Arterial Anatomy of the Tibialis Posterior Tendon

Mary Claire Manske, MD¹, Kathleen E. McKeon, MD², Jeffrey E. Johnson, MD¹, Jeremy J. McCormick, MD¹, and Sandra E. Klein, MD¹

Abstract

Background: Tibialis posterior tendon dysfunction is a common disorder leading to pain, deformity, and disability, although its pathogenesis is unclear. A vascular etiology has been proposed, but there is controversy regarding the existence of a hypovascular region that may render the tendon vulnerable. The purpose of this study was to provide a description of the arterial anatomy supplying the tibialis posterior tendon.

Methods: Sixty adult cadaveric lower extremities were obtained from a university-affiliated body donation program. Thirty specimens obtained within 72 hours of death were used for microscopic analysis. Thirty specimens were previously frozen and used for macroscopic analysis. The tibialis anterior, tibialis posterior, and peroneal arteries were injected with India Ink and Ward’s Blue Latex. The specimens used for macroscopic analysis were debrided with sodium hypochlorite to expose the extratendinous anatomy. For the microscopic analysis, the tendon was cleared using a modified Spälteholz technique to expose the intratendinous vascular anatomy.

Results: Macroscopically, an average of 2.5 ± 0.7 vessels entered the tendon proximal to the navicular insertion. In all, 28/30 (93.3%) specimens had a vessel entering 4.1 ± 0.6 cm proximal to the medial malleolus and 24/30 (80.0%) specimens had a vessel entering 1.7 ± 0.9 cm distal to the medial malleolus. Microscopically, an average of 1.9 ± 0.3 vessels entered each tendon proximal to the navicular insertion. In total, 27/30 (90%) specimens had a vessel entering the tendon 4.8 ± 0.8 cm proximal to the medial malleolus and 30/30 (100%) specimens had a vessel entering the tendon 1.9 ± 0.8 cm distal to the medial malleolus. In all specimens, a hypovascular region was observed, starting 2.2 ± 0.8 cm proximal to the medial malleolus and ending 0.6 ± 0.6 cm proximal 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.

Conclusion: The tibialis posterior tendon was supplied by 2 vessels entering the tendon approximately 4.5 cm proximal and 2.0 cm distal to the medial malleolus. A retromalleolar hypovascular region was observed.

Clinical Relevance: Improved understanding of the vascularity of the tibialis posterior tendon may be helpful in clinical practice and potentially provides a basis for further evaluation of the causative factors of tibialis posterior tendinopathy.

Keywords: vascular anatomy, tibialis posterior tendon, pes planovalgus, tibialis posterior tendon dysfunction, adult acquired flat foot deformity
rupture. Other authors have investigated the role of inflammatory cytokines, estrogen receptors and genetic markers, but no clear causative factors have been identified. Based on the complexity of this condition and the wide range of acquired deformity seen in tibialis posterior tendon dysfunction, multiple factors are likely involved. As tibialis posterior tendon dysfunction is a progressive condition, understanding the pathophysiology of this disorder may allow early, targeted interventions to delay or arrest the progress of tendinopathy and improve patient outcomes.

Impaired vascularity of the tendon has also been proposed as an explanation for this disorder, and several authors have identified a hypovascular region in the tendon adjacent to the medial malleolus. However, previous descriptions of tendon vascularity have been variable, and the location of a hypovascular region and vessels supplying the tendon has not been clearly defined. As a result, correlation of histologic changes in the degenerative tendon with the vascularity of the tendon has not been investigated. Although several authors have reported data regarding the location of tendon tears and typical location of tendinopathy, this has been assumed to be directly related to vascularity but not shown definitively.

In addition, there is controversy in the existing literature as to the existence of a hypovascular region. Prado et al reported consistent vascularity throughout the tendon, contradicting previous reports. Each of the previous studies evaluating the blood supply to the tendon uses slightly different techniques, each with limitations. A study of both the macro- and microvascularity of the tendon and the relationship between the 2 has not been performed to our knowledge. The purpose of this study was to provide a description of the arterial anatomy supplying the tibialis posterior tendon, both macro- and microscopically, and identify the location of a hypovascular region if it exists.

Material and Methods

This is a cadaveric anatomic investigation. Institutional review board exempt status was granted by our University Human Research Protection Office after a formal review of the research protocol. Sixty adult cadaveric lower extremities (30 matched pairs) were obtained from the university body donation program. Cadavers with evidence of foot or ankle deformity, surgery, or trauma were excluded from the study.

Thirty legs (15 matched pairs) that had previously been frozen, but not embalmed, were obtained and used for macroscopic evaluation. These specimens were thawed completely and amputated at the junction of the proximal and middle third of the tibia. Circumferential skin incisions were made in each toe at the level of the proximal interphalangeal joints to allow flow through the arterial network. The tibialis anterior, tibialis posterior, and peroneal arteries were identified and an 8-French triple lumen catheter was inserted into each vessel and saline injected into each artery under constant manual pressure until clear effluent was observed from skin incisions in the toes. India ink was then injected into each artery using manual pressure until the cutaneous surface of the foot was stained with ink, and ink was observed flowing from incisions in the toes. Finally, Ward’s blue latex was injected in an identical manner.

The previously frozen specimens were used for macroscopic analysis only. Following the injection the specimens were frozen for 48 hours. They were subsequently thawed completely at room temperature and the feet amputated at 10 cm proximal to the ankle joint. An axial pin was placed through the tibia, talus and calcaneus to maintain the integrity of the ankle joint and the relationship of the tibialis posterior tendon to the medial malleolus during the chemical debridement. The skin and subcutaneous fat were sharply excised from the specimens with a scalpel and chemical debridement was performed by submerging each specimen in 6% sodium hypochlorite. As the vascular walls were debrided by the sodium hypochlorite, casts of the vessels created by the latex filling the vessel lumens became visible. The specimens were checked every 20 minutes throughout the debridement, and debridement was stopped when the tibialis posterior tendon had been completely debrided by the sodium hypochlorite. The location of all vessels entering the tibialis posterior tendon below the musculotendinous junction relative to the tip of the medial malleolus and the tendon insertion on the navicular was measured and recorded. The specimens were photographed throughout the debridement (Figure 1). The measurement of the distance either proximal or distal to the medial malleolus was made relative to the line drawn perpendicular to the axis of the tibia at the tip of the medial malleolus. Any vessel proximal to this line was considered proximal to the medial malleolus. Distances were measured along the tendon relative to this line (Figure 2). Distances from the navicular insertion were measured from the most distal aspect of the tendon.
Ward’s blue latex was injected in an identical manner. Following the injection, the specimens were frozen for at least 48 hours.

The 30 fresh specimens were used for the microscopic evaluation using the Spälteholz technique.31 After thawing the specimens to room temperature, the skin and subcutaneous tissues were sharply dissected from each specimen with a scalpel. Tenotomy scissors and a scalpel were used to dissect to the tibialis posterior tendon while preserving the vasculature. A 2-0 Prolene suture was used to mark the tendon at the tip of the medial malleolus in the same location used for the macroscopic analysis (Figure 2). The tendon was then removed from the specimen by incising it proximally at the myotendinous junction and elevating it off the navicular distally. The harvested tendons were sutured to a wooden tongue blade and stored in 10% neutral buffered formalin for 48 hours in a glass test tube and then washed in running water for 2 hours before undergoing serial dehydration in increasing concentrations of ethanol (50%, 75%, 95%, 100%). The tendons were placed in ethanol for 2 hours at each concentration and an additional 2 hours in 100% ethanol. The tendons were then placed in xylene for 12 hours, followed by a 1:1 solution of methyl salicylate and xylene for 12 hours, and then stored in methyl salicylate. A stereo dissection microscope was used to evaluate each tendon and the location of the entering vessels relative to the medial malleolus measured from the prolene suture placed at the tip of the malleolus. All distances are measured along the tendon. The length of each vessel course and the length of an avascular region if present were measured and recorded. All tendons were photographed in a methyl salicylate bath.

**Results**

**Macroscopic Evaluation**

In the 30 specimens used for macroscopic analysis, a mean number of $2.5 \pm 0.6$ (range 1-4) vessels entered or crossed over each tendon proximal to the navicular insertion (Figures 1 and 2). This mean takes into consideration all vessels recorded and noted to enter or cross over the tendon from the musculotendinous junction to the tendon insertion in all specimens. The location of the vessels seen was very consistent among specimens, and no specimen had greater than 4 vessels entering proximal to the insertion. Several trends emerged. The minority of specimens (5 of the 30) had a vessel entering greater than 5 cm proximal to the tip of the malleolus, at an average of $6.3 \pm 0.02$ cm (range 6.1-6.5 cm). The remaining 25/30 had no vessel in that area. Eighteen of the 30 specimens had 1 vessel proximal and 1 vessel distal to the malleolus (*) enter the tendon with a retromalleolar hypovascular region between the entry points.

**Figure 1.** Macroscopic evaluation: 1 vessel proximal and 1 vessel distal to the malleolus (*) enter the tendon with a retromalleolar hypovascular region between the entry points.
an additional vessel that crossed over, but did not appear to enter, the tendon at 1.5 ± 0.5 cm (range 0.3-2.4 cm) proximal to the tip of the medial malleolus (12 specimens did not have this finding). Finally, 24 of the 30 specimens had a vessel entering 1.7 ± 0.93 cm (range 0.4-2.8 cm) distal to the tip of the medial malleolus (6 specimens did not have a vessel in this region). All vessels were observed to arise from the tibialis posterior artery. No other variants were noted (Figure 2) and 21/30 specimens had a total of 2 vessels entering the tendon at the 2 most common entry point regions.

A mean of 1.7 ± 0.4 vessels entered the tibialis posterior tendon at the navicular insertion. Twenty-four of the 30 specimens had 2 vessels entering the tendon at the navicular insertion and 6 of the 30 specimens had 1 entry vessel. In the 24 specimens with 2 vessels at the tendon insertion, 18 specimens had 1 vessel arising from the tibialis posterior artery (TPA) and 1 vessel arising from the tibialis anterior artery (TAA); 3 specimens had both vessels arising from the TPA and 3 specimens had both vessels arising from the TAA. In the 6 specimens with only 1 vessel entering the navicular insertion, 4 of these vessels arose from the TPA, while 2 were branches of the TAA.

Twenty-four of the 30 specimens had a vessel that entered the tendon less than 1 cm from the distal aspect of the navicular insertion, at an average of 0.3 ± 0.2 cm. Twenty-five of the 30 specimens had a vessel that entered the tendon at a distance of greater than or equal to 1 cm of the distal aspect of the insertion, at an average of 1.5 ± 0.7 cm (Figure 2).

**Microscopic Evaluation**

A mean of 1.9 ± 0.3 vessels entered the tendon proximal to the navicular insertion (Figure 3). This mean takes into...
consideration all vessels recorded and noted to enter the tendon from the musculotendinous junction to the tendon insertion in all specimens. The location of the vessels seen was very consistent among specimens and no specimen had greater than 2 vessels entering proximal to the insertion seen microscopically. Several trends emerged. In 27 of the 30 specimens, a vessel entered the tendon proximal to the tip of the medial malleolus, coursing distally. In 3 of the 30 specimens, no vessel entry point was seen in this region. This vessel entered the tendon at a mean 4.8 ± 0.8 cm (range 3.1-6.7 cm) proximal to the tip of the medial malleolus. In 30 of the 30 specimens, a second vessel entered the tendon distally and ran proximally toward the tip of the medial malleolus. This vessel entered the tendon at 1.9 ± 0.8 cm (range 0.0-3.5 cm) distal to the tip of the medial malleolus. In all specimens (100%), the vessels were observed to course toward each other but did not meet or anastomose, leaving a hypovascular region between them. The mean length of this hypovascular area was 1.5 ± 1.0 cm (range 0.5-4.1 cm), starting 2.2 ± 0.8 cm proximal to the tip of the medial malleolus and ending 0.6 ± 0.6 cm proximal to the tip of the medial malleolus.

A mean of 1.4 ± 0.7 vessels entering the tendon were observed at the navicular insertion microscopically. In 2 of the 30 specimens, no vessel entry points were identified at the navicular insertion. Thirteen of 30 specimens had 1 entry vessel, 14 of the 30 specimens had 2 entry vessels, and 1 of the 30 specimens had 3 entry vessels at the navicular insertion. The most common pattern was 1 vessel within a centimeter of the distal aspect of the tendon insertion on the navicular (mean 0.55 ± 0.3 cm) and a second vessel between 1.0 and 3.0 cm of the insertion (mean 1.8 ± 0.5 cm).

**Discussion**

Tibialis posterior tendon dysfunction causes significant pain and disability. The etiology of this common condition is not clear. Although traumatic rupture of otherwise healthy tibialis posterior tendons has been reported,5,8 these are thought to be rare.7,23 It has been experimentally demonstrated in other tendon models that normal, healthy tendons are resilient to trauma. In McMaster’s rabbit Achilles tendon model, normal healthy tendon did not rupture even when subjected to severe strain; fracture, tendon avulsion, or muscle belly ruptures all occurred prior to tendon failure. However, obstruction of the blood supply to the normal tendon resulted in tendon rupture when subjected to the same strain.21

The finding that hypoxia is associated with tendon rupture has been observed in other studies.4,19 This concept provides some basis for the consideration of hypovascularity as 1 of the factors that may contribute to tendinopathy. Tendon attrition or degeneration of the tibialis posterior tendon is believed to be more common than traumatic ruptures,7,21 and tendon degeneration may be due to intrinsic abnormality of the tendon, in what is known as McMaster’s concept of predisposition.11,21

Microscopic and histologic evaluations of diseased tibialis posterior tendons corroborate the idea that these tendons are abnormal.10,22,28,30 Microscopically, these tendons
are observed to have increased mucin content, myxoid degeneration, and fibrosis compared to nondiseased tendons, as well as disordered collagen orientation, and a shift in collagen composition from type I collagen to smaller diameter type III and type V collagen. Although it is not clear whether these histologic abnormalities are the cause or the result of subsequent tendon attrition and occasional rupture, these changes are known to decrease tensile strength within the tendon and therefore, render the tendon susceptible to injury.

There are differences in the microstructure of the tibialis posterior tendon based on the location of the tendon relative to the malleolus even within healthy tendons without evidence of degeneration. Microscopically, the retromalleolar region of the tendon immediately adjacent to the medial malleolus has the microstructure of fibrocartilage and has a high glycosaminoglycan content, typical of gliding tendons, while the rest of the tendon has the structure of dense connective tissue, as is seen in traction tendons. This difference is thought to be physiologic, rather than pathologic metaplasia or degeneration, given the tibialis posterior tendon’s function as a gliding tendon in the retromalleolar area (subject to intermittent compressive and shear stresses), rather than its function as a traction tendon throughout the rest of its course. Based on these considerations, the hypovascular region could represent a protective physiologic finding rather than a factor contributing to pathology of the tendon.

It has been considered, but never conclusively demonstrated that the tendon adjacent to the medial malleolus is a common site of tibialis posterior tendon abnormality. Given that the retromalleolar region of the tibialis posterior tendon experiences unique stresses and hypovascularity relative to the rest of the tendon, this may predispose tendons to injury. However, further clinical study is necessary to truly define the location and cause of tibialis posterior tendinopathy.

In this study, we investigated the blood supply of the tibialis posterior tendon and quantitatively described the blood supply to this tendon. We evaluated the tendon first macroscopically, to identify the location of vessel entry in situ relative to identifiable structures. The medial malleolus and navicular insertion were the most reproducible points of measurement so the branches to the tendon in each specimen were identified relative to these landmarks. The microscopic portion of this analysis provided additional data regarding the entry points of the vessels to the tendon. Based on this evaluation we confirmed that the macroscopic (in situ) location of vessels seen in each specimen correlated well to the entry points of the vessels on microscopic analysis.

We consistently observed both macroscopically and microscopically 2 branches of the tibialis posterior artery supplying the tibialis posterior tendon proximal to the insertion on the navicular. The more proximal of these vessels entered the tendon between 4 and 5 cm proximal to the tip of the medial malleolus. The second vessel entered the tendon almost 2 cm distal to the medial malleolus. The vessels coursed toward each other but did not anastomose, leaving a retromalleolar hypovascular region between them. This hypovascular area was approximately 1.5 cm in length and located immediately posterior to the medial malleolus.

More proximally, the minority of specimens had a vessel entering the tendon at the proximal aspect of the tendon, adjacent to the myotendinous junction. At the navicular insertion all specimens were observed to be well vascularized, with the majority of specimens having multiple vessels entering the tendon (range 1-3 vessels). In most specimens, the tendon at the navicular insertion was supplied by both the anterior and tibialis posterior arteries. We found good agreement between our macroscopic and microscopic results, in not only the number of vessels but also the location of entry into the tendon.

The findings of this investigation are consistent with previous studies evaluating the blood supply to the tibialis posterior tendon. In a smaller study, Frey et al evaluated 28 tibialis posterior tendons cleared by the Spalteholz technique and observed a 1.4 cm hypovascular region just distal to the medial malleolus. They compared the tibialis posterior tendon vascularity with that of the adjacent flexor digitorum longus (FDL), and found no hypovascular region in the FDL. Using an alternative technique, Petersen et al used arterial injection of radioisotope to assess intravascular volume in the tibialis posterior tendon, as well as immunohistochemistry to determine the presence of laminin, a component of the vascular basement membrane. They found both a lower intravascular volume in the retromalleolar region of the tibialis posterior tendon and no evidence of laminin in the segment of the tendon immediately adjacent and posterior to the malleolus.

Contrasting with the results of these studies, Prado et al sectioned 80 tibialis posterior tendons at 6 levels along the length of the tendon and used Masson’s trichome stain to evaluate vascular density under a light microscope. They found no difference in vascular density between the retromalleolar region and more proximal and distal sections.

The principal limitation of this and other anatomic studies is that they are observational studies, which can only describe the arterial anatomy and identify regions of relative hypovascularity. It cannot establish causality between the vascular anatomy and the development of clinical symptoms, including rupture. In addition, the ability to accurately characterize the vascular anatomy may be compromised by preexisting macro- and microvascular conditions, such as atherosclerosis and diabetes, which was not controlled for in this study. Finally, our study quantified the number and location of vessels entering the tendon, but it did not assess the vascular volume of these vessels; thus we are unable to describe the amount of blood provided to the tendon.
Our study has identified a consistent hypovascular region in the tibialis posterior tendon adjacent to the medial malleolus. The clinical significance of our findings would be enhanced by further study of the most common site of rupture or tendinopathy in the tibialis posterior tendon, and allow comparison of these pathologic changes with the vascular supply to the tendon. It is feasible that the hypovascular region may not contribute to the pathology seen clinically. The insertion site of the tendon was well vascularized in these specimens. Additional study in the future is warranted to determine if the more vascular region of the tendon is possibly a site of injury. The study provides a basis for the comparison of both clinical observation and histologic study of tendinopathy in the tendon and vascular supply.

Conclusions

- Two entry vessels supply the tibialis posterior tendon, entering approximately 4.5 cm proximal and approximately 2 cm distal to the malleolus based on macroscopic and microscopic examination.
- A hypovascular, retromalleolar region was quantitatively and qualitatively observed in 100% of the specimens microscopically.
- Concomitant hypovascularity and mechanical traction in the retromalleolar tendon may render the tibialis posterior tendon susceptible to injury.

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