

Painful Flatfoot Deformity

Bolestivé plochonoží

M. WIEWIORSKI, V. VALDERRABANO

Orthopaedic Department University Hospital Basel, Basel, Switzerland

SUMMARY

The posterior tibial muscle is the key dynamic support of the medial longitudinal arch of the foot. When it fails –typically in women older than 40 years of age– progressively, the arch slowly collapses, the heel drifts into valgus, the midfoot flattens, and the forefoot gradually abducts and supinates, resulting in painful acquired pes planovalgus abductus et supinatus. Posterior tibial tendon insufficiency (PTTI) is often misdiagnosed as a chronic ankle sprain, osteoarthritis, or collapsed arch as a result of aging or obesity, and it leaves the patient debilitated. Prompt diagnosis prevents frustration for the patient and allows treatment to be started at an earlier, more easily managed stage. The diagnosis of PTTI is largely a clinical one. An increased awareness of the existence of PTTI should serve to help patients with earlier referral and treatment and by limiting the amount of disability.

INTRODUCTION

Posterior tibial tendon insufficiency (PTTI) is accepted as the most common etiology of the acquired adult flatfoot deformity. In spite increased awareness and intensive research over the past decade, PTTI is still frequently missed. Because this slowly progressing condition is best treated at early, easier manageable stage, prompt diagnosis is necessary. PTTI is characterized by hindfoot valgus, flattening of the longitudinal foot arch, and abduction and supination of the forefoot (29, 38). This occurs most commonly in women older than 40 year of age. Since the first description of a posterior tibial tendon rupture by Key et al. in 1953 (15), methods of treatment have evolved and the pathology and function of the tendon have been extensively investigated. This article will review the current concepts with regard to the pathomechanism, diagnostics, and treatment strategy of this debilitating condition.

Anatomy and Biomechanics of the posterior tibial muscle

The posterior tibial muscle arises from the posterior surface of the interosseous membrane, the lateral portion of the posterior surface of the body of the tibia, and from the upper medial surface of the fibula. It is the most deeply seated muscle of the deep posterior compartment in the back of the leg. In the lower fourth of the leg its tendon passes in front of that of the flexor digitorum longus and lies with it in a groove behind the medial malleolus in a separate tendon sheath. It then passes under the flexor retinaculum of lower limb and over the deltoid ligament into the foot, and then beneath the spring ligament (calcaneonavicular ligament) inserting into the tuberosity of the navicular bone. Finally, it inserts with

multiple expansions spreading out across the plantar aspect of the hind- and midfoot (sustentaculum tali of the calcaneus, forward to the medial cuneiform and first metatarsal bone, and lateralwards to the other cuneiform bones and the bases of the second, third, and fourth metatarsal bones). Since it lies posterior to the axis of the ankle and medial to the axis of the subtalar joint, its function is to plantar flex the ankle, while it inverts the subtalar and oblique axis of the midtarsal joints (2). Due to its anatomical location it provides dynamic support along the plantar aspect of the foot. By passing underneath the spring ligament it stabilizes the osseous configuration at the talonavicular joint and prevents collapsing of the medial longitudinal arch. Loss of the medial longitudinal arch results in dorsiflexion of the first metatarsal. Insufficiency of the first ray may be found in early stages of PTTI (16).

The foot is a complex mechanism acting as a mobile adapter during weight acceptance and a rigid lever arm during propulsion (4). The talonavicular and calcaneocuboid joints (the midtarsal joint) seem to play an important role in this transition (20). Blackwood et al. demonstrated significantly less range of motion in the midtarsal joint when the calcaneus was maximally inverted compared to when the calcaneus was maximally everted (4). During normal gait, contraction of posterior tibial muscle limits subtalar eversion caused by the gastrosoleus complex. Without the inverting force of the posterior tibial muscle in the stance phase there is less intrinsic osseous stability at the midtarsal joint and the forward propulsive force of the complex of gastrosoleus acts at the midfoot instead of at the metatarsal heads (push-off phase) (31).

Due to its large cross-sectional area the relative strength of posterior tibial muscle is more than twice

that of peroneus brevis, its primary antagonist (21). With loss of an antagonist force due to PTTI, the unopposed pull from the peroneal tendons forces the heel into eversion.

Epidemiology

The prevalence of PTTI and posterior tibial tendon rupture parallel the degenerative processes of aging. Hypertension, diabetes mellitus, seronegative arthropathies and obesity have all been identified as risk factors for PTTI (18). Additionally, the effects of corticosteroids and local surgical procedures may further be associated with local vascular impairment and eventual rupture (13).

Pathomechanism of the adult flatfoot

A posterior tibial tendon dysfunction evolves from degeneration of the tendon due to repetitive microtrauma or chronic overuse (22).

The posterior tibial tendon is acutely angled where it passes posterior to the medial malleolus. This area of increased stress on the tendon is the most common site of degeneration of the posterior tibial tendon. The blood supply of the tendon can be divided into the proximal and distal areas. The proximal tendon is supplied by branches of the posterior tibial artery and the distal, which is at the bone-tendon interface, by branches of the posterior tibial and dorsalis pedis arteries (10). Whether degeneration of the PTTI is due to physiological hypovascularity of the tendon at the degeneration site remains controversial. Frey et al showed that there is an area of hypovascularity in the tendon about 1 cm distal to the medial malleolus (10). Contrary to these findings Prado et al. did not find decreased vascularization in the mid-portion of the tendon (26). Histologic examinations of surgical posterior tibial tendons from patients with PTTI revealed a degenerative tendinosis characterized by increased mucin content, fibroblast hypercellularity, chondroid metaplasia, and neovascularization. Those result in disruption of the linear orientation of the collagen bundles (9, 22). Although PTTI is widely accepted as a significant contributor to this deformity, the pathology and deformity involve more than the tendon itself. Basmajian and Stecko concluded from their electromyographic measurement that the ligaments and osseous configuration of the foot form the primary stabilizers of the foot

arch (3). Muscular involvement in stabilization is merely called upon during increased load (take off phase in walking). MRI studies confirmed the association of spring ligament failure with PTTI (30, 37). The superomedial calcaneonavicular ligament is most commonly involved, followed by the inferomedial calcaneonavicular and talocalcaneal interosseous ligaments. In patients with surgically proven spring ligament tear, Toye and al. demonstrated an abnormal spring ligament thickening, a fullthickness gap and an abnormal increased signal on preoperative T2-weighted MRI images (30).

The involvement of the deltoid ligament is being discussed (for clinical differentiation see Diagnostics). Hintermann and al. found PTTI in 22% of patients with medial ankle instability (11). However, it remains unclear if medial ankle instability may cause a secondary PTTI with elongation and/or rupture of the tendon, or vice versa 11. Patients with a preexisting flatfoot deformity show increased gliding resistance at the tendon-sheath interface of the posterior tibial tendon. The findings indicate a possible vicious circle of deformity and tendon dysfunction (32).

Clinical examination

The diagnosis of PTTI is essentially clinical. Systematic examination of the soft tissues needs to consider the potentially unaffected contralateral side. Both feet are inspected in a standing position with parallel feet, shoulder width apart. Inspection includes hindfoot alignment, deformity, and swelling. The medial longitudinal arch is inspected for presence of flattening (Fig. 1B). Hindfoot alignment is best inspected from behind the patient. Valgus hindfoot 11. Valgus angulation of more than 10° (or valgus in bilateral comparison) is typically found in PTTI in stage II and above (Fig. 1A). Forefoot abduction is indicated by the “too-many-toes sign” (Fig. 1A). The test is positive when more of the toes are visible lateral to the ankle joint of the involved side then on the contralateral side, when viewed from behind 14. The dynamic function of the posterior tibial tendon is determined by the single-limb heel-rise test. malalignment presents as an asymmetrical planus and pronation deformity of the affected The patient is asked to rise onto the ball of one foot while the other foot is suspended in the air. As the patient rises off the floor, the posterior tibial tendon inverts and sta-



a | b

Fig. 1. PTTI stage III. Two patients with stage III posterior tibial tendon insufficiency. Viewed from behind, a distinct valgus of the hindfoot with a positive too-many-toes-sign (→) is revealed (A). The side view shows a collapse of the medial longitudinal arch and a protruding talus head due to subluxation at the talonavicular joint (B).



Fig. 2. Heel-rise test. Physiological inversion of the hindfoot in tip-toe position (A). In patients with posterior tibial tendon insufficiency, the hindfoot fails to invert and remains in eversion (B). Patients with late stage PTTI often will not be able to perform this test.

bilizes the hindfoot (Fig. 2A). In patients with PTTI the heel fails to invert and remains in valgus (Fig. 2B). If unable to achieve the tip toe position on one foot, the patient should perform a bilateral heel-rise. In late stage PTTI patients often will not be able to perform neither test. This test is also helpful to differentiate PTTI from elongation or rupture of the deltoid ligament (medial ankle ligament instability). In cases of isolated medial ankle instability without involvement of the posterior tibial tendon, the hindfoot valgus deformity is corrected by activity of the posterior tibial muscle when in tiptoe position. Ankle joint stability must be assessed (drawer test and talar tilt test). Still, clinical differentiation between PTTI and medial ankle instability is challenging and diagnosis frequently needs to be further clarified by MRI or arthroscopy 12. Progressing PTTI is often associated with an abduction and supination deformity of the forefoot. In a patient standing fully weight-bearing on both feet, external rotation of the tibia is applied to provoke varus movement of the hindfoot. In a patient with supination deformity, the first metatarsal will rise off the floor (first metatarsal rise sign).

In flexible stage I and II of PTTI the patients show pain tenderness in the course of the posterior tibial tendon, especially at the medial malleolar groove, the spring ligament and the tarsal insertion. In later stages with fixed deformity and frequently found chronic rupture of

the posterior tibial tendon the pain maximum may shift to the lateral ankle due to fibulo-calcaneal impingement. Retromalleolar pain can be a sign of posterior ankle joint impingement. Other differential diagnosis include: tarsal coalition, osteoarthritis, and other posttraumatic, neurologic, diabetogenic or iatrogenic pathologies (19).

Radiology

Posterior tibial tendon dysfunction is essentially a clinical diagnosis. Plain radiography helps confirming the extent of the deformity and presence of osteoarthritis. Conventional radiologic imaging consists of anteroposterior and lateral weight-bearing radiographs of the whole foot and an anteroposterior radiograph of the ankle joint. If the deformity is present, the anteroposterior radiograph shows a pathological a.p. talar-first metatarsal angle (normal angle, 0 to 10 degrees) with and abduction of the forefoot at the transverse tarsal joint, with the navicular sliding laterally on the talar head (Fig 3A). In patients who have an advanced deformity, subluxation or dislocation of the talonavicular joint may occur in association with degenerative osteoarthritis of the posterior facet of the subtalar joint. The lateral radiograph shows a decrease in the lateral talar-first metatarsal angle (normal angle, 0 to 10 degrees) and flattening of the longitudinal arch (Fig 3B). The anteroposterior radiograph of the ankle joint reveals potential deformity

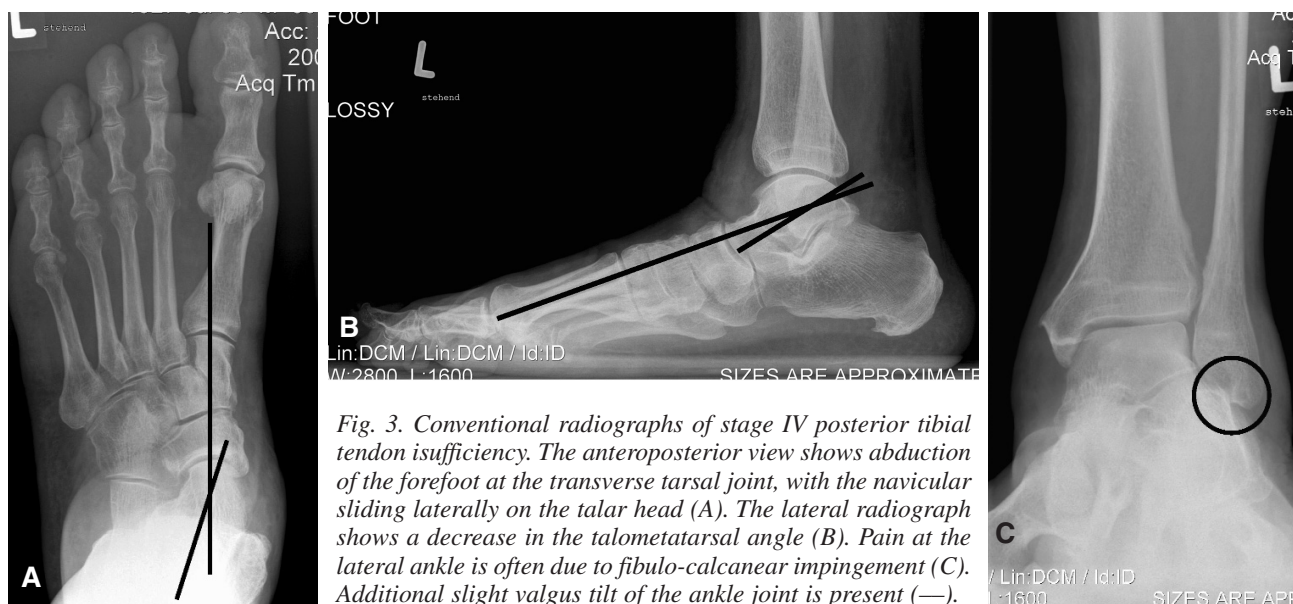


Fig. 3. Conventional radiographs of stage IV posterior tibial tendon insufficiency. The anteroposterior view shows abduction of the forefoot at the transverse tarsal joint, with the navicular sliding laterally on the talar head (A). The lateral radiograph shows a decrease in the talometatarsal angle (B). Pain at the lateral ankle is often due to fibulo-calcaneal impingement (C). Additional slight valgus tilt of the ankle joint is present (—).

Table 1. Classification and treatment recommendation for posterior tibial tendon insufficiency.

Stage	Tendon	Deformity	Pain	Single limb heel rise	Too many toes sign	Therapy
I	Peritendinitis and/or tendon degeneration	Mobile hindfoot, normal alignment	Medial: focal, mild to moderate	Normal inversion of hindfoot	–	Conservative: CRICE, NSAR, orthosis, tendosynovectomy in advanced cases
II	Elongation and marked tendon degeneration	Mobile hindfoot, valgus alignment	Medial: along TPT, moderate	No or reduced inversion of hindfoot	+	Tendon reconstruction and osteotomy of the calcaneus
III	Elongation and marked tendon degeneration	Fixed deformity, valgus position	Medial: possibly lateral, moderate	Unable to perform test, no inversion	+	Varisating subtalar arthrodesis or triple arthrodesis
IV	Marked tendon degeneration	Additional angulation of the talus and early degeneration of the ankle joint	Medial and lateral, distinct pain	Unable to perform test, no inversion	+	Triple arthrodesis with supramalleolar osteotomy Triple arthrodesis with total ankle replacement Pantalar arthrodesis

RICE = rest, ice, compression, elevation. NSAR = Non-steroidal anti-inflammatory drugs.

(talar tilt), degeneration and narrowing of the fibulo-calcaneal space (Fig. 3C). Several authors emphasize that magnetic resonance imaging (MRI) is not required to make the diagnosis and does not assist in the planning of treatment. Some authors have suggested MRI to be useful in the evaluation of PTTI (27, 28). Contrary other authors state that it is used too frequently in clinical practice and that its clinical usefulness is questionable (13, 23).

Classification

There is a continuum of PTTI ranging from tenosynovitis to fixed deformity. In 1989 Johnson and Strom described three clinical stages of dysfunction (14). Myerson et al. added a fourth to describe the most severe deformity with valgus collapse of the talus within the ankle (23) (Table 1). Stage I incorporates tenosynovitis. In this stage, the tendon is of normal length and symptoms are usually mild to moderate. Pain and swelling are present on the medial aspect of the foot. Mild weakness and minimal deformity are present. In stage II there is elongation or tearing of the tendon. The limb is weak and the patient is unable to stand on tiptoe on the affected side. There is secondary deformity as the midfoot pronates and the forefoot abducts at the transverse tarsal joint. The subtalar joint remains mobile. Stage III is characterized by a more severe deformity and a fixed hindfoot. Stage IV involves a valgus deformity and degeneration of the ankle joint.

Treatment

Treatment of PTTI intends to stop progression of the tendon dysfunction and to protect the longitudinal arch stabilizing soft tissues (e.g. spring ligament, deltoid). This can be achieved by reconstruction of the anatomic alignment and recovery of physiological biomechanics. PTTI with no or beginning deformity characterized by a flexible hindfoot foot can be treated joint-preserving by conservative and tendon reconstructive methods. Treatment of rigid hindfoot deformities in later stage III and IV intends to reconstruct the painful deformity. Osseous reposition and subsequent arthrodesis are often inevitable.

Stages I and II – The flexible foot

Stage I

A period of four to eight weeks of immobilisation in a plaster cast below the knee or a walking boot may be required to control accompanying inflammation. Complementary measures are RICE (rest, ice, compression, and elevation) and anti-inflammatory drugs. Footwear plays an important role, and patients should be encouraged to wear flat lace-up shoes, or even lace-up boots, which accommodate orthoses. Stage I patients may be able to manage with a casted insoles. The various casted, semirigid insoles support the medial longitudinal arch of the foot and either hold the heel in a neutral alignment (stage I) or correct the outward bent heel to a neutral alignment (stage II). This approach is meant to serve several functions: to alleviate stress on the posterior tibial tendon and muscle; to make gait more efficient by holding the hindfoot fixed; and thirdly, to prevent progression of deformity. When this approach has been used, two thirds of patients have good to excellent results (18). However, from our experience, conservative therapy shows poor results in the long-term. Some authors propose tenosynovectomy for patients who have advanced stage I dysfunction. Good results have been reported for either open or endoscopic technique (5, 34).

Stage II

No soft-tissue reconstructive surgical technique on its own can sufficiently contain the forces of a malaligned hindfoot. Therefore, consensus is growing that surgical treatment of stage II PTTI should include a tendon transfer in combination with corrective osteotomy (23, 25). The rationale behind this approach is that the osteotomy is required to correct the bony architecture of the foot in order to optimize the biomechanics of the reconstructed posterior tibial tendon and protect other foot stabilizing ligaments and tendons (11). According to Valderrabano et al., only about 60% of force regeneration can be expected after surgical reconstruction of a dysfunctional or ruptured posterior tibial tendon. The mag-

nitude of regained strength is primarily dependent on the duration of the dysfunction and amount of atrophy (33). Therefore PTTI needs to be surgically addressed at an early after failure of conservative treatment.

The two recommended tendon reconstruction techniques are flexor digitorum longus (FDL) tendon transfer and transfer of a split anterior tibial tendon (Cobb procedure). Rerouting a part of the anterior tibial tendon to the plantar aspect of the cuneiform allows the posterior tibial tendon to pull at its physiological insertion site. Additionally the Cobb procedure decreases the tension of the anterior tibial tendon (16), thus preventing occasionally anterior tibial tendon ruptures. This may add to correct the deformity, by reduction of the pull of the anterior tibial tendon, which is usually increased in PTTI. This dynamic correction may allow the patient to adapt the forefoot to the ground as required. Additionally, this procedure does not sacrifice the FDL tendon.

Another method is the transfer of flexor digitorum longus tendon. The FDL tendon is detached proximal of the juncture with the flexor hallucis longus (Henry's knot). The periosteum over the navicular is then dissected and a drill hole is made in the tuberosity from the dorsal to plantar aspects. The tendon is sutured side-to-side to the posterior tibial tendon and passed through the drill hole from plantar to dorsal. The use of the flexor hallucis longus tendon is not recommended due to its important role in the push-off phase off the foot (31). Intraoperative exploration of the spring ligament in PTTI is mandatory, because of its frequent concomitant degeneration or rupture (37). If found ruptured, reconstruction has to consider both components of the spring ligament complex (6). The author suggest that if a accompanying medial ankle instability is suspected upon clinical examination, an initial ankle arthroscopy needs to be performed to rule out deltoid ligament involvement. If ligament instability is found, it needs to be addressed during the following surgery.

Various osteotomies of the calcaneus can correct the pathological bony alignment. During surgery, these osteotomies should be performed prior to finalize the medial soft tissue reconstruction. In PTTI with pronounced hindfoot valgus and no or minimal foot abduction a medial sliding osteotomy is recommended (23). The lateral hindfoot incision extends from the superior border of the calcaneal tuberosity anterior to the retrocalcaneal space to the inferior border of the calcaneus superficial to the plantar fascia. An oblique transverse osteotomy is made in the calcaneus in line with the incision in the skin with use of an oscillating saw. The cut is made at a right angle to the lateral border of the calcaneus and is inclined posteriorly at an angle of approximately 45 degrees to the plane of the sole of the foot. The posterior fragment of the calcaneal tuberosity is translated medially ten or more millimeters and is secured with a cannulated headless compression screw.

In PTTI with pronounced hindfoot valgus and distinct forefoot abduction a lateral calcaneus lengthening osteotomy is recommended. By lengthening the lateral column, the medial longitudinal arch is restored secondarily to the induced adduction movement of the fore-

foot that supinates the foot at the subtalar and talonavicular joint. An osteotomy of the anterior calcaneus was originally described by Evans et al (8). Here an osteotomy of the neck of the calcaneus is performed and a tricortical bone graft impacted. Several authors have reported good results with this procedure (1, 24). Myerson et al. propose a lengthening through the calcaneocuboid joint itself, with use of a tricortical bone graft for arthrodesis of the joint (23). We favour an alternative method proposed by Hintermann et al (12). Here an osteotomy is performed from at the lateral hindfoot approximately 12 to 20 mm proximal to the calcaneocuboid joint at the "floor" of the sinus tarsi. The oscillating saw passes between the posterior and middle facet of the subtalar joint. The medial cortex is kept intact. The gap is widened with a Casper spreader and a tricortical graft or alternatively allograft bone is inserted. The amount of widening can be adjusted until the medial arch is restored sufficiently. The graft is fixed with one 3.5mm cortical screw. Preservation of the subtalar and calcaneocuboid joint offers certain advantage. One has to keep in mind that fusing the calcaneocuboid joint significantly reduces hindfoot motion (7). Maintaining hindfoot motion prevents overload on adjacent joint which may lead to osteoarthritis. Additionally, according to Knupp et al. it is easier to reduce the abducted foot if the lateral column is not further shortened by arthrodesis of the calcaneocuboid joint (17).

A recently presented surgical method for stage II PTTI is the subtalar arthroereisis. Here, the sinus tarsi is emptied and a expanding endorthesis is inserted following prior correction of the deformity and tendon repair. Good results were shown for the subtalar arthroereisis, especially in younger patients (36). However, one has to keep in mind that the fatty tissue in the sinus tarsi contains abundant nerve cell which are essential for proprioception of the hindfoot ("cerebellum pedis") and therefore should be dissected carefully and not removed.

Stages III and IV – The rigid foot

Stage III

The goal of surgical treatment of stage III PTTI is correction the deformity and pain relief. Because at this stage hindfoot deformity can not be passively reduced, joint preserving surgery frequently fails. Depending on the extent of the deformity, correction can be achieved through a varisating subtalar arthrodesis or triple arthrodesis of the subtalar, calcaneocuboid, and talonavicular articulations. In our opinion, calcaneocuboid arthrodesis can be omitted to sustain residual motion of the lateral column.

Stage IV

Stage IV PTTI has been reached when additional degenerative changes are present in the ankle joint. In such cases, a varisating triple arthrodesis together with a medial closing wedge supramalleolar osteotomy and deltoid ligament reconstruction may solidly address the deformity (Fig. 4). In very selected cases a varisating



Fig. 4. Case with PTTI stage IV. A grotesque valgus of the hind-foot with a positive too-many-toes-sign (\rightarrow) is present (A). Pedobarography (B) reveals a collapse of the longitudinal arch (\rightarrow) and medialisation of the center of pressure line (—). The anteroposterior view shows a subluxation of the talar head at the talonavicular joint (C). Anteroposterior radiographs of the ankle joint show (D) show fibulo-calcaneal impingement (O) and a valgus tilt (—). Surgery included a triple arthrodesis (involving the calcaneocuboid joint), a Ludloff osteotomy, resection of the second metatarsal head, and reconstruction of the deltoid ligament. Postoperative radiographs reveal a reconstructed foot alignment (E). Clinical examination shows physiological hindfoot valgus (F).

triple arthrodesis may be combined with total ankle replacement. However, the salvage treatment at this stage is usually a pantalar arthrodesis (ankle, subtalar, calcaneocuboid, and talonavicular articulations) (35).

Rehabilitation

Bradytroph tendon tissue requires sufficient healing time. To reach adequate stability, approximately 12 weeks for the tendon and 8–12 weeks for the bone are required. In the initial 6 weeks immobilization in a pneumatic walking brace with partial weight bearing (heel-to-toe pattern 15–20kg with crutches) is mandatory. The load can be then increased gradually until full weight-bearing is reached after 12 weeks postoperatively. Physiotherapeutic care needs to address postural hindfoot stability. From our own experience it takes 3–6 months until the rehabilitation is finished. This time span is needed to adapt the cerebellar control of balance and locomotion to the new anatomic configuration of the hind-foot.

PEARLS

- Posterior tibial tendon insufficiency (PTTI) is a progressive entity leading to painful pes planovalgus abductus et supinatus.
- If conservative treatment fails, early surgical intervention slows further progression of the disease.
- Reconstruction of the posterior tibial tendon and concomitant medial ligamentous lesions in stage II PTTI need to be accompanied by a corrective calcaneal osteotomy:
 - Distinct forefoot abduction \rightarrow lateral calcaneus lengthening osteotomy;
 - No or minimal forefoot abduction \rightarrow calcaneus medial sliding osteotomy.
- If additional medial ankle instability is suspected upon clinical examination, an ankle arthroscopy needs to rule out deltoid ligament involvement. If accompanying ligament instability is verified, an additional medial ligamentoplasty needs to be performed.

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Corresponding author:

Prof. Dr. Dr. Victor Valderrabano, MD, PhD.
Chairman, Orthopaedic Department,
University Hospital of Basel
Spitalstrasse 21
4031 Basel, Switzerland
Tel.: +4161 265 71 97, Fax: +4161 265 78 29
E-mail: vvalderrabano@uhbs.ch