CHAPTER **43**

TIBIALIS POSTERIOR DYSFUNCTION: A Critical Look at Surgical Approaches

Kieran T. Mahan, D.P.M.
Sean Walpole

Tibialis Posterior Dysfunction (TPD) is not an uncommon clinical entity, however, it is often overlooked as a diagnosis. To date there is still no single know etiology of TPD, but several authors have written on the topic. In younger patients it seems that tear or rupture of the tendon may occur secondary to some type of trauma, or in association with inflammatory arthropathy. In older individuals, the onset is usually rather insidious and not related to any single traumatic accident. These older patients tend to have a prior existing flatfoot deformity, seropositive arthritides, degenerative inflammatory process around the tendon, or another systemic manifestation such as obesity, hypertension, and/or diabetes mellitus. A key element which may predispose these individuals to rupture is a zone of hypovascularization in the tendon, posterior and distal to the medial malleolus.[1] Perhaps because the flexor tendons do not have a similar zone of hypovascularity, they rarely rupture.

HISTORY

The first documented case of TPD was published in 1953 by Key.[2] Key reported a partial tear in the posterior tibial tendon which he treated by performing a synovectomy and excision of torn and thickened tendon. Follow-up of the patient a year and a half later demonstrated that the patient suffered from a 10% to 15% disability. In 1969, Kettlekemp and Alexander reported on four cases of spontaneous rupture of the posterior tibial tendon, three of which were preceded by tenosynovitis of the tendon.[3]

Goldner et al. in 1974 attributed progressive flatfoot deformity to TPD and medial plantar ligament trauma or degeneration.[4] Treatment of the nine patients in the Goldner study was by tendon transfer of the FHL and traction of the medial calcaneal ligament, in addition to Achilles tendon lengthening. In 1982, Jahss described ten patients with suspected spontaneous rupture of the posterior tibial tendon (PTT).[5] He described the side to side suturing bridge graft of the FDL to the PTT proximal and distal to the site of injury as adequate treatment. He noted that reconstruction of the foot still allowed some residual rearfoot valgus deformity to occur, and attributed it to the inability to reproduce the natural tightness of the original tendon.

In 1983, Johnson described a number of different surgical procedures performed for TPD.[6] These included end-to-end anastomosis of the tendon, reinsertion of the tendon into the navicular, tendon transfer of the FDL, and for severely arthritic joints, subtalar arthrodesis.

In 1985, Mann and Thompson reported 17 cases of PTT rupture treated with FDL tendon transfers.[7] Citron, in 1985, described three cases of lacerated PTT.[8] One was repaired with an adjacent adherent tendon sheath, another by a residual bridge of peritenon reinforced with a plantaris graft, and a third by an end-to-end anastomoses of the stumps.

In 1986, a classification system was established by Funk et al.[9] The classification is broken into four parts based on the location of the rupture:

Group I - avulsion at the insertion of the navicular
Group II - rupture within the substance of the tendon
Group III - longitudinal partial tear
Group IV - tenosynovitis without visible tear of the tendon

Helal, in 1990, described the Cobb repair for PTT rupture.[10] This involves using half of the longitudinal section of the tibialis anterior muscle down to its insertion, routing it through the medial cuneiform traveling proximally in the posterior tibial tunnel performing a tenodesis of the tendons in an end-weave fashion. Of the six patients in Helal's study treated with a Cobb repair, five were restored to normal.

Janis et al. in 1993 studied 17 patients with a type I or II dysfunction of the PTT and found that 88% had pain relief after surgical repair with a procedure similar to Helal.[11] The study also revealed that the average time after the operation to full activity was three months. Eighty-two of the patients in the study achieved a return to full activity.

In 1994, Teasdall and Johnson studied 19 patients suffering from Type I TPD.[12] These patients underwent surgical synovectomy and debridement of the tendon. Postoperatively, fourteen of these patients (74%) had complete relief of pain.

Myerson et al. in 1995 reported on 18 adult patients who underwent surgical treatment of TPD acquired flatfoot deformity.[13] These patients were repaired with a combination of a calcaneal osteotomy and tenodesis of the FDL. The through-and-through cut of the calcaneal osteotomy is made at a right angle to the lateral border of the calcaneus and is inclined posteriorly approximately 45 degrees to the plantar surface of the hindfoot. No bone is removed and there is no attempt to tilt the tuberosity. The free tuberosity is then translated medially and fixated with a lag screw. The FDL tendon tenodesis is similar to those already reported.

Frankel et al. in 1995 reported repair of a single case of tibialis posterior rupture with a double calcaneal osteotomy.[14] The patient had a pes planovalgus deformity that was too severe for soft-tissue repair, yet there were no secondary arthritic changes noted so arthrodesis would be premature. The surgery entailed a transverse through-and-through cut made through the body of the calcaneus translating the free end medially (Koutsogiannis procedure). The second osteotomy (Evans) is made 1.5 cm proximal to the calcaneocuboid joint where a 1 cm bone graft is impacted into the site. The authors state that at early stage follow-up in the patient, the result has been favorable.

ANATOMY

The deepest muscle of the deep posterior compartment of the leg is the tibialis posterior. Arising from the proximal half of the tibial shaft laterally, the adjacent interosseous membrane, and the proximal half of the posteromedial shaft of the fibula, with contribution also from the intramuscular septum and crural fascia, the muscle courses distally becoming a tendinous band just proximal to the medial malleolus. In the distal aspect of the leg the tendon courses medially, passing deep to the FDL, and through the medial malleolar sulcus. The tendon continues distally in the first compartment of the flexor retinaculum medial to the deltoid ligament, forming fibrous attachment to the distal aspect of the sustentaculum tali.[15] As the tendon approaches its primary insertion into the navicular, it splits into two bands. The larger superficial band attaches to the navicular tuberosity and medial cuneiform. The second, deep band, extends to the bases of the second, third, and fourth metatarsals, the intermediate and lateral cuneiform, and on occasion attachment to the cuboid.

The tibialis posterior muscle has an excellent blood supply in the leg, receiving vascularization from both the posterior tibial artery and the peroneal artery. However, as with most tendons, the PTT lacks a rich supply of blood. To accentuate the malnourishment of the PTT, the PTT is without a mesotenon which supplies vascularization to other tendons in the foot. Frey et al. further elaborated to describe a zone of hypovascularity in the mid portion of the PTT starting one-and-a-half centimeters distal to the medial malleolus running proximal for approximately 14 millimeters.[1] Therefore, anatomical consideration is very important when planning and implementing treatment.

FUNCTION

The tibialis posterior muscle functions at the ankle, subtalar, and the midtarsal joint allowing for plantarflexion of the ankle, supination of the subtalar joint, and supination of the oblique axis of the midtarsal joint. As a stance phase muscle, the muscle begins to fire just after heel strike and continues to fire until just after heel lift of the ipsilateral limb. During the time that the muscle is firing, it provides deceleration of pronation of the subtalar joint and internal rotation of the tibia, acceleration of the subtalar joint supination and leg external rotation, maintenance of the oblique midtarsal joint in supination, and also assists in heel off of the ipsilateral limb in the propulsive phase of gait.

Tibialis posterior is the primary invertor of the foot, and is balanced by the function of the antagonizing peroneus brevis. Attrition or loss of muscle function will not only cause rearfoot

instability inducing an everted position of the calcaneus, but also will allow peroneus brevis to gain mechanical advantage resulting in forefoot abduction. The inability to effectively supinate at the subtalar joint will permit more stress to affect an already unstable oblique axis of the midtarsal joint forcing the forefoot into further abduction. The activity of the Achilles tendon will also maintain the rearfoot in a valgus attitude by passing on the posterolateral side of the subtalar joint. The end result is a pes planovalgus foot that is progressively deforming each day that it is left untreated.

CLINICAL FINDINGS

According to Holmes and Mann, the average patient who suffers from TPD is a female over the age of forty.[16] These patients present with the insidious onset of moderate to severe pain localized to the medial ankle or medial arch of the foot. Points of maximum tenderness are found along the course of the tendon from just proximal to the medial malleolus down to the insertion into the navicular. Cases of acute onset are seen primarily in younger patients. Both acute and insidious onset are aggravated by extensive standing, more so than walking, and are associated with loss of the medial longitudinal arch.

Physical examination reveals a valgus hindfoot and abducted forefoot. Swelling may be noted in the medial rearfoot accompanied by tenderness and mild calor. The patient will also demonstrate the "too many toes sign" and a fullness of the medial arch (Fig. 1). However, the most effective test to determine dysfunction is the inability

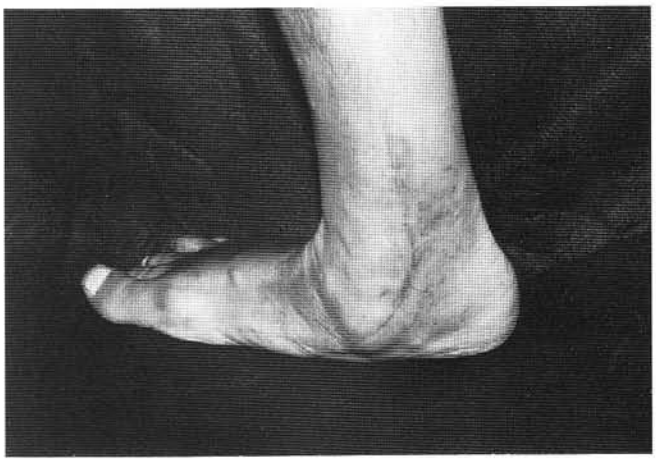

Figure 1. Medial view of foot and ankle, demonstrating severe talar bulge. This patient had already had a flexor hallucis longus transfer, which failed.

to perform the single-heel-raise test. Patients with TPD become skillful at substituting with tibialis anterior (TA). Evaluation of tibialis posterior strength requires eliminating the effect of TA. This can be done best by plantarflexing the foot prior to having the patient resist an eversion force. Frankel et al.[14] summarized the stages of TPD seen clinically and radiographically as described by others (Table 1).

Table 1

SUMMARY OF STAGES OF TPD

	CLINICAL SIGN	RADIOGRAPHIC SIGNS
Stage I -	Pain and edema along tendon Possible weak heel-raise test Relief with rest	No changes
Stage II -	Increased pain Minimum relief with rest Medial arch begins to flatten "Too many toes" sign	Forefoot abducted Subluxed T-N joint Decreased arch height MRI shows tendon irregularity
Stage III -	Increased forefoot abduction Increased heel valgus No rearfoot inversion Minimal heel rise Pain at sinus tarsi Difficulty walking *foot still reconstructible	Increased forefoot abduction Minimal arch height Arthritic changes in tarsal joint MRI shows partial or complete rupture or avulsion
Stage IV-	End stage Rapid progress to pes planus *only option is fusion	Rapid progress of DJD

As Frankel et al. have summarized, as the deformity progresses, the lateral side of the foot, especially the area of the sinus tarsi, sustains pain as a result of excessive pronation followed by arthritic changes affecting the tarsal joints.[14] Therefore, it is imperative to treat the deformity early to prevent further degeneration (Fig. 2).

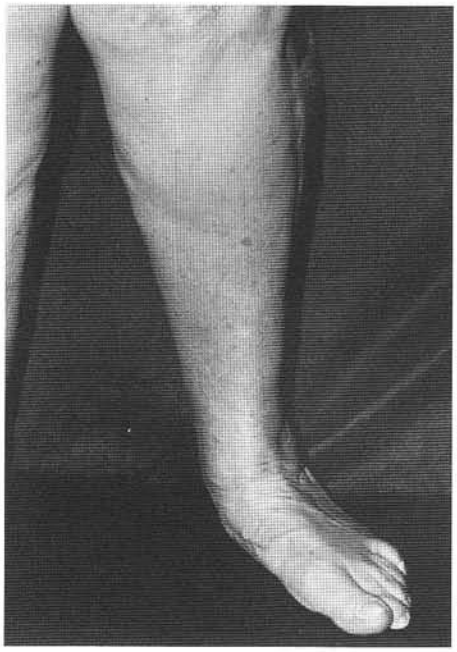

Figure 2. Tibialis posterior rupture with peroneal spasm. Note the genu valgum, which is a common clinical finding accompanying foot collapse associated with tibialis posterior dysfunction.

INVASIVE & NON-INVASIVE TESTING

Although a diagnosis of TPD can be made clinically, there are a number of techniques that can be used to help confirm a diagnosis of TPD and/or assist in the evaluation of the compensatory effects on the foot. These include plain radiographs, tenography, computed tomography (CT), and magnetic resonance imaging (MRI). According to Hogan, anterior-posterior and lateral view radiographic findings can not be diagnostic.[17] However, plain radiographs can be helpful when the TPD is due to avulsion of the navicular. Jahss also concluded that radiographs are of little diagnostic value.[5] Jahss[5] and Hogan[17] state that tenography may be of value, but it is often very difficult when the tendon can not be palpated or adhesions obliterate the synovial space. Often, when injecting the contrast media above the medial malleolus, roentgenograms demonstrate an outline of the FDL instead of the TP because the upper portion of the TP retracts posterolateral to the FDL.

CT and MRI are both choices for further study in TPD. MRI, because of its superiority in assessing soft-tissue integrity, is the study of choice (Figs. 3A, 3B). Kerr et al. concluded that CT is not as useful as MRI in evaluating tendon injuries, because it is often unable to demonstrate a longitudinal tear or differentiate tendon from adjacent fluid.[18]

TREATMENT

Conservative

Conservative care has a limited but important role in management of TPD. For young, active patients there is a clearer role for surgical management in order to restore full power and function. For

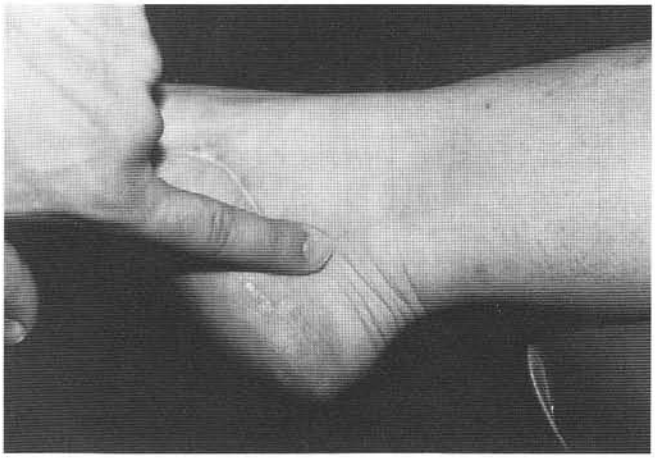

Figure 3A. This patient had two prior surgeries before this examination, a heel spur surgery and a Kidner procedure. Note the area of greatest tenderness.

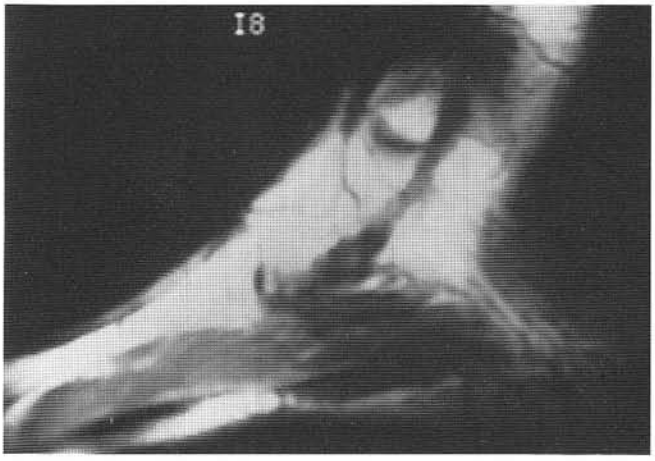

Figure 3B. Sagittal MRI demonstrating attenuation of tendon correlating with point of maximum tenderness.

patients with limited functional demands, or for patients who are not medically able to have surgery, conservative care may be the only option. Many patients with TPD are older and some of these older patients may not have sufficient functional demand or stamina to warrant surgery.

Conservative care can consist of nonsteroidal anti-inflammatories, casting, strapping, ice, physical therapy, and other modalities in the acute stage. Chronic TPD can be treated with bracing, shoe modifications, and specially designed orthotics such as those incorporating a Kirby scive and high medial flange. The type of care needed is dependent upon the patient's level and severity of deformity, age, weight, and functional demand.

Surgical

Rosenberg et al. state that chronic rupture of the PTT can be classified into three categories, all of which should be treated surgically[19] Type I is a partial rupture of the tendon associated with increased girth and focal areas of fiber rupture, degeneration, and repair. Type 2 is a more severe partial rupture in which the tendon is markedly attenuated in size. Type 3 is a complete rupture with a gap and retraction of the proximal and distal portions of the tendon.

In the past, the definitive treatment for severe TPD was considered to be triple arthrodesis. Treatment has changed a great deal as the entity has become more commonly recognized and our experience has increased. The surgical treatments can be broken down into three categories. Soft tissue procedures include tendon repair with or without graft, the Cobb procedure, flexor transfer into the navicular, and a Young's teno-suspension. A second category is a group of procedures that can be categorized as osteotomies and limited fusions. This group includes the Evans, Koutsogiannis, and reverse Dwyer calcaneal osteotomies, navicular-cuneiform, and Lapidus fusions (Figs. 4A-4D). Arthroereisis would fall into this category as well. The third category is major fusion procedures such as triple arthrodesis, subtalar fusion, and talo-navicular fusion (Fig. 5).

The category I procedures are indicated for acute ruptures, and those that are recent and without significant deformity (either compensatory or primary). Category II is indicated in those patients with more severe deformity such as abductus deformity at the midtarsal joint, or sag at the naviculo-cuneiform joint. The third category is reserved for patients with severe deformity and degenerative joint disease. Tendo Achillis lengthening may need to be performed in any of the categories.

The surgeon must learn not to rely too heavily on visual inspection of the tendon. Conti et al.[20] have demonstrated convincingly that surgeon grading based on intraoperative inspection is a poor correlate with outcome. MRI classification proved to be much more reliable as a positive correlate with outcome. The combination of clinical, radiographic, gait, and MRI information is usually necessary for best determining what type of repair is necessary.

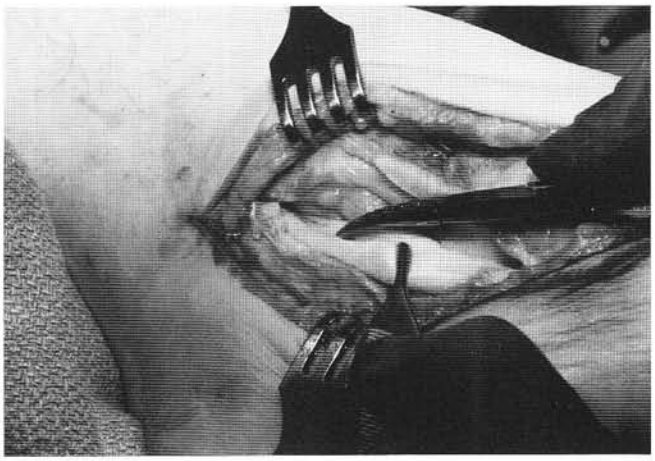

Figure 4A. Clinical photos demonstrating surgical repair. Note tibialis posterior with longitudinal tear and hypertrophy.

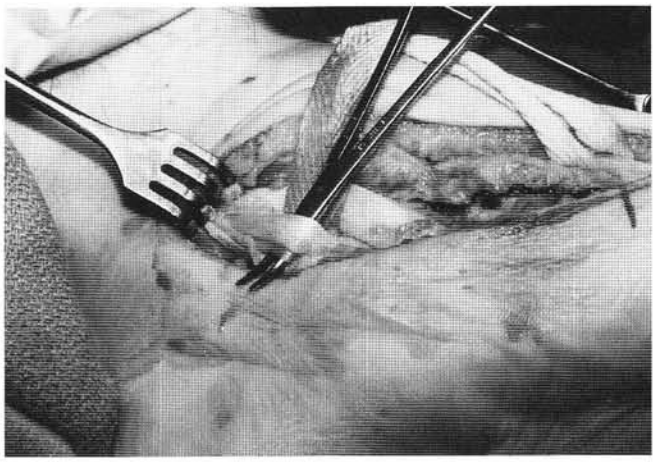

Figure 4B. Identification of flexor digitorum longus for transfer.

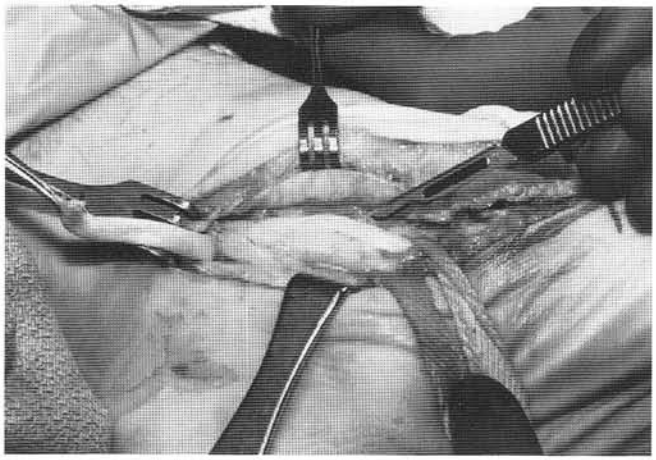

Figure 4C. Debridement of tibialis posterior.

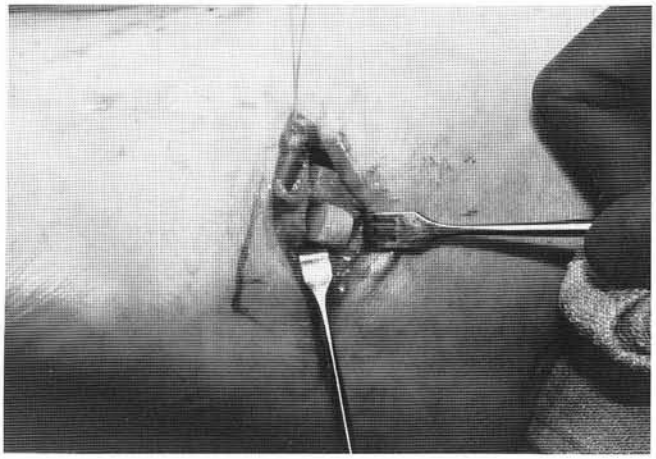

Figure 4D. Bone graft inserted for Evans calcaneal osteotomy.

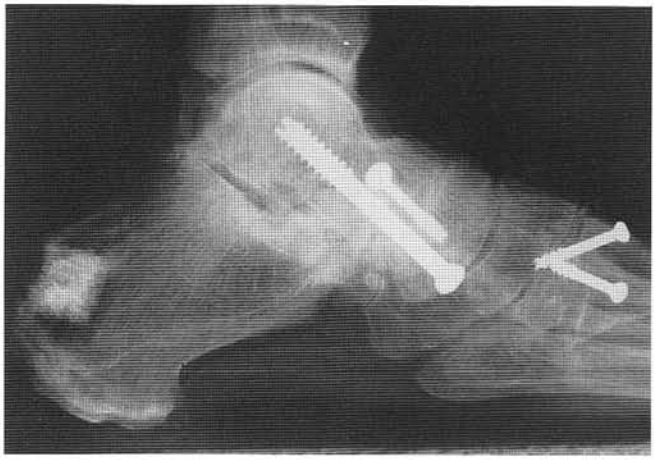

Figure 5. Postoperative lateral view of talo-navicular and Lapidus fusion for TPD in a patient with rheumatoid arthritis. Note hydroxyapatite packing for calcaneal bone graft applied to talo-navicular fusion.

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