

Tibialis Posterior and Anterior Tendons

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34.1 Introduction

Sports activities are increasing worldwide as a promoter of global health (Palacios-Cena et al. 2012). Enhanced proprioception and fine-tuned neuromuscular control around the foot and ankle are particularly required for gait in uneven ground and most types of sports (Cote et al. 2005). This includes fine balance between dorsiflexion-plantarflexion and pronation-supination. General population is presenting increased and earlier attention to foot and ankle tendon-related disorders probably led by their higher functional demand. This fact might play a role in earlier diagnosis of some condition in its primary stages thus also influencing the clinical outcome. This is of paramount relevance in the herein described conditions.

The ankle joint complex comprises the tibiotalar (talocrural), talocalcaneal (subtalar), and transverse tarsal (talocalcaneonavicular) joints (Brockett and Chapman 2016).

Plantar- and dorsiflexion occur predominantly at the tibiotalar joint (Brockett and Chapman 2016). Pronation-supination mostly depends on subtalar joint but tibiotalar and talocalcaneonavicular participate at different levels. Foot pronation is a complex movement which combines abduction of the forefoot, eversion of the hindfoot, and dorsiflexion (Gluck et al. 2010). Supination also happens in the three planes while combining internal ankle rotation, hindfoot adduction, forefoot inversion, and medial arch elevation. Pronation provides some degree of flexibility opposing to supination which increases foot stability (Cote et al. 2005).

The *tibialis anterior* (together with the *extensor hallucis longus*) produces dorsiflexion and some degree of inversion of the foot (Brockett and Chapman 2016). The *extensor digitorum longus*

only produces dorsiflexion of the foot. The *tibialis posterior* (together with the *flexor digitorum longus* and the *flexor hallucis longus*) produces plantarflexion and inversion of the foot (Otis and Gage 2001; Brockett and Chapman 2016). Conversely, peroneal muscles (PMs), including *peroneus longus*, *brevis*, and *tertius*, are the active evertors of the foot (Selmani et al. 2006). Plantarflexion of the foot is furthermore provided by the *triceps surae* and *plantaris muscles*. These muscles play a key role controlling ankle motion (Brockett and Chapman 2016).

In order to understand and properly deal with physiopathology, it is mandatory to comprehend the fundamental biomechanics of the foot and ankle. The navicular bone is considered a “key bone” providing distal support to the talus. It is the highest structure of the longitudinal medial arch in the standing position. In the static position, the balance of these structures relies on the surrounding bones and the spring ligament (calcaneonavicular). In motion, the action of the posterior tibial muscle (PTM) preserves the superior position of the navicular thus assisting in the support of the medial arch. Therefore, a weakened PTM (e.g., neurologic conditions) or an insufficient posterior tibial tendon (PTT) is incapable to sustain the navicular in place, and a collapse of the medial arch might happen (acquired flatfoot condition) (Lhoste-Trouilloud 2012). From gait analysis, one observes a dorsiflexion moment at heel strike as the dorsiflexors (e.g., *tibialis anterior*) eccentrically contract to control the rotation of the foot and avoids the foot from slapping the ground (Brockett and Chapman 2016). These basic examples are representatives of the paramount relevance of biomechanical phenomena in this field.

Some principles of clinical assessment are common to both groups of pathology. Clinical examination while standing and gait analysis are critical in any foot and ankle conditions. Moreover, global assessment, including alignment, of inferior limbs is required.

Radiological study must always include standing foot and ankle x-rays. Further views might be considered according to the clinical findings and local experience. Ultrasound and MRI might be useful (Fig. 34.1); however, limitations exist and

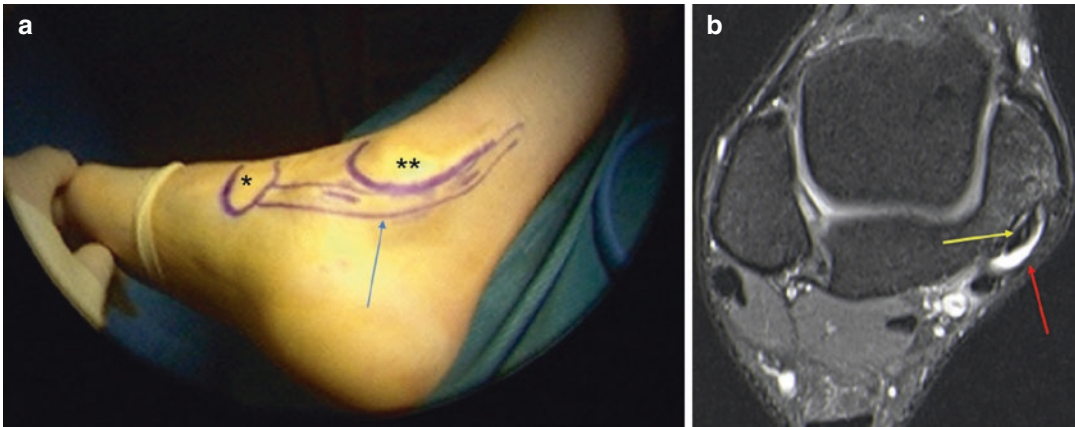


Fig. 34.1 (a) Surface anatomy with *blue arrow* representing hypovascular zone along with posterior tibial tendon course, (*asterisk*) navicular bone, and (*double*

asterisk) medial malleolus. (b) MRI axial view with intra-tendon signal changes in the hypovascular zone (*yellow arrow*) and fluid within its sheath (*red arrow*)

must be acknowledged. Ultrasound is known to be operator dependent while providing dynamic evaluation (Nallamshetty et al. 2005). MRI is considered diagnostically specific but not highly sensitive for some tendon-related disorders (Park et al. 2012; van Sterkenburg et al. 2010b). Local anesthetic injection might be helpful to confirm the origin of pain (Cooper et al. 2007). CT scans are particularly useful in the study of bony structures which might be suspected (e.g., navicular deformities, bone ossicles/spurs). Endoscopic/tendoscopic evaluation provides direct inspection and is gaining popularity in either diagnostic or therapeutic approaches (van Sterkenburg et al. 2010b; Park et al. 2012).

Shoewear inspection is also required either because of its possible contribution to pathology (e.g., shoes-related compression might cause inflammatory reaction) or because it might reflect some conditions (e.g., uneven wear of shoe soles might reflect malalignment) (Kulig et al. 2009).

Listening to the patient's complaints is critical. The targets are to identify its cause, worsening, and relief factors and understand its functional implication. Moreover, clinicians must understand patient's expectations and provide "realistic" information of therapeutic options.

Herein, the most common conditions affecting anterior tibial tendon (ATT) and PTT will be discussed. Despite being rare, simultaneous

affections of both tendons resulting in severe flat-foot and dropfoot gait have been described requiring surgical treatment (Frigg et al. 2006).

The concepts of tendinopathy are described elsewhere and thus are out of the scope of this work.

34.2 Posterior Tibial Tendon Pathology

34.2.1 Clinical Anatomy

Tibialis posterior muscle arises from the posterior aspect of the tibia and fibula and from the interosseous membrane. PTT passes immediately behind the medial malleolus, through a fibrous tunnel which is covered by the flexor retinaculum. After contouring the malleolus, the tendon begins to fan out. It has a wide insertion including the navicular, the *sustentaculum tali*, first cuneiform bones, and the bases of the second, third, and fourth metatarsals. The PTT is an important dynamic stabilizer of the medial arch and the most powerful inverter of the foot (Gluck et al. 2010). The spring ligament complex is the static soft tissue support of the talonavicular joint and also plays a key role in its biomechanics (Boss and Hintermann 2002). The PTT does not have a mesotenon.

A retromalleolar hypovascular region (Fig. 34.1) has been observed and can be implicated in degenerative changes of the tendon (Frey et al. 1990; Manske et al. 2015). The posterior tibial tendon is usually supplied by two vessels entering the tendon approximately 4.5 cm proximal and 2.0 cm distal to the medial malleolus (Manske et al. 2015).

34.2.2 Etiology and Pathogenesis

Inflammatory, degenerative, functional, and traumatic processes might lead to PTT dysfunction at different levels (Yao et al. 2015). Inflammatory diseases (e.g., lupus or rheumatoid arthritis) are more common in younger patients (Myerson 1997; Otis and Gage 2001). Chronic overuse and consequent tendon deterioration have been noticed to occur more often and more frequently in late to middle age, women, and obese (Myerson 1997; Otis and Gage 2001). However, degeneration due to overtraining with repetitive micro-trauma has also been described among young athletes, particularly in running and sports requiring repeated and rapid changes in direction (Ribbans and Garde 2013; Supple et al. 1992). Inflammatory changes, tendon tears, and tenosynovitis have been connected to activity levels around 1500 to 2000 cycles per hour (Bare and Haddad 2001).

Other risk factors include hyperpronation or anomalous anatomy, ligamentous laxity, diabetes mellitus, hypertension, and corticosteroid therapy (Yao et al. 2015). Moreover, Probasco et al. recently stated that an increased valgus orientation of the subtalar joint is more frequent among patients with adult acquired flatfoot when compared to controls (Probasco et al. 2015). Acute trauma is rarely the cause of tendon dysfunction or rupture (Trnka 2004).

The precise mechanisms leading to PTT degeneration remain unclear. The combination of the hypovascular zone and abnormal mechanical forces experienced behind the medial malleolus where the tendon acutely changes direction in several activities might play a role (Trnka 2004).

The summary of sequential events might be summarized: loss of posterior tibialis function results in collapse of the medial longitudinal arch and increased stress in the medial structures of the foot. Gradually, there is weakening of the medial ligaments: the spring ligament (connects the anterior margin of the *sustentaculum tali* to the plantar surface of the navicular) gradually ails with repetitive loading, leading to a flatfoot deformity. The deltoid ligament also might become insufficient, permitting the talus to tilt into valgus. In the hindfoot valgus, the Achilles tendon becomes an evertor due to its position lateral to the axis of the subtalar joint and also might become shortened/contracted, resulting in an equinus deformity.

34.2.3 Clinical Presentation and Imaging

A patient suffering from PTT tendinopathy might refer isolated posteromedial ankle pain. The natural history is usually slow, with insidious onset of complaints which can cause delay in searching for assistance. However, on clinical examination one can find local tenderness, medial pain (sometimes irradiating to the calf), positive PTT provocation test, or inability to walk on tiptoes. Over time, patients may notice progressive collapse of the medial longitudinal arch (acquired flatfoot) and increased hindfoot valgus. The shoe soles may show signs of increased wear on the medial side, and they may report difficulty standing on their toes due to pain and weakness.

Pain usually worsens with walking and progressively affects sports and activities of daily living (Trnka 2004). On later stages of the disease, patients often report that pain shifts laterally as the fibula begins to impinge against the calcaneus while the medial pain often disappears (Yao et al. 2015). At these stages sports activities are severely compromised if possible at all.

Examination (Fig. 34.2) should begin with inspection through all sides while standing (including podoscope for plantar view). The examiner should search for increased valgus angulation of the hindfoot and abduction of the



Fig. 34.2 (a) X-ray lateral view with characteristic changes of adult flatfoot; (b) lateral view of the foot with visible collapse of medial arch; (c) posterior view with hindfoot valgus and “to many toes sign” on the *left* foot opposing to neutral alignment of the *right* foot (*red lines*

represent hindfoot alignment); (d) the valgus alignment is not corrected when raising the heels; (e) positive single “heel raise test” on the *left* foot. The patient is incapable to raise and experiences pain, and the valgus does not correct during the attempt

forefoot. Both these features are reflected in a positive “too many toes” sign, in which more toes are visible on the lateral side of the affected foot when viewed from behind given the increased hindfoot valgus and forefoot abduction (Johnson 1983).

Next, the “heel-rise test” (Fig. 34.2) is performed to assess severity of the condition (Johnson 1983). The patient is asked to attempt to rise onto the ball of the affected foot while keeping the contralateral foot lifted off the ground. The normal foot is also tested for comparison. A positive test is considered if the affected hindfoot will either remain in valgus abduction during the heel raise (once the PTT fails to invert it) or the patient will refer important pain limiting this movement. It might be required to repeat the test a few times for proper assessment. Hintermann and Gächter have described another useful test: the first metatarsal rise sign (Hintermann and Gächter 1996). The patient is asked to stand in full weight bearing on both feet. The examiner will then externally rotate the shin of the affected foot with one hand. In the presence of marked PTT insufficiency, the head of the first metatarsal will be lifted off the floor (fixed supination of the first ray).

Subsequent clinical examination can be managed with the patient seated. The PTT should be palpated throughout its course to assess for a palpable gap, crepitation, tenderness, or swelling. The lateral side of the foot should also be palpated once subfibular tenderness may be an indication of calcaneofibular impingement. Palpation and strength assessment should be repeated with resisted inversion and compared to the opposite side. The foot is held in slight plantarflexion and eversion to isolate the posterior tibialis from the synergistic action of tibialis anterior (Myerson and Corrigan 1996). 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 painful site. The mobility of the ankle and the subtalar joint should be carefully assessed given the implication in the method of treatment. Range of motion at the subtalar joint will progressively decrease with the progression of the condition, and eventually the hindfoot will assume a

fixed valgus deformity. The forefoot and midfoot will try to compensate for this by progressively adopting a supinated position, which is best appreciated with the heel in a neutral position. This is another critical part of the examination because a fixed supination deformity of the forefoot is an important consideration in the subsequent selection of treatment (Yao et al. 2015).

It is also mandatory to assess the Achilles tendon for contracture. This is often associated with chronic hindfoot valgus once the Achilles tendon adopts an abnormal position lateral to the axis of the subtalar joint, leading to tendon shortening over time (Yao et al. 2015).

Radiographies assess global foot and ankle morphology and alignment and are helpful in later stages of these disorders. In early stages, they are often normal.

Standing anteroposterior and lateral view (Fig. 34.2) radiographs of both feet and ankles as well as mortise views of the ankle joint should be ordered. The collapse of the longitudinal arch on a lateral weight-bearing radiograph might be noticed in any of the following measurements, especially if asymmetric: calcaneal inclination angle (angle between the calcaneal inclination axis and the supporting surface, considered low if less than 20°), talometatarsal angle (angle between the axis of the talus and the axis of the first metatarsal, normally between 0° and 10°), or the distance of the medial cuneiform from the floor (normally between 15 and 25 mm) (Slovenkai 1997).

Uncovering of the talar head might be recognizable on an anteroposterior view. With increased forefoot abduction deformity, the navicular slides laterally, leading to subluxation of the talonavicular joint which is abnormal if more than 15% of the talar head is uncovered (Slovenkai 1997). Plain radiographs might also show talar tilt; calcaneofibular impingement; arthritic changes of the tibiotalar, subtalar, and talonavicular joints; and signs of enthesopathy in the form of bony irregularities and hypertrophic changes at the navicular insertion site of the posterior tibialis tendon (Kong and Van Der Vliet 2008).

MRI (Fig. 34.3) and ultrasound, despite known limitations in low-grade PTT disease, are

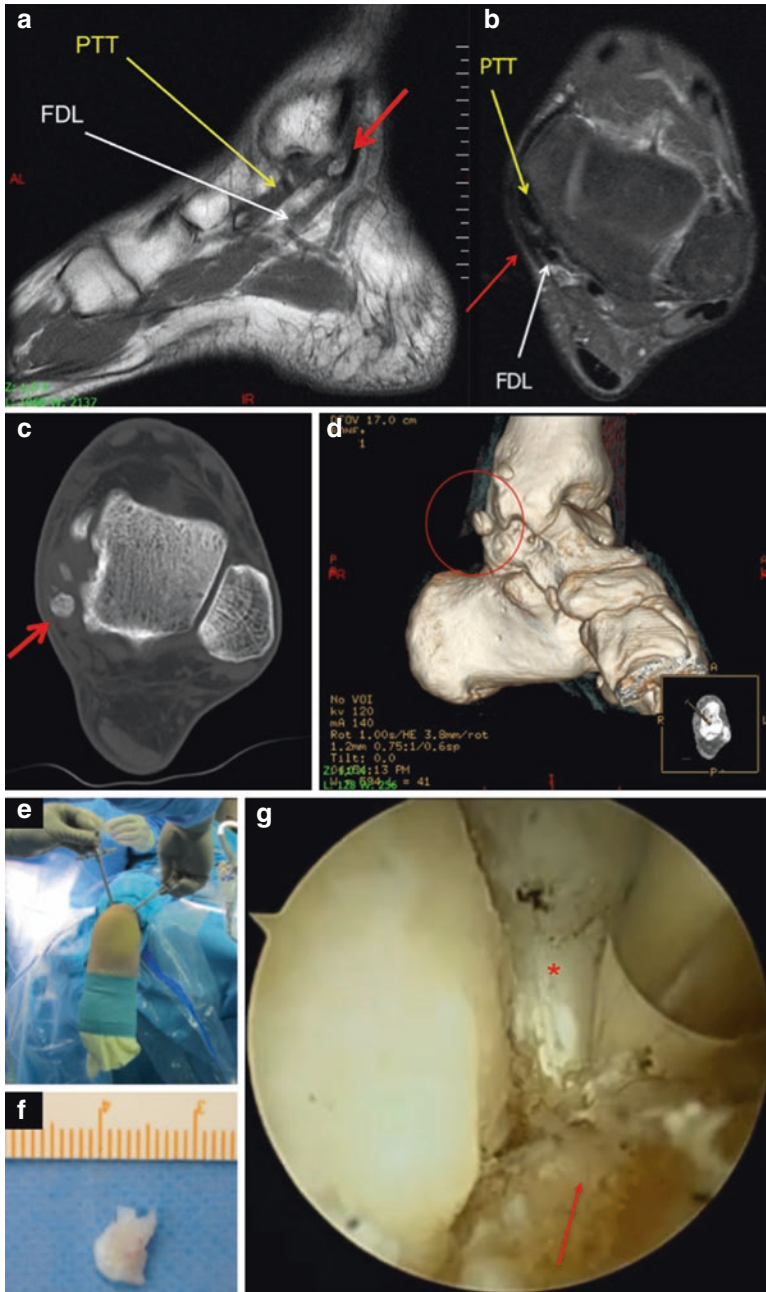


Fig. 34.3 (a and b) represent MRI views (sagittal and axial, respectively) of posterior tibial tendon (PTT) and flexor digitorum longus (FDL). (c and d) show CT axial and 3D images. Red arrows and red circle indicate bone ossicle causing tendon's irritation. (e) shows posterior

ankle arthroscopic approach. (f) The removed ossicle. (g) Arthroscopic view where PTT (red *) and the ossicle (red arrow) are visible, with distal tibia on the left and surgical instrument on the right

useful in identifying different pathologies, such as tenosynovitis (longitudinal) ruptures, degenerative changes, or adhesions (Cooper et al. 2007; Lhoste-Trouilloud 2012). Moreover, MRI can be useful in identifying spring and deltoid ligament injuries (Kong and Van Der Vliet 2008; Nallamshetty et al. 2005).

CT scan is useful in identifying bone ossicles (Fig. 34.3) and deformities, namely, secondary to fractures which might cause persistent damage to the tendon (Kong and Van Der Vliet 2008).

When dealing with diagnosis of PTT disorders, one must also consider and rule out subtalar arthritis, Müller-Weiss disease, Lisfranc joint pathology, consequences of fractures or bone ossicles, PTT subluxation, coalition, Charcot arthropathy, spring ligament injury, and deltoid ligament injury.

34.2.4 Principles for Treatment

Johnson and Strom proposed a classification system correlating the severity of PTT dysfunction

and subsequent adaptations of the foot to treatment recommendations (Johnson and Strom 1989). This classification was further refined by Myerson (Myerson 1997) and Bluman (Bluman et al. 2007) (Table 34.1 summarizes one of the most popular classification systems). Further classifications have been proposed, and all try to correlate major clinical findings and treatment options (Haddad et al. 2011). Treatments of PTT disorders include conservative and surgical options depending on the severity of the condition.

In the early stages of the disease, conservative treatment is recommended. This comprises modification of activity, cryotherapy, physiotherapy, medication, insoles, corrective shoes (medial heel and sole wedge), and orthosis (Kulig et al. 2009).

In common patients, when a trial of 3–6 months of conservative treatment has failed with progression of symptoms (Ribbans and Garde 2013; Bare and Haddad 2001), tendoscopy represents an option with increased popularity (Khazen and Khazen 2012; van Dijk et al. 1997). Tendoscopy (Fig. 34.4) is considered in grade I

Table 34.1 PTT disease classification of Johnson and Strom (1989) modified by Myerson and Bluman (Bluman et al. 2007)

	Clinical findings	Physical examination	Radiographs
Stage I	<ul style="list-style-type: none"> • Tenosynovitis • No deformity 	<ul style="list-style-type: none"> • Single-heel raise+ • Too many toes sign(–) 	<ul style="list-style-type: none"> • Normal
Stage IIA	<ul style="list-style-type: none"> • Flatfoot deformity • Flexible hindfoot • Normal forefoot 	<ul style="list-style-type: none"> • Single-leg heel raise(–) • Too many toes sign+ • Mild sinus tarsi pain 	<ul style="list-style-type: none"> • Medial arch collapse deformity
Stage IIB	<ul style="list-style-type: none"> • Flatfoot deformity • Flexible hindfoot • Forefoot abduction (“too many toes;” >40% talonavicular uncoverage) 		
Stage IIC	<ul style="list-style-type: none"> • Flatfoot deformity • Flexible hindfoot • Forefoot abduction • Medial ray instability (not corrected with ankle plantarflexion) 		
Stage III	<ul style="list-style-type: none"> • Flatfoot deformity • Rigid forefoot abduction • Rigid hindfoot valgus 	<ul style="list-style-type: none"> • Single-leg heel raise(–) • Too many toes sign+ • Severe sinus tarsi pain 	<ul style="list-style-type: none"> • Medial arch collapse deformity • Subtalar arthritis
Stage IV	<ul style="list-style-type: none"> • Flatfoot deformity • Rigid forefoot abduction • Rigid hindfoot valgus • Deltoid ligament injury 	<ul style="list-style-type: none"> • Single-leg heel raise(–) • Too many toes sign+ • Severe sinus tarsi pain • Ankle pain 	<ul style="list-style-type: none"> • Medial arch collapse deformity • Subtalar arthritis • Talar tilt

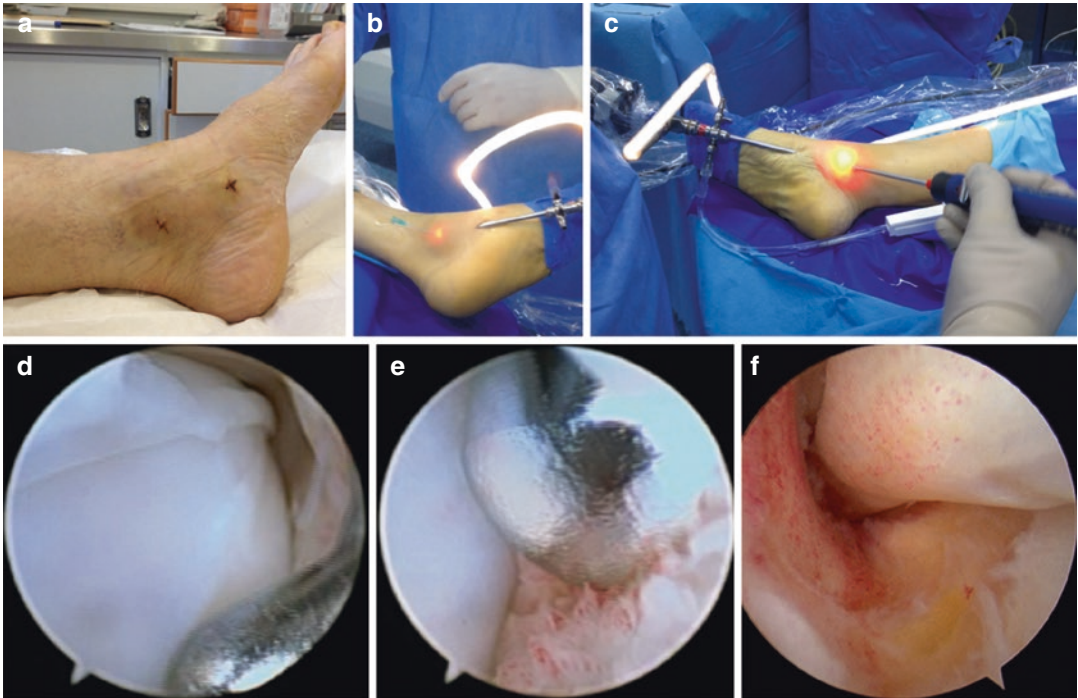


Fig. 34.4 Posterior tibial tendon tendoscopy (PTT) with portal placement (a), arthroscope introduced by the distal portal and proximal portable created using transillumination and a needle (b). With arthroscope and shaver (c) the

debridement of partial longitudinal ruptures (d) or synovectomy is possible (e). Inflammatory changes in the synovia and the tendon are often found in rheumatoid patients (f)

(Khazen and Khazen 2012; Chow et al. 2005; van Dijk et al. 1997) and selected cases of early grade II cases (Lui 2007). However, one must consider that particularly in patients with inflammatory diseases, the process of tendon degradation secondary to synovitis might be more aggressive, and surgical treatment is considered earlier (6-week trial of conservative treatment) (Myerson et al. 1989).

More complex approaches include the combined treatment of spring and deltoid ligaments as recently described by Lui et al. (Lui 2015, 2016a). Posterior arthroscopic approach (Fig. 34.3) has also been described and might also be used for the removal of bone ossicles in post-traumatic cases (Hua et al. 2015). However, these approaches should be further validated by clinical evidence before widespread recommendation.

Currently, indications for posterior tibial tendon tendoscopy (van Dijk et al. 1997; Pereira et al. 2014) include:

- Tenosynovectomy
- Tendon sheath release
- Tendon debridement and cleaning of partial rupture
- Resection of pathological vincula
- Removal of exostosis/irregularity of posterior tibial sliding tunnel
- Endoscopic removal of implants (screws/anchors) from medial malleolus
- Adhesiolysis
- Diagnostic procedure

PTT tendoscopy enables diagnostic confirmation of symptomatic partial tendon tear (false positives and false negatives have been described on MRI and ultrasound) (van Sterkenburg et al. 2010b). Partial tendon tears can be definitively treated endoscopically. However, if that is not the case, it will help to diminish open surgical approach thus contributing in the worst-case scenario to lower the incision, diminish postoperative

pain, and contribute to earlier rehabilitation. The tendoscopic procedure can be converted to a “guided” mini-open approach, which is still less invasive than the standard open procedure (Pereira et al. 2014).

34.3 Technique for Posterior Tibial Tendoscopy (van Dijk et al. 1997)

Patient is positioned supine. For superficial landmarks the patient should be questioned before being anesthetized to actively invert the foot. In this way, the painful site can be identified as well as the course of the PTT facilitating the placement of the portals. The distal portal is performed first, 2.5 cm distal to the posterior edge of the medial malleolus. A 2.7 or 4.0 mm arthroscope is introduced, and the complete tendon sheath should be inspected. The proximal portal is made under direct vision. Tissue debridement by means of shaver or tendon repair by using recently developed suture tools can be used. At the end of the procedure, the portals are sutured, and a bandage is applied. Active range of motion of the ankle (plantarflexion-dorsiflexion) is encouraged since the first day. Partial weight bearing is advised for 2–3 days and gradually resumption of daily activities as tolerated. Sutures are removed from 10 to 14 days.

34.4 Operative Treatment Guidelines

The goals for treatment depend on the severity of the disease and include: control of symptoms, restore function, stop progression of the deformity, and obtain adequate alignment.

- Stage I—Endoscopic or open (McCormack et al. 2003) tendon debridement or synovectomy (Fig. 34.4) is a valid option in symptom controls after failed conservative treatment. Tendon repair with or without augmentation by flexor tendon transfer (most commonly using the flexor digitorum longus) might be considered (Vora et al. 2006). Some authors combine medial sliding calcaneal osteotomy (Fig. 34.5) when in the presence of a flatfoot (Vora et al. 2006).
- Stage II—Upon PTT insufficiency and subsequent deformity, flexor tendon transfer (Fig. 34.6) combined with a calcaneal osteotomy (most commonly of the medial slide type) is used. In the presence of a fixed elevation of the first ray, the medial cuneiform open-wedge osteotomy (Cotton osteotomy) is considered (Aiyer et al. 2016). If the first ray is unstable, a first metatarsal-tarsal fusion is considered. Fusion of the navicular cuneiform joint is rarely required and difficult to achieve (Mosier-LaClair et al. 2001). Treatment of Stage IIB

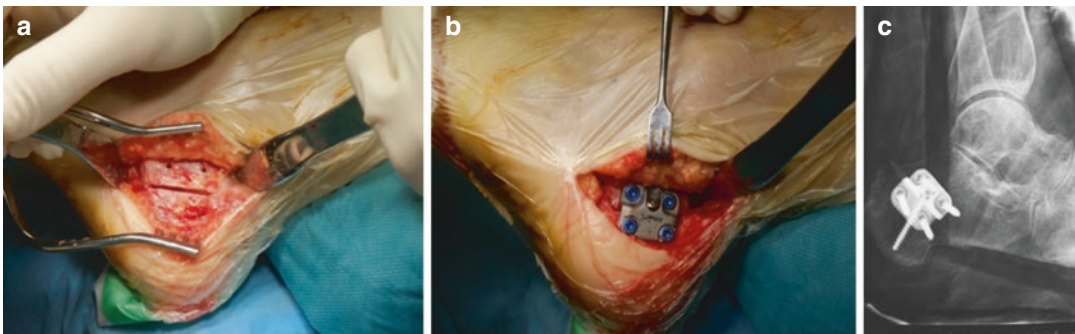


Fig. 34.5 Surgical approach (a and b) and x-ray (c) of medial sliding calcaneal osteotomy

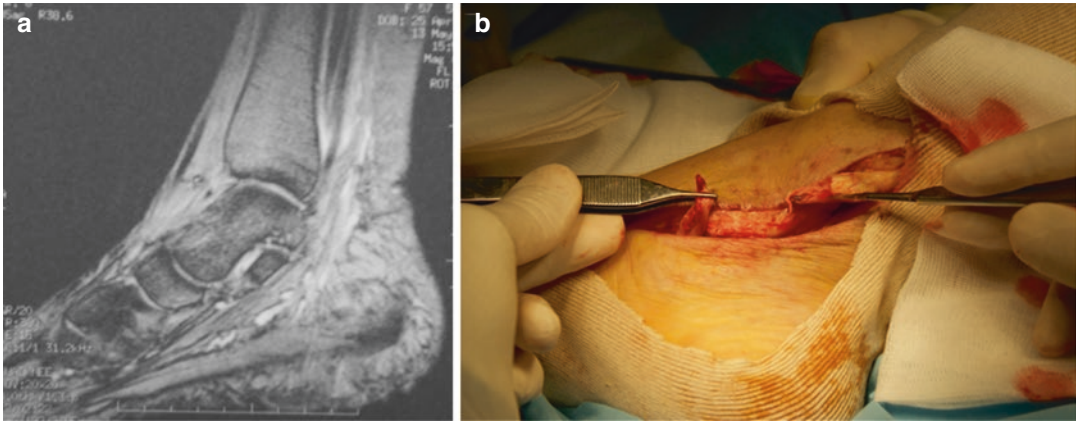


Fig. 34.6 Posterior tibial tendon rupture on MRI (a) and direct surgical repair (b) which was further augmented by *flexor digitorum longus* transfer

deformity is particularly controversial, with some clinicians performing a lateral column lengthening for this stage while others choose other options (Lee 2005). The amount of abduction requiring a lateral column lengthening has not been established (Chi et al. 1999). Besides the previously described, spring ligament repair/reconstruction can also be used when required (Palmanovich et al. 2015). The formerly described correction can be combined with arthroereisis in selected cases (Soomekh and Baravarian 2006). Subtalar fusion with correction of alignment represents another valid option in more severe cases (Yao et al. 2015).

- Stage III—The most frequent treatment at this stage involves a triple arthrodesis (Catanzariti et al. 2014) (triple joint complex—subtalar, talonavicular, and calcaneocuboid). Overcorrection of deformity should be avoided. The talonavicular joint should be fused in neutral position without excessive abduction, but also without supination (varus of the midfoot and forefoot). The metatarsal heads should be even to the floor with the heel in neutral position. If the excessive heel valgus remains after triple arthrodesis, a medial heel slide is added to provide good alignment and avoid excessive stress on the deltoid and progression to Stage IV.

- Stage IV—There is limited clinical evidence on the outcome of Stage IV patients (Deland et al. 2004). Correction of alignment in the foot is critical. Flexible deformities in the foot, if present, should be corrected as described for Stage II. Correction of fixed deformity should be addressed as described for Stage III. Consideration for reconstruction of the deltoid ligament with tendon graft can be done and may give correction to the alignment at the ankle (Catanzariti et al. 2014). When symptomatic ankle arthritis develops, ankle fusion is required to relieve pain (Toullec 2015). Total ankle replacement is also an option (Ketz et al. 2012). However, inadequate function of the deltoid ligament after total ankle replacement can result in failure of the arthroplasty (Toullec 2015; Yao et al. 2015).

As previously described, in all stages, assessment of the Achilles tendon is critical. If shortened, an Achilles lengthening or gastrocnemius recession should be considered. Furthermore, we emphasize that instability or hypermobility of the first ray must also be considered. If that is the case, a first metatarsal-tarsal fusion or Cotton osteotomy should be considered (Yao et al. 2015).

34.5 Anterior Tibial Tendon Pathology

34.5.1 Anterior Tibial Tendon (ATT) Clinical Anatomy

The tibialis anterior muscle (Fig. 34.7) originates from the lateral tibial condyle and interosseous membrane. The musculotendinous junction occurs at the transition of the middle and distal thirds of the tibia. The ATT has its

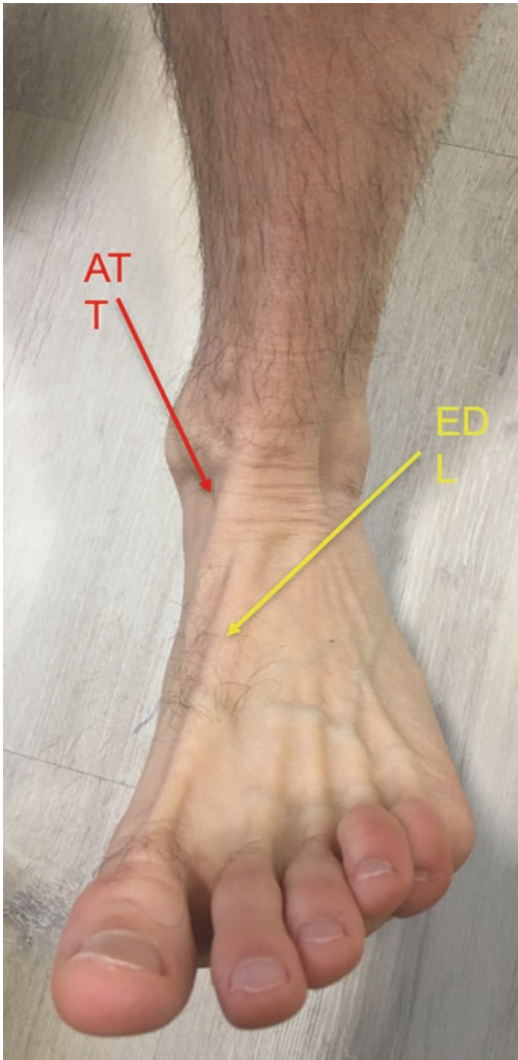


Fig. 34.7 Surface anatomy where anterior tibial tendon (ATT) and extensor digitorum longus (EDL) are identified

main insertions at the medial cuneiform and the inferomedial base of the first metatarsal; however, different types of distal insertions have been described in the literature (Varghese and Bianchi 2014). The ATT courses within a synovial sheath during most of its course, deep to the extensor retinaculum of the ankle and foot. It is the strongest dorsiflexor of the foot (accounts for more than 80% of the strength required for this movement) while also contributing to inversion (Ouzounian and Anderson 1995). It also controls deceleration of the foot after heel strike. It is innervated by the deep peroneal nerve. Peterson et al. described a zone of hypovascularity (Petersen et al. 2000); however, Geppert et al. had opposed to this theory stating that ATT has no sustainable hypovascular zones (Geppert et al. 1993). It receives vascular supply from the anterior tibial artery proximally and the medial tarsal arteries distally. The ATT's bursa is found close to its insertion, between the tibialis anterior tendon and the medial cuneiform bone.

34.5.2 Etiology and Clinical Presentations

Lesions of the tibialis anterior muscle and tendon are not frequently found in international literature (Varghese and Bianchi 2014). However, most likely being unreported, tibialis anterior tendinopathy or rupture might not be that rare. Pathology can be spontaneous, trauma related, associated with arthropathy or more generalized conditions (Varghese and Bianchi 2014). Either local or systemic diseases can be considered as risk factors: corticosteroid injection, diabetes mellitus, hyperparathyroidism, or aging. In most cases, a careful history and a thorough physical examination are sufficient for achieving the diagnosis. Clinical assessment alone, however, may not be sufficient for distinguishing conditions like tendinopathy, tears (Fig. 34.8), bursitis, calcifications, and cysts (Fig. 34.8), among others (Ouzounian and Anderson 1995). Insertional tendinopathy has been described, more often in ballet dancers or jumpers. Tendinopathy might derive from repetitive compression from footwear

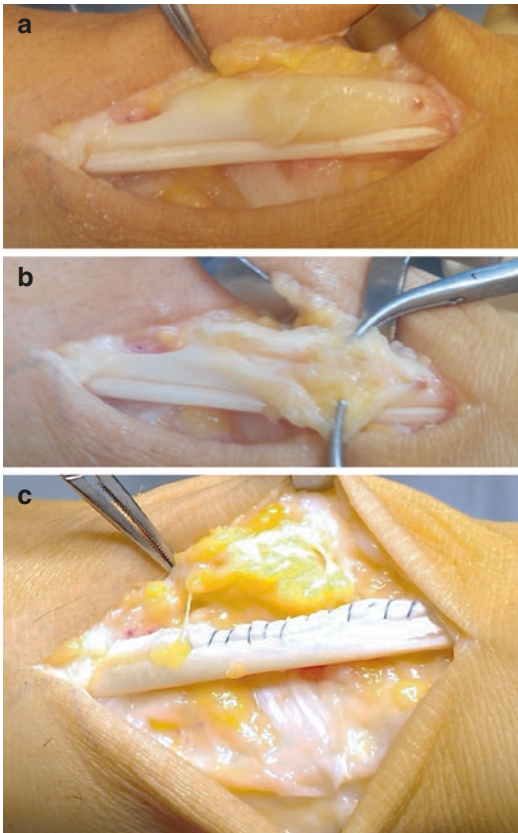


Fig. 34.8 Cystic degeneration with rupture of the anterior tibial tendon (a and b). After debridement, tubulization and direct repair have been performed (c)

or overuse leading to inflammatory changes. Patients describe pain on the medial cuneiform particularly when loading the foot immediately after heel strike or during swing phase of gait (Ritter and Moore 2008). Bursitis can occur as a result of excessive local friction, infection, or direct trauma.

The tibialis anterior tendon may rupture spontaneously or by open or closed trauma. TAT rupture may present as an acute lesion or as a chronic, painless foot drop which is usually late diagnosed (Varghese and Bianchi 2014). Younger patients without degenerative tendon changes rarely suffer spontaneous rupture. Their most frequent injury mechanism is laceration following penetrating trauma or tibial fracture (Sammarco et al. 2009). Spontaneous ruptures typically occur in older patients with tendon degeneration. Minor

trauma by plantarflexion-eversion might be the precipitating event.

Clinical examination includes inspection for swelling or palpable mass. Closed tibialis anterior ruptures might present with a bulbous mass at the anterior medial aspect of the ankle joint due to the proximal retraction of the ruptured tendon (Dooley et al. 1980). A gap might be felt indicating rupture. Analysis of gait pattern in chronic ruptures might range from slight changes, with limitation only on uneven ground, up to slap-foot gait or foot drop. Incapacity to walk on heels suggests ATT dysfunction. During the swing phase of gait patients might hyperflex the hip and knee to compensate loss of ankle dorsiflexion. Diminished ankle dorsiflexion strength is another sign. When asked to dorsiflex the ankle, patients will exhibit toe hyperextension. In chronic cases, Achilles contracture might also occur. A minimum of 10° dorsiflexion must be preserved in order to permit ATT repair. For this reason, in some cases, repair is combined with Achilles lengthening (Ouzounian and Anderson 1995).

Radiography can be useful in conditions like *myositis ossificans* or to assess arthropathy. Magnetic resonance imaging, due to its excellent tissue contrast, allows simultaneous assessment of the muscle, joint, and bone. It remains a second-line study due to its high cost (Varghese and Bianchi 2014). Ultrasound, which enables dynamic evaluation, despite the fact that it is operator dependent, can also be useful for final diagnosis (Varghese and Bianchi 2014).

Differential diagnosis includes peroneal nerve palsy, lumbar radiculopathy, or peripheral neurologic conditions affecting ATT function.

34.5.3 Principles for Treatment

Concerning ATT tendinopathy, the principles of treatment are common to other tendons. Conservative treatment is the first line of treatment. The former includes rest, ice, immobilization, analgesics, appropriate physiotherapy protocol, change in footwear, injection therapy, etc. Upon failure of conservative treatment,

endoscopy is being presented as a promising possibility, including bursectomy, synovitis, or debridement (Lui 2016b). However, in this less reported group of pathology, increased experience and evidence are required before more definitive conclusions.

Although the treatment for ATT ruptures is somewhat controversial, most literature recommends conservative management for most long-standing chronic ruptures, as well as ruptures occurring in the elderly or sedentary patients (Funk et al. 2016). Treatment of this clinical entity must be tailored to the individual.

Conservative therapy may consist of shoe modifications, bracing, and a non-weight-bearing short leg cast with the foot in a dorsiflexed and inverted position for 4 to 6 weeks. An ankle foot orthosis may be indicated in these patients after therapy due to some loss in muscle strength and ankle joint dorsiflexion (Markarian et al. 1998).

Surgical management is indicated for younger, more active patients, who present an acute ATT rupture, or preferably within 3 to 4 months after injury (Trout et al. 2000; Christman-Skieller et al. 2015). The age of the patient, functional status and demands, stage of rupture, and chronicity should be considered when developing a treatment plan (Trout et al. 2000; Christman-Skieller et al. 2015).

Some authors recommend direct primary repair for acute tibialis anterior ruptures (Dooley et al. 1980; Markarian et al. 1998). Primary tendon repair may be accomplished with nonabsorbable suture (Fig. 34.8) using Bunnell, Krakow, or modified Kessler suture techniques (Harvey and Rockett 2000). Fixation using EndoButton® has also been described (Funk et al. 2016). In case it is not possible to perform a direct suture of the tendon or reinsert the tendon (Mao and Xu 2015) into its native insertion, a tendon reconstruction technique can be used. The extensor retinaculum should always be reconstructed to prevent the bowstringing phenomenon and cicatricial adhesions to the subcutaneous tissue (Sammarco et al. 2009). After surgery, a period of 6 weeks of immobilization with plaster cast is recommended before the patient begins active mobilization and physical therapy rehabilitation.

Several surgical techniques have been described to treat anterior tibial tendon ruptures. Available options include direct primary repair (less viable in chronic cases), augmentation by tendon grafts, tendon transfers, and tendon reconstruction. Considering the latter, Trout et al. utilized the central one third of the anterior tibial tendon to bridge the ruptured gap (Trout et al. 2000). The flap was rotated posteriorly and distally and tenodesed into the medial cuneiform using a 4–0 mm cancellous screw with washer. Another option is interposing an allograft (Huh et al. 2015) or autologous graft, such as the *plantaris* tendon, *extensor digitorum longus* tendon, *peroneus tertius* tendon, Achilles tendon, or semitendinosus tendon (Christman-Skieller et al. 2015). *Extensor hallucis longus* transfer to medial cuneiform is probably the most popular tendon transfer/augmentation technique (Sammarco et al. 2009; Christman-Skieller et al. 2015).

Studies have shown that patients treated surgically were more likely to develop a normal gait, with more dorsiflexion strength and motion compared to patients treated conservatively (Markarian et al. 1998; Christman-Skieller et al. 2015). Surgical intervention appears to achieve a better functional outcome when compared to conservative treatment. Mild to moderate flatfoot deformity, decrease in ankle joint range of motion, lack in coordination or a slapping type gait, and Achilles tendon contraction were more frequent among patients treated conservatively (Trout et al. 2000).

However, we must emphasize the paucity of the available literature and methodological limitation of the small sample series reported.

34.6 Injection Therapy in Tendinopathies

Injection treatments for controlling painful tendon-related disorders (Fig. 34.9) have been increasing its popularity, particularly among populations with higher functional demand.

Through time, several agents have been studied such as corticosteroids (Johnson et al. 2011),



Fig. 34.9 Injection therapy around in the posterior tibial tendon (PTT) sheath. The PTT is visible (*white circle*) by asking the patient to actively invert the foot against resistance from the examiner. Ultrasound can be a useful tool to perform these treatments under visual control

polidocanol, platelet-rich plasma (PRP), high-volume injections, hyperosmolar dextrose, brisement, aprotinin, and low-dose heparin (van Sterkenburg and van Dijk 2011).

One must understand that each agent and technique presents its specific implications, and until now there is no such thing as a securely effective and harmless percutaneous “panacea” capable to cure all sources of pain.

There is controversial data in literature related to percutaneous treatment/prolotherapy. Often, the cause-effect rationale supporting its application, particularly in tendons, is not completely understood, so despite being open-minded, we must evaluate them with necessary caution.

Many limitations persist concerning clinical and biological aspects of tendon healing. The main goal in tendon repair is to achieve a faster rehabilitation with tissue healing with similar or better characteristics than those of normal tendons. Tissue engineering and regenerative medicine research envisions new perspectives for the future (Hogan et al. 2011).

Corticosteroids are powerful anti-inflammatory medications which were previously injected around tendons. Its use in tendinopathies is currently discouraged given the risk of subsequent tendon degeneration and/or rupture (Speed 2001).

Polidocanol has been proposed as a method to abolish neovascularity within and around inflamed

tendons (Ohberg and Alfredson 2002). However, the role of this neovascularization phenomenon is not completely comprehended. Moreover, there have been inconsistent results obtained from the method (van Sterkenburg et al. 2010a).

Currently, the most popular injectable therapy is probably PRP in one of its different preparations (with inherent different effects) (Papalia et al. 2012). It proposes a wide range of favorable effects in several tissues including tendon pathologies, muscle injuries, or even cartilage and joint arthritis. However, besides its proven safety profile, there has been some controversy around clinical outcome (Tinsley et al. 2012). Results have been obtained from pathologies, considering different tissues and different preparations of PRP (e.g., with/without leukocyte and platelet concentration) which limits the possibility for further assumptions regarding clinical use (Martinez-Zapata et al. 2012). It should be acknowledged that this is a most promising technique. Growth factors have provided consistent laboratorial results; however, it has not been easy to replicate such results in the clinics (Boswell et al. 2013). Improved research methodology and more clinical trials assessing outcome of PRP technology are still required (Engebretsen and Schamasch 2012).

“It is necessary to develop appropriate guidelines and increase evidence-level prior to its widespread application as treatment option for joint” (Sheth et al. 2012).

Conclusions

Tendinopathies around the foot and ankle are frequently associated with overuse and/or traumatic events. Anterior and posterior tibial tendon pathologies require a complete understanding of foot and ankle anatomy and biomechanics. Tendoscopy has been significantly developed in recent years. Posterior tibial tendon insufficiency requires diagnosis on an early phase of the disease in order to diminish further consequences and limitations. In later stages the prognosis is significantly worst and therapeutic options more aggressive and of more uncertain outcome.

On the other hand, ruptures of the anterior tibial tendon are unusual injuries but probably occur with greater frequency than reported. The fact that there is regularly a delay of several months in the diagnosis of ATT rupture indicates the need for an increased awareness of this entity. Ruptures of the ATT might have significant implications on gait and ambulation. The proper treatment option must be tailored to individual aspects. However, there seems to be improved outcome in the surgically treated group.

Injection therapy is under development aiming to control degeneration and achieve or enhance healing, particularly in the early stages of tendon's disease.

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