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Modern Theory of the Development of Adult Acquired Flat Foot and an Updated Spring Ligament Classification System

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Abstract

Traditionally tibialis Posterior Insufficiency is still considered the commonest cause of acquired adult flat foot. This is still considered as the primary cause and has influenced both treatment and the diagnosis of the condition. Foot surgeons are now questioning the whole classification and the fundamental errors within this that have not been challenge for over 30 years. In this review article we examine the emerging evidence that suggests a new and alternative pathogenesis to this disease process centred on failure of the spring ligament.

Keywords: Flat foot; Spring ligament; Planovalgus foot; Chopart's joint

Introduction

AAFD has now become synonymous with the term Posterior tibialis Tendon Dysfunction (PTTD). Our current stance on AAFD (adult acquired flat foot deformity) secondary to tibialis dysfunction is almost entirely influenced by the work of Johnson and Strom. In 1989, Johnson and Strom proposed a sequence of stages with progressive failure of the tibialis posterior through the stages resulting in synovitis, subsequent elongation and tears and eventual rupture of the tendon in stage 3 deformities [1].

Their classification system which was both anatomic clinical was for the first time able to look at a spectrum of deformities and allow them to be graded and communicated and has now been accepted as the standard to which we base our diagnosis and treatment. They presented a series of clinical findings on the state of the foot and then related this to the state of the tibialis posterior tendon. They specifically stated that in stage 2 that there is elongation of the tendon which results in the characteristic planus deformity thus implying that the tibialis posterior tendon is the primary dynamic stabiliser of the medial longitudinal arch. The subsequent assumption has always been that it is the primary dysfunction of this tendon that then results in a cascade of events that leads from a spontaneous primary synovitis and then secondary stretching and tears of the tendon and then rupture causing a sequence of structural changes in the foot with fixed planovalgus deformity being the end point.

At the time of writing this article our understanding of this condition continues to be influenced by Johnson and Stroms description but we challenge this fundamentally flawed position and attempt to elucidate the true pathogenesis of this condition.

Presentation

Patients can present with a constellation of symptoms and signs. It typically presents with medial foot pain, lateral foot impingement pain and swelling and a sensation of instability. Patients may even complain of an inability to tolerate uneven surfaces and have a progressive collapse of the medial longitudinal arch.

Clinically the patients may have collapse of the medial longitudinal arch and have an inability to single stance leg raise [1,2]. It is more common in females with high BMI [3,4].

The Traditional Theory for the Plano Valgus Foot

Traditionally the functional failure of the tibialis posterior tendon was assumed to occur for 2 reasons. Firstly the tendon becomes a spontaneously synovitic and the secondarily stretches and renders the tendon ineffective in maintaining the medial longitudinal arch. In fact Johnson and Strom suggested substitution of the tibialis posterior tendon with FDL as part of their treatment protocol in their treatment of stage 2 disease.

The tibialis Posterior is the primary dynamic support for the arch and functions as a hind foot invertor. It adducts and supinates the foot to lock the midfoot and allow it to progress in stance. It also acts as a secondary plantar flexor of the ankle. Its importance in acting as an antagonist to peroneus brevis in maintaining the balance of the foot is important and its over activity due to the lack of PB is an important cause of pes cavus [5].

The Tibialis Posterior musclotendinous unit is second only to that of the Achilles tendon in its strength in the leg. During locomotion, the tendon moves through a distance of 1 to 1.5 cm and small increases in its length due to synovitis is thought to lead to a significant breakdown in its function. This is thought to make it ineffectual in supporting the medial longitudinal arch of the foot leading to the collapse of the medial arch and AAFD [5].

The plantar fascia, plantar ligaments and the spring ligament complex must also fail prior to the collapse of the arch. It is important to remember that the plantar fascia has three fold strength in maintaining the medial arch compared to tibialis posterior. Some authors believe that intrinsics can also play an additional role in the maintenance of the arch [6]. The final deformity has several components including planus, hindfoot valgus and forefoot abduction. Fixed joint changes and degeneration are a later stage phenomenon [1,2,5].

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Stage	Deformity
Stage 1	Tenosynovitis of tibialis posterior without foot deformity
Stage 2	Flatfoot with forefoot abduction (too many toes sign).
Stage 3	Flatfoot deformity with rigid forefoot abduction and rigid hind foot valgus (X-Ray shows subtalar arthritis).
Stage 4	Flatfoot, rigid forefoot and hind foot deformity with deltoid ligament compromise (X-ray shows subtalar arthritis with talar tilt on ankle mortise views).
Stage 4 disease was added later by Myerson [7].	

Table 1: The original staging of plano valgus foot.

The Original Staging of Plano-Valgus Foot

Table 1 shows the tibialis posterior insufficiency has four grades

Challenging the Existing Theory

Within the orthopaedic community, the terms adult acquired flat foot(AAFD) and tibialis posterior Insufficiency are used interchangeably inappropriately reinforcing our belief that Plano-valgus foot exists only as a consequence to Tibialis Posterior insufficiency.

The role of the spring ligament and its contribution to the medial longitudinal arch have has been underrepresented and often ignored as part of the treatment planning [8,9].

In 2001, Yeap et al published a key paper describing the results of tibialis posterior tendon transfers. These procedures were performed as a treatment for drop foot and in a series of 12 patients. None developed planovalgus deformities that we might have expected [10]. The mean follow up was 90 months (range 24 to 300). Whilst the scale of the study was modest and the age range of the patients significantly different from the more mature population we normally associate with AAFD, the failure of the collapse of the medial longitudinal arch does force us to question traditional view point.

A similar study by Mizel [11] et al looked at ten patients with complete traumatic common peroneal nerve palsy, with no previous foot or ankle surgery or trauma distal to the knee, who had undergone anterior transfer of the posterior tibial tendon to the midfoot. Six had a transfer to the midfoot and four had a bridle procedure with tenodesis of half of the posterior tibial tendon to the peroneus longus tendon. At 74.9 months follow-up (range, 18-351 months) all patients' feet were assessed for muscle strength, the longitudinal arch, and motion at the ankle, subtalar, and Chopart's joint. Weightbearing lateral radiographs and Harris mat studies were done on both feet. In no case was any valgus hindfoot deformity associated with the lack of the tibialis posterior was found. Their conclusions were that seems that the AFFD associated with a posterior tibial tendon deficient foot will not manifest itself if peroneus brevis function is absent.

In our unit, 10 cases of tibialis posterior transfer for pes cavus and drop foot in both phasic and non-phasic transfer usage have failed to result in a single case of flat foot over a follow up period of 2 to 8 years. Despite the use of lateral translation of the foot as a guide to spring ligament failure/strain, there was no demonstrable increase in lateral translation in 9 feet and no clinical presence of planovalgus in any foot.

These studies question the essential role of the tibialis posterior and clearly demonstrate that its absence does not necessarily lead to planovalgus foot.

Increasing evidence has emerged establishing the primary pathology of this disease is in fact entirely due to the failure of the static restraints and most importantly the failure of the spring ligament [8,9,12].

Deland et al. [13] in a cadaveric study showed that the planovalgus deformity was recreated by systematically cutting key ligamentous structures. Later, the deformity was corrected by reconstructing the spring ligament alone using a bone/tendinous graft.

Isolated spring ligament failure in the absence of tibialis tendinopthy has been demonstrated. Saxby et al demonstrated cases of spring ligament failure without posterior tibial tendon synovitis leading to planovalgus foot. Orr et al. [14] described 6 patients, all female who presented with isolated rupture of the spring ligament and apparently normal tibilais posterior tendons. All of the patients achieved normal foot positions following surgery to the spring ligament itself and/or bony fusion.

Crucially Jennings et al. demonstrated in 5 cadaver specimens using a 3-dimensional kinematic system and a custom-loaded frame in the in vitro model, and quantified the rotation of the talus, navicular, and calcaneus before and after sectioning the spring ligament complex. They did this whilst incrementally tensioning the posterior tibial tendon [15]. After sectioning the spring ligament complex significant changes in talar, navicular, and calcaneal rotations were demonstrated. Importantly they demonstrated that spring ligament complex sectioning alone created instability in the foot, which crucially the intact posterior tibial tendon was unable to subsequently compensate for. They concluded that the spring ligament was the major stabilizer of the arch during mid-stance. Correctly they concluded that the spring ligament complex should be evaluated and - if indicated-repaired in flatfoot reconstruction.

The classification system presented to us by Johnson and Strom can be criticised on multiple levels. There has never been a study both anatomical and cadaveric which demonstrates the progression of one stage to the other. Yet these assumptions have become part of our traditional thinking. It also focuses upon the tendon erroneously and therefore bypasses the focus from other structures that fail to allow AFFD.

The authors seem to link conclusions regarding the state of the foot with the state of the tibialis tendon. At each stage of the deformity there is a physical change in the state of the foot and yet conclusions drawn from this clinical picture on the state of the tendon which may not always occur. Some of this would be impossible to prove such as the lengthening of the tendon and may be a long standing false assumption.

The Johnson paper also begins with the assumption that the foot begins in a neutral posture which then goes onto planovalgus but a number of authors have described an association of tibialis posterior dysfunction with a pre-existing flat foot [16]. Johnson's system does not take this into account of the degree of pre-existing planovalgus [17,18]. Jahss noted a pre-existing flatfoot in 100 per cent of his own series. The paper contains no actual data and no subsequent publication has examined the reliability and reproducibility of the Johnson system or indeed how it influences clinical decision taking.

Ultrasound and MRI are increasingly being used to diagnose PTTD. Ultrasound however is more operator dependent. No studies have been able to link ultrasound findings of the tendon with prognostic evaluation. The foot might also be in planovalgus but have no synovitis around the tendon. It is also not possible to state if the tendon is stretched and no US study has yet been able to prove this.

The classification system finally fundamentally also oversimplifies stage 2 disease which can be broken down into the loss of 4 components: tarso-metatrsal instability, fixed supination deformity,

tight gastrocsoleus/ tendoachillies and failed spring ligament. These components need to be identified and assessed individually. These can be evaluated clinically and a recent clinical test has also been described for the assessment for the spring ligament complex [8].

Towards a New Theory of Plano Valgus Foot

The authors believe the spring ligament is the most important issue in Acquired Adult flat foot and tibialis posterior synovitis occurs as a secondary synovitis. It is most likely the primary failing structure in the AAFD. Biomechanical factors may influence (poor collagen state and obesity and pre-existing planovalgus foot) its early failure. This then drives a mechanically overload of the tibialis posterior leading to its synovitis/dysfunction. This is akin to peroneal overload/dysfunction in pes cavus where peroneous brevis tendon becomes synovitic due to biomechanical overload. We therefore believe that stage 2 flatfoot– as described by Johnson et al-cannot occur without spring ligament attenuation and/or rupture. This event would then be followed by the failure of the other ligaments and cause a secondary biomechanical synovitis of tibialis posterior [8,9].

This position is reaffirmed by Singh et al, who showed that patients without tibialis Posterior function iatrogenic transfer of tibialis posterior tendon transfer for neurological feet do not necessarily develop a flat foot, even in the presence of peroneus brevis function [10].

The idea of stage 1 disease can be challenged as the development of spontaneous synovitis is unlikely. The overall incidence of planovalgus feet in patients who have extensive primary synovitis can be assessed in the rheumatoid population. Patients with rheumatoid arthritis have only 11% incidence of planovalgus in some studies. This suggests that despite inflammation in the tendon and the ligaments the foot fails to constantly develop planovalgus. The tendon is more likely to become synovitic as a result of abnormal biomechanical environment around which it acts [16].

Dyal also showed that 70% of patients with unilateral symptomatic tibialis tendon had a contra-lateral flat foot, implying that the symptomatic foot was probably flat to start with [19]. We believe that this biomechanical profile subsequently allows easier failure of the spring ligament and is the subsequent strain that allow the foot to go from a state of stable to unstable planovalgus. MRI of the symptomatic flat feet show abnormalities in all the spring ligaments as well as the tibialis posterior tendon in nearly all feet. Most showed abnormalities in the superficial deltoid, interosseous and talocalcaneal ligaments [19]. Crucially radiographic imaging cannot differentiate the difference between the stable planovalgus foot and the unstable planovalgus foot which is painful. We believe that in the non-painful planovalgus foot the spring ligament has not failed and there is no TMT instability and the foot is statically restrained.

In Table 2, we propose an altered classification. Here, the existing system has now been revisited to centre on the spring ligament.

We believe that if a tendinopathy occurs due to the unstable flat foot, this would suggest that there must be a pre-tendinopathic stage where the Spring Ligament ruptures and the tendon have not yet become overactive or synovitic. Early spring ligament failure can often be difficult to diagnose [17]. Early spring ligament failure can now be isolated and tested for using the neutral heel lateral push test. Pasapula believes that this early failure uses the talonavcular axis and the first ray to amplify the strain that develops in the spring ligament. The spring ligament is largely a medial structure which results in a largely lateral plane deformity far before the development of planovalgus which requires further failure of the medial column in the stretching of the plantar fascia and the development of first TMT instability. Pasapula described this state as stage 0 disease where the Spring Ligament has failed as detected by excessive lateral translation of the foot but the foot has not yet progressed to a planovalgus state and the tibialis posterior has not become synovitic [8,9]. This initial stage would then be followed by stage 1 disease in which there is attenuation or rupture of the Spring Ligament with a secondary synovitis around tibialis posterior as described by Johnson and Strom.

Stage	Deformity
Stage 0	Spring ligament laxity but no tendinopathy or planovalgus
Stage 1	Spring ligament laxity/failure with tendonopathy but normal tendon length and no deformity
Stage 2	Spring ligament failure with tendon lengthening and flexible planovalgus deformity
Stage 3	Spring ligament failure with tendon lengthening, and fixed planovalgus deformity

Table 2: Altered classification.

Pasapula proposed a reclassification of the Johnson and Stroms 1989 original classification with more focus around the spring ligament. Its aim was to recognise the failure of the spring ligament in the asymptomatic flat foot. Crucially, this new system demonstrates the asymptomatic stage or Stage 0 disease [8,9].

Stage 2 disease is a mixture of complex problems with progressive failure of the medial column starting with 4 associated complexities of which the first 3 are progressive deformities.

a. spring ligament failure (identified by the neutral heel lateral push test)

b. TMT instability

c. TMT instability with permanently dorsiflexed first ray or a fixed supination deformity once the hindfoot has been put back into neutral

d. Primary or Secondary tight gastrocsoleus (impossible to differentiate)

This new perspective has implications in the management of AFFD. Earlier recognition of the strain that develops in the spring ligament may lead to earlier intervention and this intervention may lead to the prevention of further failure and instability of more structures in the foot.

The failure to address the spring ligament effectively intra operatively, may lead to high recurrence rates particularly in large corrections. Niki et al reported their results of 25 patients who underwent a calcaneal osteotomy with FDL transfer and showed that just two radiographic parameters improved. In this series, the authors concluded that this procedure alone had limited effectiveness except to treat small corrections [20].

More recently cadaveric work on modelling different reconstruction models of the spring ligament by Pasapula et al has showed that spring ligament reconstruction is best done through an augmented device such as the arthrex internal brace. This is far superior to a non-augmented reconstruction. He also demonsatrated in the biomechanical model that an FDL transfer with load applied fails to improve lateral translation of the foot [9].

Conclusions

In conclusion we believe that AAFD is primarily a disease of the spring ligament and a failure to address this intra-operatively is essential. Procedures performed on the posterior tibial tendon at an

early stage have probably had no effect on the natural history of the disease. Bony fusion is effective but is associated with a loss of dynamic movement in the hind foot and increased pressure on adjacent joints leading to further degenerative changes. The emphasis should be on early detection and repair of the spring ligament and other static restraints.

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