TIBIALIS POSTERIOR DYSFUNCTION

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INTRODUCTION

Tibialis posterior dysfunction has gained increased recognition and appreciation as a disabling cause of progressive flatfoot deformity. Spontaneous rupture may occur, but the cause is usually a degenerative inflammatory process where the tendon loses its ability to support the medial arch and resist the peroneus brevis. Recent literature hypothesizes an area of hypovascularity in the posterior tibial tendon as it courses posterior and distal to the medial malleolus.

Patients with seropositive arthritides, particularly rheumatoid arthritis, are also more susceptible to tendon dysfunction in general. The posterior tibial tendon is no exception. It is likely that degenerative changes, tenosynovitis, constriction under the flexor retinaculum, hypovascularity and the anatomical course of the tendon all contribute to this disease.

SYMPTOMATOLOGY

The symptomatology is variable. The chief complaint is usually pain and/or swelling, localized warmth along the medial arch and visible abduction and collapse of the forefoot. Physical examination reveals valgus deformity of the rearfoot and inability to invert the affected heel when raised on their toes. The "too many toes" sign is also evident (Fig.1). When viewing the patient posteriorly, more toes are seen on one foot versus the other, a factor of forefoot abduction.

Radiographic signs of progressive pes plano valgus become noticeable with the course of the dysfunction. The navicular may sublux off of the head of the talus on the dorsoplantar view and on the lateral view, talonavicular or naviculocuneiform sagging may be evident. Imaging of questionable tibialis posterior tendon pathology ranges from computed tomography to ultrasound. The most specific modality is magnetic resonance with the results corresponding most accurately with surgically identified pathology (Fig. 2A - 2C).

MRI soft tissue enhancement is superior to CT scanning but both studies tend to underestimate tendon abnormality. Mild heterogeneity of the tendon such as longitudinal splits may be



Fig. 1. Illustration depicting the "too many toes" sign as seen in posterior tibial dysfunction. More toes are visible on this right foot because of abduction of the forefoot.



Fig. 2A. Morphologic types of tibialis posterior tendon dysfunction. Acute ruptures may occur with distraction of tendon ends.



Fig. 2B. Attenuation of tendon ends may occur with long term strain.



Fig. 2C. Hypertrophy of the tendon may occur in early dysfunction, after partial tears or with in-situ tears.

difficult to differentiate. Attention must be paid to medial malleolar cortical thickness when evaluating tendon size as well. Diagnostic tenography is possible but difficult. If the tendon is not palpable, localizing the tendon sheath will be almost impossible.

Although ultrasound of the foot is not common practice, its ability to evaluate soft tissue abnormality throughout the body is excellent. The use of this for posterior tibial tendon imaging needs further documentation. These studies are all of some diagnostic value, but management and surgical intervention still depend on clinical examination and the experience of the doctor. Physical examination and manual muscle testing will usually provide the diagnosis. Attention to the action of adjacent muscles is important. The tibialis anterior and the flexor digitorum longus actions should be negated to accurately assess posterior tibial function. Tibialis anterior is frequently used as a non-weightbearing invertor of the foot and will make inversion strength appear normal to the unpracticed eye.

There are several classifications of tibialis posterior dysfunction. They each attempt to capture the stage of the disease and correlate such with treatment. Generally, in the degenerative situation, the tendon will hypertrophy initially in an attempt to reinforce itself, and then subsequently attenuate leading to frank rupture. Conservative treatment consisting of shoe modifications and local therapy usually meets with limited success. Most recommendations reserve orthotics and injection therapy for the nonoperative patient.

Acute posttraumatic rupture seen immediately will do very well with primary repair. However, treatment of the more frequently seen degenerative tendon scenario is made more difficult because the diagnosis may not be made for a year or two. In these patients, the fundamental question is whether to perform fusions or soft tissue reconstruction. Flexor digitorum longus is the most widely recognized transfer choice and may provide symptomatic relief. The basic foot architecture however, remains unchanged with soft tissue procedures. Arthrodesis of either the subtalar joint alone or triple arthrodesis provide more permanency and structural correction. Author's opinions vary regarding the need for isolated subtalar fusion or triple fusion. Since tibialis posterior dysfunction inherently results in pes plano valgus, or vice versa, it appears triple arthrodesis is the most efficacious manner to gain permanent, triplane correction and prevent further breakdown at the midtarsal joint.

Dysfunction of the posterior tibial tendon is now a more recognized entity as a cause of progressive flatfoot deformity. Treatment requires early diagnosis and prevention of further deformity. The definitive treatment for each stage is still uncertain. Greater experience is needed before we can prognosticate accurately with these patients.

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