Posterior Tibialis Tendon Dysfunction: Overview of Evaluation and Management

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educational objectives

As a result of reading this article, physicians should be able to:

1. Recognize posterior tibialis tendon dysfunction and begin to include it in differential diagnoses.

2. Recall the basic anatomy and pathology of the posterior tibialis tendon.

3. Assess a patient for posterior tibialis tendon dysfunction with the appropriate investigations and stratify the severity of the condition.

4. Develop and formulate a treatment plan for a patient with posterior tibialis tendon dysfunction.

ABSTRACT

The posterior tibialis is a muscle in the deep posterior compartment of the calf that plays several key roles in the ankle and foot. Posterior tibialis tendon dysfunction is a complex but common and debilitating condition. Degenerative, inflammatory, functional, and traumatic etiologies have all been proposed. Despite being the leading cause of acquired flatfoot, it is often not recognized early enough. Knowledge of the anatomical considerations and etiology of posterior tibialis tendon dysfunction, as well as key concepts in its evaluation and management, will allow health care professionals to develop appropriate intervention strategies to prevent further development of flatfoot deformities. [Orthopedics. 2015; 38(6):385-391.]

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The posterior tibialis is a muscle of the lower limb that plays several key roles in the ankle and foot: it plantarflexes the ankle, inverts the sub-talar joint, and is integral to the maintenance of the medial longitudinal arch of the foot. Although exact epidemiological data are unavailable, posterior tibialis tendon dysfunction (PTTD) is a common and well-established source of pain and walking disabilities.\(^1,2\) It is also the leading cause of acquired flatfoot and can lead to debilitating foot and ankle dysfunction.\(^3,4\) However, the dysfunction is often not recognized early enough,\(^5\) is under-diagnosed, and is not well understood by the majority of health care professionals. Also, the amount of literature on this topic is minimal, and the most recent review of this dysfunction was conducted in 2004.\(^5,6\) The current review aims to concisely summarize the available literature on PTTD and the recent updates in the diagnosis and treatment of this dysfunction.

**ANATOMY AND FUNCTION**

The posterior tibialis is the deepest of the 3 muscles found within the deep posterior compartment of the calf. It originates from the interosseous membrane and the adjoining proximal surfaces of both the tibia and fibula. The muscle descends between the flexor hallucis longus and flexor digitorum longus, forming its tendon in the distal third of the calf before passing posterior to the medial malleolus and then changing its direction to enter the plantar surface of the foot. It is here that the tendon demonstrates several changes in its tissue structure: it experiences a region of relative hypovascularity due to a watershed pattern of blood supply\(^6,7\) and also exhibits increased fibrocartilage.\(^8,9\) The posterior tibialis eventually inserts via 3 main tendinous insertions: an ante-rior insertion onto the navicular tuberosity and 2 other insertions that extend across the plantar aspects of the remaining tarsal bones and the bases of the middle 3 metatarsals.\(^10,12\)

The position of the posterior tibialis tendon medial to the axis of the subtalar joint and posterior to the axis of the ankle joint facilitates powerful supination of the hindfoot and plantarflexion of the ankle. The posterior tibialis also plays a role in stabilizing the medial longitudinal arch of the foot: its contraction during normal gait produces inversion at the subtalar joint and locks the transverse tarsal joint in place, thus supporting the medial longitudinal arch and preventing it from collapsing.\(^5\)

**ETIOLOGY AND PATHOGENESIS**

The etiology of PTTD includes inflammatory, degenerative, functional, and traumatic processes. Inflammatory causes, often secondary to systemic inflammatory diseases like lupus and rheumatoid arthritis, appear to be more common in young patients.\(^10,13\) Posterior tibialis tendon dysfunction secondary to chronic overuse and subsequent tendon degeneration has been noted to occur more frequently in late-middle-aged women who are obese.\(^10,13\) Functional causes of PTTD, such as abnormal biomechanical forces due to excessive pronation\(^14\) or anomalous anatomy, have also been described. Acute trauma is rarely the cause of tendon dysfunction or rupture.\(^5\) Other risk factors include ligamentous laxity, diabetes mellitus, hypertension, and corticosteroid therapy.\(^10,15-18\)

Posterior tibialis tendon dysfunction most commonly occurs in the perimalleolar region and tends to involve tenosynovitis (tendon degeneration) rather than tendinitis (tendon inflammation); the posterior tibialis tendon undergoes fibrotic changes after being subjected to repeated minor trauma.\(^19\) Although the precise underlying mechanisms that predispose the tendon to degeneration remain unclear, it has been postulated that the zone of hypovascularity and the abnormal mechanical forces experienced by the tendon as it acutely changes direction behind the medial malleolus may contribute to the pathogenesis.\(^5\)

In PTTD, loss of posterior tibialis function results in collapse of the medial longitudinal arch and increased strain in the medial structures of the foot. Progressively, there is attenuation of the medial ligaments: the spring ligament, which connects the anterior margin of the sustentaculum tali to the plantar surface of the navicular, gradually fails with cyclical loading, promoting a flatfoot deformity. The deltoid ligament also eventually becomes insufficient, allowing the talus to tilt into valgus. As the hindfoot goes into valgus, the Achilles tendon becomes an evertor due to its position lateral to the axis of the subtalar joint and, with time, frequently becomes contracted as well, resulting in an equinus deformity.

**CLINICAL FEATURES**

Symptoms of PTTD are often present for months or years before patients decide to seek help. On presentation, patients often describe an insidious onset of pain and swelling along the course of the tendon in the medial ankle and foot. Occasionally, the pain may radiate into the medial proximal calf. Over time, patients may notice changes in the shape of their foot, with gradual collapse of the medial longitudinal arch and increasing hindfoot valgus. The soles of their shoes may show signs of abnormal wear on the medial side, and they may report difficulty standing on their toes due to pain and weakness. Walking aggravates the pain, and participation in sports becomes almost impossible.\(^13,20\) As the dysfunction of the tendon progresses, patients often report that pain shifts laterally as the fibula begins to impinge against the calcaneus.\(^13\) The medial pain often disappears.

Examination should begin with inspection from above and behind the standing patient’s feet. In PTTD, the examiner will note increased valgus angulation of the hindfoot and abduction of the forefoot. These features may present as the “too many toes” sign, in which more toes are visible on the lateral side of the affected foot when viewed from behind if the forefoot is pathologically abducted (Figure 1).\(^21\) Next, the patient should perform the single-heel-raise test, which reflects the
function of the hindfoot and is a sensitive indicator of PTTD. This is done by asking the patient to attempt to rise onto the ball of his or her affected foot while keeping the contralateral foot lifted off the ground. The test is then repeated with the normal foot for comparison. In this test, the affected hindfoot will either remain in valgus abduction during the heel raise due to the failure of posterior tibialis to invert it or the patient will experience difficulty rising onto the forefoot due to pain. It may be necessary for the patient to perform repeated heel raises to reveal subtle weakness of the tendon. Another useful test is the first metatarsal rise sign, first described by Hintermann and Gächter. The patient is required to stand full weight bearing on both feet. The examiner then externally rotates the shin of the affected foot with one hand. In PPTD, the head of the first metatarsal will be lifted off the floor.

The rest of the examination can be conducted with the patient seated. The posterior tibialis tendon should be palpated through its course along the medial side of the ankle to assess for its integrity and for the presence of any tenosynovitis, which may manifest as tenderness or swelling. The lateral side of the foot should also be palpated because tenderness in the subfibular region may be an indication of calcaneofibular impingement. The strength of the posterior tibialis tendon can also be evaluated at this point. The foot is first held by the examiner in slight plantarflexion and eversion to isolate the posterior tibialis from the synergistic action of tibialis anterior. The patient is then asked to invert and further plantarflex the foot against resistance while the examiner palpates for the posterior tibialis tendon to determine its integrity and the site of maximum tenderness. Then, the mobility of the ankle and the subtalar joint should be carefully assessed because the degree of passive hindfoot inversion will affect the method of treatment. Movement at the subtalar joint will become increasingly limited with worsening dysfunction, and eventually the hindfoot will fix in a valgus deformity. The forefoot and midfoot compensate for this by progressively adopting a supinated position, which is best appreciated with the heel in a neutral position. This is a critical part of the examination because a fixed supination deformity of the forefoot is an important consideration in the subsequent selection of treatment, as well as the design of any subsequent foot orthosis. The Achilles tendon should also be assessed for contracture, which is often associated with chronic hindfoot valgus due to the Achilles tendon adopting an abnormal position lateral to the axis of the subtalar joint, with the tendon shortening over time.

**INVESTIGATIONS**

**Plain Radiographs**

Plain radiographs are not required for the diagnosis of PTTD. However, abnormal alignment and bony changes, which can be appreciated on plain radiographs, are important in helping to determine the degree of deformity and hence the selection of treatment. Anteroposterior and lateral weight-bearing radiographs of the foot, as well as mortise views of the ankle joint, should be ordered. Radiographs of early dysfunction may appear normal. However, several features may become prominent as dysfunction progresses. One of these is the collapse of the longitudinal arch on a lateral weight-bearing radio-

Figure 1: “Too many toes” sign on the right foot.

Figure 2: Pes planus with collapse of the longitudinal arch.

**Ultrasound**

Ultrasound is a sensitive and cost-effective investigation to assess deformities of the posterior tibialis tendon. The normal posterior diameter of the tendon ranges from 4 to 6 mm, and it has a hyperechoic appearance. Thickening and...
heterogeneous hypoechoic texture is an indication of low-grade posterior tibialis tendon injury. Other clues to the dysfunction include thickening of the peritendinous soft tissues or thinning, splitting, or rupture of the tendon. A view of the surrounding soft tissues is also used to evaluate the dysfunction. Large amounts of fluid would be present around the tendon in cases of tenosynovitis. Classic signs of tenosynovitis include a target sign on the transverse view and hypoechogenic trim on the longitudinal view. It is now widely recognized that ultrasound is a useful and noninvasive methodology to investigate PTTD to the extent that it has even been recommended as a tool for routine screening for early dysfunction. The sensitivity and specificity of ultrasound is similar to that of magnetic resonance imaging (MRI). In addition, ultrasound is relatively cheaper, may be more convenient, and is easily accessible. However, this technique is highly operator dependent and is best performed by an expert musculoskeletal sonographer.

**Magnetic Resonance Imaging**

Magnetic resonance imaging is recognized as the gold standard investigation to assess PTTD. A classification of tendon tears based on MRI results has been developed by Conti et al. Type I partial tear features include hypertrophy, rounding, increased intrasubstance signal, and longitudinal splits. Magnetic resonance imaging features of a type 2 partial tear are tendon atrophy to less than the size of the adjacent flexor tendons, wider longitudinal splits, and intramural degeneration. Absence of the tendon is classified as a type 3 complete tear. There is usually more diffuse swelling and uniform degeneration, replacement of the tendon with fibrous tissue, or a complete gap. In cases of paratendinitis, fluid is present around the tendon, but there is no evidence of a tear.

Magnetic resonance imaging is considered the best imaging methodology due to its soft tissue resolution and multiplanar imaging ability. This modality of imaging is especially good for detection of bony edema, bony changes, and malalignment. It has been demonstrated that MRI has a sensitivity of 95%, specificity of 100%, and accuracy of 96% in diagnosing PTTD. However, compared with other imaging modalities, MRI is relatively costly and may not be as easily accessible. It is also difficult to obtain good-quality images from uncooperative and claustrophobic patients due to motion artifacts.

**CLASSIFICATION**

Posterior tibialis tendon dysfunction can be classified into 4 stages depending on the extent of the deformity. Stage I includes paratendonitis. There is evidence of tendon degeneration but the tendon remains at a normal length. Patients can experience ankle pain, swelling, and mild weakness. Stage II includes features of tendon elongation or tear. Patients will have difficulty performing a single-heel raise on the affected side. There is subtle flatfoot deformity on weight bearing, but the hindfoot remains flexible. Stage III is characterized by a rigid flatfoot deformity with a fixed hindfoot. Stage IV was added by Myerson and involves early degenerative changes of the ankle that occur as a result of the valgus tilt of the talus in the ankle mortise.

**TREATMENT**

The treatment of PTTD is based on the severity and stage of the dysfunction and includes both nonoperative and operative treatment. In the majority of patients, symptoms can normally be relieved with appropriate conservative treatment. The first step in all patients is to rest the tendon through minimizing or ceasing activity that worsens the pain by switching to low-impact exercise (eg, swimming and bicycling), which is generally well tolerated by patients. Patients should also be encouraged to ice the most painful area of the posterior tibialis tendon 3 to 4 times daily and especially after activity by applying cold packs to decrease swelling and inflammation, but they should be advised not to apply ice directly to the skin. Analgesia can normally be achieved through the judicious use of simple analgesics, including paracetamol and non-steroidal anti-inflammatory drugs such as ibuprofen and naproxen. Non-steroidal anti-inflammatory drugs help reduce pain and inflammation and can be used as prophylaxis prior to exercise and activity. However, particular care should be taken by physicians to ensure patients who are at high risk of gastrointestinal bleeding, renal failure, and polypharmacy are appropriately medicated.

If the deformity progresses, orthotics and braces may benefit patients by modifying the biomechanical stressors on the
tendon by correcting the flexible component of the deformity and providing arch support. Neville and Houck\textsuperscript{29} reported that reducing flatfoot deformity while allowing ankle movement may minimize the progression of PTTD. For patients with associated mild pes planus, an over-the-counter orthotic may prove adequate in relieving pain related to the posterior tibialis tendon. In patients with associated moderate-to-severe pes planus, a custom-fitted orthotic may prove necessary, such as a molded ankle-foot orthosis. Semi-rigid, rigid, and articulated foot-ankle braces have been used in the treatment of PTTD. These can help maintain the heel in neutral alignment and support the medial longitudinal arch of the foot in patients with stage I and II deformity to reduce forces acting on the posterior tibialis tendon.\textsuperscript{13} Patients with stage III deformity and pain can benefit from a rigid-type brace; articulated braces should be reserved for patients with asymptomatic stage III deformity.\textsuperscript{5} Any brace or orthosis that is used should fit well and have a soft, protective surface to avoid causing abrasion and ulceration of the skin.

Along with an orthosis, a regime of aggressive physiotherapy and rehabilitation with structured exercise has also been shown to be effective in treating patients with early PTTD. Chao et al\textsuperscript{3} reported that 67\% of patients treated with an orthotic alone had good-to-excellent subjective and functional results, and, in a more recent study, Alvarez et al\textsuperscript{29} reported that 83\% of patients with early PTTD who underwent an orthosis and exercise program had successful subjective and functional outcomes, with 11\% requiring surgery. Lin et al\textsuperscript{40} reported that after 7 to 10 years of follow-up in patients with stage II PTTD who underwent nonoperative treatment, 69.7\% avoided surgery and remained brace free, with 60.6\% of patients being satisfied with their outcome. Kulig et al\textsuperscript{11} recently performed a randomized, controlled trial that showed patients who wore an orthosis, stretched, and underwent an eccentric progressive resistive exercise program had better outcomes than patients who only wore an orthosis and stretched. Rehabilitation may be home based or center based; Bek et al\textsuperscript{12} reported both to be similarly effective in relieving pain and improving function in patients with stage I to III PTTD.

Operative management of PTTD is normally reserved for patients in whom a trial of at least 6 months of conservative therapy has failed or who have progressive progression of function. Operative management is normally based on the stage of the deformity and location of the pain. A study by O’Connor et al\textsuperscript{13} found that patients with a higher body mass index, longer symptom duration, prior cortisone injections, and prior orthotic use were more likely to require surgery. Commonly performed procedures include gastrocnemius recession and lengthening of the Achilles tendon, open and endoscopic tenosynovectomy, tendon transfer, calcaneal osteotomy, and arthrodesis.

Stage I deformity can normally be managed conservatively, but, if that fails, tenosynovectomy, tendon relief, and debridement should be performed. Teasdall and Johnson\textsuperscript{34} reported that 74\% of patients with stage I PTTD who underwent a posterior tibialis tendon release, tenosynovectomy, and debridement reported complete pain relief, and 84\% had return of function of the posterior tibialis tendon. Chow et al\textsuperscript{35} found tendoscopic debridement to be beneficial for 6 patients with stage I PTTD, with no patient progressing to stage II deformity.

Stage II deformity can be treated with heel cord lengthening, tendon transfer, and calcaneal osteotomy because the deformities are still flexible (Figure 5). The transferred tendon is normally the flexor digitorum longus. Pomeroy and Manoli\textsuperscript{36} initially reported that 17 patients who underwent heel cord lengthening, flexor digitorum longus tendon transfer, lateral column lengthening, and medial displacement calcaneal osteotomy had significantly improved foot rating scores. Myerson and Corrigan\textsuperscript{23} reported that 97\% of 129 patients with stage II PTTD who underwent a calcaneal osteotomy and flexor digitorum longus tendon transfer to the navicular experienced pain relief, 94\% showed improvement of function, and 84\% were able to remain orthotic free. Wacker et al\textsuperscript{37} reported similar results, with 43 of 44 patients who underwent calcaneal osteotomy and flexor digitorum longus tendon transfer for stage II PTTD reporting improvements in function and pain after 3 to 5 years. Fayazi et al\textsuperscript{38} reported that, at an average of 35 months, 96\% of patients with stage II PTTD who underwent tendon transfer and calcaneal osteotomy maintained their improvement.

The flexor hallucis longus can also be transferred. Sammarco and Hockenbury\textsuperscript{39} reported no patients complaining of donor deficit, with good clinical results and high patient satisfaction rate. Alternatively, a Cobb reconstruction using a split tibialis anterior musculotendinous graft and involving a bone tunnel in the navicular rather than medial cuneiform can be performed. Parsons et al\textsuperscript{40} reported that 29 of 32 patients who underwent this procedure for stage II PTTD had good outcomes at 5 years. Haeseker et al\textsuperscript{41} recently reported that patients with stage II PTTD who underwent calcaneal osteotomy had significantly better outcomes than patients who underwent a calcaneocuboid distraction arthrodesis.

Treatment of stage III and IV PTTD is by either subtalar, double, or triple ar-
thorodes, all of which are supported by the literature. In patients with a fixed deformity but a transverse tarsal joint that remains mobile and pain free, isolated subtalar fusion is indicated. Tibiocalcaneal arthrodesis and plantar arthrodesis are commonly used to treat stage IV deformity. However, up to 50% of patients with stage III and IV deformity may have persisting pain after arthrodesis.

**CONCLUSION**

The posterior tibialis tendon is an important source of support of the medial longitudinal arch of the foot, and PTPTD can lead to debilitating pain and foot and ankle dysfunction but may be misdiagnosed or mismanaged as ankle arthritis or flatfoot. Increased awareness of this dysfunction by health practitioners may lower the threshold for organizing appropriate investigations and referrals for patients with PTPTD. This will lead to earlier recognition and diagnosis of the deformity, subsequently leading to earlier nonoperative or operative intervention for early-stage PTPTD, which can improve subjective and objective patient outcomes in terms of improved pain and function and prevent progression of the deformity to late-stage PTPTD, which can be difficult to manage.

**REFERENCES**

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