Ultrasound evaluation of adult-acquired flatfoot deformity: Emphasis on the involvement of spring ligament

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Abstract

Adult-acquired flatfoot deformity (AAFD), a condition commonly caused by tibialis posterior tendon (TPT) dysfunction, has recently been recognised to encompass a spectrum of other deformities, including the disruption of the spring ligament complex. This case series reviews eight examples of chronic AAFD, outlines the sonographic assessment of the TPT and spring ligament and depicts various abnormalities of the TPT and spring ligament that are relevant to AAFD. The importance to always include the spring ligament during the assessment of AAFD will be addressed, as misdiagnosis of the involvement of the spring ligament could lead to inappropriate operative management.

Keywords: adult acquired flatfoot deformity, spring ligament, TPT dysfunction, ultrasound.

Introduction

Adult-acquired flatfoot deformity (AAFD) is well recognised by podiatrists and foot/ankle surgeons.[1] The condition is known to have a progressive nature.[2] It is characterised by a flattened longitudinal arch, the development of a valgus heel with a gradually abducting and supinating forefoot.[3] Often patients complain of pain and swelling of the medial hindfoot.[2,4] Tibialis posterior tendon (TPT) dysfunction is the commonest cause of AAFD, but is not a sole pathological entity.[3,5] More recently, the condition has been recognised to involve not only compromise of the TPT as the dynamic stabiliser of the longitudinal medial arch but also the calcaneo-navicular (spring) ligament as the primary static stabiliser. A spectrum of abnormalities may contribute to AAFD including isolated tears of the TPT, TPT tears in association with spring ligament failure or isolated spring ligament rupture.[6–9] Misdiagnosis or failure to recognise the condition may increase patient morbidity and lead to adverse clinical outcomes.[3] This review examines a series of eight cases of chronic AAFD patients, discusses the anatomy and function of the TPT and spring ligament and the aetiology and pathophysiology of AAFD. The ultrasound approach for assessment of the TPT and spring ligament will be outlined, and various abnormalities of these structures that are relevant to AAFD will be discussed. This article is intended to generate more discussion and greater awareness to the efficacy of ultrasound in the evaluation of this common debilitating condition.

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Anatomy and function

The tibialis posterior (TP) muscle is buried deep in the posterior calf, arising from the posterior surfaces of the tibia, fibula and interosseus membrane.[2,10] The distal tendon is enclosed within a synovial sheath, formed in the distal third of the leg, where it courses immediately posterior to the medial malleolus (MM) and is firmly held down by the flexor retinaculum.[2,11,12] Distal to this region the tendon exhibits a more flattened orientation and divides into multiple fibrous slips, the biggest component inserts into the navicular tuberosity with branches inserting into the cuneiform bones and the bases of the first to fourth metatarsals.[11,12] TP is the prime dynamic stabiliser of the hindfoot, its contraction causes subtalar inversion and plantar flexion of the foot, and locking of the calcaneocuboid and talonavicular joints, making the hindfoot and mid-foot rigid, hence allows the gastrocnemius muscle to act with much greater efficiency during gait and resulting in the elevation of the medial longitudinal arch.[2,13]

The calcaneo-navicular (spring) ligament complex fills the bony gap between the anterior part of the calcaneus and the navicular bone.[14] It is composed of three distinct groups. The larger and superomedial is a hammock-shaped ligament, it courses along the medial margin of the talonavicular articular surface, it arises from the under surface of the sustentaculum tali and has a fanlike insertion on the inferior and medial

surfaces of the navicular bone.[15,16] The second much smaller inferoplantar component is located lateral and plantar to the superomedial ligament. It is a striplike bundle of short and thick fibres and originates in the notch between the middle and anterior calcaneal facets, spreading out and terminating on the inferior beak of the navicular bone.[15] The third ligament is the thinnest, having a medioplantar oblique course running independently of the other two ligaments and originates from the coronoid fossa at the anterior aspect of the calcaneus and attaches to the navicular beak[14] (Figure 1). The spring ligament is a static support of the head of the talus, and it serves as a slinglike structure that prevents talus plantar rotation and the calcaneus from undergoing valgus angulation.[8,9,17]

Aetiology and pathophysiology

Several factors have been suggested to attribute to the aetiology of TPT dysfunction. They include degenerative tendinosis, particularly within a zone of 14 mm length behind the MM, which has been postulated to be due to hypovascularity.[2,13,18] The histopathological characteristic of TPT obtained from the specimens of AAFD further shows the concept of degenerative tendinosis without inflammation as believed to be the underlying pathological mechanism in chronic TPT dysfunction.[19] Others have proposed the condition is due to chronic overuse or rheumatoid arthritis.[13,20,21] TPT predisposition to rupture has also been noted in association with acute trauma, obesity, hypertension, diabetes, prolonged oral intake of corticosteroids, repeated local steroid injections and seronegative arthropathies.[4,13,22] Jahss[23] hypothesised TPT dysfunction is due to the impingement mechanism at the level of the fibro-osseous groove posterior to the MM.[23] Lopez-Larrea and Diaz-Pena[24] advocate that the increased level of shear and/or compression at the site of functional enthesis leads to microdamage of TPT. In summary, the aetiology for TPT dysfunction can be grouped into four categories: direct injury, pathological rupture, functional rupture and idiopathic rupture.[25]

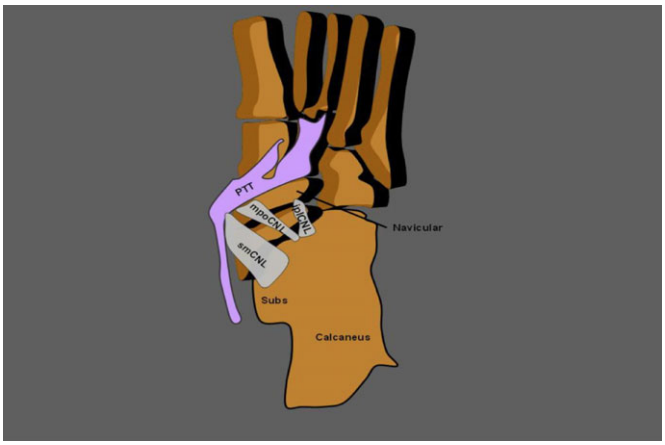


Figure 1: Schematic Diagram of Spring Ligament; smCNL, Superomedial Calcaneonavicular (Spring) Ligament; mpoCNL, Medioplantaroblique Calcaneonavicular (third) Ligament; ipICNL, Inferoplantar Longitudinal Calcaneonavicular Ligament; PTT, Posterior Tibial Tendon; Subs, Sustentaculum Tali of Calcaneus.[15]

Dysfunction of TPT can lead to progressive failure or tears of the static stabilizer, the spring ligament. Once the static stabilizer ruptures, the head of the talus collapses due to lack of supporting mechanism, resulting in collapse of the medial longitudinal arch hence flat foot deformity.[9] Isolated spring ligament tears range from interstitial sprain injury to frank tear.[15] The superomedial component has been established to be commonly associated with tears and plays the most significant role in the spring ligament's contribution to ankle stability.[3,17] Histological and biomechanical analysis of the spring ligament fibrocartilage complex has shown that the structure is purely collagenous with no elastic properties.[26] The term "spring" is a misnomer and is based on a thought that the ligament acted as spring for the longitudinal arch of the foot.[26] Macroscopic evidence suggests that the central portion of the superomedial fibres shows a poor or absence of blood supply, which could be a factor predisposing to injury.[27]

Case reports

The eight cases are summarised in Table 1. The patient ages ranged from 45 to 78 years and presented to our clinic between July 2014 and October 2015. The patients were referred from the same podiatrist, and they all presented with progressive flattening of the arch, chronic pain and swelling of the medial ankle, with moderate to severe pes planovalgus deformity. These patients have difficulty performing "single limb heel rise" test. With no improvement in patient's symptoms after eight months of conservative orthoses, the podiatrist referred for ultrasound assessment of any tear in the TPT and assessment of spring ligament morphology. Table 2 demonstrates the ultrasound finding of the eight cases. The images illustrate various degrees of dysfunction in the TPT, ranging from tendinosis to tenosynovitis to complete rupture, as well more specifically it shows the abnormalities such as partial tear to complete tear of the spring ligament.

Ultrasound technique

Prior to imaging, pertinent history should be reviewed and a physical examination performed. The ultrasound scan was tailored to the medial ankle. Anatomy is assessed in orthogonal planes. The patient is in lateral oblique position, knee flexed to 45° and the foot resting on its lateral margin. Place the transducer in a transverse position, with the anterior margin on the medial surface of the medial malleolus (MM). TPT lies superficial and adjacent to MM, and it appears largest in the medial group tendons. Follow and evaluate the TP proximally to the musculotendinous junction and then distally to its navicular

Table 1: Summary of the eight cases of the chronic acquire flatfoot deformity due to TPT or TPT and spring ligament dysfunction.

Patient	Side	Sex	Age	Clinical notes	Treatment regime	TPT morphology	Spring ligament morphology
1	Left	F	46	Medial ankle pain and swelling	Orthoses treatment failed	Tendinosis, tenosynovitis with longitudinal tear	Thinning & heterogeneous change
2	Right	F	76	Severe pes planovalgus deformity Painful medial ankle	Orthoses treatment failed	Complete rupture with retraction	Near complete tear, thinning and heterogeneous change
3	Right	F	75	Tenderness anterior to the medial Malleolus with flatfoot deformity	Orthoses treatment failed	Tendinosis	Complete tear
4	Left	M	60	Worsening flatfoot deformity	Orthoses treatment failed	Tendinosis with longitudinal tear	Thickening, loss of the normal fibrilar internal structure
5	Left	F	78	Swelling in medial ankle, Pes planovalgus deformity	Orthoses treatment failed	Tendinosis and tenosynovitis	Thinning, loss of the normal fibrilar internal structure
6	Left	F	68	Ongoing medial rear foot pain	Orthoses treatment failed	Tendinosis and tenosynovitis	Thinning, heterogeneous change and split tear
7	Left	F	71	Tibialis posterior tenosynovitis Pain & swelling in medial ankle	No improvement with orthoses treatment	Tendinosis, tenosynovitis with longitudinal tear	Heterogeneous change with increase vascularity
8	Right	M	73	Medial ankle pain, ? TPT dysfunction	Orthoses treatment failed	Tendinosis and tenosynovitis	Heterogeneous change, increase vascularity with high grade partial tear

insertion in short axis. The curved path of the TPT requires the transducer angle be continually adjusted around the MM to prevent anisotropy and remain in true short axis. Avoid excessive transducer pressure so as not to overcompress a potential intrasubstance tear or synovial sheath fluid. Complete the assessment of the TPT in longitudinal axis as this will assist in demonstrating any delamination tears.

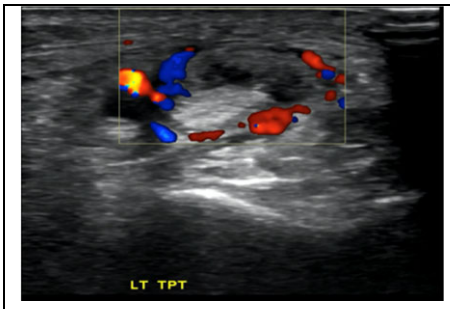
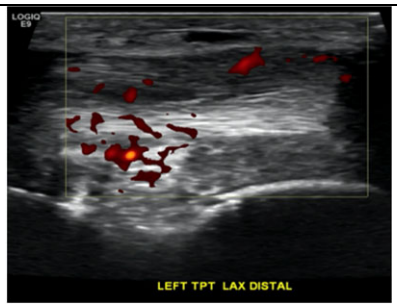
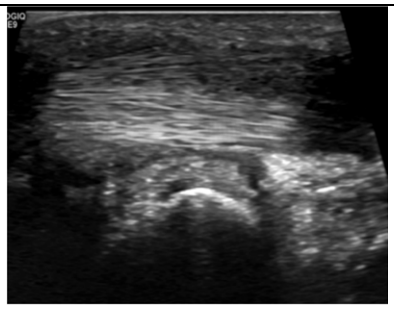
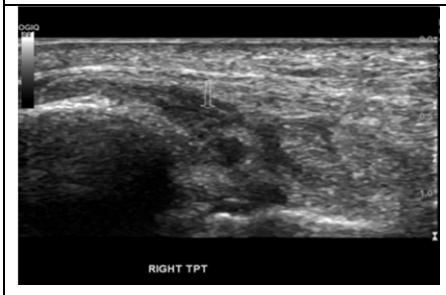
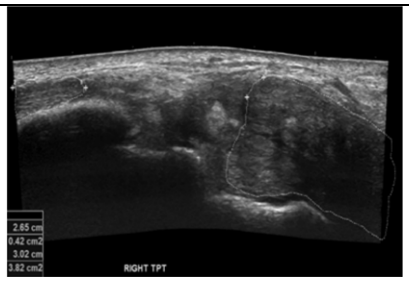
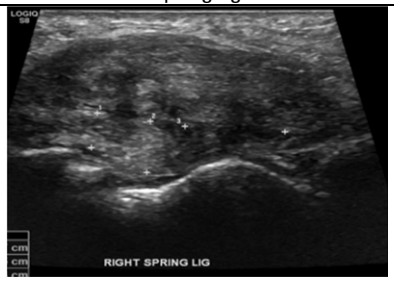
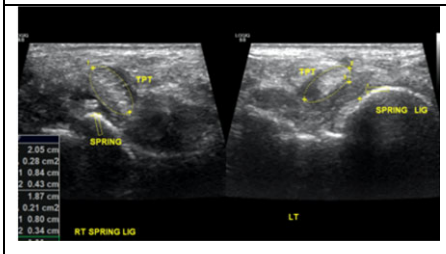
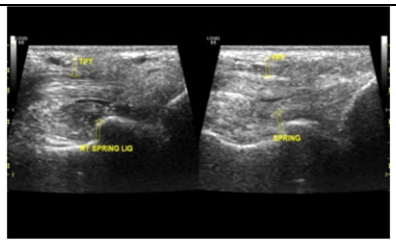
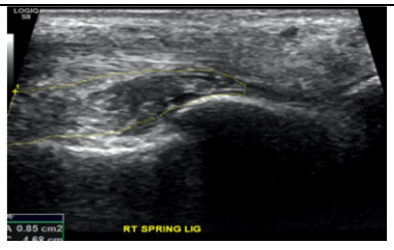
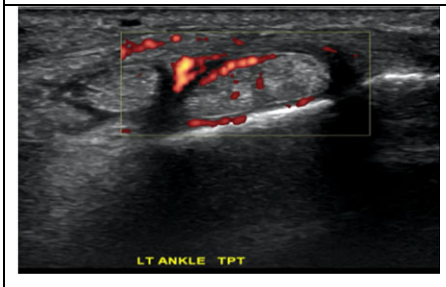
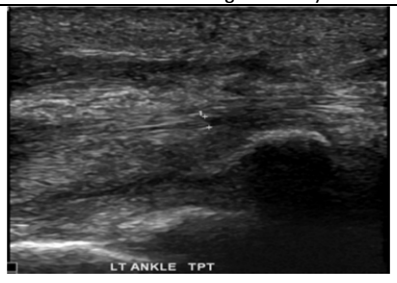
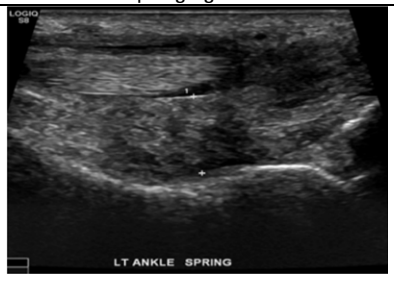
An entity worth keeping in mind during the assessment of TPT is the presence of an intratendinous ossicle in the region of the tuberosity of the navicular, known as os tibiale externum.[12] Os tibiale externum has been reported in 14% of the population.[28] Three distinct types have been described: type I small, 2–3 mm round and embedded within the TPT; type II most common, large, triangular shape ossicle adjacent to navicular, connected by a synchondrosis, measuring 9–12 mm; type III an enlarged medial horn of the navicular itself, also known as a cornuate navicular. These findings should be considered within the spectrum of normal appearances. Nevertheless, the presence of either type II or type III increases the risk for TPT tendinopathy.[29]

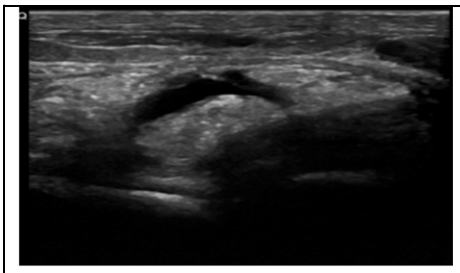
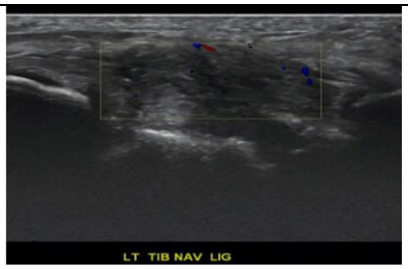
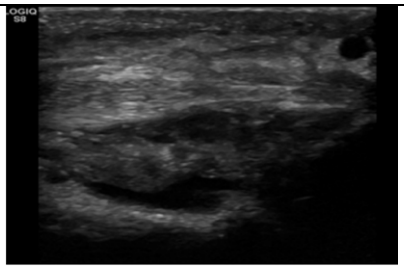
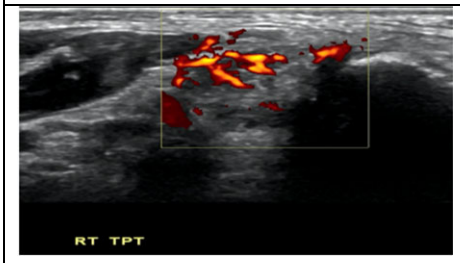
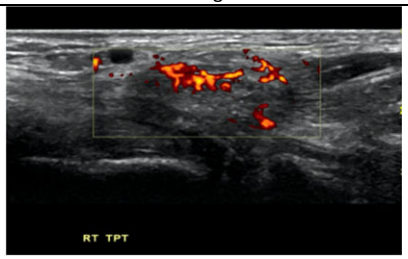
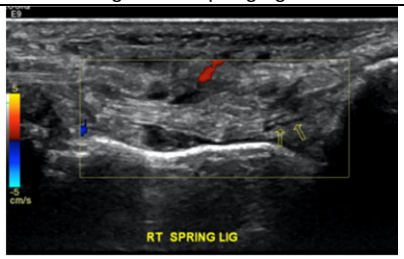
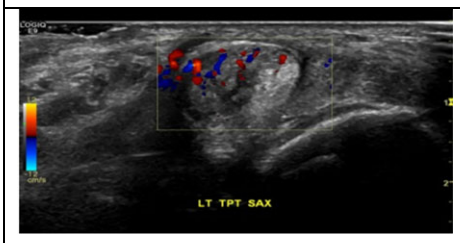
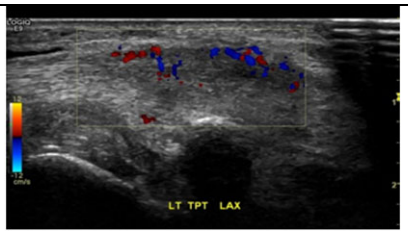
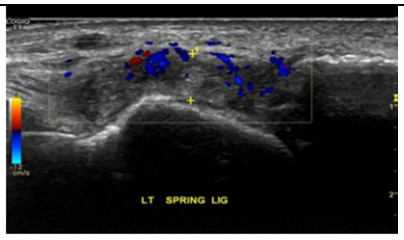
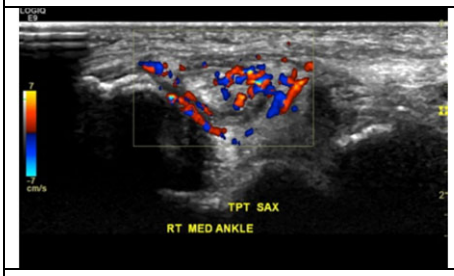
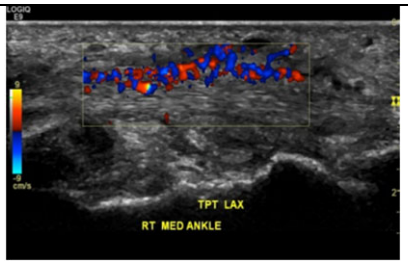
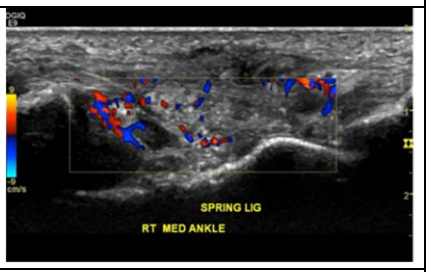
For the spring ligament complex, leave the patient in a similar position as that for scanning the TPT but slightly abduct and pronate the foot place the spring ligament under tension. Start with the transducer inferior to the MM and parallel to the plantar surface of the foot to place one end of the transducer over the sustentaculum tali and the other end slightly tilted superiorly over the talar head in the direction of the superomedial aspect of the navicular bone. The ligament can be visualised deep to the distal fibres of TPT, bridging the sustentaculum tali of the calcaneus and the navicular bone.

Discussion

This case series shows the efficacy of ultrasound in the evaluation of chronic AAFD, and it demonstrates chronic AAFD is often associated with an abnormality of the TPT. Table 2 illustrated that in chronic AAFD, TPT is always abnormal, which

Table 2: Ultrasound images for the eight cases of chronic AAFD.

		
Case 1a TPT Short axis. Colour Doppler demonstrates an increase in vascularity of the TPT and its tendon sheath	Case 1b TPT Long axis. Power Doppler and B-mode show a longitudinal tear within the TPT	Case 1c Spring ligament. B-mode shows thinning and heterogeneous change in the spring ligament
		
Case 2a TPT Short axis. Ultrasound of medial compartment of the anklesshows an absent of TPT	Case 2b TPT Long axis. Ultrasound of medial compartment of the ankle shows retraction of a completely ruptured TPT with distal stump	Case 2c Spring ligament. Ultrasound of medial compartment of the ankle shows thinning and heterogeneous change in the spring ligament, almost full thickness tear
		
Case 3a TPT Short axis. Comparison ultrasound of medial ankle shows an absent of spring ligament	Case 3b TPT Long axis. Comparison ultrasound of medial ankle shows an absence of spring ligament with the TPT almost touching the bony surface	Case 3c Spring ligament. Comparison ultrasound of medial ankle shows acomplete rupture of the distal spring ligament
		
Case 4a TPT Short axis. Ultrasound of medial compartment shows increase in power Doppler of the TPT and its tendon sheath	Case 4b TPT Long axis. Ultrasound of medial compartment of the ankle shows a longitudinal tear within the TPT	Case 4c Spring ligament. Ultrasound of medial compartment of the ankle shows thickening and heterogeneous change in the spring ligament

		
Case 5a TPT Short axis Ultrasound of medial compartment shows tenosynovitis in TPT	Case 5b TPT Long axis Ultrasound of medial compartment of the ankle shows heterogeneous and thickening of TPT	Case 5c Spring ligament Ultrasound of medial compartment of the ankle show a thinning and heterogeneous change in the spring ligament
		
Case 6a TPT Short axis. Ultrasound of medial compartment shows increase in power Doppler of the TPT and its tendon sheath	Case 6b TPT Long axis. Ultrasound of medial compartment of the ankle shows heterogeneous change in TPT with increased vascularity	Case 6c Spring ligament. Ultrasound of medial compartment of the ankle shows thinning and split tear in the distal portion of spring ligament
		
Case 7a TPT Short axis. Ultrasound of medial compartment shows increase in colour Doppler of the TPT and tendon sheath and split tear	Case 7b TPT Long axis. Ultrasound of medial compartment of the ankle shows heterogeneous change in TPT with increase vascularity and split tear	Case 7c Spring ligament. Ultrasound of medial compartment of the ankle shows heterogeneous change and increased vascularity of the spring ligament
		
Case 8a TPT Short axis. Ultrasound of medial compartment shows increase in colour Doppler of the TPT and tendon sheath	Case 8b TPT Long axis. Ultrasound of medial compartment shows increase in colour Doppler of the TPT and tendon sheath	Case 8c Spring ligament. Ultrasound of medial compartment of the ankle shows heterogeneous change and increased vascularity and grade partial tear of proximal spring ligament

ranges from tendinosis, tenosynovitis and/or tear. More importantly, the condition is commonly associated with spring ligament failure. In the setting of chronic AAFD, various disruptions of the spring ligament were frequently evidenced, which itself can compromise the stability of the longitudinal arch. This case series demonstrates the high specificity and sensitivity of ultrasound detecting abnormality of the spring ligament as associated with chronic AAFD. It reinforces the importance for evaluating and including the spring ligament when assessing chronic AAFD.

TPT dysfunction affects multiple patient groups. In women over the age of 40, the prevalence is 3.3%, and in elderly women, the condition may be present in 10%.[1] A female-to-male ratio of three to one is seen in isolated TPT dysfunction.[7,30] Concurrent TPT dysfunction and spring ligament tear is seen most commonly in middle-aged women,[17,19] typically affecting only one side; bilateral disease is rare.[4] This series of case reports highlight the finding of more female-dominant disease; it demonstrated six of the eight cases were female.

Scientific literature usually considers TPT dysfunction as the commonest cause of AAFD.[5,6] Yet, there are cases of AAFD presenting with symptoms at the TPT with pre-existing flat foot deformity.[5] Several traumatologists believe that in most cases, the medial longitudinal arch flattens prior to the damage of the TPT.[5] As a matter of fact, apart from TPT dysfunction, the condition has been recognised to encompass a spectrum of others deformities, such as disruption of the spring ligament complex or an isolated spring ligament rupture causing AAFD.[6–9] Tryfonidis *et al.*, in a series of nine cases of AAFD related to acute trauma, demonstrated isolated rupture of the spring ligament as the only pathological finding.[9] Another recent study by Gallardo *et al.*[31] of 60 cases, in 49 patients with AAFD and dynamic ultrasound evaluation, concluded that the spring ligament degeneration is more frequently visualised than TPT tendinosis in the initial phases of disease. Paying attention merely to TPT disease during the assessment of AAFD is not sufficient; it is paramount to always extend the examination to routinely cover the integrity of the spring ligament complex. Despite a small sample size, this case series serves to remind us that in chronic AAFD apart from TPT dysfunction, disruption of the spring ligament is frequently evident. Tendon degeneration has been known to begin far before clinical disease is apparent.[4] Earlier detection of TPT degeneration, and/or abnormality of the spring ligament complex, is the key to avoiding progression of the disease. If left undetected, surgical reconstruction with osteotomy and arthrodesis becomes necessary.[4]

The clinical manifestation of AAFD is as variegated and complex as its pathophysiology. Four clinical stages have been established describing the severity of the deformity. Stage 1 occurs when a patient presents with pain and swelling on the medial aspect of the foot, but there is no alteration to the tendon length.[32] Stage 2 is characterised by a dynamic deformity of the hindfoot where the patient is unable to stand on tiptoe on the affected foot and there is elongation or tearing of the tendon seen in conjunction with a valgus heel, clinically observed as the "too many toes" sign.[32] Stage 3 patients have developed a rigid deformity with compensatory supinated forefoot and loss of mobility of the subtalar joint.[32] In stage 4, there is early degenerative change in the ankle and evidence of valgus deformity.[33] Patients with stages 3 and 4 deformity gradually lose shock absorption whilst walking as the ankle is at peak eversion. The valgus alignment of the heel and abduction at the talonavicular joint creates tension in the spring ligament and superficial portion of the deltoid ligament apparatus.[34] Clinical staging of the condition has been well accepted.[32] Research has been focused on further understanding the morphology and integrity of the TPT and spring ligament.[30] For instance, antero-posterior and lateral X-ray of the foot with weight bearing will demonstrate an increased talometatarsal angle and decreased calcaneoplantar angle, indicating the extent of the planovalgus deformity.[35] Radiography also helps to exclude subtalar and ankle articulation degenerative changes.[1,12]

The usefulness of MRI in the diagnosis of musculotendinous pathology of the ankle has been well established.[13] MRI is able to detect the spectrum of early partial tendon tear and complete tear. Partial tears are classified as (type I) hypertrophic with fine longitudinal splits, (type II) degenerative change with wider longitudinal splits and (type III) when the tendon demonstrates a complete tear. MRI is able to show high intrinsic soft tissue contrast.[13] Whilst the usefulness of MRI compared to other modalities is known, a study of 16 fresh cadaveric foot and ankle specimens concluded ultrasound has a higher specificity and the same sensitivity and accuracy when compared to MRI for the detection of TPT intrasubstance longitudinal tears.[36] In another study of 17 patients with surgically confirmed TPT tenosynovitis, sonography successfully diagnosed the condition with similar sensitivity and specificity reported by MRI.[37]

Ultrasound has gained acceptance for evaluating a broad range of musculoskeletal disorders.[13,36] Direct multiplanar capability allows it to interrogate and follow the direction of complex tears without the restriction of axial, sagittal or coronal planes. It has the ability to assess tendons in dynamic real time, allowing tendons and ligaments to be stressed or manoeuvred demonstrating tethering or instability. The lack of ionising radiation, low cost, non-invasiveness and availability of higher-resolution transducers make it an ideal screening modality for TPT dysfunction.[2,13,38]

The normal TPT will exhibit hyperechoic texture with an AP diameter between 4 and 6 mm[2]. Collagen fibres in the distal 1.5 cm of the TPT appear heterogeneous due to diverging direction and anisotropy artifact. The deep fibres of the TPT that merge with the underlying spring ligament undergo sesamoid fibrocartilage transformation resulting in a "particle

board" echotexture as opposed to the "laminated wood" pattern which associate with healthy tendon structure in long-axis assessment (Stephen Bird, private correspondence, 2017). Tenosynovitis will present with a large amount of fluid within the tendon sheath, swelling of the tendon and hyperaemia of the sheath. The abnormal tendon and surrounding sheath appear as a target sign in the short-axis view, and hyperaemia of the synovium is present on colour Doppler.[2,10,13] Three classifications have been described in regard to TPT tears with increasing severity: type I, a thickened tendon accompanied with longitudinal splits, as demonstrated in cases 1, 3, 4, 6, 7 and 8; type II, an abnormal echogenicity of the tendon due to areas of tearing and fibrosis and the tendon appears thin and focally elongated, which is demonstrated in case 5; and type III as seen in case 2, an empty retromalleolar groove with complete rupture of the tendon and stump retraction, the focal gap filled with hypoechoic fluid or echogenic granulation tissue in the grove.[39,40] Dysfunction of the dynamic stabiliser TPT has the potential to cause stretching or tears of the static stabilizer spring ligament, resulting in a flattened longitudinal arch.[15]

In the setting of a normal TPT and healthy patient, a normal spring ligament has a hyperechoic "cotton wool" echo pattern. The spring ligament however has a different echotexture to the other ankle ligaments as it is made of fibrocartilage and hence has an ultrasound appearance similar to the triangular fibrocartilage complex of the wrist or the medial meniscus of the knee (Stephen Bird, private correspondence, 2017). The superomedial component of the spring ligament when normal measures 4 mm proximally and 3.6 mm distally.[9] A patient with typical TPT dysfunction often will show a thickened spring ligament with loss of fibrillar echopattern, especially in the distal portion over the talar head.[9] Mansour et al[41] demonstrated an abnormal spring ligament measuring 5.1 mm and 6.1 mm at the proximal and distal sites, respectively; the same authors also observed in their studies that five of the nineteen patients were found to have increased power Doppler of the spring ligament when it was deficient.[41] The superomedial component of spring ligament that blends with the deltoid ligament is often noted to have ruptured in patients with AAFD.[42]

There are three types of spring ligament tear; type I, partial tears either at the sustentaculum tali or navicular insertion as single or multiple small delamination tears within the mid-substance, which is demonstrated in case 4; type II, obvious evidence of ligament laxity with or without the finding of a tear, demonstrated in cases 2, 5, 6, 7 and 8; and type III correlated to case 3, complete rupture of the ligament.[43] The spring ligament with a partial tear will appear hypoechoic and thickened with loss of granular echotexture; increased vascularity may be seen on power Doppler.[44] When the spring ligament ruptures, the head of the talus collapses due to lack of a supporting mechanism. This results in flattening of the medial longitudinal arch hence the flatfoot deformity.[8,15]

Conclusion

AAFD is well recognised by podiatrists and foot/ankle surgeons.[1] Although the condition has been well published in literature, it is often misdiagnosed as a chronic ankle sprain or arthritis by the general practitioner.[1,3] The clinical staging of the severity of the condition has been well accepted.[32] Much progress has been made to improve diagnostic imaging of AAFD with ultrasound and MRI. The high specificity and sensitivity of ultrasound detecting abnormality of the spring ligament as associated with AAFD is seldom mentioned in literature. This article re-enforces the efficacy of ultrasound in detecting TPT dysfunction, as well as the frequently associated spring ligament involvement. Despite a small sample volume, the case series serves to provide an insight into the significance of incorporating the spring ligament during the ultrasound evaluation of AAFD. However, larger-scale studies are needed to make statistically significant conclusions on the prevalence of spring ligament involvement in AAFD. An early recognition with detailed diagnosis of the deformity can improve patient outcome and prevent progression of the deformity to late-stage AAFD.

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