FOOT AND ANKLE

Tibialis posterior dysfunction


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Summary
Tibialis posterior dysfunction is the commonest cause of adult acquired flat foot. Although awareness is increasing, the diagnosis is frequently missed and referral for specialist treatment delayed. Early treatment with simple orthotic devices can often prevent progression. This review outlines the diagnosis, treatment and surgical outcomes, and highlights controversies and recent advances.

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Introduction
Partial rupture of the tibialis posterior tendon was first described in 1953.1 Kettlekamp later reported four cases of spontaneous rupture of the tendon and concluded that satisfactory results could not be achieved with delayed repair.2 Mann described the clinical findings and successful results of repair following tibialis posterior dysfunction using a flexor digitorum longus transfer in 1983.3 As tibialis posterior dysfunction is the commonest cause of acquired flat foot deformity in adults, a thorough knowledge of it is required by all general orthopaedic surgeons assessing foot and ankle problems in a clinical setting. Its prevalence has been reported to be up to 10% in elderly patients but it is classically seen in middle aged women.4 While awareness of tibialis posterior dysfunction has improved, the diagnosis is often missed. As prompt diagnosis and early treatment can prevent progression, delay can result in progression of the condition and a poor outcome.5–8 Altered foot shape may not be the presenting complaint, rather pain and swelling around the medial hindfoot, lateral sided ankle pain or a reduced walking distance may prompt the patient to seek medical advice.5

The tibialis posterior tendon
The tibialis posterior muscle is innervated by the tibial nerve. It originates from the posterior interosseous membrane and the proximal 2/3 of the adjacent posterior tibia and fibula, forming the deep posterior compartment with popliteus, flexor hallucis longus and digitorum longus. It condenses to form a tendinous structure in the distal third of the calf before passing behind the medial malleolus. Unlike ankle muscles of similar power, a larger part of tibialis posterior is tendinous proximal to the malleolus, where it runs along a groove deep to the deltoid ligament, changing direction in its sheath towards the navicular tuberosity. It then inserts into the navicular tuberosity and the plantar surface of the medial cuneiform by an anterior slip. The posterior slip inserts into the plantar surfaces of the cuneiforms and the base of the second to fourth metatarsals.

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Proximally the tendon is supplied by branches of the posterior tibial artery, which continue distally within the tendon; distally by branches from the dorsalis pedis. Four centimeters from its insertion there is a watershed in the vascular supply. It is thought that the acute change in direction of the tendon as it hugs the medial malleolus causes a zone of relative hypovascularity rendering the tendon susceptible to degenerative change.9,10

Tibialis posterior is the primary dynamic stabiliser of the medial longitudinal arch. It elevates the medial longitudinal arch by inverting and plantar flexing the foot, locking the mid-tarsal joints, inverting the subtalar joint and stabilising the hindfoot. The now rigid mid and hindfoot enables a more effective action of gastrocnemius/soleus during mid stance and propulsion in the gait cycle.5

The tendon has a relatively large cross sectional area, twice that of peroneus brevis, it’s primary agonist.11 As it has only a short excursion, it will fail in its principle function as the primary stabiliser of the medial longitudinal arch if elongation of as little as 10 mm occurs12 and is then functionally useless. If, as a consequence, the midfoot does not lock during propulsion, contraction of the gastrocnemius/soleus causes excessive forces at the midtarsal joints leading to collapse of the medial arch and eversion of the subtalar joint. To keep the foot plantigrade, abduction takes place at the talonavicular joint, giving rise to the radiographic appearance of an ‘uncovered navicular’ (Fig. 1a).

Patients with tibialis posterior dysfunction have significantly reduced stride length, cadence and walking speed,13

Figure 1  Antero-Posterior and lateral weight bearing radiograph of patient with tibialis posterior dysfunction. (a) AP view shows ‘uncovering’ of the talo-navicular articulation. (b) Lateral view showing loss of the medial longitudinal arch and an obtuse talo-navicular angle.

Figure 2  Posterior view of non-pathological double heel raise. In the resting position the hindfoot angle is five degrees of valgus. When the heel raise is performed, the heel swings into a varus position.
which explains why patients may present with reduced function rather than altered foot shape as their primary complaint.

Aetiology of tibialis posterior dysfunction

We now have greater insight into the pathological process and treatment options for tibialis posterior dysfunction. However, the aetiology of tibialis posterior dysfunction remains controversial and is still not completely understood, but is likely to be a multifactorial.

Middle age women are commonly affected, and the incidence increases with age. Other risk factors include hypertension, obesity, diabetes mellitus, and seronegative arthropathies. There are a number of reports in the literature associating both local injection of steroids and oral corticosteroid intake with rupture of the tendon. The presence of an accessory navicular may interfere with the function of tibialis posterior and be a risk factor; an MRI study of 27 patients with an accessory navicular and tibialis posterior dysfunction found that the insertion of tibialis posterior was solely into the accessory navicular without slips to the plantar surfaces of the metatarsals in the majority. Fourteen percent of the adult population have an accessory navicular, but only one in a thousand will be symptomatic and it has recently been suggested that the flatfoot deformity may precede the dysfunction of the tibialis tendon. Imhauser et al showed that dysfunction of the tibialis posterior tendon caused a posterior shift in the centre of pressure and abnormal loading of the medial structures and concluded that it unclear whether the flat foot precedes or results from the tendon dysfunction.

The pathological process in tibialis posterior tendon dysfunction may be degenerative, inflammatory or repeated microtraumatic in nature. Histological findings include mucinous degeneration, vascular hyperplasia and tendon sheath hyperplasia. Additionally, disruption of collagen fibers, metaplasia of fibrocartilage and calcific deposits are common findings in the tendons of tibialis posterior tendinopathy.

Figure 3  The examiner can quantify the degree of loss of the medial longitudinal arch clinically by the number of fingers that can be passed underneath the midfoot.

Figure 4  Posterior view of a patient with tibialis posterior dysfunction on the left displaying ‘too many toes sign’.

Figure 5  Double heel raise displaying failure of the hindfoot to swing into a varus position on the left.

Figure 6  Loss of the medial longitudinal arch of the left foot.
foci have been described. Again, it is not clear whether these changes precede or appear after the clinical changes.

**Classification**

Johnson and Strom classified posterior tibial tendon dysfunction, later modified by Myers to add a fourth stage. The key points in this classification are whether the tendon is still functional and mobility of the subtalar joint.

- **Stage I.** The arch is maintained but tibialis posterior tendon is inflamed
- **Stage II.** The tendon is no longer functional and there is an acquired flat foot.
- **Stage III.** There is an acquired flat foot and the subtalar joint valgus cannot be passively reduced.
- **Stage IV.** There are arthritic changes in the ankle.

This classification is used widely as a guide to management (Table 3).

**Diagnosis of tibialis posterior dysfunction**

In the early stages the patient will often describe medial ankle discomfort along the course of the tendon. If there is an element of tenosynovitis, there may be swelling around the medial malleolus. As the condition progresses, tenderness initially present along the tendon may disappear, and patients may complain of fatigue and aching in the leg and a reduction in walking distance. With further progression they may notice a change in the shape of the foot with a loss of the medial arch and the heel drifting into valgus, but commonly patients complain of loss of function rather than a change in the shape of their foot. They may also have symptoms of instability, a limp and inability to walk on uneven surfaces. In the later stages the patient may complain of lateral ankle pain as the fibula impinges on the calcaneum. Patients find standing on tiptoe difficult, and notice asymmetrical wear along the medial sole of their shoes. One must always consider tibialis posterior tendon dysfunction in middle aged or elderly patients with lateral hindfoot pain and not forget that other foot pathologies may initiate medical attendance, and incorrect initial diagnosis of hallux valgus, hallux rigidus and metatarsalgia may be the initial diagnosis in primary care.

While the typical patient is a middle aged female, there is a distinct subset of patients who are young athletes. These 20–30 year old patients are often involved in impact sporting activities and may recollect a distinct injury. The forefoot is classically significantly pronated in this group of patients.

**Figure 7**  Lateral radiograph of extreme loss of medial longitudinal arch and adult acquired flat foot.

**Figure 8**  a) T2 weighted magnetic resonance imaging in axial and sagittal sections showing increased signal around the tibi- alis posterior tendon. (b) Ultra-sound cross sectional image, with hypo-echogenic area around tibialis posterior tendon.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Differential diagnosis of painful flat foot</th>
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<tbody>
<tr>
<td>Arthritis</td>
<td>Degenerative arthritis of the ankle, talonavicular or tarsometatarsal joints secondary to inflammatory arthropathies, osteoarthritis or post trauma.</td>
</tr>
<tr>
<td>Neuropathic Foot</td>
<td>Diabetes Mellitus, peripheral neuropathy or leprosy.</td>
</tr>
<tr>
<td>Failure of supporting foot anatomy</td>
<td>Tibialis posterior dysfunction. Spring/deltoid ligament dysfunction. Rupture of tibialis anterior.</td>
</tr>
<tr>
<td>Avascular necrosis</td>
<td>Kholler's disease (painful avascular necrosis of the navicular in childhood)</td>
</tr>
<tr>
<td>Entrapment neuropathies</td>
<td>Tarsal tunnel syndrome (poorly localized burning pain over the medial ankle and foot).</td>
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The diagnosis is made clinically; thus careful examination is of paramount importance, which must expose the entire lower limbs as valgus deformity of the knees can accentuate the appearance of pes planus. The feet should be examined from above and behind. In health the hindfoot is in 5 degrees of valgus, which is best assessed from behind (Fig. 2). In stage I tibialis posterior dysfunction the medial longitudinal arch will be maintained and there may be swelling and tenderness along the course of the tendon behind the medial malleolus. Passing a hand under the arch of both feet using the examiners finger creases as a reference allows quantification of any discrepancy in the medial arch (Fig. 3). In the later stages there will be an acquired flat-foot deformity, which initially will be correctable, with free subtalar movements. The ‘too many toes sign’ may be present; when viewing from behind more than two lesser toes are seen along the lateral border of the foot (Fig. 4).

The patient should undertake both double and single heel raise tests. A double heel raise, performed when the patient rises from standing plantigrade onto the balls of the feet is described as being without upper limb support. However some elderly patients are unable to do this and allowing them to rest their hands against a wall will often increase their sense of stability without significantly affecting the clinical findings - this is a test of motor power, not balance. Viewing the limb from behind, a normal heel will move into five degrees of varus as the medial longitudinal arch and hindfoot lock allowing gastrocnemius/soleus to plantarflex the ankle. Loss of the heel varus movement is characteristic of tibialis posterior dysfunction (Fig. 5). The single heel raise should then be performed; the patient will be unable to do this on the affected side. Note however that in a younger sporting patient, these tests may need to be repeated up to twenty times to demonstrate fatigue of the tendon, particularly comparing with the normal side. The patient should be then asked to walk and examined from behind and from the front. During gait, the differences may become even more apparent.

The appearance of acquired flat foot caused by tibialis posterior dysfunction must be distinguished from that of non-acquired flat foot. Tibialis posterior dysfunction causes collapse of the midfoot, apparent clinically by inspecting the talonavicular joint angle from the medial side. When inspecting the medial border of the foot the talo-navicular angle will be obtuse in tibialis posterior dysfunction (Fig. 6).

The power of tibialis posterior is assessed by sitting the patient seated higher than the examiner with his/her knee bent and the affected foot resting on the examiners lap. The foot is held everted and plantarflexed to eliminate the synergistic action of tibialis anterior.25 The patient is then instructed to ‘swing the foot inwards’ against the resistance of the examiners hand. After palpating tibialis anterior tendon to confirm that it is not being recruited, the tibialis posterior tendon is palpated along its length. Subtalar and ankle movements are also assessed as many patients will also have chronic tendoachilles or calf tightness exacerbating the hindfoot valgus. An accurate assessment of ankle dorsiflexion with the knee flexed and extended should be made to identify if the tightness is within the gastrocnemius or both the gastrocnemius and soleus (Silfverskiöld’s test). The position of the forefoot must be assessed in relation to the hindfoot, because as the hindfoot valgus...

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**Table 2** MRI classification of tibialis posterior tendon tears27

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
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<tbody>
<tr>
<td>Type 1</td>
<td>One or two fine longitudinal splits in the tendon.</td>
</tr>
<tr>
<td>Type 2</td>
<td>Wider longitudinal splits and intramural degeneration (grey areas in tendon). Variable diameter.</td>
</tr>
<tr>
<td>Type 3</td>
<td>Diffuse swelling and uniform degeneration. Most of tendon is replaced by scar tissue.</td>
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**Table 3** Classification of the clinical stages of tibialis posterior dysfunction.23,17 Surgical Treatment options

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Inflammation and/or degeneration of the tendon without compromise of the medial longitudinal arch.</td>
<td>Decompression of tendon*&lt;br&gt;Synovectomy +/- FDL Transfer</td>
</tr>
<tr>
<td>II</td>
<td>Diseased tendon elongates.&lt;br&gt;Flexible flatfoot deformity.</td>
<td>FDL Transfer and medial displacement calcaneal osteotomy*&lt;br&gt;FDL Transfer&lt;br&gt;LateraL column lengthening (e.g. calcaneo-cuboid distraction arthrodesis)&lt;br&gt;Medial column fusion (navicular-cuneiform or/meta-tarsal-cuneiform joint)&lt;br&gt;Isolated hindfoot fusions&lt;br&gt;Arthroereisis</td>
</tr>
<tr>
<td>III</td>
<td>Rigid flatfoot deformity.</td>
<td>Triple fusion*&lt;br&gt;Isolated hindfoot fusions (eg. subtalar fusion) or medial column fusion</td>
</tr>
<tr>
<td>IV</td>
<td>Rigid flatfoot deformity with degenerative ankle arthrosis.</td>
<td>Pan-talar arthrodesis*</td>
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* Authors preferred method and the most widely used techniques.
increases, compensatory forefoot supination occurs to create a plantigrade foot.

Radiographic evaluation

Although the diagnosis is clinical, radiographic evaluation is necessary to assess deformity and the presence of ankle and subtalar arthritis and to exclude other causes of acquired flatfoot deformity (Table 1, Fig. 7). Standing plain antero-posterior and lateral radiographs of foot and ankle (Fig. 1a and 1b) will show uncovering of the navicular head and a flatfoot deformity. The talometatarsal angle on standing lateral radiographs may be less than 0 degrees (normal 0–10 degrees).17

Magnetic Resonance Imaging has a sensitivity of 95%, a specificity of 100% and accuracy of 96% in the diagnosis of disorders of the tibialis posterior tendon26 and will demonstrate tendon integrity, size, shape and internal signal.11 Axial sections allow the best evaluation of tenosynovitis (Fig. 8a). MRI findings have been classified by Conti (Table 2).27 Ultrasound is a cost effective alternative to MRI with specificity and sensitivity close to that of MRI (Fig. 8b).27,28

Treatment

Conservative management is indicated initially in almost all patients, irrespective of the stage. Immobilisation in a below knee walking cast or walker for six to eight weeks, with non-steroidal anti-inflammatory if there is an acute tenosynovitis. After immobilization, semi rigid orthoses should be used to support the medial arch and correct the hindfoot valgus. The aim of orthoses in stage I and II is to support the medial arch and correct the hindfoot valgus. As the disease progresses and deformities become fixed, orthoses should accommodate the foot shape and prevent progression. There are many variants of ankle-foot orthoses (AFOs), but these often need to be custom made to correct and/or accommodate the range of deformities. Footwear has an important role in the conservative treatment of tibialis posterior dysfunction.5 Lace up flat shoes or boots should be encouraged, but alterations may necessary to accommodate the foot deformity and orthoses.

There is no role for local or systemic steroids, owing to the risk of causing tendon rupture.15,17 The role of physiotherapy is controversial; many regimes have been suggested including cryotherapy and massage.11

Surgical treatment can produce good to excellent results in more than 80% of patients at up to 5 years.17 The surgical procedure indicated depends on the clinical stage of the disease (Table 3). However treatment of this complex disorder is evolving and much of the literature relating to surgery reports only small series with short to medium term follow up.

Stage I. For isolated tenosynovitis refractory to conservative measures, open synovectomy and debridement of diseased tissue has been recommended by Teasdall.29,30 The results beyond 5 years are not known. Some authors have suggested that the abnormal tendon should be augmented with a flexor digitorum longus (FDL) tendon transfer.21

Stage II. The operative treatment of stage 2 tibialis posterior dysfunction remains the main area of controversy in the orthopaedic literature and the answer is yet to be fully defined. There are several surgical options including tendon transfer, osteotomy, arthrodesis or various combinations. The most common tendon transfer utilises flexor digitorum longus, first popularized by Mann.16 The FDL is harvested close to the knot of Henry and passed through a plantar to dorsal drill hole in the medial cuneiform. This helps to cradle the navicular bone. In order to address the hindfoot valgus and redirect the vector of the Achilles tendon, Myerson added a medial displacement osteotomy of the calcaneus.17,31 The redirected pull of the gastrocnemius/soleus complex increases the varus pull at the subtalar joint correcting the hindfoot valgus. This extra articular osteotomy of the calcaneus is cut perpendicular to the lateral wall of the calcaneus, angled 45 degrees from the sole of the foot and is displaced medially by approximately one centimeter and fixed with a screw (Fig. 9a and 9b). The broad cancellous surfaces promote healing and union rates are high. Myerson recently reported the midterm results to 5.2 years.
of FDL transfer with calcaneal osteotomy for stage 2 disease. High patient satisfaction was reported in 129 patients. There is increasing biomechanical data suggesting that additional superior translation of 0.5 cm of the calcaneal osteotomy may create lower forefoot plantar pressures and theoretically reduce postoperative lateral forefoot pain.32

As the flatfoot deformity progresses, the lateral column of the foot becomes relatively shortened with respect to the medial column. Lateral column lengthening has therefore been suggested for stage II dysfunction. This can be performed by distraction arthrodesis of the calcaneocuboid joint, or by an opening wedge osteotomy of the calcaneum 1.5 cm proximal to the calcaneocuboid joint (Fig. 10). There is a high incidence of non union with both these techniques. Additionally increasing lateral plantar pressures can cause post operative failure.21

Stage III and IV. Patients with rigid deformities of stage three and four particularly in the presence of lateral pain commonly require triple arthrodesis (subtalar, calcaneocuboid and talonavicular fusion) (Fig. 11).

Realignment of the hindfoot with a plantar grade foot allowing full weight bearing is the goal of surgery. Myerson reported high patient satisfaction at 5.7 years.17 The concept of limited fusion to correct the deformity, with preservation of maximal joint movement is attractive,11 and isolated subtalar or talonavicular joint fusions have been suggested, but only low volume short term follow up has been reported. Thus the place of an isolated arthrodesis is not yet clear.

![Figure 10: Postoperative AP weight bearing radiograph of lateral column lengthening osteotomy.](image1)

**Figure 10** Postoperative AP weight bearing radiograph of lateral column lengthening osteotomy.

**Figure 11** Postoperative lateral radiograph of triple arthrodesis (subtalar, calcaneocuboid and talonavicular fusion).

**Novel surgical treatments.** Subtalar arthroereisis involves insertion of a ‘plug’ or bone block into the sinus tarsi to restrict subtalar eversion. A number of techniques have been described, and early results are encouraging.33,34 Further research is necessary to define the optimum treatment.

**Conclusion**

Tibialis posterior tendon dysfunction is a common condition that is often misdiagnosed. It causes a progressive, painful flatfoot deformity. Early diagnosis and staging is important to prevent disease progression and increasing disability. Early conservative treatment is often successful in preventing progression of the dysfunction and deformity. Surgery is often successful, but controversy continues about the optimal intervention for stage II tibialis posterior dysfunction.

**References**