

Anatomy, pathophysiology and classification of posterior tibial tendon dysfunction

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Abstract. – OBJECTIVE: Adult acquired flatfoot deformity is generally associated with a collapsing medial longitudinal arch and a progressive loss of strength of the posterior tibial tendon (PTT). This condition is commonly associated with PTT dysfunction or rupture, which can have an arthritic or a traumatic etiology. Several causes have been proposed to explain the clinical evidence of tendon degeneration observed at the time of surgery including trauma, anatomical, mechanical, inflammatory and ischemic factors.

MATERIALS AND METHODS: In this review, we analyzed anatomy, pathophysiology and existing classifications of posterior tibial tendon dysfunction.

RESULTS: Anatomical features, and in particular vascularization, expose PTT to major degenerative disorders until rupture. A literature overview showed that a low blood supply of the gliding part of the tendon is linked to a dysfunction and/or a rupture of the PTT in the region located behind the medial malleolus.

CONCLUSIONS: PTT low blood supply causes a dysfunction resulting in an abnormal loading of the foot's medial structures. This may be the reason why PTT dysfunction leads to an acquired flatfoot deformity. Conversely, flatfoot deformity may be a predisposing factor for the onset of PTT dysfunction.

Key Words

Adult Acquired flatfoot, Flatfoot deformity, Posterior tibial tendon dysfunction, Vascular density.

Introduction

The posterior tibial tendon (PTT) is the largest and the most anterior of the medial ankle tendons, blocked by the retinaculum behind the medial malleolus. PTT acts as a primary dynamic stabilizer of the medial longitudinal arch and as the main inver-

ter of the midfoot^{1,2}. PTT dysfunction or rupture is the most common cause of adult acquired flatfoot deformity, resulting in the collapse of the medial longitudinal arch and a progressive loss of the strength of the tendon. This progressive collapse of the medial longitudinal arch leads to the development of many secondary deformities typical of the flatfoot such as the abduction of the forefoot, calcaneus valgus, plantar drop of the talus and fixed forefoot varus-supination deformity³⁻⁵.

Several studies investigated the epidemiological factors involved in this condition: anatomic, micro-traumatic and systemic factors⁶⁻⁸.

Tendon degeneration begins far before clinical symptoms appear and frequently comes to our observation when flatfoot deformity is already present, due to a delayed diagnosis.

The aim of this review is to provide recent insights on anatomy, pathophysiology, and classification of PTT dysfunction, in order to better understand this disorder and to help surgeons in choosing the best treatment.

Anatomy

Macroscopic aspect

The anatomy of the PTT is known: the tibialis posterior muscle originates from the interosseous membrane, the posterolateral tibia and the postero-medial fibula. It descends between flexor hallucis longus and flexor digitorum longus. The muscle tendon junction is located in the medial posterior part of the distal third of the calf. At intermediate portion, PTT flexes about 80° anteriorly rotating behind the medial tibial malleolus. Distal insertion is placed on the tubercle of the navicular bone, with plantar expansions reinforcing medial and plantar talo-navicular joint capsule (*Coxa Pedis*)⁹⁻¹⁰. The



Figure 1. Anatomy of posterior tibial tendon (*black arrows*; retinaculum has been resected at dashed line).

tendon length ranges from 12 to 15 cm and the cross section has an oval shape with a diameter ranging from 12 to 6-7 mm (Figure 1).

Histologic features

Where PTT turns around medial malleolus, it is characterized by specific microscopic and macroscopic features: for this reason, it is also called “Gliding Tendon” (Figure 2). The gliding par is characterized by the presence of fibrocarti-

lage¹¹, differentiating by a typical traction tendon structure. In the past, some authors considered the presence of the fibrocartilage as a metaplasia or degeneration. Actually, many reports showed that the fibrocartilage within the gliding part of the PTT is a physiologic component¹².

Vascular findings

In 1990, Frey et al¹³ described micro-vascularization using conventional injection methods

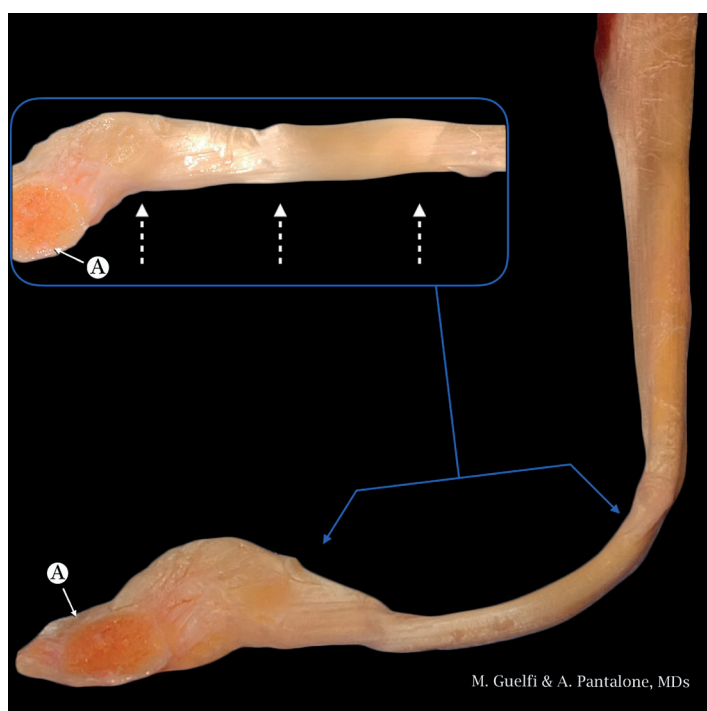


Figure 2. The gliding par, characterized by fibrocartilage (*white arrows*), is just behind the malleolus and before to attach to the navicular tubercle (*A*).

(Spalteholz's technique), without any evidence of an avascular zone. However, they suggested that the tendon has a reduced blood supply zone in the retromalleolar region.

Early as 2002, Petersen et al¹⁴ showed with immunohistochemical tests that there was no immunostaining of laminin in the anterior part of the tendon, where it passes behind the medial malleolus: stating that this region is avascular. The authors used the technique of a simultaneous injection of a solution of Technetium 99, Indian ink and gelatin into the anterior and posterior tibial artery of the leg in fresh frozen bodies. This study showed that most of "blood supply of the posterior tibial tendon is by posterior tibial artery" and that the portion of the tendon that curves around the malleolus is not vascularized.

Afterward another study by Prado et al¹⁵ evaluated a possible correlation between the most frequently affected area by degenerative lesions of the PT tendon and an area of decreased vascularization in this tendon. The most commonly used methods for analyzing vascularization of a structure are intra-arterial injections of dye followed by radiographic contrast (microarteriography) or the direct observation of the vascular tree under light microscopy. In this study, the vascular density of the PT tendon was calculated after the direct observation under a light microscope of histologic cuts stained with Masson's trichrome. According to this study, no differences among the different sites of the PT tendon were observed.

Manske et al¹⁶ studied the arterial anatomy of the PTT injecting anterior tibialis, posterior tibialis and peroneal arteries with India Ink and Ward's Blue Latex. The specimens used for the macroscopic analysis were debrided with sodium hypochlorite to expose the extratendinous anatomy. For the microscopic analysis, the tendon was cleared using a modified Spalteholz's technique, in order to expose the intratendinous vascular anatomy. The Authors reported that macroscopically, an average of 2.5 ± 0.7 vessels entered the tendon proximally to the navicular insertion. In 28 out of 30 specimens (93.3%), vessels entered 4.1 ± 0.6 cm proximally to the medial malleolus and in 24 specimens (80.0%) vessels entered 1.7 ± 0.9 cm distally to the medial malleolus. From a microscopic point of view, an average of 1.9 ± 0.3 vessels entered each tendon proximally to the navicular insertion. Twenty-seven specimens (90%) had a vessel entering the tendon 4.8 ± 0.8 cm proximally to the medial malleolus and all 30 specimens (100%) had a vessel entering the tendon 1.9 ± 0.8 cm distally to the medial malleolus. In all specimens, a hypovascular region was observed, starting 2.2 ± 0.8 cm proximally to the medial malleolus and ending 0.6 ± 0.6 cm proximally to the medial malleolus, with an average length of 1.5 ± 1.0 cm. The insertion of the tendon was well vascularized both on microscopic and macroscopic specimens. The Authors concluded that PTT is supplied by 2 vessels, which enter the tendon approximately 4.5 cm proximally, and 2.0 cm distally, to the medial malleolus. Furthermore, the retromalleolar region has a low blood supply (Figures 3, 4 and 5).

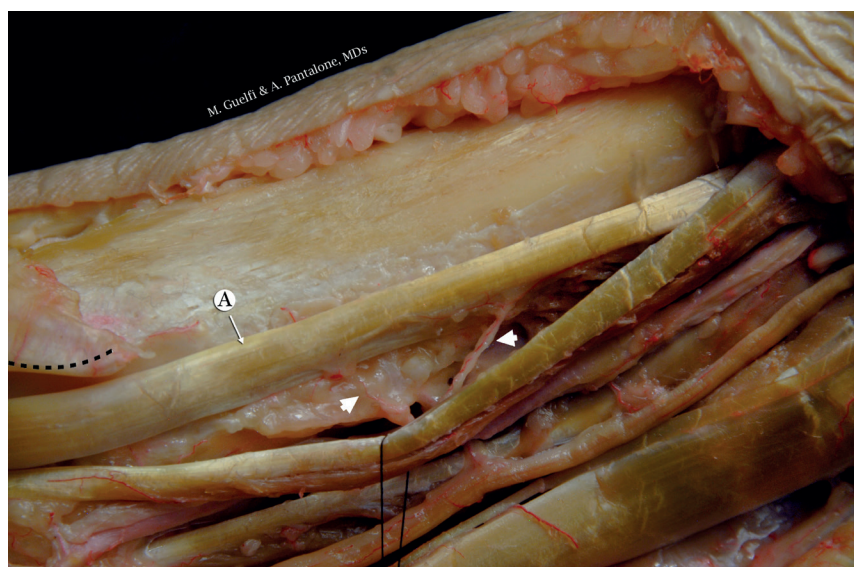


Figure 3. White arrows show the vascularization of PTT (A) proximal to medial malleolus (dashed line); FDL is retracted posteriorly.

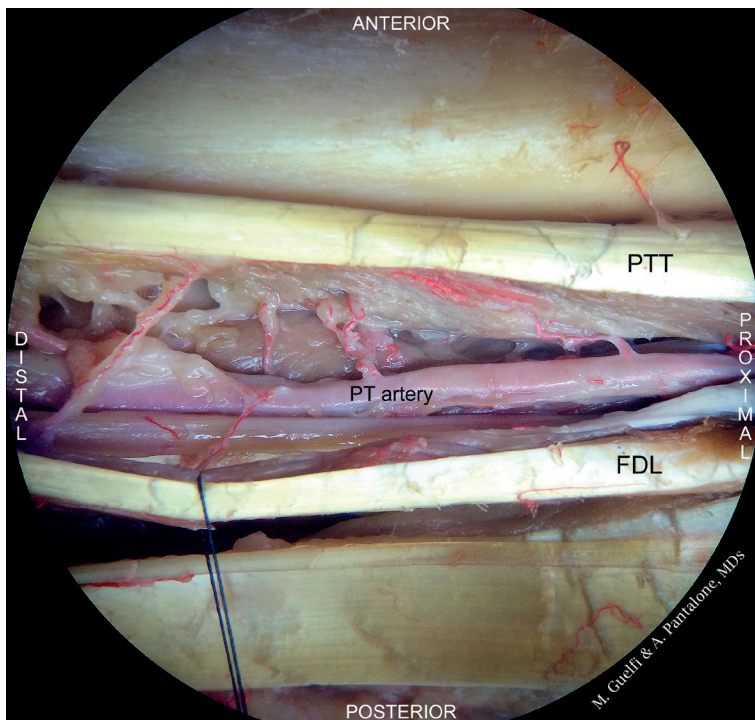


Figure 4. Optical microscopic view of proximal vascularization of PTT (FDL is retracted posteriorly).

Pathophysiology

Scott et al¹⁷ described the insights of tendon pathophysiology relevant for clinicians, including:

- a) A better characterization of the overuse injury process and the consequent structural and functional damage in chronically painful tendons¹⁸;
- b) Improved understanding of the pathomechanics associated with chronic tendon injury;
- c) A better knowledge on the influence of lifestyle factors and drugs on tendon pathology.

Posterior tibial tendon dysfunction (PTTD) is the most common degenerative process, which begins far before clinical symptoms appear, leading to tendinosis and elongation of the tendon. This influences PTT structure and function, determining an inflammatory tendinopathy characterized by fissures unable to heal. Failure of clinical management may lead to a worsening of the tendinosis, resulting in partial or complete tears. Being the primary dynamic stabilizer, PTTD with or without rupture results in abnormal foot biomechanics and contributes to the onset of the acquired flatfoot deformity^{19,20,21}.

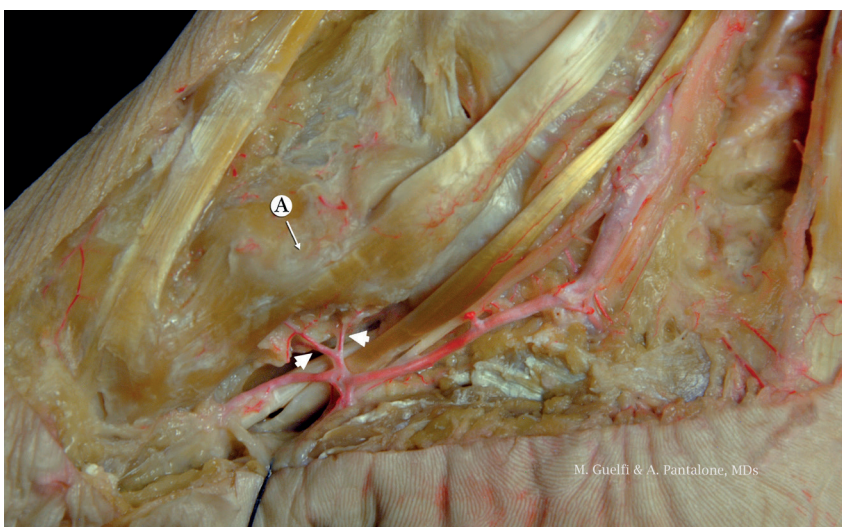


Figure 5. Distal vascularization of PTT (white arrows); vessels enter the tendon just proximally to the navicular insertion (A).

Classification

Posterior tibial tendon dysfunction is often misdiagnosed and overlooked. Johnson and Strom²² described three stages of PTTD, universally accepted for a long time: Stage 1 – Peritendinitis and tendon degeneration without tendon elongation; symptoms include pain and swelling along the posterior tibial tendon. Stage 2 – Posterior tibial tendon elongates and a supple flat foot deformity appears. Stage 3 – Hind foot rigid in a valgus position with rigid flatfoot deformity.

In 1996, Myerson published his own classification, adding a fourth stage that involves tilting of the ankle joint¹. Bluman et al²³ updated this classification in 2007 with the propose to provide a more descriptive and comprehensive system, including treatment recommendations for each of the described stages. This is actually the most used classification:

- **Stage 1: Tenosynovitis without deformity.** Pain is over because PTT is inflamed, but the overall continuity of the tendon is maintained.

- **Stage 2: Ruptured of PTT with flexible flatfoot.** PTT is elongated or damaged, as shown by the flatfoot deformity and the weakness of the plantar flexion. Stage 2 is further divided into 3 categories depending on forefoot features.
- **Stage 3: Rigid hindfoot valgus.** Associated to an advanced course of tendon rupture. The deformity is characterized by a rigid hindfoot valgus.
- **Stage 4: Ankle Valgus.** Longstanding PTT rupture and insufficiency of medial complex ligament lead to a tibiotalar joint valgus deformity.

Table I. summarizes the classification and treatment recommendations.

Recently, Smith et al²⁴ and Peterson et al²⁵ proposed a splitting of stage IV into stage IV-A and IV-B. In stage IV-A, the ankle is valgus without significant tibiotalar arthritis; in stage IV-B, the ankle is valgus, rigid or supple, with important osteoarthritis. This splitting is crucial for choosing the correct surgical approach: in fact, stage IV-A is treated with ankle joint-sparing procedures, while stage IV-B is managed with ankle joint “destructive” procedures.

Table I. Classification for PTTD according to Bluman & Myerson. Stages are associated to the recommended treatment.

Bluman & Myerson classification for PTTD			
Stage	Substage	Characteristic	Treatment
I	A	Tenderness along PTT	Conservative or Tenosynovectomy
	B	Tenderness along PTT	Conservative or Tenosynovectomy
	C	Slight HF valgus (<5°) Tenderness along PTT	Conservative or Tenosynovectomy
II	A1	HF valgus	Orthoses
		Flexible forefoot varus +/- pain along PTT	MDCO + FDL transfer +/- Achilles Tendon lengtening
	A2	HF valgus	Orthoses
		Fixed forefoot varus +/- pain along PTT	MDCO + FDL transfer Cotton Osteotomy
	B	HF valgus	MDCO + FDL transfer
	C	Forefoot abduction	Lateral Column Lengthening
		HF valgus	MDCO + FDL transfer
		Fixed forefoot varus Medial Column Instability First ray dorsiflexion with HF correction Sinus tarsi pain	Cotton Osteotomy or Medial Column Fusion
III	A	Rigid HF valgus Sinus tarsi pain	Triple arthrodesis
	B	Rigid HF valgus	Triple arthrodesis
		Forefoot abduction Sinus tarsi pain	Lateral Column Lengthening
IV	A	Tibiotalar valgus	Surgery for HF valgus and associated deformity Deltoid reconstruction
	B	Rigid tibiotalar valgus	TTC fusion or pantalar fusion

Abbreviations: PTT, Posterior Tibial Tendon. PTTD, Posterior Tibial Tendon Dysfunction. HF, hindfoot valgus. MDCO, Medial displacement Calcaneal Osteotomy. FDL, Flexor Digitorum Longus.

Another clinical classification, reported by Richter in 2013²⁶, divided the disease into four stages:

- **Stage 1:** single leg heel rise possible, heel moves to varus during heel-rise.
- **Stage 2:** single leg heel rise possible, heel moves to neutral during heel-rise.
- **Stage 3:** single leg heel rise possible, heel stays in valgus during heel-rise.
- **Stage 4:** single leg heel rise is not possible.

This classification is specific for PTTD; antedated it does only assess the function of the posterior tibialis tendon independently from the stiffness of the joints. In Richter's idea, there is a difference between the PTT insufficiency and flexibility of the deformity. Therefore, stage IV posterior tibialis tendon insufficiency is not necessarily associated with a fixed deformity. As confirmation of this, it is not unusual to observe collapsed flat feet that are not stiff at all, and *vice versa*, there are stiff feet without any PTT insufficiency²⁶.

Conclusions

Tibialis posterior tendon dysfunction is a common but often misdiagnosed condition. It causes a progressive, painful flatfoot deformity²⁷. Recent classifications of PTT disorder²³ include not only clinic features but also instrumental images, helping the choice of the best surgical treatment. Anatomy and vascularization expose posterior tibial tendon to major degenerative disorders until the rupture. In the last decades, literature data showed a low blood supply zone of the gliding part of the tendon, which seems to be directly related to the site of rupture or lesion.

Conflict of Interest

The authors declare that no conflicts of interest exist.

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