CHAPTER 40

TIBIALIS POSTERIOR DYSFUNCTION: Conservative Treatment Considerations

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Tibialis posterior dysfunction (TPD) is a relatively common disorder encountered in podiatric practices. It is often misdiagnosed by several physicians and/or under-treated prior to the patients's presentation to the office. Treatment of early tibialis posterior pathology is often not initiated due to the delay in proper diagnosis.

Tibialis posterior dysfunction occurs as an insidious course of repetitive trauma and overuse, resulting in inflammation, degeneration, and eventual failure of the tendon. The sequential pathologic changes frequently continue until a unilateral, severe flatfoot with marked rearfoot eversion and forefoot abduction results.

Surgical treatment of TPD has been the mainstay of therapy over the past decades. Recently, conservative treatment has gained attention as an alternative to surgical intervention, for poor surgical candidates, and as a preoperative treatment trial. As with most progressive pathologies, conservative treatment of TPD is most successful when initiated in its early stages. Conservative treatment options for early and late stage TPD will be discussed.

ANATOMY

The tibialis posterior muscle originates from the superior aspect of the posterior surface of the tibia, the medial aspect of the posterior surface of the fibula, the posterior surface of the interosseous membrane, and the deep transverse septum. The muscle lies in the deepest portion of the posterior compartment of the leg between the flexor digitorum longus and the flexor hallucis longus. The tendon crosses deep to the flexor digitorum longus tendon as it courses medially. It lies in its own synovial tendon sheath as it courses posterior to the medial malleolus in the retromalleolar groove.

The tibialis posterior tendon passes deep to the flexor retinaculum, which prevents bowstringing of the tendon, and lies superficial to the deltoid ligament as it enters the foot. As the tendon passes inferior to the plantar calcaneal navicular ligament, it divides into two slips. The major and more superficial slip inserts into the tuberosity of the navicular. A posteriorly directed slip may insert into the sustentaculum tali of the calcaneus, while a more distal slip may continue from the navicular, to insert on the medial cuneiform and occasionally the base of the first metatarsal. The deeper, smaller slip courses a groove in the undersurface of the navicular, to insert on the plantar surfaces of the bases of the central three metatarsals and the intermediate cuneiform. There are many described anomalies and variations of insertions of this tendon.[1-4]

BIOMECHANICS

The tibialis posterior muscle is the most powerful subtalar joint supinator of the foot, and strong supporter of the medial longitudinal arch.[5] The tibialis posterior tendon is ideally located medial to the subtalar joint (STJ) axis and posterior to the ankle joint axis to invert the foot at the ankle. The muscle also acts as the major antagonist for the peroneus longus and peroneus brevis muscles.[4,6] The tibialis posterior tendon has two effective pulleys along its course. The first, at the medial malleolus, provides an effective angle of pull on the subtalar and ankle joints. The second, at the navicular tuberosity, provides an effective angle of pull on the oblique midtarsal joint in the supination direction.[4] The tibialis posterior tendon insertions at the cuneiform, and the central three metatarsal bases serve to stabilize the lesser tarsus.[7]

The primary site of action of the tibialis posterior tendon is a subject of ongoing debate. The talonavicular and calcaneocuboid joints,[6,8] the subtalar joint,[4] and the "rearfoot",[3] have all been cited as sites of primary action. The tibialis posterior tendon most likely functions at all of the above sites. It is a stance phase muscle that contracts from heel contact to just after heel lift. At

heel contact, the tibialis posterior muscle contracts to decelerate subtalar joint pronation and internal leg rotation. Through midstance phase of gait, it supinates the subtalar joint and externally rotates the leg. It also assists in heel lift by its ankle joint plantar flexion force, and by allowing the gastrocnemius-soleus complex to lift the heel while the tibialis posterior tendon stabilizes the rearfoot.[4]

PATHOLOGY

Different classification systems have been developed to describe the associated pathology with TPD. Funk et al.[8] identified four types of lesions in 19 patients with the clinical diagnosis of tibialis posterior dysfunction that underwent surgical exploration. Group I consisted of avulsion of the tendon at the insertion; Group II consisted of tendons with mid-substance ruptures; Group III consisted of tendons with an in-continuity tear; and Group IV consisted of tendons with tenosynovitis only and no tear.

Johnson and Strom[9] proposed a staging system. Stage I demonstrates normal tendon length with mild weakness on single heel rise, and no significant rearfoot deformity. Stage II demonstrates an elongated tendon, increased pain, "too many toes sign," marked weakness and difficulty with single heel rise, and a flexible valgus position of the rearfoot. Stage III shows elongation of the tendon, rigid valgus of the rearfoot, pain now over the sinus tarsi, no rearfoot inversion on single heel rise or inability to rise on the ball of the foot, and severe flatfoot with "too many toes sign."

Mann[10] suggested that the mechanism for the progressive unilateral flatfoot results from a weakening of the tibialis posterior muscle, which gives the peroneus brevis muscle a much greater mechanical advantage to pronate the foot. This persistent pronation, along with a loss of ligamentous support, eventually collapses the arch, everts the rearfoot, and abducts the forefoot.

The pathologic changes that occur with TPD include rupture, hypertrophy, degeneration, cystic changes, and tenosynovitis.[11] The physiologic and pathologic changes occur within the tendon as a result of microtears, inflammation, and rupture that ensues as a consequence of repetitive loading. During normal activity, a tendon probably does not exceed a tensile load of 25% of its physiologic maximum. If tendon fibers stretch no more than 40% of their length, the original wave pattern of tendon fibers will return. If the tendon fibers are stretched more than 40% of their length, the fibers are stressed and may begin to fatigue and tear.[4]

If the overuse and/or abnormal biomechanics of the tibialis posterior tendon continues, and the tendon lengthens or ruptures, the plantar ligaments of the midtarsal joint may also fail. The talus will plantarflex and adduct and the talar head will become prominent between the calcaneus and the navicular. The calcaneus will evert and the unopposed peroneus muscles will abduct the forefoot. Eventually, the adult, unilateral, acquired flatfoot with forefoot abduction, flexor substitution stabilization, and an apropulsive gait develops.[1,9,12]

ETIOLOGY

There are multiple causative factors of TPD. Systemic causes include the arthritides, systemic lupus erythematosus, sero-negative spondyloartrhopathies, and collagen diseases.[6]

Mueller also described and classified local etiologies as follows:

> Type I, Direct: direct injury to the tendon, resulting in dysfunction.
>
> Type II, Pathologic rupture: tendon degeneration associated with systemic conditions such as rheumatoid arthritis.
>
> Type III, Idiopathic rupture: etiology unknown.
>
> Type IV, Functional rupture: the tibialis posterior tendon is intact but not functioning well.

The concept of functional rupture is important. Abnormalities of function that result in a pronated foot may predispose a patient to TPD. The majority of TPD patients have an intact tendon that may be hypertrophic, but functions as if there is a complete rupture of the tendon. The loss of function of the tendon is presumed to occur from the tendon healing in a lengthened position. The hypertrophy occurs from scarring in the healing process of the tendon.[13]

Iatrogenic causes of TPD may include tendon trauma secondary to surgery at the medial ankle region, or repetitive steroid injections into the

tendon or tendon sheath. Steroid injections into and around the tibialis posterior tendon are cautioned against by many authors.[1,12,14,15] Mueller, in his 1984 report of 25 cases of ruptures and lacerations of the tibialis posterior tendon, used steroid injections along the tendon, but with little relief.[16] Ford and DeBender reported on 15 various ruptured tendons in 13 patients injected with a steroid in or about the tendon.[17]

Other, more physiologic etiologies, have been described and continue to be investigated. Kannus and Jozsa[18] studied tendons from 891 patients and concluded that spontaneous rupture occurred in tendons showing degenerative changes. It is theorized that these degenerative changes occur in TPD from the long-standing inflammation within and around the tibialis posterior tendon.

Frey[19] has described a zone of hypovascularity within the tibialis posterior tendon located posterior and distal to the medial malleolus. This area of hypovascularity and the degenerative changes seen in overuse and abnormal functioning of the tibialis posterior tendon, may be the predisposing factor for TPD and rupture.[13]

Mann[13] reported an unusually high incidence of os tibiale externum in TPD. Normally, 10% to 15%, this author has described an incidence of 60% in TPD. He suggests that the mechanical disadvantage caused by the insertion of the tendon fibers onto the ossicle may be a contributing factor in developing TPD in many patients.

CLINICAL PRESENTATION AND DIAGNOSIS

Initially, patients presenting with tibialis posterior symptomatology complain of medial arch or medial ankle pain. Their complaints may be associated with swelling and localized warmth posterior and inferior to the medial malleolus. Pain on palpation along the course of the posterior tibialis tendon is usually present from the posterior medial malleolus to the insertion into the navicular.

In the early stage of TPD, pain aggravated by weight bearing, may be the only presenting factor. Pain is usually noted with palpation along the course of the tendon. In this early stage, no clinical weakness or associated change in foot structure may be noted. As the problem continues, or goes untreated, the inflammatory cycle progresses. Localized edema and calor may now be seen. The continued inflammation leads to degeneration, hypertrophy, and eventual "functional rupture" of the tibialis posterior tendon. As this occurs, muscle weakness is noted.

Banks and McGlamry describe manual muscle testing of the tibialis posterior tendon by exerting an abductory force to the patient's supinated foot against resistance. Normally, a patient should be able to resist such a force, and the examiner should not be able to overpower the tendon. With an inflamed or ruptured tendon, the patient will be unable to maintain the supinated position and can often easily be overpowered.[14]

Johnson describes a single leg toe rise test. The patient is asked to stand only on the affected extremity. He or she is then asked to attempt to rise on the ball of the affected foot. The tibialis posterior tendon must be used to bring the rearfoot into a locked position so that the gastrocnemius-soleus complex can plantarflex at the ankle and rise on the ball of the foot. A normal test will demonstrate ability to rise on to the ball of the foot with simultaneous inversion of the rearfoot. A positive test will show continued rearfoot valgus with no inversion noted and little or no ability to rise on the ball of the foot.[15] Both of these tests will reproduce the pain that the patient experiences if the tendon remains intact, but not functioning.

In late stages of TPD evidenced by such described muscle weakness, loss of the longitudinal arch is usually present. The heel will be in a valgus position and the forefoot is usually abducted. The "too many toes" sign may be present, in which more than the normal number of toes will be noted laterally when the affected foot is viewed from the posterior position.[15]

Radiographs may show signs consistent with pronation, including an increased talocalcaneal angle, diminished articulation of the talonavicular joint, anteriorly displaced cyma line, and the lateral process of the talus abutting the floor of the sinus tarsi. If present for an extended period of time, degenerative changes of the medial column, midtarsal, subtalar, and ankle joints may be noted. A valgus alignment of the talus within the ankle mortise may also occur.

The diagnosis of TPD is usually made based on the history and clinical signs and symptoms described previously. Tenography, CT scans, and MRI have all been utilized to aid in the diagnosis. Of these diagnostic modalities, MRI offers the

physician the best and most accurate information. If a true rupture is suspected, with a palpable defect along the tendon course, an MRI will show the extent and exact location of the rupture. In a "functional rupture," an MRI typically shows hypertrophy and effusion about the affected tendon. Perhaps the best advantage of the MRI is in planning a soft tissue procedure to address the TPD. If the surgeon plans a procedure that relies on the integrity of the tibialis posterior tendon, a pre-operative MRI would be most beneficial.[13] Recently, ultrasound has been reported to be a promising method of evaluating rupture of the TPD.

CONSERVATIVE TREATMENT

Conservative treatment of TPD is seldom reported. There are many patients with this clinical disorder that are poor surgical candidates. Morbid obesity, peripheral vascular disease, cardiac disease, advanced age, and high-risk deep venous thrombosis patients are only some examples of poor operative candidates. Other patients may elect for nonsurgical treatment modalities. It is generally accepted that the late stage tibialis posterior dysfunctional foot with secondary structural changes is best addressed with surgery. Conservative treatment is best utilized for the early to midstage TPD patient with no structural changes. It is often necessary to attempt some conservative means of treatment before proceeding with surgical intervention. Lastly, conservative treatment modalities are the only treatment modalities at the physician's disposal for some patients.

In Stage I TPD as described by Johnson and Strom, the patient may have purely a peritendinitis characterized by amber synovial fluid and synovial proliferation. This stage may also present with longitudinal split tears, bulbous enlargement, and degeneration of the tendon.[9] It is this stage that a perceptive practitioner will make an early diagnosis. The Stage I patient presents with pain along the medial ankle and arch, possible edema and calor along the tendon course, and pain with attempts at single leg toe rise test.

Stage I TPD operative patients should be treated conservatively for three to six months. Relative rest, ice, compression, nonsteroidal anti-inflammatory medications, and orthoses or shoe modifications should be initiated. Often, the patient best benefits from immobilization on initial presentation combined with a nonsteroidal anti-inflammatory medication. In patients with peritendinitis, without clinical signs of weakness, complete resolution of symptoms is possible, in the author's experience, utilizing a removable walking cast and anti-inflammatory medication for three to six months. A custom-molded functional orthotic device can be utilized in conjunction with the removable walking cast for added support of the longitudinal arch. This same orthotic device is then continued after discontinuance of the walking cast and return to normal shoes. Continued weight bearing is preferable as the stress encourages tendon repair by helping to organize new collagen fibers in the direction of the stress.[4] Steroid injections in and around the tendon should be avoided.

Stage I non-operative patients can often be maintained comfortably with custom fabricated functional foot orthoses. Blake[4,20,21] has written extensively on the use of orthoses rather than surgical intervention for TPD and flatfeet. Orthotic control will decrease the length of time the tibialis posterior muscle fires, reduce rearfoot eversion and resulting load on the muscle-tendon complex, and allow for better function of the foot. This will allow for enhanced healing of the tibialis posterior muscle-tendon.[1] After the "tendinitis" has resolved, formal physical therapy can be initiated for restrengthening.

In Stage II TPD, the tendon shows marked degeneration, enlargement, and multiple longitudinal tears.[9] The patient exhibits increased pain, difficulty with ambulation, inability to perform single leg toe rise test, and "too many toes" sign. Conservative treatment for operative patients in this stage, should be carried out as previously described for Stage I patients. However, conservative trial can be shortened in light of already existent muscle weakness. Nonoperative Stage II TPD patients may also be managed long term with the use of functional orthotic devices. If the forefoot abduction and/or forefoot varus is severe, a custom molded shoe should be considered.

Stage III TPD is marked by rigid flatfoot deformity. The structural changes associated with loss of tibialis posterior tendon integrity have become fixed with coexisting degenerative arthrosis. These patients present with pain in the medial and lateral ankle, apropulsive gait, positive

single leg toe rise test with inability to lift the heel, and rigid pes planus with accompanying rearfoot eversion, forefoot abduction, and often forefoot varus. No conservative treatment will benefit the operative patient. The patient will best benefit from a stabilizing, realigning arthrodesis procedure. The difficulty arises in this group of patients that do not desire surgical intervention or are poor surgical candidates. Ankle-foot orthoses have been successfully used in these cases.[22,23] Lee and associates used the ankle-foot orthoses combined with a UCBL (University of California Biomechanics Laboratory) orthotic device successfully in 53 tibialis posterior tendon ruptures.[23] Custom-molded shoes can also be used in this group of patients with accommodations for coexisting equinus and forefoot varus.[14] A double upright brace attached to a custom-molded shoe has proven effective in maintaining ambulation in patients with gross end stage deformities of TPD.

CONCLUSION

Tibialis posterior dysfunction is a disabling condition for many patients. Even though early surgical intervention may be the best treatment for many patients, conservative therapy may be the only consideration in some patients. Patients with early signs of TPD can be treated successfully with short courses of conservative care, followed by physical therapy. End-stage TPD patients can also be managed at a comfortable level with aggressive conservative care.

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