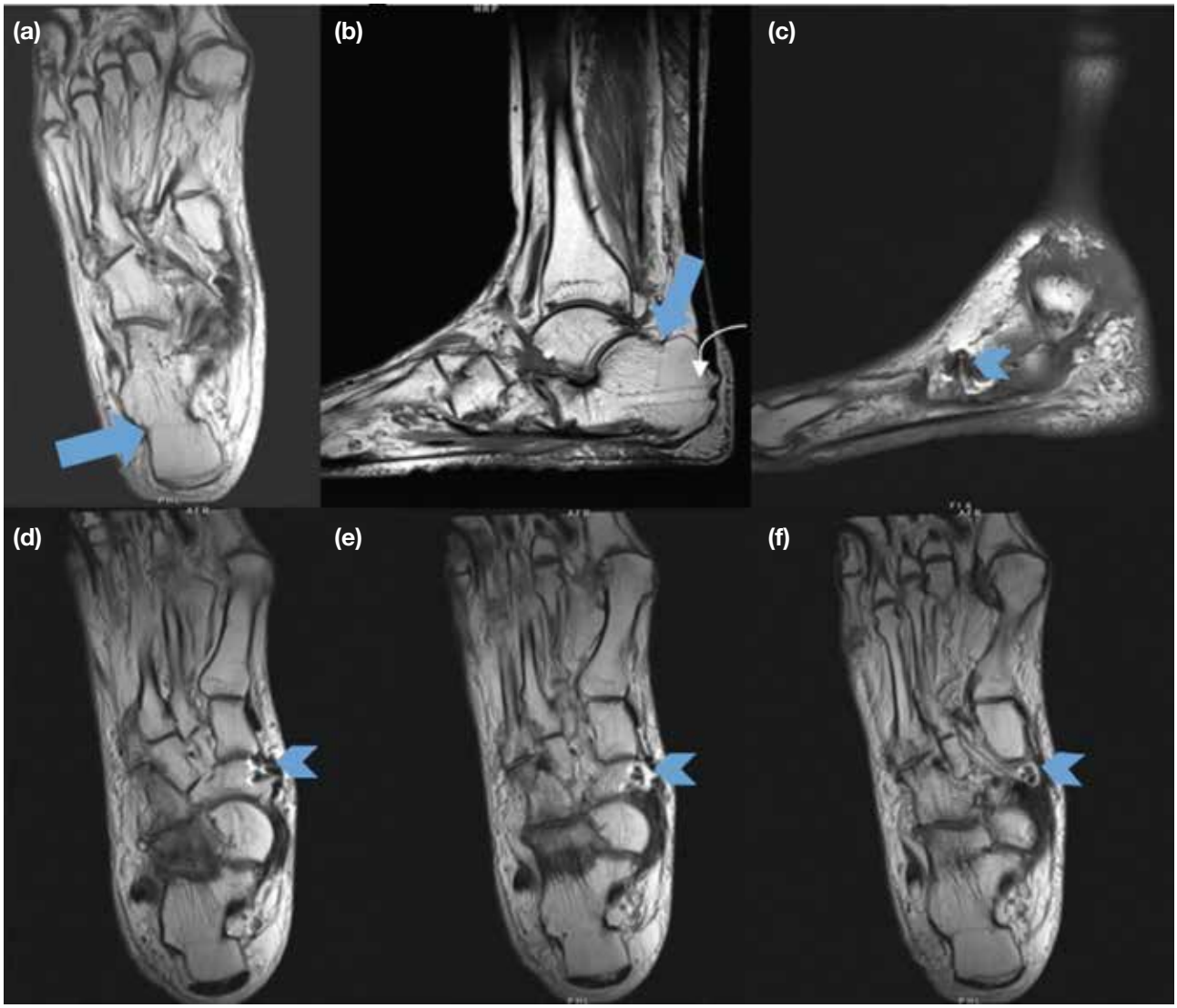

Figure 11. Postoperative magnetic resonance imaging (MRI) of the right ankle of a 67-year-old man who had flexor hallucis longus (FHL) tendon transfer and medialising calcaneal osteotomy for rupture of posterior tibial tendon 8 years ago. (a) Axial proton density (PD)–weighted and (b) sagittal T1-weighted MRI of the right ankle showing satisfactory bony reunion from medialising calcaneal osteotomy (arrows). There is evidence of prior screw insertion from proximal to distal calcaneum for fixation (curved arrow in [b]), which had already been removed. (c) Sagittal T1-weighted MRI and (d-f) axial PD-weighted MRI from proximal to distal showing surgical materials at the tunnel created at the navicular bone (arrowheads), where the transferred FHL tendon is attached.

CONCLUSION

Imaging findings of PTTD include a spectrum of changes ranging from tenosynovitis, tendinosis, and partial to complete tear. A myriad of related secondary findings may be seen as PTTD increases strain on other supporting structures, causing progressive malalignment and deformity of the foot. Familiarisation with the imaging features of PTTD allows early diagnosis, guides appropriate treatment, and aids surgical planning in advanced cases.

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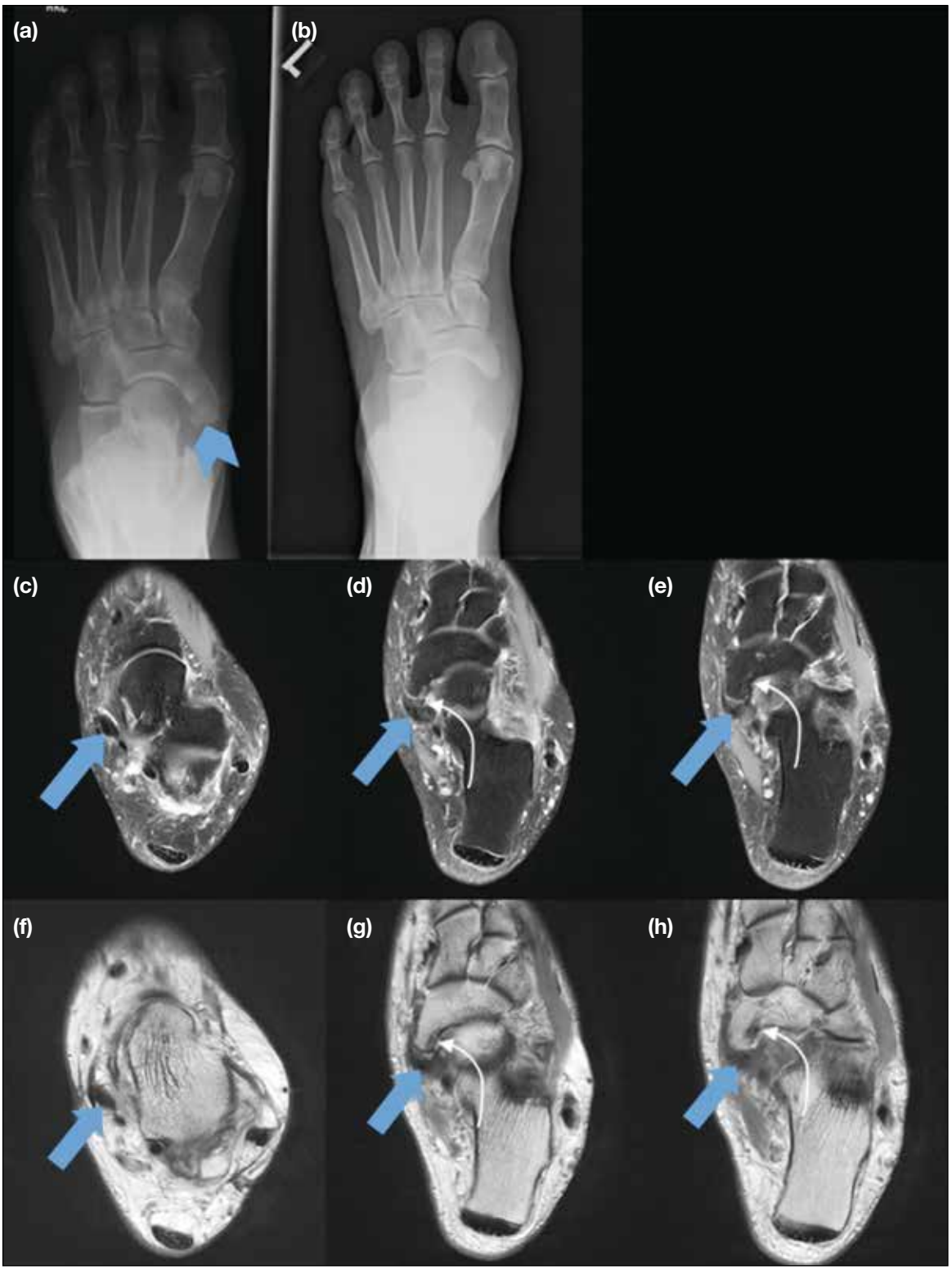

Figure 12. Radiographs and magnetic resonance imaging (MRI) of a 50-year-old woman with a history of left posterior tibialis tendonitis and accessory navicular, who underwent a Kidner procedure and later re-excision of bony overgrowth of the navicular. (a) Preoperative dorsoplantar (DP) radiograph of the left foot showing a prominent navicular bone with type II accessory navciular (arrowhead). (b) Postoperative DP radiograph of the left foot demonstrates that the accessory navicular and part of the parent navicular have been excised. (c-e) Postoperative axial fat-saturated proton density (PD)–weighted MRI of the left ankle from proximal to distal show the distal tibialis posterior tendon (arrows) reattached to the navicular body by a suture anchor (curved arrows in [d] and [e]). (f-h) Axial PD-weighted MRI corresponding to Figure 12c to 12e reveals similar postoperative findings. The distal tibialis posterior tendon (arrows) was reattached to the navicular body by a suture anchor (curved arrows in [g] and [h]), which is better appreciated in this sequence.

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