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CLINICAL VIGNETTE

Getting a Leg Up on Posterior Tibialis Tendon Dysfunction

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Case Presentation

A 79-year-old male presented for an annual wellness exam. During the visit, he mentioned having pain at the medial aspect of the right ankle for the past 3-4 years, mainly during weight-bearing and especially during running. He reported completing 20 marathons but stopped marathons in the last few years due to the ankle pain, which limited running. He was still doing a combination of walking and jogging for 4-5 miles per day, 4-5 days per week, and he used a lace-up, Velcro ankle brace which seemed to alleviate his ankle pain slightly.

Physical exam was notable for moderate to severe pes planovalgus, worse on the right than the left. No tenderness or edema was noted, including along the course of the posterior tibialis tendon. Manual strength testing revealed 4/5 strength at the right ankle against resisted plantar flexion and inversion. A positive "too many toes sign" (Figure 1) was noted on the right during stance. Moderate right calf atrophy was present, as well as weakness and associated calcaneal valgus on attempted right heel raises. Gait analysis revealed markedly excessive pronation bilaterally, more on the right than the left.

Anatomy and Function of the Posterior Tibialis Muscle and Tendon

This patient presented with classic features of posterior tibialis tendon dysfunction (PTTD). The posterior tibialis muscle has a broad origin, arising from the posterior aspect of the interosseous membrane, the proximal two thirds of the posteromedial portion of the fibula, the posterosuperior aspect of the tibia, and the intermuscular septum in the posterior compartment of the leg. The posterior tibialis muscle gives rise to the posterior tibialis tendon (PTT) at the level of the distal third of the tibia. Coursing distally just posterior to the posteromedial aspect of the tibia, the PTT runs just posterior to the medial malleolus and then makes an abrupt anterior turn, changing its line of force from primarily vertical to nearly horizontal. After passing inferior to the plantar calcaneonavicular (spring) ligament, the PTT splits into 2 slips. The superficial slip inserts onto the navicular tuberosity at the medial aspect of the midfoot, whereas the deep slip continues on to give rise to expansions that attach to the plantar aspects of the second through fourth metatarsals and the middle cuneiform. It is innervated by the tibial nerve (from the L4 and L5 nerve roots) and supplied by muscular branches of the sural, peroneal, and posterior tibial arteries. A vascular watershed exists approximately between the level of the medial malleolus and just proximal to the navicular, thus pre-disposing this stretch of tendon to relative hypovascularity and impaired healing. The posterior tibialis muscle-tendon unit serves as a plantar flexor and inverter of the ankle, as well as an important supporter of the arches of the foot.[1-3]

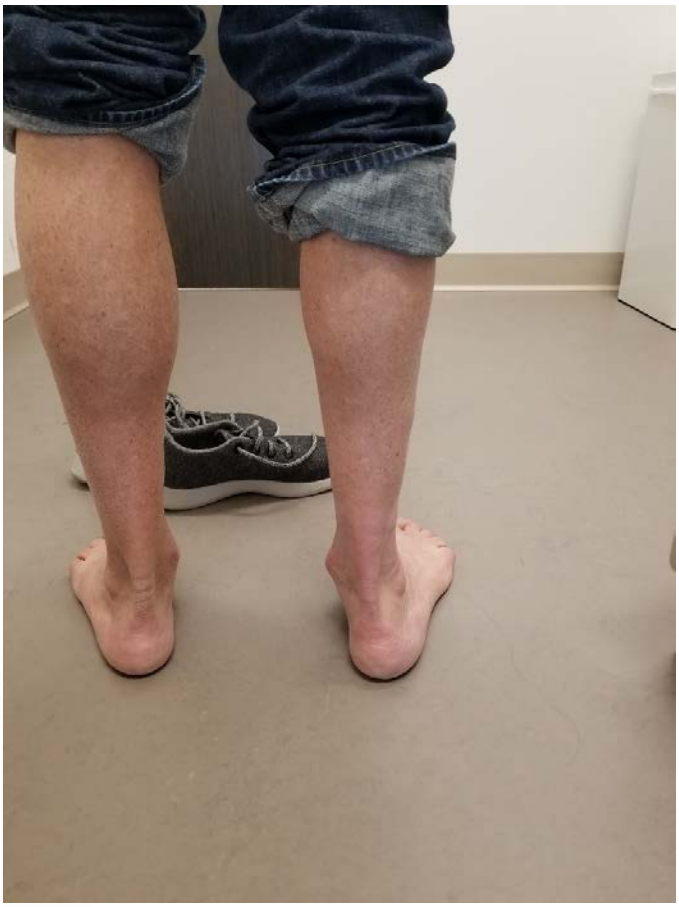

Figure 1

Posterior Tibialis Tendon Dysfunction: Clinical Manifestations, Epidemiology, and Classification

PTT dysfunction (PTTD) is most commonly due to repeated microtrauma, and typically manifests with the insidious onset of mild pain along the course of the distal tendon, between the medial malleolus and the navicular. Patients often report pain and swelling in this region, usually worse with weight-bearing and especially with running, jumping, or other activities placing

dynamic strain upon the ankle and foot. Rising up on the toes is often particularly painful, especially if performed unilaterally on the involved side. Middle-aged women have been considered the population with the highest incidence of PTTD. However, multiple risk factors have been implicated in the development of PTTD, including older age, obesity, female gender, diabetes mellitus, hypertension, seronegative spondyloarthropathies, local steroid injections, and prior foot or ankle surgery.[4] Acute trauma to the foot or ankle can acutely rupture or injure the PTT, but this is a relatively uncommon.[5]

If the injury is not properly treated, it typically follows a progressively worsening course, with the eventual development of an acquired flatfoot deformity, chronic pain, and persistent dysfunction of gait and activity. Different classification systems have been designed to reflect these clinicopathologic stages. No universal agreement exists as to how best to classify the latter stages of dysfunction, when multiple other structures (ie; the spring ligament and associated static and dynamic arch stabilizers) begin to fail and flatfoot deformity develops.[6] The original three stage classification system was published by Johnson and Strom in 1989.[7] Myerson's system adds a separate fourth stage, although Johnson and Strom described this as a possible stage in their original article. Additional classification systems have since been described, with sub-divided stages to reflect the wide variation in clinical presentations.[8-10] For simplicity, the basics of the modified Myerson classification system are presented below:

Stage I: Pain and swelling along the distal course of the PTT. Normal alignment, no deformity yet. Weakness, pain, and difficulty with single foot heel raise.

Stage II: Worsening pain and swelling, more impairment of function. Flexible pes planus now developed. Marked weakness on single foot heel raise. The "Too Many toes Sign" may now be present. Weight-bearing X-rays of the ankle and foot reveal forefoot abduction and talonavicular subluxation.

Stage III: Severe medial ankle/foot pain, with possible secondary pain laterally at the sinus tarsi due to chronic compression of the lateral structures. Worsening flatfoot deformity, now fixed rather than flexible. Worsening pain and weakness on single foot heel raise. X-rays may now show talonavicular, subtalar, and calcaneocuboid degeneration.

Stage IV: Tibiotalar arthritis laterally, and deltoid ligament insufficiency medially, respectively due to worsening compression of the lateral structures and ongoing loss of support medially.

Imaging of PTTD

The initial diagnosis of PTTD is clinical, but imaging can be very helpful in delineating disease severity and helping with surgical planning. In Stage I disease, plain x-rays are not useful, although they are sometimes obtained for baseline structural assessment, as well as to assess for other conditions (ie; stress fracture, pre-existing arthritis, neoplasms, etc.). In Stage II disease and beyond, bilateral weightbearing plain x-rays of the foot and ankle are helpful to further assess the deformity and the extent and severity of arthrosis. Ultrasound is non-invasive, relatively inexpensive, involves no ionizing radiation, and can show tendinosis (thickening, abnormal fiber structure) of the PTT, as well as tendon tearing and fluid collections. Recently, advances in high-resolution musculoskeletal ultrasound in expert hands, may rival or even surpass MRI in accuracy for detecting PTT pathology. However, ultrasound is extremely operator dependent, so it needs to be performed (and then interpreted) by providers with appropriate expertise. MRI has generally been considered the "gold standard" for tendon evaluation, and can show associated soft tissue, bony, and cartilaginous pathology as well. It also is non-invasive and involves no ionizing radiation, but is expensive. [11]

Treatment of PTTD

If the process is detected early in Stage I, a conservative approach may be successful. Initially, a regimen of relative rest, icing, compression, and elevation (RICE) is instituted to alleviate symptoms and allow the injured tendon to heal. Avoidance of painful or provocative activities is very important. For overweight or obese patients, weight loss is extremely important, as this unloads the tendon and its supporting structures. For athletes, it is especially crucial to design a pain-free cross-training program to allow them to remain active and fit while allowing their injury to heal. This usually involves low to non-impact activity, such as pool running, swimming, weightlifting, and cycling. Supporting the arch with appropriate footwear is recommended, and the addition of either over the counter arch supports or customized orthotic devices may be warranted. For patients with more severe symptoms, use of a cam walker boot and/or crutches may be necessary. Physical therapy may be useful to further reduce symptoms and swelling, as well as to promote a gradual return to safe activity in a supervised environment.

Patients in more advanced stages of PTTD may benefit from early referral to a sports medicine specialist or foot and ankle specialist. Patients with Stage II disease are often treated surgically, although initial conservative management may be attempted. If symptoms and activity impairment persist despite conservative measures, surgery is warranted. At this stage, the deformity is not yet fixed and significant joint degeneration has not developed and surgical interventions correct the flatfoot deformity and dynamically stabilize the arch. In recent years, the use of subtalar arthroereisis (insertion of an implant into the subtalar joint to correct flexible flatfoot deformity and relieve associated symptoms) has gained popularity and with generally good results.[12] Patients who have progressed to Stage III or IV have a rigid flatfoot deformity and arthrosis. Conservative measures can help pain management and support function (ie; ankle foot orthosis). Surgical candidates with Stage III disease typically undergo fusion of the mid/hindfoot (triple arthrodesis of the talonavicular, calcaneocuboid, and subtalar joints),

whereas Stage IV disease usually requires additional fusion of the ankle.[13]

Summary

The natural history of untreated PTTD is slowly progressive dysfunction and development of fixed deformity, foot and ankle arthrosis, chronic pain and disability. However, if the process is identified and properly treated early, severe outcomes may be prevented, and patients may be able to return to their sports and activities rather than require joint fusions and/or permanent loss of mobility and function. Thus, it is crucial that providers be aware of the signs and symptoms of PTTD and institute appropriate workup, management, and referral.

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