CHAPTER 47

EVALUATION OF THE TIBIALIS POSTERIOR AND ACHILLES TENDONS

M. Jay Groves, D.P.M.

The Achilles and tibialis posterior tendons are the most commonly affected in the foot and ankle. Injury, degeneration and dysfunction of these tendons can result from both metabolic conditions and mechanical forces.

Systemic conditions such as rheumatoid and seronegative arthritis, as well as chronic steroid ingestion, may cause pathologic tendon degeneration or rupture. Familial hypercholesterolemia and hyperlipidemia may create xanthoma deposition in the Achilles tendon. These asymptomatic and painless lesions do not predispose the tendon to rupture, but are important in the diagnosis and early treatment of these diseases.

Pathologic mechanical forces can also be responsible for tendon injury. When the Achilles and tibialis posterior tendon pathology remain undiagnosed, foot and ankle function usually deteriorates, causing irreversible deformity. Early diagnosis and treatment allows the most favorable and functional outcome. Accurate evaluation of Achilles and tibialis posterior tendinopathy involves a thorough understanding of the anatomy, kinesiology, patient history, physical examination, and imaging of these tendons.

ANATOMY & KINESIOLOGY

Achilles Tendon

The calcaneal tendon is one of the thickest and strongest tendons in the body. In addition to providing heel lift during gait, the soleal and gastrocnemius muscles assist with knee extension and flexion. The soleus also supinates the subtalar joint and stabilizes the lateral column.

The tendon arises from the converging gastrocnemius aponeurosis and soleal tendon. The blending of these structures forms the superior aspect of the tendo Achilles, approximately 15 cm above the calcaneus. The soleus continues to contribute anterior muscle fibers to the tendon almost to the level of calcaneal insertion.

The distal tendon is ovoid in shape and is ensheathed by a vascular paratenon (Fig. 1). The Achilles is covered more superficially by the crural fascia, superficial fascia and skin; anterior to the tendon is the pre-Achilles fat pad (of Kagar's triangle) (Fig. 2). The tendon measures 5 mm to 6 mm thick at the ankle, and 1.2 cm to 2.5 cm in width at the insertion.

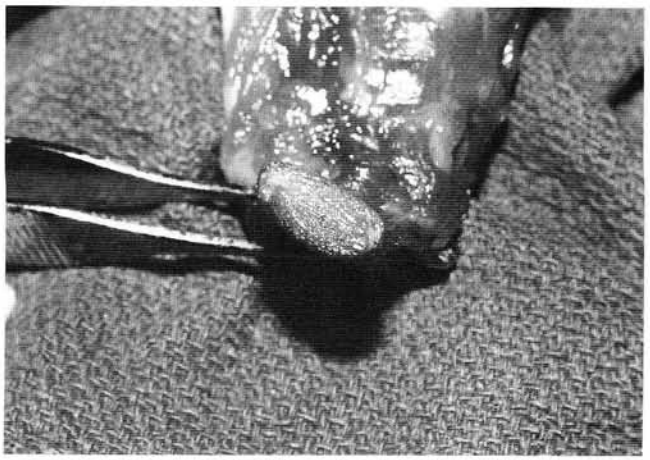

Figure 1. Note the oval appearance of the Achilles tendon near its insertion.

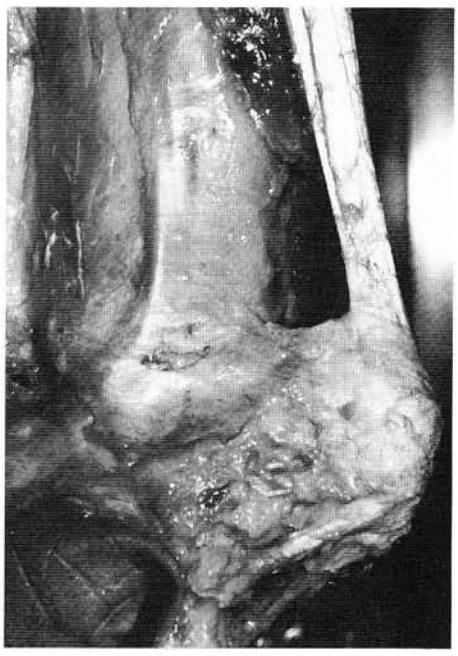

Figure 2. Medial view of Kagar's triangle with excised preachilles fat pad. Note the distal soleal muscle belly insertion, the plantaris tendon, and the proximity of the Achilles insertion and plantar fascia.

Rotation of tendinous fibers occurs from proximal to distal. Although slight variations exist, the gastrocnemius fibers insert laterally, and the soleal fibers medially at the posterior calcaneus. The tendon inserts into the middle one-third of the posterior calcaneal surface. Some of the tendon fibers are continuous with the plantar aponeurosis. The superior one-third of the calcaneus is separated from the tendon by the deep retrocalcaneal bursae. A retro-Achilles subcutaneous bursa may also be present.

Tibialis Posterior

The influences of the tibialis posterior (TP) tendon on the foot are diverse. The gliding TP tendon crosses the ankle, subtalar and midtarsal joints thereby creating plantar-flexion, supination and adduction/plantarflexion of these joints, respectively, during propulsion. The primary function of the TP tendon at the midtarsal joint appears to be adduction. Thus, it is a direct antagonist to the peroneus brevis muscle.

The tendon's multiple proximal, distal and lateral insertional slips provide ligamentous-like support to the tarsal and lesser metatarsal bones. The propulsive supinatory action of the TP tendon's gliding component acts to stabilize the midfoot through these diverse ligamentous-like attachments. The gliding component is that part of the TP which is proximal to the navicular insertion.

During the contact phase, the TP acts to decelerate the rate and extent of plantarflexion, pronation and abduction of these joints. Ultimately, it is the ligaments which prevent extremes of motion, and provide persistent structural stability at these joints during this phase of gait.

The TP tendon arises from the deepest and largest muscle of the deep posterior leg compartment. The muscle belly originates from the posterior tibial and fibular surfaces, interosseous membrane, deep transverse intermuscular septum and adjacent fascia. The tendon begins proximal to the medial malleolus in the distal quarter of the leg. It then courses inferiorly and medially deep to the flexor digitorum longus (FDL) tendon to reside in the retromalleolar sulcus and first compartment of the flexor retinaculum (Fig. 3). Superior to the medial malleolus, the FDL is medial in relation to the TP; at the level of the medial malleolus, the TP is medial. The TP tendon continues beyond the flexor retinaculum into the medial and plantar aspect of the foot (Fig. 4).

The tendon sheath of TP begins proximal to the flexor retinaculum approximately 6 cm superior to the medial malleolar colliculus, and ranges from 7 mm to 9 mm in length. The sheath nearly reaches the navicular tuberosity, and extends more on the osseous surface and less on the superficial surface. Furthermore, the TP tendon has no mesotenon.

There are several insertions of the TP tendon (Fig. 5). The tendon's primary insertion is the navicular tuberosity. However, it divides into three tendinous slips which collectively insert into all the tarsal bones except the talus. The anterior slip courses distally past its navicular attachment to insert into the plantar aspect of the medial cuneiform. The posterior component arises from the tendon proximal to its navicular insertion, and courses lateral and posterior to insert on the anterior part of the sustentaculum tali. The lateral

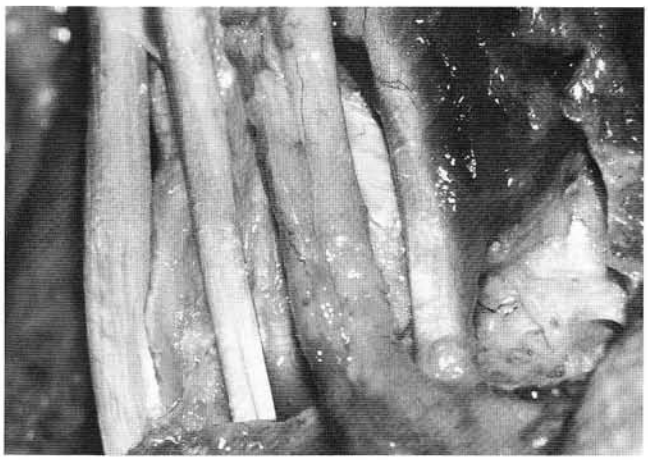

Figure 3. Contents of the flexor retinaculum from left to right: TP, FDL, posterior tibial neurovascular structures and flexor hallucis longus. Note the relative sizes of the TP and FDL tendons.

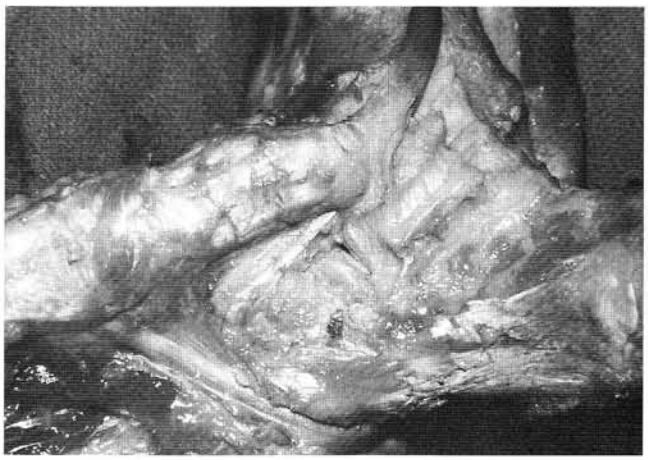

Figure 4. The TP tendon as it reaches the medial and plantar aspect of the foot. The peroneus longus is seen coursing across the plantar aspect to its insertion.

Figure 5. The diverse insertions of the TP with the peroneus longus tendon transected. The anterior, lateral and posterior components can be easily identified.

slip provides insertions into the intermediate and lateral cuneiforms, cuboid, the fibrous covering of peroneus longus and the bases of metatarsals two, three, four, and frequently five. The second through fifth metatarsal bases are reached by slips passing dorsal to the peroneal tendon.

CLINICAL EVALUATION

The patient's history may be the most important means of evaluating Achilles and TP tendon disorders. The physical exam often confirms the suspected pathology. However, advanced imaging may be necessary.

Achilles Tendon

Patients with Achilles tendon ruptures, whether partial or complete, usually recall the initial trauma. The patient often relates feeling or hearing a "pop" in the posterior leg region. Initial pain may be minimal, and loss of function variable. However, continued weakness, swelling, and persistent pain usually motivate the patient to seek medical treatment. In addition to mechanical rupture, laceration may cause Achilles tendon discontinuity (Fig. 6).

Mechanical rupture of the healthy Achilles tendon is rare. In the absence of tendinopathy,

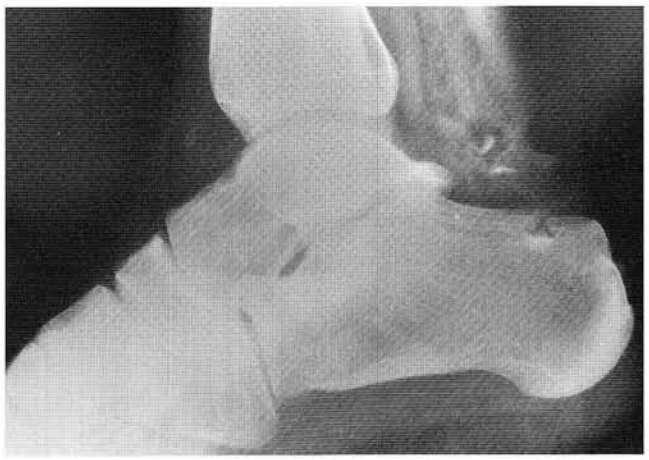

Figure 6. Laceration of the Achilles tendon. The tendon was severed by a sharp metal object which contacted the posterior aspect of this patient's leg. The superior calcaneus was also damaged in this injury.

excessive force or stretch to the posterior leg usually results in muscle belly tears, tears at the myotendinous junction, or osseous avulsion. However, it has been suggested that the healthy Achilles tendon may be forcibly ruptured. Support for the pre-existing tendinopathy theory is based upon patient history as well as histological examination of ruptured Achilles tendons. Chronic inflammatory and reparative processes of the Achilles tendon with areas of micronecrosis has been demonstrated microscopically. Rupture of the degenerative tendon occurs primarily in an area of micro-ischemia and micronecrosis.

Physical examination provides valuable information in diagnosing Achilles tendon ruptures. Visually, a defect of the Achilles tendon may be present (Figs. 7, 8). Localized discomfort and unilateral edema is a common finding. A palpable gap may be present in the Achilles tendon, and may best be detected by applying a viscous gel before examination. Diminished or absent plantarflexion with squeezing of the calf (a positive Thompson's test) may be present. Unilateral decrease in plantarflexory strength may be noted with manual muscle testing, and during gait examination.

In acute total tears, the physical exam is usually diagnostic. When examination is delayed, inflammatory changes occur which may obscure the diagnosis, This delay may be responsible for the reported 20% to 30% misdiagnosis rate. Moreover, partial ruptures may be difficult to discern from tendinitis, and in these cases imaging may be indicated.

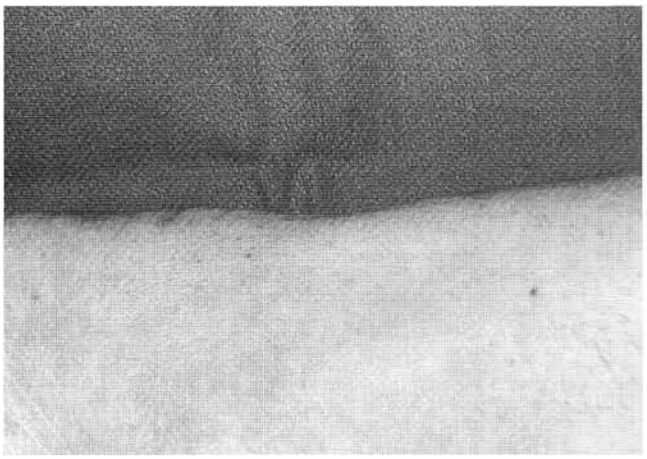

Figure 7. Achilles tendon defect noted with lateral visual examination (patient is prone with heel to left). A palpable defect was present at this site.

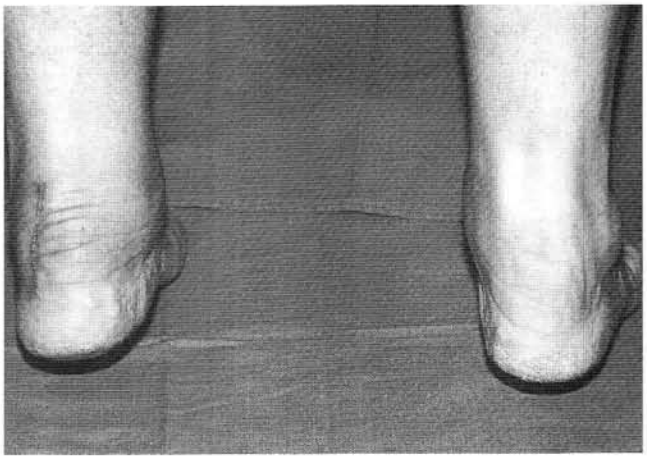

Figure 8. Abnormal tendon contour and transverse skin creases are present on the right. Diminished heel lift is also present on the right.

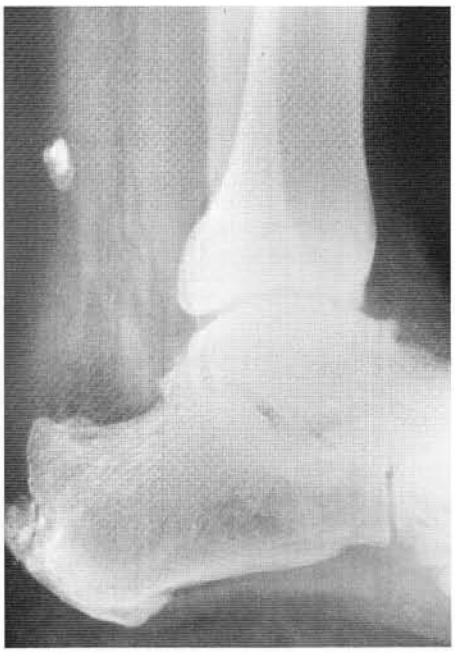

Figure 9. Complete rupture of the Achilles tendon with calcification of the proximal tendon stump. Note the distortion of Kagar's triangle (compare with Figure 6).

Imaging of the Achilles tendon region may delineate the tendon pathology. The primary study is a conventional lateral radiograph of the foot and lower leg to rule out osseous injury of the calcaneus. Kagar's triangle (bordered by the Achilles tendon, calcaneus and deep flexors) may suggest Achilles injury by loss of the normal configuration. The lateral radiographs of a patient with a rupture may reveal calcified tendon at the rupture site (Fig. 9). Other diagnostic imaging modalities are ultrasonography and magnetic resonance imaging (MRI).

Ultrasonography is an excellent method to evaluate the Achilles tendon. The sensitivity and specificity of this modality are higher than 92% for experienced investigators. Other advantages include no ionizing radiation dose, good tendon resolution, real-time examination, evaluation of healing, and relatively low cost when compared to MRI. Disadvantages are lack of availability, and a dependance on the technician's experience.

Ultrasonographic examination is best performed with the patient in a prone position, with the feet extending beyond the end of the table. A 1 cm to 2 cm thick silicone pad is applied to the Achilles area to allow for better contact of the transducer. A 7.5 Mhz linear array transducer provides a superior image of the tendon. The tendon is imaged in longitudinal and transverse projections in both static and dynamic views of the tendon (i.e., dorsiflexion and plantarflexion). Tendon abnormalities such as discontinuity, thickening, sonolucency, and edema may be evaluated.

Although it is more expensive than sonography, MRI examination of the Achilles tendon is an excellent modality for evaluation. Its most valuable application may be in differentiating paratendinitis from partial rupture as well as evaluating chronic injury and persistent pain (Fig. 10). The Achilles can be visualized in the sagittal, coronal, and axial planes. The sagittal and axial planes are best for evaluation, however, optimal assessment is achieved through bilateral studies using both T1- and T2-weighted images.

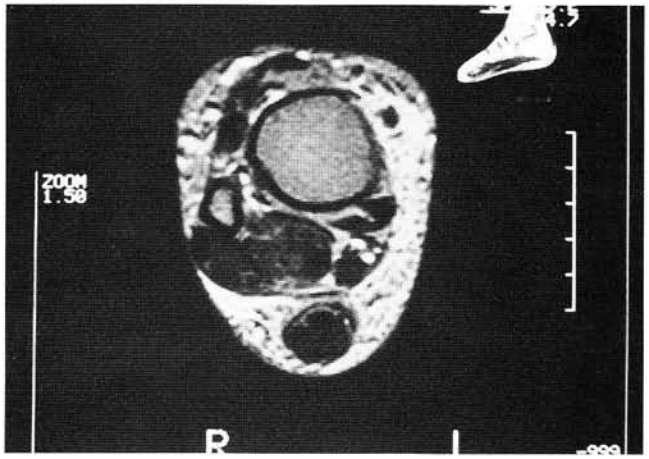

Figure 10. Achilles paratendinitis. Note anterior edema between the Achilles and the paratenon on this axial T2-weighted image.

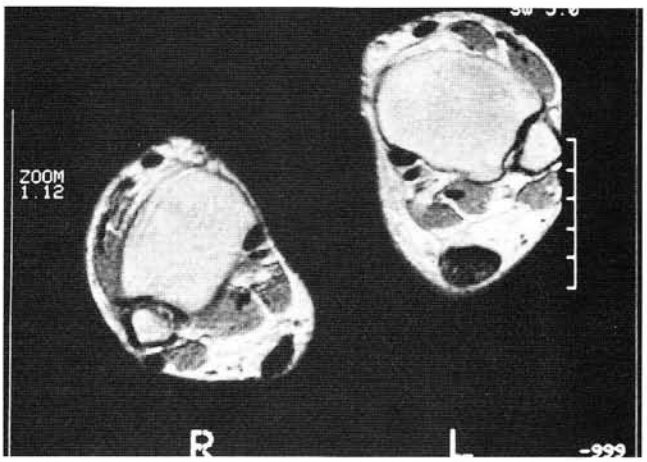

Figure 12. Axial image of a partially ruptured left achilles tendon. Differences in signal intensity, tendon shape, and size are apparent.

Several patterns of tendon injury are identified by changes in anatomical contour and signal intensity. Normally, the Achilles tendon is uniformly shaped, with a homogeneously low signal intensity. Recognized pathological changes include loss of tendon continuity, tendon thickening, multiple longitudinal intratendinous tears, increased signal intensity, irregular or wavy tendon appearance, and focal or diffuse fusiform tendon swelling (Figs. 11, 12).

Tibialis Posterior

Tibialis posterior disorders are most often insidious in onset. Although uncommon, acute trauma such as direct tendon laceration, sudden forceful contraction, and medial malleolar fracture may cause TP rupture.

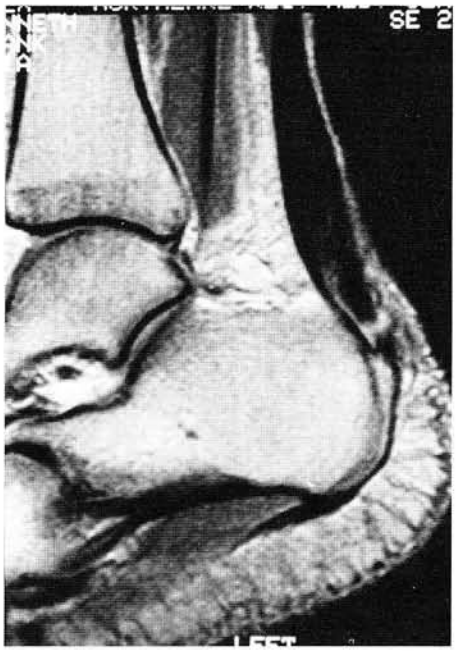

Figure 11. Sagittal plane T2 weighted image of a partial Achilles tendon rupture. Increased intratendinous and posterior signal intensity is detected.

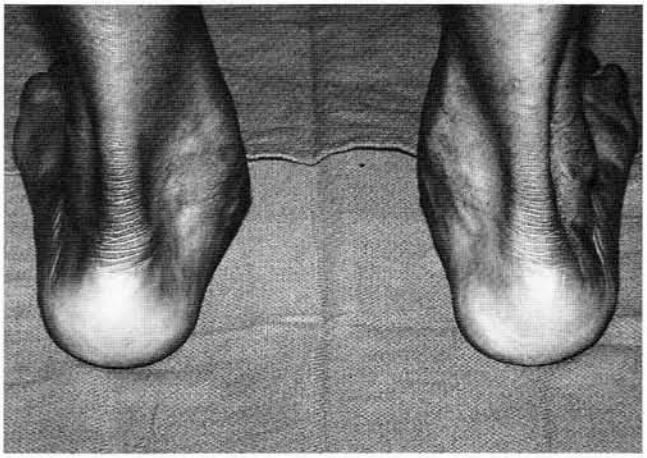

Figure 13. Tibialis posterior dysfunction is most commonly associated with bilateral pes valgus. The patient's left side is symptomatic in this case.

Progressive TP dysfunction is most commonly seen in the pes valgus foot type (Fig. 13). In the pathologic foot, the ligamentous structures are subjected to chronically abnormal tension. As these tissues elongate, the TP is called upon to increase its muscular activity. It is this pathological recruitment which leads to its dysfunction. These de-stabilized soft tissues allow for subluxation of the midtarsal joint, and potential development of degenerative joint disease at the subtalar, talo-navicular, cuneonavicular, first metatarsophalangeal joint, and anterior ankle.

Historically, the patient may describe ankle and medial arch pain of variable duration. The patient occasionally recalls an initial injury at the medial ankle or foot, and describes progressive deformity with pain. Muscular fatigue, as well as arthritic pain, may be present.

Consistent physical findings are present with TP dysfunction, However, they may vary, relative to the available compensation and primary deformity. Visually, severe pes planovalgus with forefoot abduction and rearfoot valgus is present. Medial arch collapse with talar head prominence, tendon enlargement and inflammatory changes may be present (Fig. 14). Posteriorly, the "too many toes" sign and decreased rearfoot varus with heel elevation is often present unilaterally.

Manual examination usually provides the most valuable information. The TP tendon should be palpated from its insertion to the posterior aspect of the medial malleolus. The course of the tendon may be tender, warm, and edematous. Rarely, a palpable tendon defect may be identified. When the tendon has ruptured, manual muscle testing reveals a decrease in adduction and inversion strength of the foot (Fig. 15). Unilateral accentuation of the TP tendon, ankle equinus and increased midtarsal joint motion may be appreciated.

Gait analysis reveals a non-supinatory apropulsive foot upon which the lateral border is not fully loaded. Forefoot abduction and calcaneal eversion are classic findings with or without antalgia during gait.

In addition to the physical exam, baseline radiographs are required to further evaluate TP dysfunction. On the AP view, increased talocalcaneal angle and abduction of the navicular on the talus are common findings. The lateral view may show subluxation, or a sagittal plane break at the talonavicular joint. Navicular avulsion or an accessory navicular may be identified.

The history and physical will provide definitive diagnosis in the majority of TP dysfunction cases. Occasionally, specialized studies, particularly MRI, will be required to accurately evaluate the patient's condition. Ultrasonography is not used regularly in diagnosing TP dysfunction. The indications for MRI examination of the TP tendon at the author's institution involves the following principles: if the tendon is ruptured clinically, then scanning is not performed, patients who do not respond to conservative treatment may be selected for scanning, acute ruptures usually undergo MRI examination.

The TP tendon is best evaluated on sagittal and axial MRI views. Normally, on the axial view the TP is usually twice the diameter of the FDL and flexor hallucis longus (FHL) at the medial malleolar level. In pathologic states, the singular finding may be increased signal intensity of the tendon sheath representing tenosynovitis. Further, the TP tendon may be several times larger than the FDL and FHL. This is considered to be a type 1 partial rupture (Fig. 16). A type 2 TP tendon rupture is a partial rupture with an attenuated tendon region. Type 3

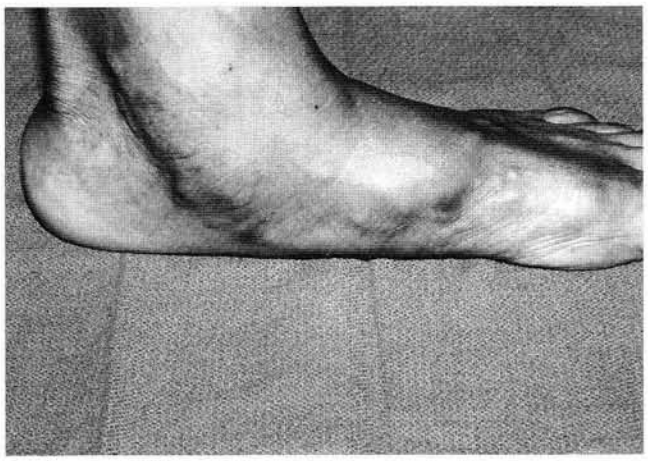

Figure 14. Medial arch collapse with associated edema is often present in TP dysfunction.

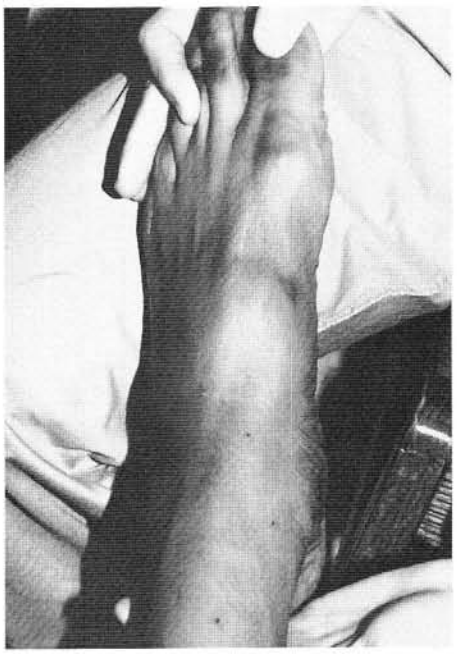

Figure 15. Manual muscle testing of TP is best performed with the foot in a plantarflexed position to eliminate tibialis anterior substitution.

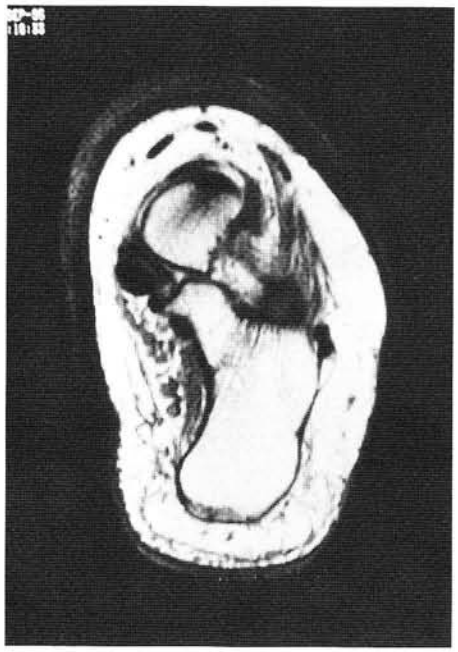

Figure 16. Axial image of the TP tendon. The cross-sectional diameter of the TP tendon is much greater than FDL's indicating a type 1 partial rupture (compare with Figure 12).

ruptures involve a complete discontinuity of the tendon with possible retraction of the tendon ends. Increased signal intensity (representing fluid) may be present about or within the tendon.

CONCLUSION

Once the diagnosis of Achilles or tibialis posterior dysfunction has been determined, treatment may be initiated. Treatment is based on the severity, and the acute or chronic nature of the deformity. Conservative treatment is directed towards reducing acute symptoms and returning the patient to acceptable function through supportive treatment. Surgical treatment may be directed towards tendon reanastomosis acutely, whereas long-standing TP dysfunction may require bony fusion with tendon support of the medial arch. In any case, avoidance of progressive compensatory deformity and arthrosis is the primary goal.

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