



Sonographic and radiographic findings of posterior tibial tendon dysfunction: a practical step forward

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Abstract

The purpose of this article is to describe the sonographic and radiographic findings in the diagnosis and treatment of posterior tibial tendon dysfunction. Ultrasound and radiographs play a crucial role in the diagnosis of posterior tibial tendon dysfunction and in imaging the postoperative changes related to posterior tibial tendon dysfunction. Early detection and diagnosis of posterior tibial tendon dysfunction is important in helping to prevent further progression of disease, obviating the need for more invasive and complex procedures.

Keywords Posterior tibial tendon dysfunction · Musculoskeletal ultrasound · Pes planus · Hindfoot valgus · Sinus tarsi syndrome · Spring ligament insufficiency · Lateral hindfoot impingement · Subfibular impingement

Introduction

Posterior tibial tendon dysfunction (PTTD) is the most common cause of adult acquired pes planus, and if not diagnosed and treated early, can result in significant pain, disability, and a foot and ankle deformity [1, 2]. PTTD refers to a spectrum of clinical and imaging findings related to abnormalities of the posterior tibial tendon (PTT), causing dysfunction and eventual loss of its functionality. At one end of the spectrum, this includes pain and instability related to posterior tibial tendinosis, tenosynovitis or subluxation. At the other end, this includes complete rupture of the tendon with a marked clinical

deformity and associated imaging findings, which are discussed in detail. Early detection is therefore crucial and can often slow the progression of disease, allowing the use of conservative treatments such as physical therapy, braces, orthotics, anti-inflammatory medications, and ultrasound-guided injections with reported good results [3–5]. The purpose of this article is to describe the pathophysiology and the radiographic and sonographic findings of PTTD. Radiographic findings of PTTD, including pes planus, a talonavicular fault, uncovering of the talonavicular joint, distal medial tibia changes, subtalar and tibiotalar joint osteoarthritis, hindfoot (HF) valgus, and HF impingement, are discussed and illustrated in detail. Although ultrasound cannot be reliably used to evaluate many of the osseous changes and other causes of pes planus, such as tarsal coalition, knowledge of its use in conjunction with the clinical and radiographic findings, is critical in the evaluation of the PTT and in the diagnosis and staging of PTTD [4]. Sonographic findings of PTTD including posterior tibial tendinosis, tenosynovitis, tears, subluxation, and other important associated sonographic findings are reviewed. Treatment options and their associated radiographic and sonographic findings are also discussed.

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Epidemiology

Posterior tibial tendon dysfunction most often occurs in middle-aged to elderly women [2, 4–6]. It is predominantly the result of tendon ischemia and most commonly involves the PTT at the level of the medial malleolus [1, 6–8]. Those with a history of obesity, diabetes, chronic steroid use, congenital flatfoot deformity, surgery, trauma, and inflammatory arthropathies are at increased risk [2, 6, 9].

Athletes may also be affected, commonly presenting with posterior tibial tenosynovitis. Those in sports and activities including soccer, basketball, running, ice hockey, gymnastics, and ballet dancing, which involve high stress or repetitive activities, are most commonly affected [10–12]. Although rare, if PTT rupture does occur in this younger athletic population, it typically occurs acutely and near the navicular insertion [13]. These less common distal PTT tears can also occur in patients with an underlying inflammatory arthropathy [7].

The presence of a type II or type III accessory navicular increases the risk of PTT tears and tendinosis at the navicular insertion; however, most patients with an accessory navicular do not have posterior tibial tendinopathy [1, 8]. An ossified accessory navicular (os naviculare accessorium) is present in up to 25% of individuals [14]. Based on the Geist classification, there are three distinct accessory navicular entities that exist (Fig. 1) [15]. Type I is a small round ossicle adjacent to the navicular tubercle and embedded within the PTT. A type I navicular can also be referred to as an os tibiale externum. Type II is a large triangular or slightly rounded ossification adjacent to the navicular tubercle, with an associated synchondrosis. Pain can occur at the synchondrosis because of abnormal motion between the ossicle and the native navicular known as accessory (os) navicular syndrome [7]. Type III is an enlarged medial horn of the navicular at the tibial tubercle known as a cornuate navicular. In type II and type III navicular configurations, the bulk of the PTT inserts onto the accessory navicular rather than its normal insertion sites, altering and weakening the insertion point [1, 6–8, 14].

Anatomy

The PTT lies in a concave area along the posteromedial edge of the distal tibia known as the retromalleolar groove. The flexor digitorum longus tendon lies just posterior and adjacent to the PTT. The PTT extends proximally to the tibialis posterior muscle. This muscle originates at the proximal third of the lower leg from the interosseous membrane and the adjacent posterior tibial surface. The PTT continues inferiorly distally to the medial malleolus within the tarsal tunnel. Just proximal and superficial to the tarsal tunnel resides the flexor retinaculum, an important structure for preventing subluxation, dislocation, and bowstringing of the flexor tendons of the ankle.

Distal to the tarsal tunnel, the bulk of the PTT inserts onto the medial aspect of the navicular at the navicular tubercle. However, there are additional slips of the PTT that insert at the cuneiforms and the second, third, and fourth metatarsal bases [1, 16–18].

The PTT is surrounded by a sheath, but it is important to note that this synovial sheath ends at the mid-portion of the talus.

The proximal PTT receives its arterial supply via the posterior tibial artery whereas the distal tendon and insertion are supplied by both the posterior tibial and dorsalis pedis arteries. The mid-portion of the tendon, at the medial malleolus, however, has a relatively poor blood supply. This area is prone to ischemia, and is therefore, the most common site of posterior tibial tendinopathy and eventual rupture in adults [1, 2, 6, 16–18].

The PTT is the primary stabilizer of the longitudinal arch of the foot. The muscle and tendon act to plantarflex the ankle and invert the foot [1, 16–18]. Dysfunction of the tendon results in eventual loss of the medial longitudinal arch of the foot with resultant pes planus, eversion at the subtalar joint, and a valgus alignment of the heel leading to an eventual HF valgus deformity [2].

Diagnosis

History and clinical findings

Patients with PTTD often present with medial foot pain, weakness, difficulty weight-bearing or a developing flatfoot deformity [2, 19]. On physical examination, these patients may present with the “too many toes” sign because of the pes planus and HF valgus (HF planovalgus) deformities [2]. They also have difficulty and pain standing on their toes, with an inability to perform a single heel rise test [2]. Initially, the HF planovalgus deformity is clinically flexible and not rigid. As the PTT loses functionality and associated osseous changes develop, the HF planovalgus deformity becomes clinically rigid [20–23]. As patients gradually develop severe HF valgus in the later stages of PTTD, they may feel as if they are walking on the medial ankle and complain of excessive wear involving the sole of their shoes [24]. Also, in the end stages of PTTD, as the HF planovalgus deformity worsens, the distal fibula abnormally contacts the adjacent lateral calcaneus, resulting in complaints of lateral ankle pain [2, 25, 26]. Diabetics with neuropathy often present late owing to a lack of the typically associated sensation of pain related to posterior tibial tendinopathy [1]. Rather than presenting with the early clinical and imaging findings of PTTD, including pain related to posterior tibial tendinosis, these patients can present late with PTT rupture and a rigid flatfoot deformity [21, 23].



Fig. 1 Classification of accessory navicular entities demonstrated on anteroposterior radiographs of the foot. **a** Normal navicular (*white arrow*). **b** Type I navicular with a small ossicle adjacent to the navicular

tubercle (*black arrow*). **c** Type II with a triangular or rounded ossification (*star*). **d** Type III with an enlarged medial horn of the navicular (*triangle*)

Imaging techniques

Initially findings of PTTD are determined by the presenting clinical symptoms, physical examination, and findings on the weight-bearing radiographs of the foot and ankle. Anteroposterior, oblique, and lateral weight-bearing radiographs of both the foot and ankle should be obtained initially, allowing for evaluation of HF planovalgus and other associated osseous changes, which will alter PTTD staging and determine appropriate management and treatment [25]. Foot radiographs are required to evaluate for the previously discussed accessory navicular types, pes planus, and additional radiographic findings, which are subsequently discussed. Ankle radiographs are needed to better evaluate the subtalar and tibiotalar joints for osteoarthritis, which is crucial in the staging of PTTD. Ankle radiographs also allow better evaluation for tarsal coalition, another cause of pes planus. Weight-bearing is necessary to demonstrate the osseous structures and alignment in a functional position. These radiographs require the patient to be standing on their feet allowing weight distribution across both feet and ankles and allowing accurate evaluation of the longitudinal arch of the foot and the joint spaces of the subtalar and tibiotalar joints in particular [27]. A clinically suspected pes planus or HF planovalgus that is confirmed on weight-bearing radiographs is termed “fixed” [26–28]. If the patient cannot weight-bear, non-weight-bearing radiographs should still be obtained and can, in conjunction with the clinical findings, contribute to the diagnosis and staging of PTTD. Weight-bearing can also be simulated by applying plantar foot pressure using a plastic board [27].

Ultrasound or magnetic resonance imaging (MRI) play a crucial secondary role in the clinical and radiographic findings [4, 25]. At our institution, musculoskeletal ultrasound is most often used to diagnose PTTD after radiographs are obtained. Ultrasound is recommended as the imaging modality of choice for the diagnosis of PTTD after radiographs [4, 29]. Ultrasound has been shown to have a reported sensitivity of 100%, an accuracy of 93%, and a specificity of 88% in detecting PTT tears [25]. The dynamic capabilities and high resolution of ultrasound are crucial in diagnosing instability by evaluating for PTT subluxation and excluding important clinical mimickers of PTTD, such as crystalline deposition disease

including gout [30]. It is also critical in the evaluation of the PTT and in helping to accurately stage PTTD [2, 14, 25]. Specific details of PTTD staging and the sonographic findings of PTTD are discussed in a subsequent section.

Ultrasound of the foot and ankle should be performed using a high-frequency linear-array transducer, typically 12 MHz or higher [14, 30]. A higher frequency compact linear transducer may also be beneficial to evaluate the superficial PTT and allows easier maneuvering around the medial malleolus with minimal loss of contact. Ultrasound has shown similar accuracy to that of MRI in diagnosing posterior tibial tendinopathy [25]. In one study, high resolution ultrasound has even been shown to be slightly more accurate than 3 Tesla MRI in the diagnosis of PTTD [29].

There are many benefits of using ultrasound over MRI:

1. The ability to perform dynamic assessment while interacting directly with the patient.
2. Ultrasound has a higher spatial resolution than MRI and is excellent for imaging superficial structures.
3. The ability to perform real-time Doppler analysis and compare with the contralateral side.
4. The ease of accessibility and the lower cost of ultrasound compared with MRI.
5. The ability to perform ultrasound in patients with contraindications to MRI [31].

However, it is critical that the ultrasound is performed by a sonographer trained in musculoskeletal ultrasound as it is an operator-dependent imaging technique. Also, some referring clinicians and surgeons, especially if surgery is contemplated, may not be comfortable with the use of musculoskeletal ultrasound and may prefer MRI in its place or in conjunction with an ultrasound.

During the routine ultrasound evaluation of the ankle, the PTT is followed from the musculotendinous junction proximal to the medial malleolus, inferior to the navicular insertion on both the long and the short axis (Fig. 2). The PTT on the long axis normally appears homogeneous with a defined hyperechoic fibrillary pattern. On the short axis, it has a uniform echogenic appearance [14]. More superficially, the flexor retinaculum is also evaluated just above the medial

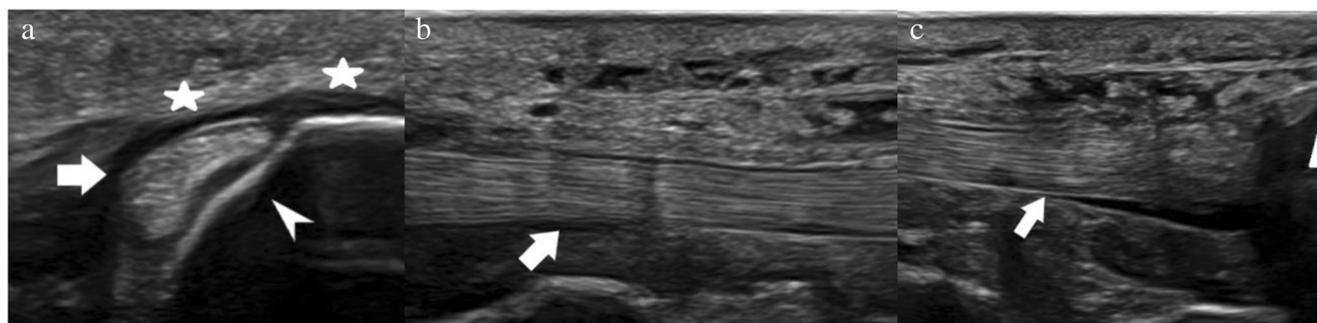


Fig. 2 Normal sonographic appearance of the posterior tibial tendon (PTT) in a 39-year-old man. **a** Short-axis sonographic image of the PTT (*arrow*) at the level of the medial malleolus in the retromalleolar groove (*arrowhead*) demonstrates the normal uniform echogenic appearance. Superficial to the PTT, the flexor retinaculum (*stars*) is seen attaching to the tibia and appearing uniformly thin and hypoechoic. **b** Long-axis

sonographic image of the PTT (*arrow*) obtained at the level of the medial malleolus demonstrating the normal homogenous, defined, hyperechoic, fibrillary pattern. **c** Additional long-axis sonographic image of the PTT (*arrow*) inferior to the medial malleolus demonstrating the normal appearance and the normal navicular insertion (*triangle*)

malleolus, attaching to the tibia and appearing uniformly thin and hypoechoic (Fig. 2) [32]. Dynamic evaluation for PTT subluxation is especially unique to ultrasound and can be performed by two means. One method is by placing the patient's foot in slight plantarflexion and inversion and instructing the patient to point his/her forefoot toward the ceiling (dorsiflexion) against the sonographer's manual resistance, while evaluating the PTT in the short axis at the level of the medial malleolus [30]. A second maneuver is to place the foot in eversion and instruct the patient to actively perform circumduction while assessing the PTT in the same way [25]. Subluxation is diagnosed when there is anterior displacement of the tendon over the medial malleolus. Diagnosis of PTT subluxation is important in the evaluation of tendon function, as chronic subluxation causes instability, which can lead to tendinosis or tenosynovitis and eventual PTT rupture [25, 30, 32]. As PTTD is based on PTT functionality, this unique dynamic feature of ultrasound allows the radiologist to determine function through imaging.

Staging and classification

In 1989, Johnson and Strom developed a classification describing three stages of PTTD. Although Johnson and Strom briefly alluded to a possible fourth stage, in 1996, Myerson and Corrigan formally added a final fourth stage [26, 33]. Using a combination of the clinical, radiographic, and ultrasound or MRI findings, PTTD is placed into one of these four stages (Table 1) [2, 25, 26, 33]. In stage I, there is inflammation of the PTT due to tendinosis, tenosynovitis or subluxation, evident on ultrasound or MRI, with no associated clinically rigid or radiographically fixed pes planus or HF planovalgus deformities. As a fixed pes planus develops on weight-bearing radiographs and as the ultrasound or MRI demonstrates severe posterior tibial tendinosis, a partial

thickness tear or partial tendon rupture, the patient is upgraded to stage II [12]. At this stage, Johnson and Strom and Myerson and Corrigan used the phrase “tendon elongation,” referring to the pathological findings of tendon degeneration with partial tearing, resulting in tendon lengthening and laxity and a subsequent radiographically fixed pes planus [26, 33]. As subtalar joint eversion worsens because of eventual PTT rupture, stage III commences with the development of a clinically rigid and radiographically fixed HF planovalgus and subtalar joint osteoarthritis. This is also known as talocalcaneal impingement. The final stage, stage IV, is applied when the rigid and fixed HF planovalgus progresses, the talus tilts within the tibiotalar joint, approximating the lateral aspect of the tibial plafond and resulting in tibiotalar joint osteoarthritis. This stage can also include deltoid ligament dysfunction due to the severe HF planovalgus deformity [12, 24–26, 33, 34].

As the patient progresses from stage III to stage IV, extra-articular lateral HF impingement develops. This includes the stage III finding of talocalcaneal impingement and what is referred to as subfibular impingement. As the severe HF planovalgus worsens, the lateral aspect of the talus, in addition to the lateral calcaneus, begin to abnormally contact the distal fibula, resulting in subfibular impingement. This subfibular impingement can also affect the adjacent peroneal tendons [24, 25, 34–36].

Radiographic findings of PTTD

Accessory navicular type

Anteroposterior radiographs of the foot are required to evaluate for a type II or type III accessory navicular that can increase the risk of PTTD (Fig. 1). Navicular morphology cannot be determined by physical examination and its presence

Table 1 Summarized findings of posterior tibial tendon dysfunction by stage

Stage	Findings
I	Inflammation of the posterior tibial tendon with no radiographically fixed or clinically rigid deformity
II	Severe posterior tibial tendinosis, partial thickness tearing, and tendon lengthening (i.e., severely dysfunctional to nonfunctional tendon). Fixed pes planus on weight-bearing radiographs, although clinically flexible
III	Posterior tibial tendon rupture (i.e., nonfunctional tendon). Fixed and rigid hindfoot planovalgus with subtalar joint osteoarthritis
IV	Talar tilt within the tibiotalar joint with tibiotalar joint osteoarthritis

on radiographs can help to diagnose PTTD, especially if pain corresponds to this area.

Pes planus

Lateral weight-bearing radiographs of the foot are used to evaluate for a clinically suspected pes planus, which is present in stages II–IV of PTTD. One method of evaluating for pes planus is using the calcaneal inclination (pitch) angle. This angle is made between the plane of support and the calcaneal inclination axis on the lateral weight-bearing radiograph of the foot (Fig. 3). It is normally 20–30° and is decreased in pes planus and increased in pes cavus [37].

Talonavicular fault (“talar sagging”)

As pes planus worsens, the talus can undergo excessive plantar flexion and result in the radiographic finding of a talonavicular fault [1]. This is diagnosed on the weight-bearing lateral foot or ankle radiograph and manifests as an inferiorly located talar head in conjunction with pes planus (Figs. 3a and 4a). Talonavicular fault is particularly common in spring ligament injuries owing to a high association between injuries of this ligament and PTT injuries [6, 32, 38].

Subtalar joint osteoarthritis

In stages III and IV, lateral weight-bearing radiographs of the ankle and foot best demonstrate the usual findings of

osteoarthritis at the subtalar joint, including joint space narrowing, subchondral sclerosis, subchondral cyst formation and osteophytosis (Fig. 4) [25].

HF valgus

Also, in stages III and IV, HF valgus is present and can be diagnosed radiographically using the talocalcaneal (Kite’s) angle. This angle is obtained on anteroposterior weight-bearing radiographs of the foot and is made by the intersection of lines drawn through the axis of the talus and through the axis of the calcaneus (Fig. 5). The talocalcaneal angle is normally between 25 and 40°. As the foot pronates with the HF valgus, the calcaneus abducts and rotates away from the talus, resulting in an increased talocalcaneal angle [27, 37, 39, 40].

Although typically obtained by MRI or CT, the HF valgus angle may also be obtained on HF alignment view radiographs [41]. The ankle normally has a HF valgus angle of 0–6°. This is measured by the angle created by drawing a line along the medial calcaneal wall and another line drawn parallel to the longitudinal axis of the tibia [35, 41]. As reported by MRI results, HF valgus can be graded as mild (7–16°), moderate (17–26°), and severe (> 26°) [36].

Uncovering of the talonavicular joint (“unroofing of the talus”)

Multiple radiographic findings are seen as the HF valgus worsens and as the patient transitions from stage III to stage



Fig. 3 Lateral weight-bearing radiographs of the foot show the calcaneal inclination angle in a 53-year-old woman with pes planus and a talonavicular fault before and after corrective surgery. **a** A decreased calcaneal inclination angle consistent with pes planus before surgery. The angle is obtained between the plane of support and the calcaneal inclination axis. There is also excessive plantar flexion of the talus

consistent with a talonavicular fault. **b** A normal calcaneal inclination angle obtained in the same patient after undergoing a calcaneal osteotomy with a partially threaded cannulated screw and a calcaneal plate-and-screw fixation device with resolution of the previously seen pes planus and talonavicular fault



Fig. 4 Findings of posterior tibial tendon dysfunction (PTTD) in a 77-year-old woman with stage IV PTTD presenting with chronic ankle pain. **a** Lateral weight-bearing radiograph of the ankle demonstrates severe osteoarthrosis at the posterior facet of the subtalar joint (*white solid arrow*) and a talonavicular fault, as seen in stages III and IV of PTTD. **b** Anteroposterior weight-bearing ankle radiograph of the same patient shows findings of stage IV PTTD, including hindfoot valgus with talar tilt and tibiotalar joint osteoarthrosis (*black solid arrow*) and uncovering of the talonavicular joint (*empty triangle*). Extra-articular lateral hindfoot

impingement is also seen with neofacets (*star*) secondary to abnormal contact and remodeling between the distal fibula and the lateral talus (talocalcaneal impingement) in addition to the lateral calcaneus (subfibular impingement). A distal medial tibial hypertrophic ridge (*empty white arrow*) is also noted compatible with posterior tibial tendinopathy. **c** Anteroposterior weight-bearing radiograph of the foot in a 36-year-old male patient also demonstrates hindfoot valgus with uncovering of the talonavicular joint (*empty black arrow*)

IV. One important radiographic finding that can be seen is the uncovering of the talonavicular joint. This is best diagnosed on the anteroposterior weight-bearing radiographs of the foot and ankle by identifying lateral subluxation of the navicular at the talonavicular joint, resulting in a non-articulating portion visualized at the medial aspect of the talar head (Fig. 4b, c). This is the result of a severely dysfunctional PTT and unopposed pull by the peroneus brevis tendon resulting in lateral subluxation of the navicular at the talonavicular joint. Typically in the finding of the uncovering of the talonavicular joint, less than 85% of the articular surface of the talus remains covered by the navicular [1, 27, 42].

Tibiotalar joint osteoarthrosis

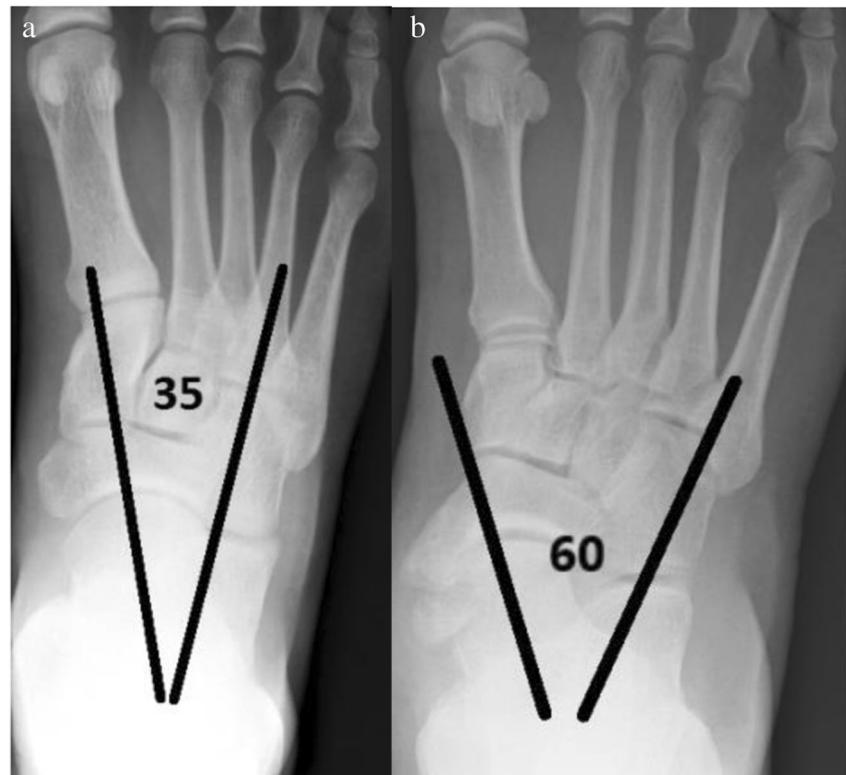
In the final stage, stage IV PTTD, typical findings of osteoarthrosis at the tibiotalar joint become evident. This includes talar tilt and joint space narrowing between the lateral

aspect of the talar dome and the lateral aspect of the tibial plafond with resultant subchondral sclerosis, osteophytosis, and subchondral cyst formation (Fig. 4) [2].

Extra-articular lateral HF impingement

As extra-articular lateral HF impingement develops, including talocalcaneal and subfibular impingement, abnormal facets or “neofacets” may form, resulting in cortical remodeling and flattening at the bony contours (Fig. 4). This is best diagnosed on the anteroposterior and oblique weight-bearing ankle radiographs. The neofacets typically first develop at the area of abnormal contact between the calcaneus and the lateral aspect of the talus in talocalcaneal impingement and as the HF valgus worsens, at the area of abnormal contact between both the lateral aspect of the talus and the lateral calcaneus with the distal fibula in subfibular impingement [35, 36].

Fig. 5 Anteroposterior weight-bearing radiographs of the foot demonstrating the normal and abnormal talocalcaneal angle, which is measured by lines drawn through the axis of the talus and calcaneus. **a** Normal talocalcaneal angle measurement in a 24-year-old woman. **b** Increased talocalcaneal angle in a 47-year-old woman with hindfoot valgus. A type II accessory navicular is also noted



Avulsion fracture

Another associated radiographic finding that may be seen in PTTD is an avulsion fracture at the flexor retinaculum attachment at the distal medial tibia (Fig. 6a). PTT dislocations are rare and typically occur in athletes secondary to trauma or due to repetitive microtrauma. They occur in conjunction with a tear of the flexor retinaculum, which keeps the tendon in place within the retromalleolar groove [1, 30, 43].

Distal medial (medial malleolus) tibia changes

Another associated radiographic finding that can also suggest PTTD is a small hypertrophic ridge (spur), an area of cortical irregularity or slight periosteal reaction (periostitis) of the distal medial tibia along the superior aspect of the medial malleolus (Fig. 6). This is thought to be related to PTT chronic subluxation, inflammation, chronic tenosynovitis or a chronic or remote flexor retinaculum injury [1, 43–45].

Sonographic findings of PTTD

PTT tendinosis

The normal PTT is approximately twice the diameter of the adjacent flexor digitorum longus tendon when imaged by

ultrasound in the short axis [32, 46]. In PTTD stages I and II, the PTT loses its normal homogenous and hyperechoic appearance. When tendinotic, the PTT becomes thickened, enlarged, and appears focally or diffusely hypoechoic. The tendon also loses its normal defined fibrillary architecture when imaged on the long axis (Fig. 7). There may also be associated hyperemia [14, 47].

Tenosynovitis

In stage I, particularly in the younger athletic population, PTTD may present with acute symptomatic tenosynovitis [12, 13, 35]. A small amount of compressible, anechoic, physiological fluid can normally be seen in the PTT sheath; however, this should be less than 1–2 mm thick and never circumferential. Additionally, there should not be any fluid at the distal 1–2 cm of the tendon, as it is devoid of a tendon sheath [1, 14]. Alternatively, tenosynovitis may appear as a hypoechoic rind surrounding the tendon within the PTT sheath, resulting from complex fluid with associated echogenic debris or due to thickened synovium. This may occur with or without hyperemia (Fig. 7).

PTT partial thickness and full-thickness tears

As tendinosis worsens, ultrasound can be used to detect partial-thickness and full-thickness tears of the PTT.



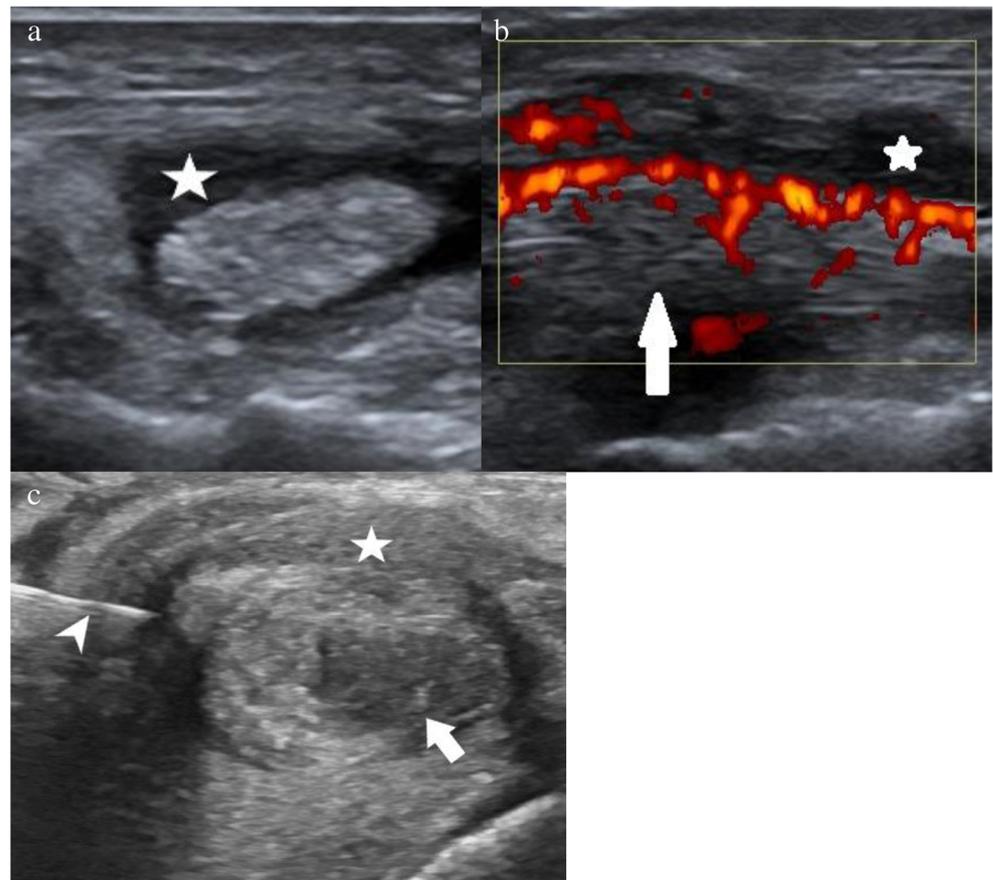
Fig. 6 Anteroposterior radiographs of the ankle demonstrating distal medial tibial (medial malleolus) changes associated with PTTD. **a** A 27-year-old man with a history of ankle trauma and previous surgery displays evidence for a remote flexor retinaculum avulsion fracture (*arrow*). **b** A hypertrophic ridge is noted at the distal medial tibia

(*arrowhead*) in a 52-year-old woman with a history of trauma, previous surgery, and posterior tibial tendinosis. **c** Periostitis is noted along the distal medial tibia (*star*) in a 61-year-old man with known posterior tibial tenosynovitis

Intrasubstance cystic changes can be present within the PTT that are compatible with mucoid degeneration. Partial-thickness tears and longitudinal split tears present on ultrasound as linear hypoechoic or anechoic clefts (Fig. 8a–c). A full-thickness tear or rupture manifests as a definable gap with associated proximal and distal retraction of the opposing tendon stumps (Fig. 8d, e). Utilizing the ultrasound benefits of dynamic maneuvers and

compression, these opposing stumps can be further delineated and full-thickness versus partial-thickness tears can be validated [14]. The most common location of partial- and full-thickness tears is at the perimalleolar zone, which represents the site of tendon-relative hypovascularity [1]. In younger athletes, patients with inflammatory arthropathies and in patients with a type II or type III navicular, these tears can be identified sonographically at the less

Fig. 7 Ultrasound images of posterior tibial tendinosis and tenosynovitis in a 58-year-old woman with severe medial ankle pain. **a** Short-axis and **b** long-axis ultrasound images of the PTT demonstrate tendinosis, as indicated by a hypoechoic, thickened tendon (*arrow*) and tenosynovitis (*star*) with hyperemia associated with the tendinosis and tenosynovitis. Tenosynovitis is noted by the hypoechoic rind surrounding the tendon in which the hyperemia is present. **c** Short-axis ultrasound image during an ultrasound-guided PTT sheath injection (needle—*arrowhead*) with underlying posterior tibial tendinosis (*arrow*) and tenosynovitis (*star*)



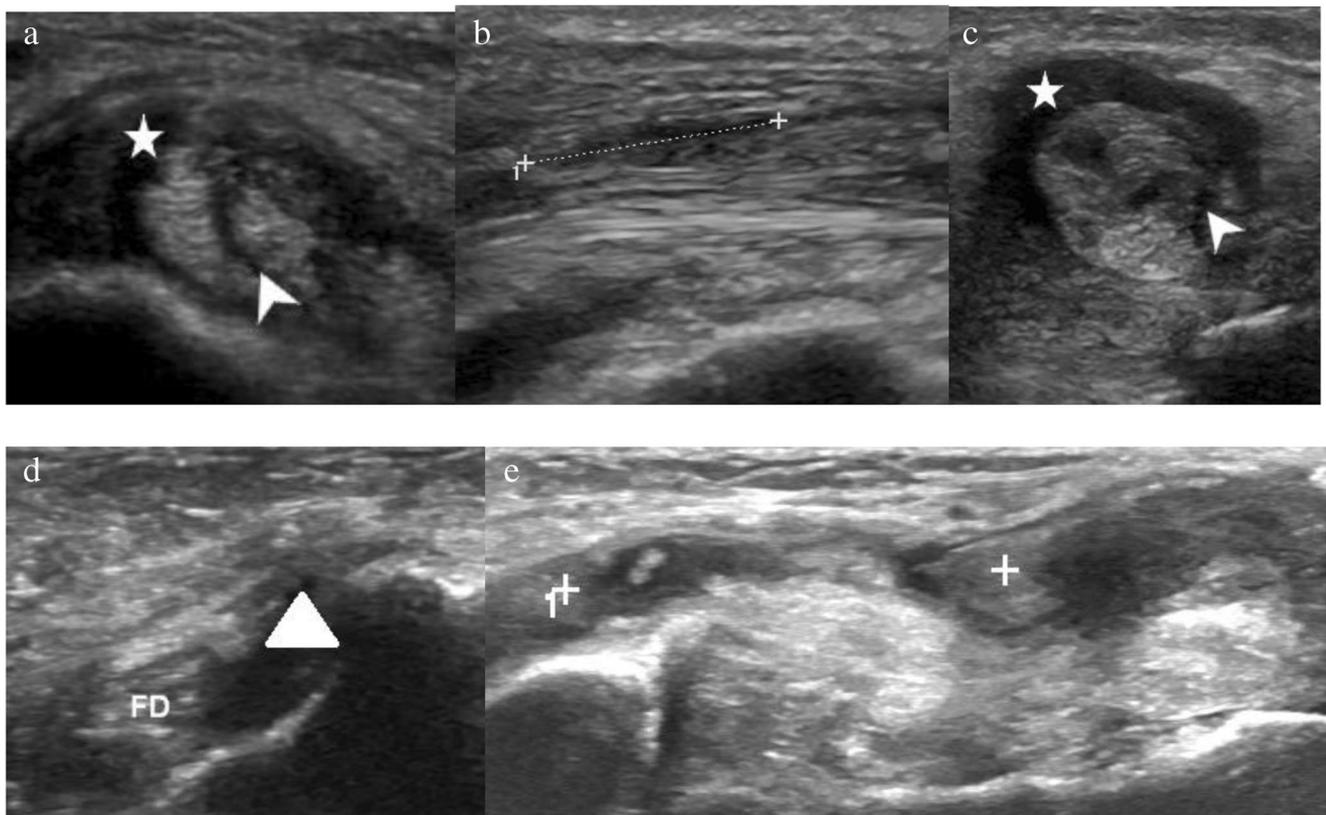


Fig. 8 Ultrasound images of longitudinal split, partial-thickness, and full-thickness tears of the PTT. **a** Short- and **b** long-axis ultrasound images in a 77-year-old man demonstrating a PTT longitudinal split tear. The split tear is noted by the *arrowhead* on the short-axis image and the caliper measurement on the long axis image. Tenosynovitis is noted by the *star* on the short-axis image. **c** In another 69-year-old woman, posterior tibial tendinosis, tenosynovitis (*star*) and an intrasubstance partial-thickness

tear (*arrowhead*) are demonstrated on this short-axis ultrasound image. **d** Short-axis and **e** long-axis ultrasound images of a 53-year-old woman with an acquired flatfoot deformity reveal a full-thickness tear with absence of the PTT (*triangle*) in the perimalleolar region, with the adjacent intact flexor digitorum longus tendon (*FD*). Long-axis image demonstrates the full-thickness tear of the PTT with retracted tendon stumps indicated by the calipers

common site of the navicular insertion. Ultrasound can also be utilized to identify a type I, type II or type III accessory navicular (Fig. 9) [1, 7].

Subluxation/dislocation of the PTT with or without a flexor retinaculum tear

Although PTT dislocation is rare, it is the second most commonly dislocated ankle tendon after the peroneal tendons. It can be due to a shallow retromalleolar groove with or without a lax or torn flexor retinaculum [43]. Chronic subluxation or an acute dislocation of the PTT can lead to posterior tibial tendinosis or tears and subsequently PTTD. Ultrasound is superior to MRI in diagnosing a subluxing or dislocating PTT due to the added benefit of real-time dynamic imaging (Fig. 10a) [14]. It is most commonly seen in athletes and is often the result of a traumatic event or repetitive microtrauma [30]. The flexor retinaculum is evaluated immediately superior to the medial malleolus, where it attaches to the tibia and appears uniformly thin and hypoechoic [32]. Ultrasound demonstrates a thickened,

hypoechoic or absent flexor retinaculum (Fig. 10). Occasionally, an associated cortical avulsion can be seen as an adjacent, usually linear, echogenic shadowing focus compatible with an avulsed fracture fragment (Fig. 10c). In chronic or remote injuries of the flexor retinaculum, chronic subluxation, or chronic tenosynovitis, the sonographic finding of cortical irregularity, may be seen along the distal medial tibia corresponding to the hypertrophic ridge or associated periosteal reaction [1, 30, 43–45].

Subfibular impingement

The distal fibula should not extend inferiorly and abut the lateral cortex of the calcaneus [36]. In the later stages of PTTD, as the HF planovalgus deformity worsens, abnormal contact can occur between both the lateral aspect of the talus and the calcaneus with the distal fibula, resulting in subfibular impingement [35]. Although talocalcaneal impingement cannot be reliably diagnosed sonographically, subfibular impingement can be identified as a low-lying fibula adjacent to the lateral cortex of the calcaneus

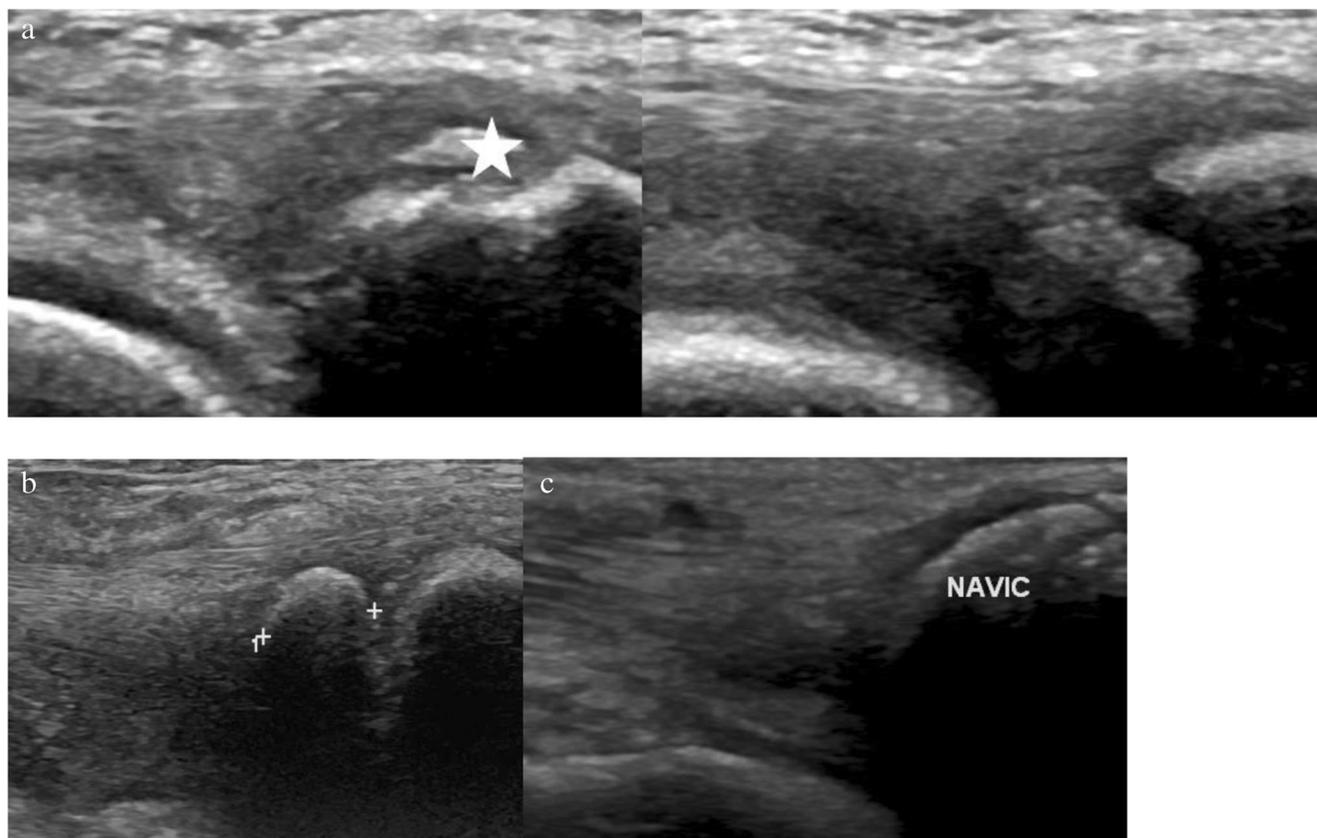


Fig. 9 Sonographic images of the normal and accessory navicular types. **a** Long-axis ultrasound image demonstrates a type I navicular (*star*) within the right PTT, with a contralateral left normal navicular. **b** Long-axis

image displays a type II navicular (calipers). Note tendon fibers of the PTT inserting onto the type II navicular. **c** Long-axis image shows a type III navicular (*NAVIC*) with PTT tendon fibers inserting onto it

(Fig. 11). Also, edema and fluid may be seen within the soft tissues between the distal fibula and the adjacent lateral calcaneus. Peroneal tendinosis, with potential impingement of the peroneal tendons between the fibula and calcaneus, may also be diagnosed sonographically because of the HF planovalgus deformity [25, 36]. Given this manifestation, when an ankle ultrasound for PTTD is requested, whether the pain is isolated to the medial ankle or the study is ordered solely to evaluate the PTT, we recommend always evaluating the lateral aspect of the ankle to exclude associated subfibular impingement.

Sinus tarsi syndrome

Sinus tarsi syndrome is an important secondary sign of posterior tibial tendinopathy and is highly associated with PTTD [48]. As PTT insufficiency worsens, the pes planus deformity also worsens. This is in part due to the associated dysfunction of other structures that play a role in maintenance of the arch of the foot, including the ligaments within the sinus tarsi and the spring ligament complex [6]. The sinus tarsi is readily evaluated by ultrasound by placing the transducer at the lateral aspect of the ankle, in a

coronal–oblique plane (Fig. 12). The sinus tarsi can be identified as a triangular space between the anterior–superior surface of the calcaneus and the talar neck [49, 50]. An abnormally hypoechoic appearance of the sinus tarsi is compatible with sinus tarsi syndrome (Fig. 12b) [51]. In our experience, the sinus tarsi may also appear narrowed or completely obliterated.

Spring ligament insufficiency

The spring ligament complex also plays a crucial role in the stability of the longitudinal arch by providing subtalar stability and maintaining alignment of the talus and calcaneus [6]. It is common to see spring ligament insufficiency in association with PTTD [6, 32, 38]. As pes planus progresses in the later stages of PTTD, there is repetitive loading on the spring ligament leading to attrition with laxity or rupture [38]. One study showed that 92% of patients with an advanced PTT injury also had an abnormality of the spring ligament [6]. Ultrasound has been shown to be a reliable method of evaluating the superomedial calcaneonavicular ligament of the spring ligament complex [32, 52]. Placing the transducer in an anteroinferior orientation, the spring

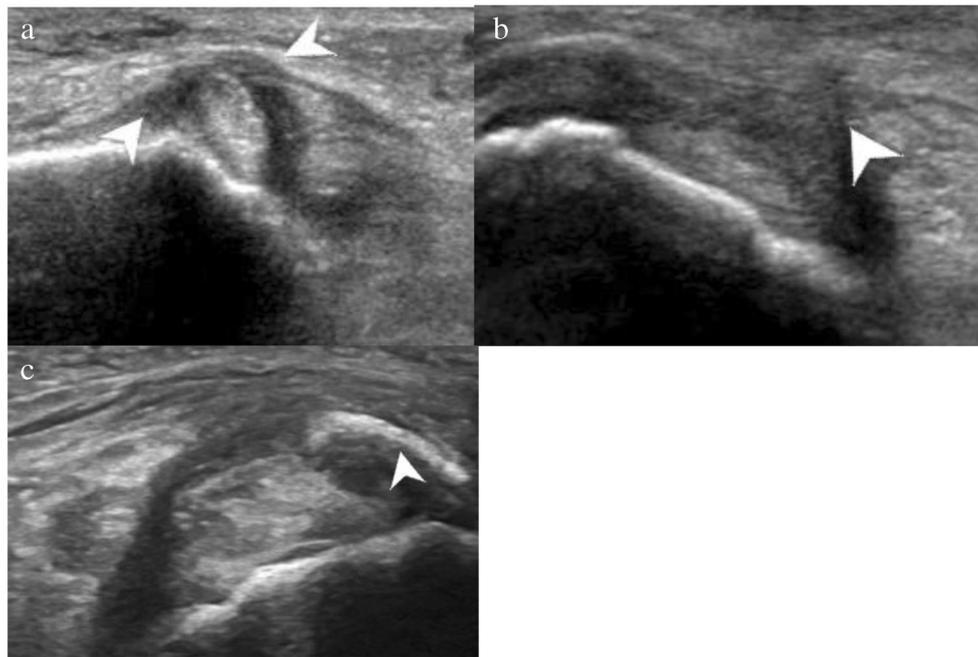


Fig. 10 Ultrasound images of subluxation of the PTT and associated abnormalities related to the flexor retinaculum. **a** Short-axis ultrasound image of a 28-year-old female athlete with a history of a previous ankle injury and instability demonstrates mild anterior subluxation of the PTT at the medial malleolus. Flexor retinaculum (*arrowheads*) is slightly thickened and irregular, but intact. **b** Short-axis ultrasound image at the level of the distal medial tibia of a 43-year-old man with a history of ankle trauma

demonstrates a flexor retinaculum tear (*arrowhead*) as noted by a hypoechoic cleft in the retinaculum with a diffusely thickened and irregular retinaculum. **c** Short-axis ultrasound image at the level of the medial distal tibia in a 59-year-old man with a history of an old ankle injury demonstrates a linear hyperechoic focus within a thickened, ill-defined flexor retinaculum (*arrowhead*) consistent with a chronic tibial avulsion fracture fragment

ligament is seen in the long axis as a hyperechoic fibrillary structure originating from the medial side of the sustentaculum tali of the calcaneus and extending to the superomedial aspect of the navicular (Fig. 13a) [32, 38, 53]. An abnormal appearance is defined as a hypoechoic

appearance or thickening of greater than 4 mm in the midsubstance of the ligament (Fig. 13b) [38]. Although the normal spring ligament can usually be readily identified, it is important to note that in our experience, when there is significant pathology or degeneration, the spring ligament can be challenging to visualize.

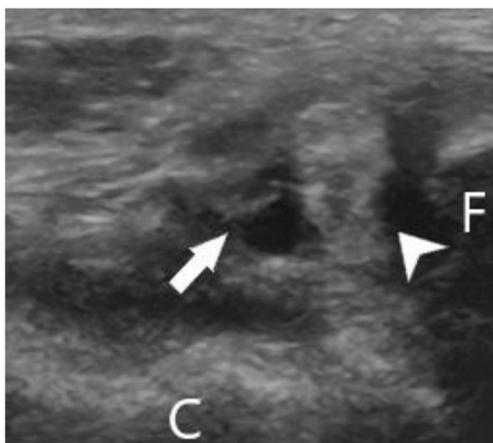


Fig. 11 Short-axis ultrasound image in a 66-year-old woman presenting with lateral ankle pain demonstrating the sonographic findings of subfibular impingement, including inferior extension of the distal fibula (*F*) to close to the level of the lateral cortex of the calcaneus (*C*) with interposed fluid and edema (*arrow*). Notice the adjacent peroneal tendons (*arrowhead*), which are susceptible to associated pathological conditions

Treatment

Radiographic and sonographic findings of PTTD treatments by stage

Stage I

Early detection and diagnosis of PTTD is important to prevent disease progression and to limit the need for more invasive and complex procedures [4]. As stage I findings are localized to the PTT with no rigid clinical deformity and no arthritic changes, treatment is initially non-operative. Conservative treatments at this stage are aimed at alleviating pain and slowing progression of the disease. Treatment options include physical therapy, brace immobilization, custom-made orthotics, and medications such as nonsteroidal anti-inflammatory drugs. Ultrasound-guided corticosteroid and analgesic

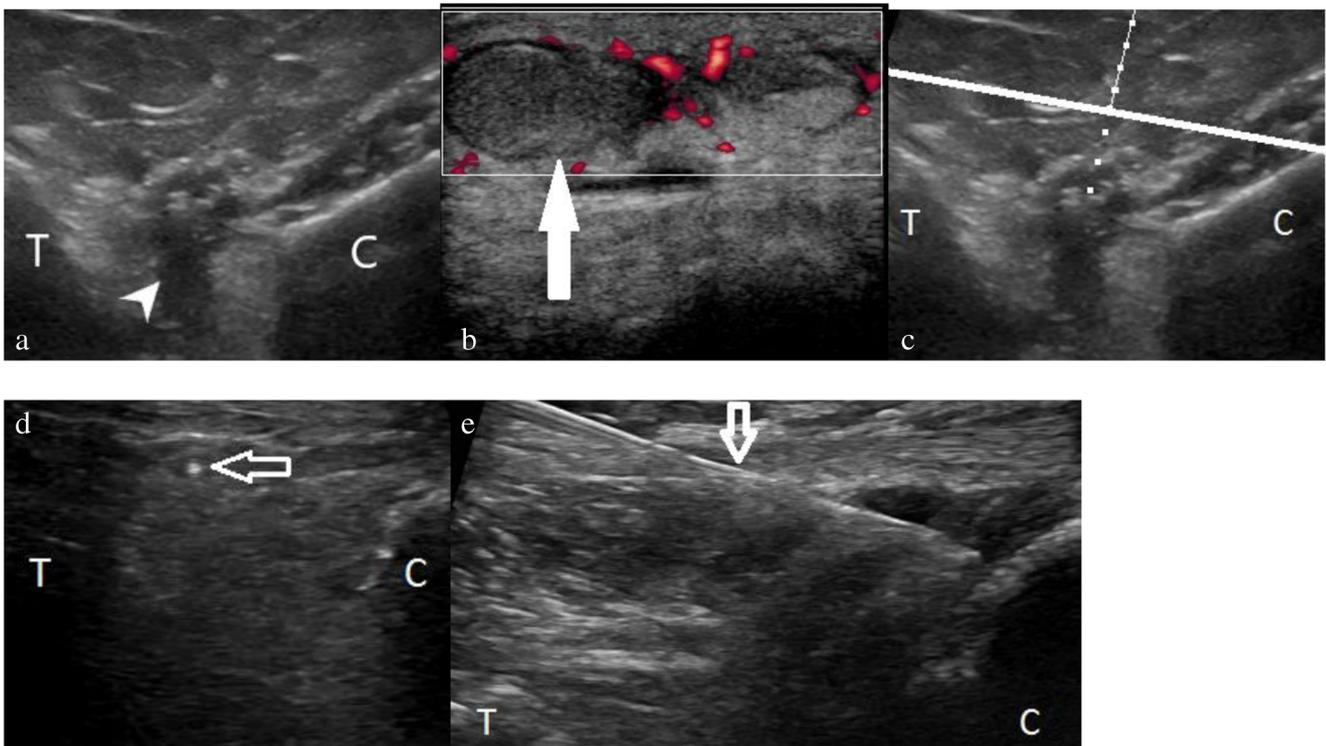


Fig. 12 Ultrasound appearances of the normal and abnormal sinus tarsi with techniques for ultrasound-guided sinus tarsi injections. **a** Normal ultrasound appearance of the sinus tarsi (*arrowhead*), talus (*T*), and calcaneus (*C*). **b** In a 71-year-old man with lateral ankle pain, there is a complex, noncompressible, hyperemic, and hypoechoic area in the sinus tarsi compatible with sinus tarsi syndrome with a complex ganglion (*solid arrow*). **c** Ultrasound image of the sinus tarsi demonstrating the orientation of the probe transducer footprint (*solid line*) held in the coronal-

oblique plane in relation to the talus (*T*) and calcaneus (*C*). The needle (*dotted line*) is depicted as introduced into the short axis. **d** Ultrasound-guided sinus tarsi injection with the needle (*empty arrow*) introduced into the short axis utilizing a lateral approach, with the transducer held in the coronal-oblique plane. **e** Ultrasound-guided sinus tarsi injection utilizing the same lateral approach and the transducer held in the coronal-oblique plane with the needle (*empty arrow*) introduced in the long axis

injections of the PTT sheath can also be utilized (Fig. 7c) [2, 3, 24, 34, 49]. Ultrasound-guided injection of the sinus tarsi may also be considered in patients with sinus tarsi syndrome (Fig. 12). The needle can be introduced into either the long or short axis. Approximately 80% of patients presenting with early PTTD respond well to these conservative treatments [5]. After 3–4 months, if these more conservative measures are

unsuccessful, endoscopic or open PTT debridement or tenosynovectomy may be performed [2, 5, 24, 34].

Stage II

At stage II, the tendon becomes dysfunctional and a fixed pes planus develops on weight-bearing radiographs although it is

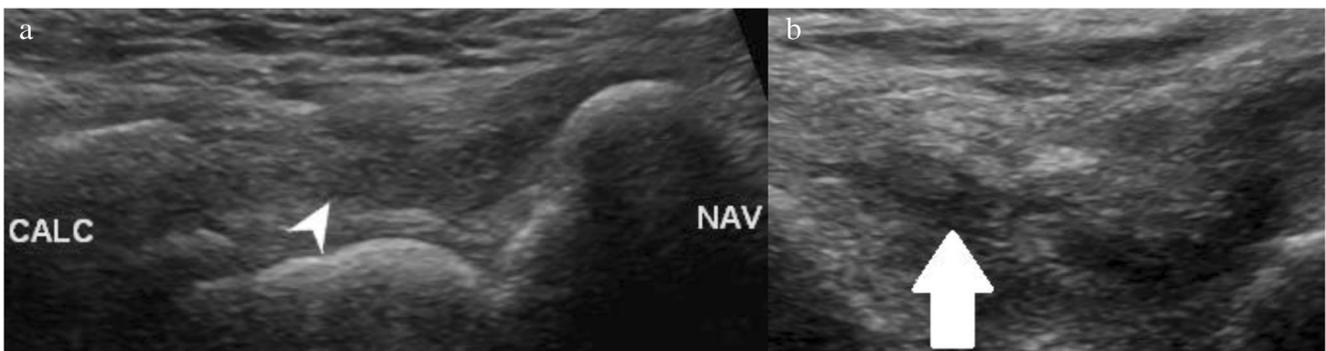


Fig. 13 Ultrasound appearance of the spring ligament complex. **a** Long-axis ultrasound image of a normal spring ligament complex (*arrowhead*) in a 21-year-old woman extending from the sustentaculum tali of the calcaneus (*CALC*) to the navicular (*NAV*). **b** Ultrasound image of a 45-

year-old woman with an acquired flatfoot deformity demonstrates a torn spring ligament, as indicated by a thickened ligament with a focal hypoechoic defect within it (*arrow*)

clinically not rigid. The more conservative treatments, although still attempted, often fail in these patients [34]. Surgical procedures are often required at this stage, aimed at correcting the clinically flexible deformity and stabilizing the medial longitudinal arch while preserving the joints, which again demonstrate no arthritic changes [2, 23]. These operative procedures may include PTT excision or reconstruction. The flexor digitorum longus is the most commonly transferred tendon used for reconstruction. If there is a sufficient amount of viable PTT fibers remaining they can be interweaved in a tenodesis with the flexor digitorum longus tendon transfer (Fig. 14a, b) [20, 28, 54]. Otherwise, a direct osseous fixation of the transferred flexor digitorum longus tendon to the navicular can be performed (Fig. 14c, d) [5, 8, 34]. In conjunction with the tendon transfer and in an effort to correct the HF malalignment, an osteotomy is often performed, most commonly a medial displacement (medializing) calcaneal

osteotomy (Figs. 3b and 15a). Percutaneous or open Achilles tendon lengthening is also often performed, which can help to address the loss of ankle dorsiflexion and avoid proximal migration of the posterior calcaneal fragment following a medial displacement calcaneal osteotomy due to excess Achilles tendon tautness [2, 5, 6, 8, 24, 34, 55]. If spring ligament insufficiency is also present, it is recommended that a spring ligament complex reconstruction be performed [6, 52, 56].

As an alternative option to more invasive osseous procedures in stage II patients, some suggest the use of a sinus tarsi implant known as subtalar arthroereisis (Fig. 16). By placing a bioabsorbable or metallic implant in the sinus tarsi, subtalar joint motion can be limited to help resist increased valgus deformity. This is typically done in combination with a flexor digitorum longus tendon transfer to the PTT [8, 28, 54, 57, 58].

If a type II or III accessory navicular is present and found to be contributing to the PTTD, especially in



Fig. 14 Ultrasound and radiographic findings of a PTT reconstruction with tendon transfer. **a** Short and **b** long axis ultrasound images in a 64-year-old woman of a PTT (*arrow*) tenodesis intertwined with a flexor digitorum longus (*FD*) tendon (*arrowheads*) transfer. **c** Anteroposterior

and **d** lateral radiographs of the foot of a 74-year-old woman demonstrate hardware and a lucent bone tunnel related to a PTT transfer to the navicular and medial cuneiform



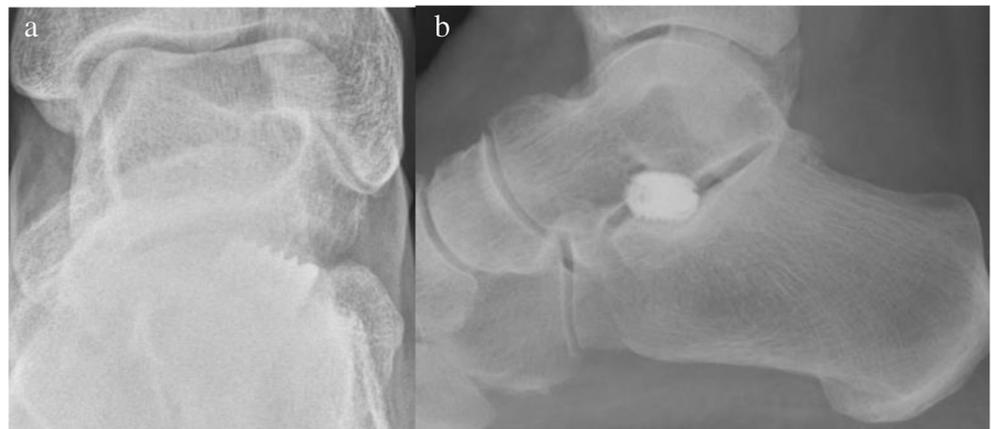
Fig. 15 Sonographic and radiographic imaging related to a Kidner procedure. **a** Harris calcaneal radiograph of a 55-year-old woman with stage II posterior tibial tendon dysfunction demonstrates a suture anchor (*arrow*) in the navicular from a previous Kidner procedure. Also, note the healed calcaneal osteotomy with two partially threaded cannulated screws in the calcaneus. Anteroposterior weight-bearing radiographs of the foot in a 33-year-old man **b** pre- and **c** post-Kidner procedure, with resection

of a symptomatic type II accessory navicular (*triangle*) and placement of a corkscrew anchor. Also, notice the preoperative hindfoot valgus evident by an increased talocalcaneal angle (angle not shown). **d** Short and **e** long axis ultrasound images of a 51-year-old woman show the postoperative changes associated with a Kidner procedure at the level of the navicular (*arrowheads* indicate the suture anchor)

patients with accessory (os) navicular syndrome, a Kidner procedure may be attempted. This procedure

provides pain relief by resecting the painful accessory navicular. This can be performed in conjunction with

Fig. 16 **a** Anteroposterior and **b** lateral ankle radiographs in a 44-year-old woman demonstrate a hardware implant in the sinus tarsi consistent with subtalar arthroereisis



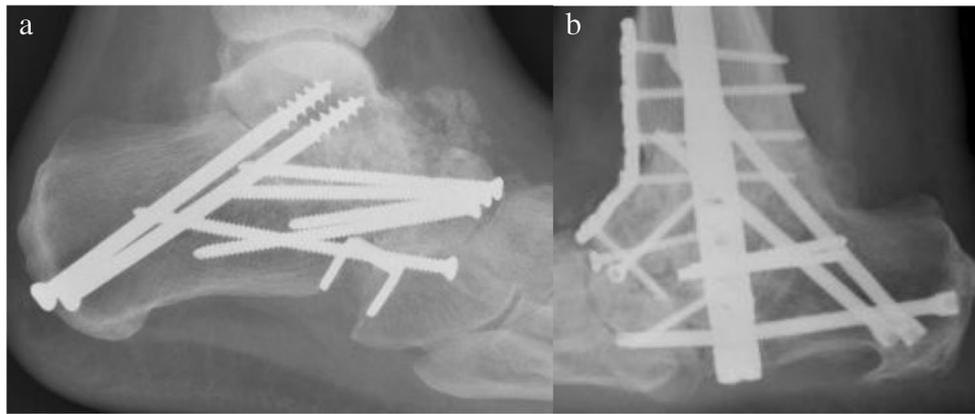


Fig. 17 Radiographic findings of arthrodesis changes related to stages III and IV PTTD. **a** Lateral ankle radiograph in a 62-year-old woman with stage III PTTD demonstrates extensive hindfoot hardware, including multiple screws associated with a triple arthrodesis of the subtalar, talonavicular, and calcaneocuboid joints. **b** Lateral ankle radiograph in a

79-year-old man with stage IV PTTD and a history of triple arthrodesis demonstrates extensive hardware associated with a tibiotalar calcaneal arthrodesis, including a retrograde intramedullary nail traversing the calcaneus, talus, and tibia in addition to a bridging plate-and-screw fixation device along the anterior aspect of the distal tibia and dorsal talus

resection of the area of posterior tibial tendinopathy and reconstructing the tendon by anchoring the healthy viable distal PTT fibers with suture anchors to the native navicular (Fig. 15) [8, 20, 28, 54].

Stages III and IV

Stages III and IV require more invasive surgical osseous procedures to correct the rigid and fixed HF planovalgus deformity, address the associated arthritis changes, and alleviate pain. Joint-sparing procedures such as tendon reconstruction and osteotomies are not sufficient. In stage III, treatment typically involves a triple arthrodesis including fusion of the

subtalar, talonavicular, and calcaneocuboid joints (Fig. 17a) [21, 59–61]. In stage IV, a triple arthrodesis is performed in conjunction with a tibiotalar calcaneal arthrodesis (Fig. 17b) [2, 4, 13, 19, 20, 36, 62, 63]. Also, in stage IV, deltoid ligament reconstruction may be performed (Table 2) [5].

Conclusion

Ultrasound and radiographs play a crucial role in the diagnosis of PTTD and in facilitating medical and surgical guidance in the management of patients with PTTD. Early detection and diagnosis of PTTD is important to prevent further progression

Table 2 Summarized findings and treatments of posterior tibial tendon dysfunction by stage

Stage	Findings	Treatment
I	Inflammation of the posterior tibial tendon with no radiographically fixed or clinically rigid deformity	Conservative treatments including ultrasound-guided injections. If unsuccessful after 3–4 months, endoscopic, open debridement or tenosynovectomy can be considered
II	Severe posterior tibial tendinosis, partial-thickness tearing and tendon lengthening (i.e., severely dysfunctional to nonfunctional tendon). Fixed pes planus on weight-bearing radiographs although clinically flexible	Non-operative treatment often fails. Joint-sparing procedures include the Kidner procedure (in the setting of an associated accessory navicular), posterior tibial tendon excision, reconstruction of the posterior tibial tendon with a flexor digitorum longus tendon transfer and Achilles tendon lengthening, subtalar arthroereisis, calcaneal osteotomy, or spring ligament reconstruction
III	Posterior tibial tendon rupture (i.e., nonfunctional tendon). Fixed and rigid hindfoot planovalgus with subtalar joint osteoarthritis	Surgical treatment with a triple arthrodesis
IV	Talar tilt within the tibiotalar joint with tibiotalar joint osteoarthritis	Surgical treatment with a tibiotalar calcaneal (ankle) arthrodesis with possible deltoid ligament reconstruction

of disease and avoid more invasive and complex procedures. Ultrasound can also be utilized for image-guided injections of the PTT sheath and the sinus tarsi. It is also important to know the radiographic and sonographic findings of the different surgical procedures related to PTTD and the associated structures.

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Compliance with ethical standards

Source of support None.

Conflicts of interest The authors declare that they have no conflicts of interest.

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