

Surgical treatment for tibialis posterior dysfunction

Dysfunction of the tibialis posterior tendon is the most common cause of acquired flatfoot deformity in adults. Early treatment with orthotics may often prevent progression of the dysfunction into a fixed deformity. Late presentation or failure of conservative measures may need to be corrected by surgical means. In this review we will discuss the anatomy, etiology, diagnosis and treatment of tibialis posterior dysfunction. Surgical options for dealing with patients who have failed nonoperative measures will be covered, outlining outcomes, recent advances and future directions in the treatment of this complex disorder.

KEYWORDS: arthroereisis ■ hindfoot fusion ■ medial displacement osteotomy
■ surgical treatment ■ tibialis posterior dysfunction

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Tibialis posterior dysfunction (TPD) is the most common cause of adult-acquired flatfoot deformity. Its prevalence in the elderly population has been estimated to be up to 10% [1,2]. Symptoms including pain and swelling of the tendon around the medial malleolus, lateral ankle impingement pain or reduced walking distance ability are what may prompt the patient to seek medical advice [3]. Early diagnosis and nonoperative intervention may relieve the symptoms and result in cessation of progression of the deformity [4]. However, diagnosis is often missed and referral for specialist treatment is commonly delayed. This may be associated with a negative outcome for the patient [2,5–7].

Knowledge of the surgical interventions available for TPD will help rheumatologists, general practitioners and physiotherapists assessing podiatric problems in the timely referral to an orthopedic surgeon with a special interest in foot and ankle surgery to maximize the possible outcome for their patients.

Anatomy & function of the tibialis posterior tendon

The tibialis posterior muscle runs in the posterior compartment of the leg, originating from the posterior interosseous membrane and the proximal two thirds of the tibia and fibula. The muscle runs in a groove behind the medial malleolus and inserts onto the tarsal navicular and the plantar surface of the medial cuneiform. There is an area of relative hypovascularity approximately 4 cm from its insertion, leaving the tendon susceptible to degenerative change [8–10]. The tendon is the primary dynamic stabilizer of the medial

longitudinal arch. It elevates the medial longitudinal arch by inverting and plantarflexing the foot. This locks the midtarsal joints, inverts the subtalar joint and stabilizes the hindfoot via the windlass mechanism [11,12]. The stabilized mid- and hind-foot enables more effective action of the gastrocnemius during mid-stance and propulsion in the gait cycle [3]. The tendon will fail in its function as the stabilizer of the medial longitudinal arch if elongation of only 10 mm occurs [13,14]. The tendon is functionally useless once it has been permanently stretched and its function can only be surgically augmented by a tendon transfer. If the midfoot is unable to lock during propulsion, contraction of the gastrocnemius causes excessive forces at the midtarsal joints, leading to collapse of the medial arch and eversion of the subtalar joint. To create a plantigrade foot, abduction occurs at the talonavicular joint, creating the radiographic appearance of an 'uncovered navicular' (FIGURE 1).

Patients primarily seek a medical opinion when they suffer from loss of function and pain rather than for an altered foot shape; however, the resultant foot deformity may lead the patient to seek advice with a history of preceding loss of function. Patients have significantly reduced stride length, cadence and walking speed [15].

Etiology of tibialis posterior dysfunction

The etiology of TPD is not fully understood and remains controversial [1]. Middle-aged women are commonly affected and the incidence increases with age [16]. Patients are primarily

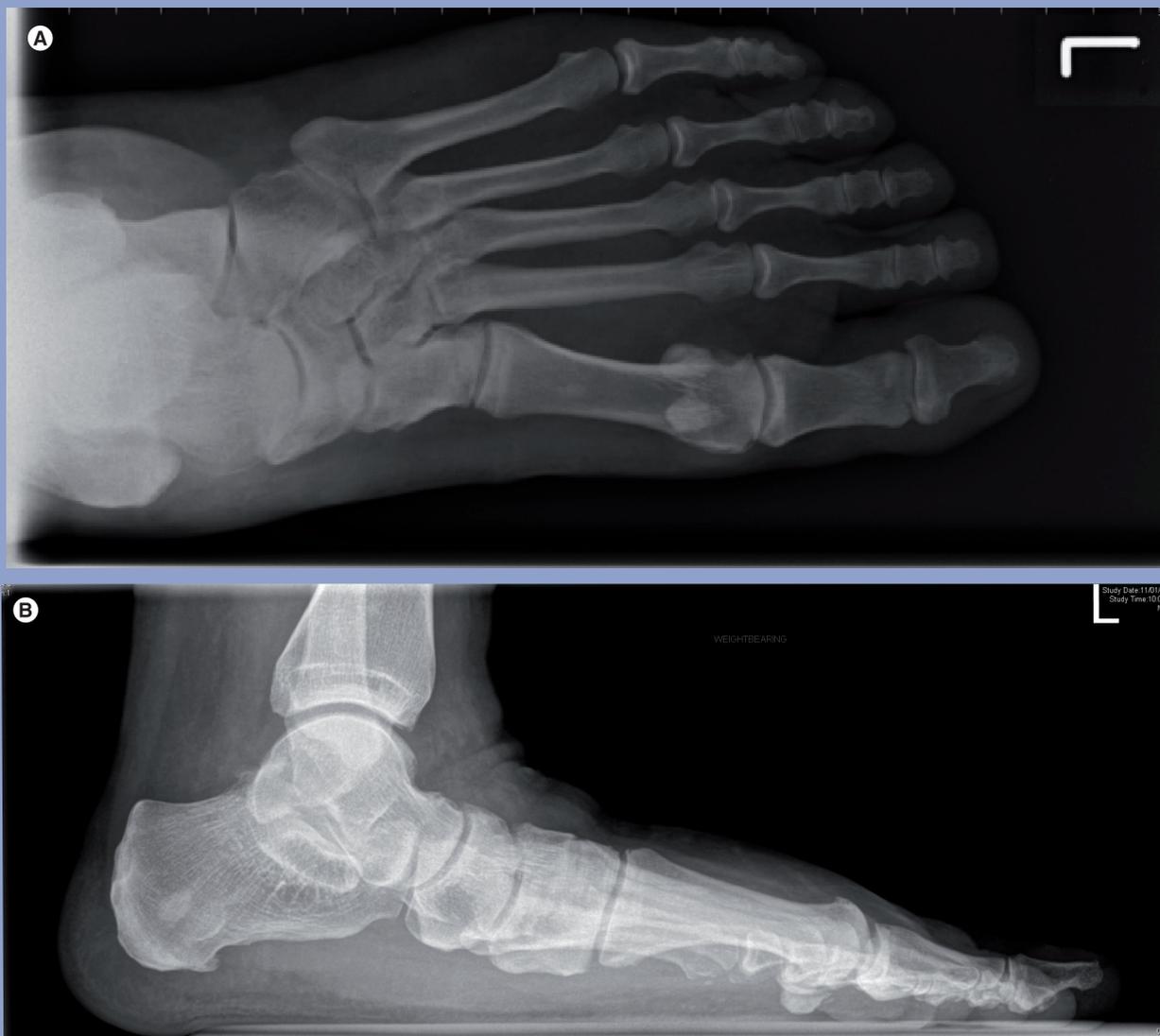


Figure 1. Antero-posterior and lateral weight-bearing radiograph of patient with tibialis posterior dysfunction.

The uncovered navicular and loss of medial arch are revealed.

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female with a ratio of 8:1, but this predominance is not fully understood [1,16]. Other risk factors include diabetes mellitus, hypertension, obesity, seronegative arthropathies and oral and locally injected steroids [16–18].

Histological findings include mucinous degeneration, vascular hyperplasia, tendon sheath hyperplasia, disruption of collagen fibers, metaplasia of fibrocartilage and foci of calcification [19,20]. These changes represent tendinosis rather than tendonitis. It is not clear whether these changes precede or appear after the clinical symptoms and signs occur. The chronology of the symptomatic progression has been questioned by some authors who believe that failure of the tibialis posterior tendon

occurs as a consequence of, rather than as a cause of, flatfoot [20]. Friction of the tendon around the medial malleolus, failure of the spring ligament and subluxation of the talocalcaneal joint have also been implicated in the etiology [21–23]. The exact cause of TPD is still not completely understood, but is likely to be a multifactorial process.

Classification

Myersons' classification is widely used and acts as a guide to surgical management; it is outlined in TABLE 1 [18]. It has recently been updated by Myerson to include stage IIa and IIb, to differentiate between a functionally impaired tendon and an incompetent one [24].

Table 1. Myerson's modification of Johnson and Strom's classification.

Stage	Clinical findings
I	Swelling and pain along course of tendon. Power of tendon intact and no deformity.
II	Mainly medial symptoms with mild medial arch collapse. Single stance heel raise still possible; the deformity is correctable.
III	Rigid flatfoot deformity.
IV	Rigid flatfoot deformity with ankle arthritis.

Data taken from [24].

Diagnosis of tibialis posterior dysfunction

Patients with stage I dysfunction complain of pain and swelling along the course of the tendon. Patients may report fatigue and aching in the calf and notice a reduction in their walking distance. Progression will lead to a change in the shape of the foot, with loss of the medial arch and the heel moving into valgus. The patient may have symptoms of instability, a limp and inability to walk on uneven surfaces [1,2]. Late symptoms may relate to lateral ankle pain as the fibula impinges on the calcaneum. Patients find standing on their toes difficult.

The hindfoot is best examined from behind. In healthy individuals, a total of 5 degrees of valgus should correct to a varus position when the patient performs a double-heel raise (FIGURE 2). The loss of this movement is characteristic of TPD (FIGURE 3). In the early stages, a single-heel raise will identify the dysfunction; however, as the disease progresses the patient will be unable to perform a single-heel raise and a double-heel raise may be utilized to demonstrate the pathology. In the later stages of TPD, an acquired flatfoot deformity will

form. Initially, this will be correctable with free subtalar movements. The 'too many toes sign' refers to the increased number of lateral lesser toes seen from behind (FIGURE 4).

Imaging of the patient with TPD should include weight-bearing radiographs of the foot and ankle to detect the presence of ankle and subtalar arthritis and to exclude other etiologies. The presence of subtalar and ankle arthritis may be either the primary pathology or a secondary resultant of advanced TPD (TABLE 2).

Weight-bearing plain radiographs of the foot and ankle are required when a patient is thought to have TPD (FIGURE 1). These radiographs allow measurement of the first talometatarsal angle, calcaneal pitch, distance from the medial cuneiform base to the floor and talonavicular coverage angle. As a flatfoot deformity develops, the arch sags at the naviculocuneiform or talonavicular joint, causing a decrease in calcaneal pitch, a decreased lateral first talometatarsal angle and depression of medial cuneiform height. The forefoot moves laterally into abduction, causing lateral subluxation of the talonavicular joint and an increase in the



Figure 2. A nonpathological double-heel raise. In the resting position, the hindfoot angle is 5 degrees of valgus. When the heels are raised, the hindfoot swings into a varus position. Reprinted from [1] with permission from Elsevier.



Figure 3. Pathological heel raise. Failure of the hindfoot to swing into a varus position (left) is characteristic of tibialis posterior dysfunction. Reprinted from [1] with permission from Elsevier.

talonavicular coverage angle [25]. MRI allows evaluation of the tendons and is both accurate and sensitive for confirming the diagnosis [26].

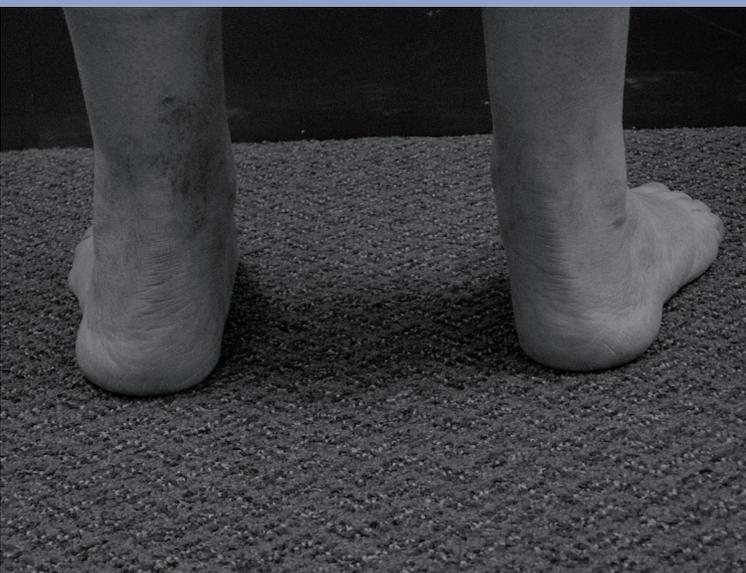


Figure 4. The 'too many toes sign'.

Specific MRI findings and classification systems are used by many foot and ankle surgeons to determine a final surgical plan. Ultrasound can be used as a cost-effective alternative to MRI (FIGURE 5) [27].

Conservative treatment

Conservative management is recommended as the initial treatment in almost all patients who present with TPD, irrespective of the stage. Immobilization is indicated using a below-the-knee walking cast or walker for 6–8 weeks, along with oral NSAIDs, if there is an acute tenosynovitis [18]. After immobilization, semi-rigid functional orthoses should be used to support the medial arch and correct the hindfoot valgus. The aim of orthoses in stage I and II dysfunction 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, but these often need to be custom made to correct or accommodate the range of deformities. Footwear has an important role in the conservative treatment of TPD, and modification of the patient's own shoes is often useful [3,4].

There is no role for local or systemic steroids in the treatment of TPD owing to the risks of causing tendon rupture [16,18].

The role of physiotherapy is controversial; many regimes have been suggested, including cryotherapy and massage [1,18]. The aims of physiotherapy regimes are based on dispersal of inflammatory mediators and products by non-toxic means. The combination of articulated ankle–foot orthoses and a structured exercise regime has proved successful in 89% of patients with type I and II dysfunction, and a recent randomized, controlled trial found significant benefit of ankle–foot orthoses using eccentric and concentric progressive resistive exercises. [28,29]

Surgical treatment

Treatment by surgery for patients with TPD should be reserved for patients who fail conservative measures. Tendon transfers to augment the failing tibialis posterior are popular. Osteotomies (cutting the bone and realigning it) of the calcaneus can correct the shape of the hindfoot and alter the biomechanics to aid normal function. Finally, if there are significant secondary degenerative changes in the joints, fusions allow correction of deformities and good relief of pain.

Table 2. Differential diagnosis of painful flatfoot.

Differential	Specific conditions
Arthritis	Degenerative arthritis of the ankle, talonavicular or tarsometatarsal joints secondary to inflammatory arthropathies, osteoarthritis or post trauma.
Neuropathic foot	Diabetes mellitus, peripheral neuropathy or leprosy.
Failure of supporting foot anatomy	Tibialis posterior dysfunction. Spring/deltoid ligament dysfunction. Rupture of tibialis anterior.
Abnormal bony anatomy	Tarsal coalition. Congenital vertical talus.
Avascular necrosis	Kohlers disease (painful avascular necrosis of the navicular in childhood).
Entrapment neuropathies	Tarsal tunnel syndrome (poorly localized burning pain over the medial ankle and foot).

Data taken from [1,3,5].

Surgery can produce good to excellent results in more than 80% of patients for up to 10 years [18,24]. However, long-term results are yet to be defined. Controversy remains as to what is the correct operation to perform at what time; however, the classification acts as a good guide (TABLE 3).

The majority of patients with grade I dysfunction will respond well to conservative measures with a rest period of immobilization and nonsteroidals. However, if this fails, the simple procedure of decompression of the tendon sheath and synovectomy has been demonstrated to produce excellent results for up to 5 years [30,31].

Tendon transfer commonly involves the flexor digitorum longus. The original function of the tendons can be sacrificed to allow it to be used as an alternative to the failed tibialis posterior. Flexor digitorum longus tendon (FDL) in divided distally, passed through a drill hole in

the navicular and appropriately tensioned prior to it being fixed. This tendon has been shown in many series to function well as an alternative to the failed tendon [24]. To correct the hindfoot valgus and redirect the pull of the gastrocnemius, an extra-articular medial displacement osteotomy can be used. The redirected pull of the gastrocnemius complex increases the varus pull at the subtalar joint, therefore correcting the hindfoot valgus [32]. This is an extra-articular osteotomy of the calcaneus; it is fashioned perpendicular to the lateral wall, angled 45 degrees from the sole of the foot. This is displaced medially by approximately 1 cm and fixed with a screw (FIGURE 6). The broad cancellous surfaces provide a good environment for bone healing and union rates are high after this osteotomy.

The combination of FDL transfer and medial displacement osteotomy has become the most common operation for stage II disease;

Table 3. The surgical treatment options for tibialis posterior dysfunction.

Stage	Classification of the clinical stages of tibialis posterior dysfunction	Surgical treatment options
I	Inflammation and/or degeneration of the tendon without compromise of the medial longitudinal arch	Decompression of tendon* Synovectomy +/- FDL transfer Tendoscopic debridement
II	Diseased tendon elongates Flexible flatfoot deformity	FDL transfer and medial displacement calcaneal osteotomy* Spring ligament repair Cobb procedure Lateral column lengthening (e.g., calcaneocuboid distraction arthrodesis) Achilles tendon lengthening Gastrocnemius recession (open or endoscopic) Arthroeresis
III	Rigid flatfoot deformity	Triple fusion* Isolated hindfoot fusions (e.g., subtalar fusion) or medial column fusion
IV	Rigid flatfoot deformity with degenerative ankle arthrosis	Pan-talar arthrodesis*

*Authors preferred method and the most widely used techniques.
FDL: Flexor digitorum longus tendon.
Classification of the clinical stages of tibialis posterior dysfunction data from [18,24].

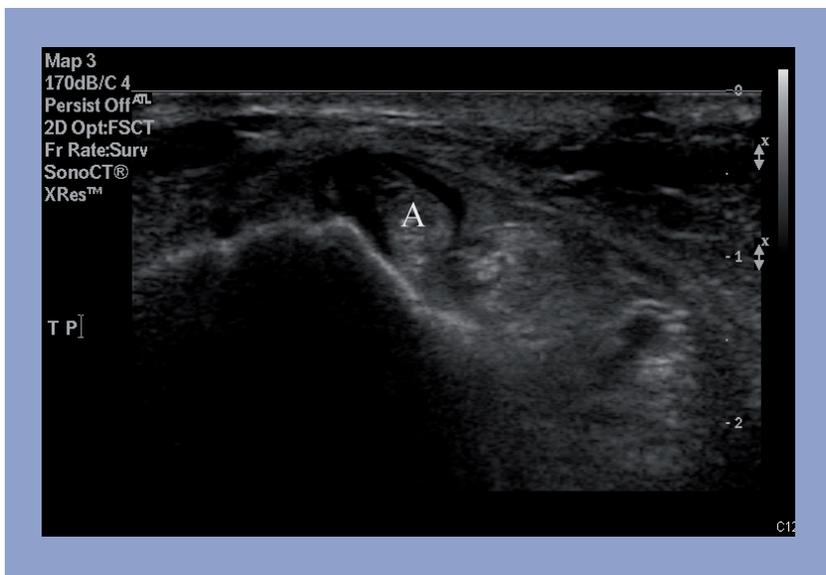


Figure 5. Ultrasonic image showing hypoechoic fluid around the tibialis posterior tendon. Label (A) highlights hypoechoic fluid around the tibialis posterior tendon.

however, controversy still remains since many other operations are reported in the literature. Myerson recently reported the midterm results into 5.2 years of FDL transfer with calcaneal osteotomy for stage II disease. High patient satisfaction was reported in 129 patients [32].

Other surgical techniques for stage II disease include a tendon transfer, splitting the tibialis anterior and attaching it to the proximal stump of the tibialis posterior (the Cobb procedure), spring ligament repair/reconstruction to reform the medial arch, Achilles tendon lengthening, gastrocnemius recession (open or endoscopic) or a combination of the previously described procedures. The long-term outcomes of these procedures are yet to be defined and the best treatment for stage II disease is not clearly defined at present.

Planal dominance refers to the plane of the foot with the greatest deformity. Deformity may be most obviously present in the coronal, sagittal or axial planes (or combinations of the three). Some foot and ankle surgeons suggest that the plane of the greatest deformity can dictate the surgical approach, especially relating to stage II disease [33].

As the flatfoot deformity progresses, the lateral column of the foot becomes relatively shortened with respect to the medial column. Therefore, lateral column lengthening has been suggested for stage II dysfunction, with or without a plantar flexion osteotomy of the medial column. This type of procedure may be most appropriate for those patients with coronal

planal dominance. Lateral column lengthening can be performed by distraction arthrodesis of the calcaneocuboid joint, or opening wedge osteotomy of the calcaneum 1.5 cm proximal to the calcaneocuboid joint. There are high incidences of nonunion with both of these techniques, as well as increasing lateral plantar pressures causing postoperative failure [34].

As the surrounding joints become degenerate and stiff, arthrodesis (joint fusions) are often required. Fusions can be created by removal of the remaining degenerate articular cartilage, preparation of the underlying cancellous bone and compression by screws or other metalwork, allowing the two surfaces to unite, similar to a fracture. Isolated foot fusions provide predictable control of pain at the expense of loss of movement, and fusions can also be fashioned to correct deformity. Rigid deformities of stage III and IV commonly require triple arthrodesis (subtalar, calcaneocuboid and talonavicular fusion) (FIGURE 7). The presence of lateral pain in patients with grade III or IV disease is an indication for triple arthrodesis. Realignment of the hindfoot with a plantar grade foot allowing full weight bearing are the goals of surgery. Myerson reports high patient satisfaction at 5.7 years [32]. The role of isolated arthrodesis is not yet completely clarified; however, the concept of limited fusion to correct the deformity with preservation of maximal joint movement is attractive. Isolated subtalar or talonavicular joint fusions have been suggested and early results are encouraging [24].

TPD with secondary osteoarthritis of the ankle and hindfoot is treated with tibio-talar-calcaneal or pan-talar arthrodesis. This may be achieved with a hindfoot intramedullary fusion nail, with or without large cannulated screws.

Conclusion

Tibialis posterior tendon dysfunction is a very common condition that can often be misdiagnosed. It produces a progressive, painful flatfoot deformity. Early diagnosis can help the clinician to utilize conservative measures to treat and prevent progression of this poorly understood condition, which can be debilitating in its latter stages.

The correct initial treatment for nearly all patients, irrespective of age, disease stage or disability, is conservative. Orthoses should be used to correct or accommodate the deformity depending upon the stiffness of the hindfoot. Local injections of steroids are detrimental and should not be used. The role of physiotherapy

is controversial, but there is increasing evidence to suggest that the combination of orthoses and specific exercise regimes may be beneficial in the early stages of the disease.

Surgical treatment of TPD should be reserved for those patients who have failed conservative measures. Surgery can produce excellent mid-term results as reported in many studies; however, there remains some debate as to the best form of intervention. The results of surgery on earlier stages produces better functional outcomes than later stages, as fusions are commonly required in stages III and IV.

The longer-term results of current techniques are not known; however, midterm results are very encouraging. Early referral to an orthopedic surgeon with a specialist interest in surgery of the foot and ankle for patients whom conservative measures fail will allow planning for surgery to optimize functional outcome.

Future perspective

Newer techniques for the surgical treatment of TPD are currently evolving. The understanding of the pathology is increasing but it is not completely understood at present. Newer, minimally invasive techniques have recently been evaluated and are becoming commonly performed procedures for patients with TPD. Arthroereisis involves insertion of a bony block, sylastic implant or metallic plug into the sinus tarsi to restrict subtalar eversion. Short- to mid-term results for early-stage disease are encouraging (FIGURE 8) [35,36]. The Topaz coblation technique utilizes a percutaneous radiofrequency probe to alter degenerative changes within the tendon. The evidence for this novel technique is very limited. Research into open and endoscopic gastrocnemius recession is currently being undertaken but its use in stage II disease is yet to be completely clarified.

Further research into the etiology, biomechanics and pathology of TPD will produce a greater insight and direct clinical understanding over the next decade. The optimum treatment for stage II TPD remains controversial at present but the variety of techniques currently used will be reported during their mid- and long-term follow-up. This clarification will allow foot and ankle surgeons to provide the most appropriate surgical interventions for their patients. There are very few randomized, controlled trials relating to dysfunction of the tibialis posterior tendon, but the next decade will provide informative trials relating not only to surgical interventions, but conservative ones too [29].

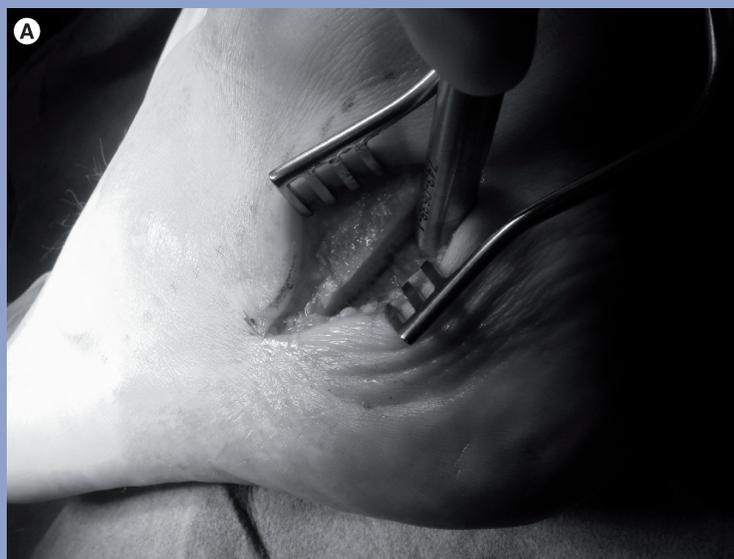


Figure 6. Intra-operative and postoperative images of oblique medial displacement calcaneal osteotomy. (A) Intra-operative photograph of lateral calcaneal incision showing medial displacement of the calcaneal osteotomy. **(B)** Post-operative lateral radiograph showing the screw fixation of the medial displacement calcaneal osteotomy.

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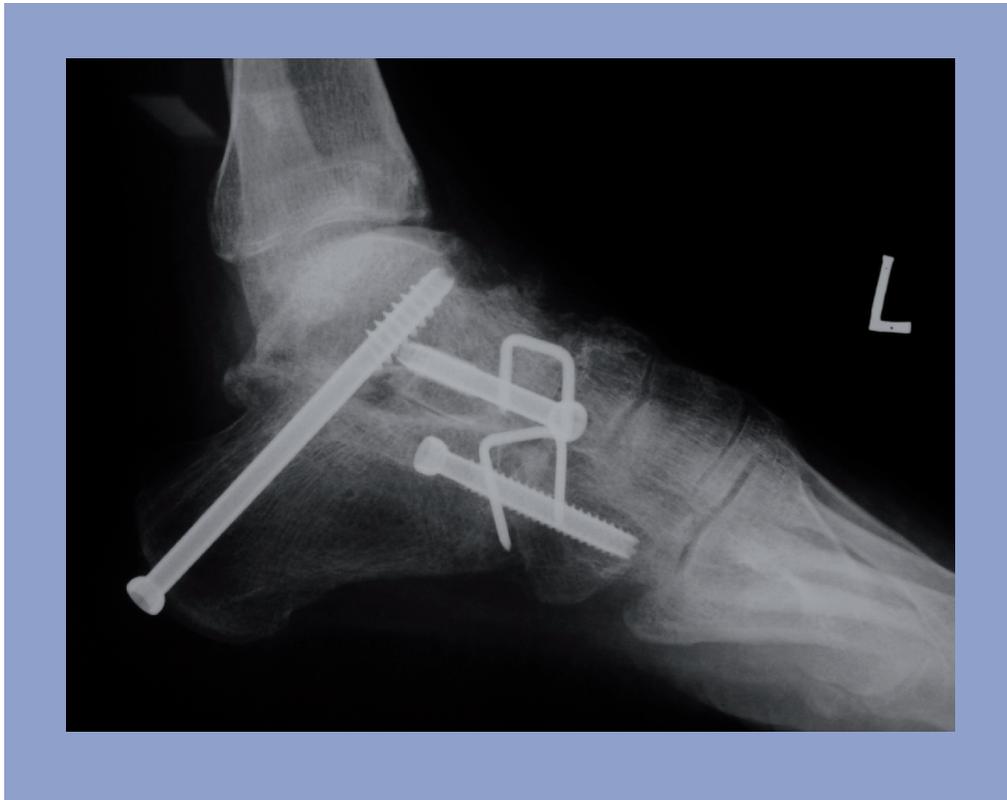


Figure 7. Postoperative radiograph of triple arthrodesis. Reprinted from [1] with permission from Elsevier.

Executive summary

Anatomy, function & etiology of tibialis posterior tendon dysfunction

- Tibialis posterior dysfunction is a common condition causing acquired flatfoot.
- The condition is often misdiagnosed as ‘ankle sprain’ or ‘tendinosis’, leading to a delay in initiation of conservative treatment modalities.
- As the condition progresses the dysfunction leads to progressive flatfoot deformity that eventually becomes fixed, leading to adjoining joint osteoarthritis.

Conservative treatment

- Many patients will respond to conservative treatment with corrective or accommodative orthotics. All patients should undergo a course of conservative treatment prior to considering surgical intervention.
- There is no role for systemic or locally administered steroids.
- Physiotherapy is considered controversial, but a recent randomized, controlled trial advocated the use of ankle–foot orthoses with eccentric and concentric progressive resistive exercises for patients with stage I and II disease. Physiotherapy may reduce symptoms; however, it is not known whether this prevents progression.

Surgical treatment

- If conservative treatments fail, surgery can involve decompression and synovectomy of the tendon, tendon transfers, osteotomies and fusions or combinations of these techniques.
- Good to excellent results can be achieved in 80% or more of patients in the medium term after surgery for tibialis posterior dysfunction.
- Controversy remains regarding the correct surgical interventions, especially concerning stage II disease.
- The long-term results for many of the surgical techniques currently used for treatment of dysfunction of posterior tibial tendon remain unclear; however, the body of literature reporting short- to medium-term results is growing and encouraging.

Future perspective

- Newer techniques, such as arthroereisis, percutaneous radiofrequency ablation, isolated joint fusions and open or endoscopic gastrocnemius recession, are currently being evaluated. These minimally invasive techniques may prevent progression and, thus, prevent patients from requiring hindfoot fusions.

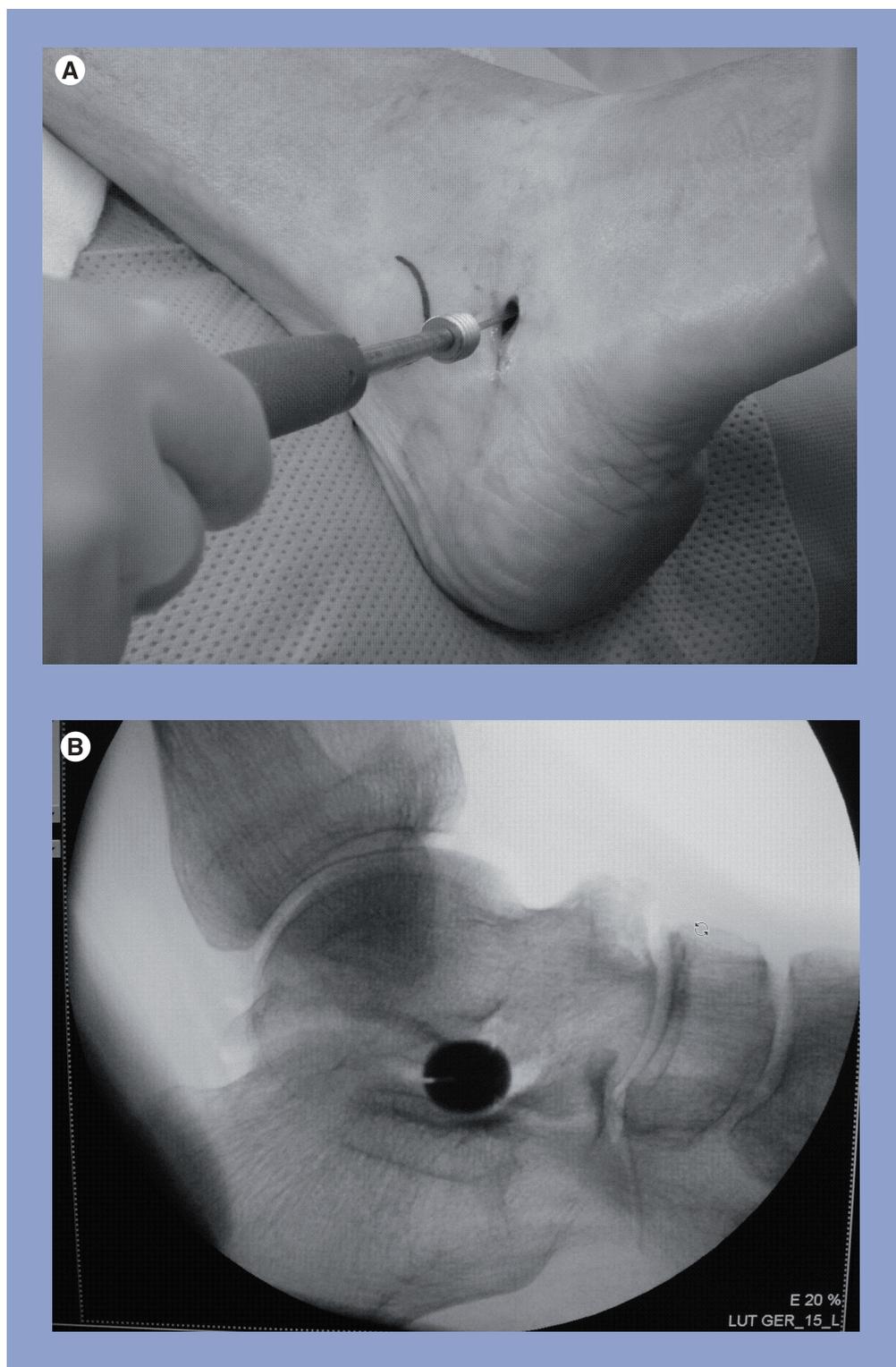


Figure 8. Clinical and radiological images of sinus tarsi arthroereisis. (A) Intra-operative placement of the subtalar arthroereisis through a minimally invasive technique. **(B)** Intra-operative radiograph of the final position of the subtalar arthroereisis. The metallic plug prevents excessive hindfoot valgus.

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