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Vascular Anatomy of the Tibiofibular Syndesmosis

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Background: Injuries to the tibiofibular syndesmosis commonly cause prolonged ankle pain and disability. Syndesmotic injuries are associated with slower healing rates compared with rates for other ankle ligament injuries and typically result in longer time away from sports. To our knowledge, the vascular supply to the syndesmosis and its clinical implication have not previously been studied. The purpose of this study was to describe the vascular supply to the tibiofibular syndesmosis with use of a method of chemical debridement of cadaveric specimens.

Methods: Twenty-five matched pairs of adult cadaver legs, fifty legs total, were amputated below the knee. India ink, followed by Ward Blue Latex, was injected into the anterior tibial, peroneal, and posterior tibial arteries under constant manual pressure to elucidate the vascular supply of the ankle syndesmotic ligaments. Chemical debridement was performed with 6.0% sodium hypochlorite to remove soft tissue, leaving bones, ligaments, and casts of the vascular anatomy intact. The vascular supply to the syndesmosis was evaluated and recorded.

Results: The anterior vascularity of the syndesmosis was clearly visualized in forty-three of fifty specimens. The peroneal artery supplied an anterior branch (the perforating branch) that perforated the interosseous membrane, an average of 3 cm proximal to the ankle joint. This branch provided the primary vascular supply to the anterior ligaments in twenty-seven specimens (63%). The anterior tibial artery provided additional contribution to the anterior ligaments in the remaining sixteen specimens (37%).

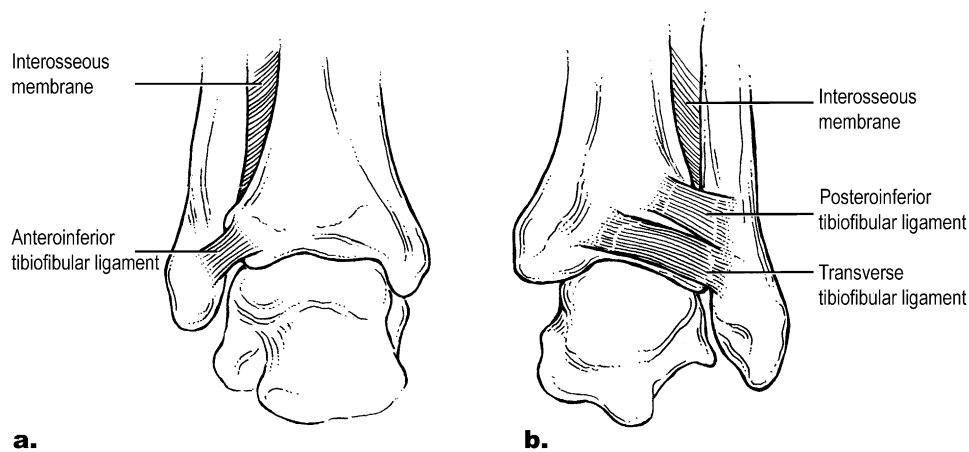
Conclusions: The location of the perforating branch of the peroneal artery places it at risk when injury to the syndesmosis extends to the interosseous membrane 3 cm proximal to the ankle joint. In the majority of specimens, injury to this vessel would result in loss of the primary blood supply to the anterior ligaments.

Clinical Relevance: The vascular supply to the anterior syndesmotic ligaments may be damaged in ankle syndesmotic injuries and may explain the delayed healing that is seen clinically.

Injuries to the distal tibiofibular syndesmosis account for up to 16% of all ankle sprains¹. This injury, commonly referred to as a high ankle sprain, is associated with a worse prognosis compared with other ligamentous injuries around the ankle. Patients frequently experience increased time away from sports, chronic pain, heterotopic ossification, decreased ankle motion, and long-term disability after a syndesmotic injury²⁻⁴. In contrast to the treatment of nonsyndesmotic ankle sprains, nonoperative treatment of syndesmotic injuries necessitates prolonged periods of non-weight-bearing, immobilization, and rehabilitation^{5,6}. Early recognition and treatment of these injuries may improve prognosis. However, the reason for prolonged recovery time after syndesmotic ligament injury is unclear.

The distal tibiofibular syndesmosis comprises four distinct ligaments (Fig. 1). The interosseous tibiofibular ligament is continuous with the interosseous membrane at its most distal aspect and runs obliquely from the tibia to the fibula in a lateral-distal-anterior direction. The ligament is pyramidal in shape and spans 2 to 3 cm before it terminates approximately 1 cm above the level of the ankle joint⁷. The anteroinferior tibiofibular ligament extends obliquely from the relatively large anterior tubercle of the distal tibia (the Chaput tubercle) to the anterior tubercle of the distal fibula. The posteroinferior tibiofibular ligament extends from the posterior malleolus to the posterior tubercle of the fibula and runs more horizontally than its anterior counterpart⁷. The transverse tibiofibular ligament has been described as either a separate ligament or a

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a.
Fig. 1

Line drawing showing anterior syndesmosis (**Fig. 1-A**) and posterior syndesmosis (**Fig. 1-B**).

deep component of the posteroinferior tibiofibular ligament⁸. The fibers of the transverse ligament lie deep to the posterior ligament and run in a similar direction. In a biomechanical study, Ogilvie-Harris et al. found the following contributions to ankle stability: the anteroinferior tibiofibular ligament, 35%; the interosseous ligament, 22%; the deep portion of the posteroinferior tibiofibular ligament or the transverse tibiofibular ligament, 33%; and the superficial fibers of the posteroinferior tibiofibular ligament, 9%⁹.

To our knowledge, no previous study has shown the vascular supply to the tibiofibular syndesmosis, although the branching pattern of the peroneal artery at the level of the ankle syndesmosis has been described^{8,10,11}. In 1941, Huber reported a perforating, or anterior, branch of the peroneal artery (**Fig. 2**) that pierces the interosseous membrane and runs across the anteroinferior tibiofibular ligament beneath the peroneus tertius tendon¹⁰. Bartoníček also mentioned a perforating branch of the fibular artery that penetrates the interosseous membrane⁷.



Fig. 2



Fig. 3

Fig. 2 Photograph showing the anterior syndesmosis in which the perforating branch (A) of the peroneal artery (B) travels through the interosseous membrane. **Fig. 3** Photograph showing the peroneal artery (A) branching into a perforating branch (B) and a posterior branch (arrow). The perforating branch penetrates the interosseous membrane, an average of 3 cm proximal to the ankle joint.

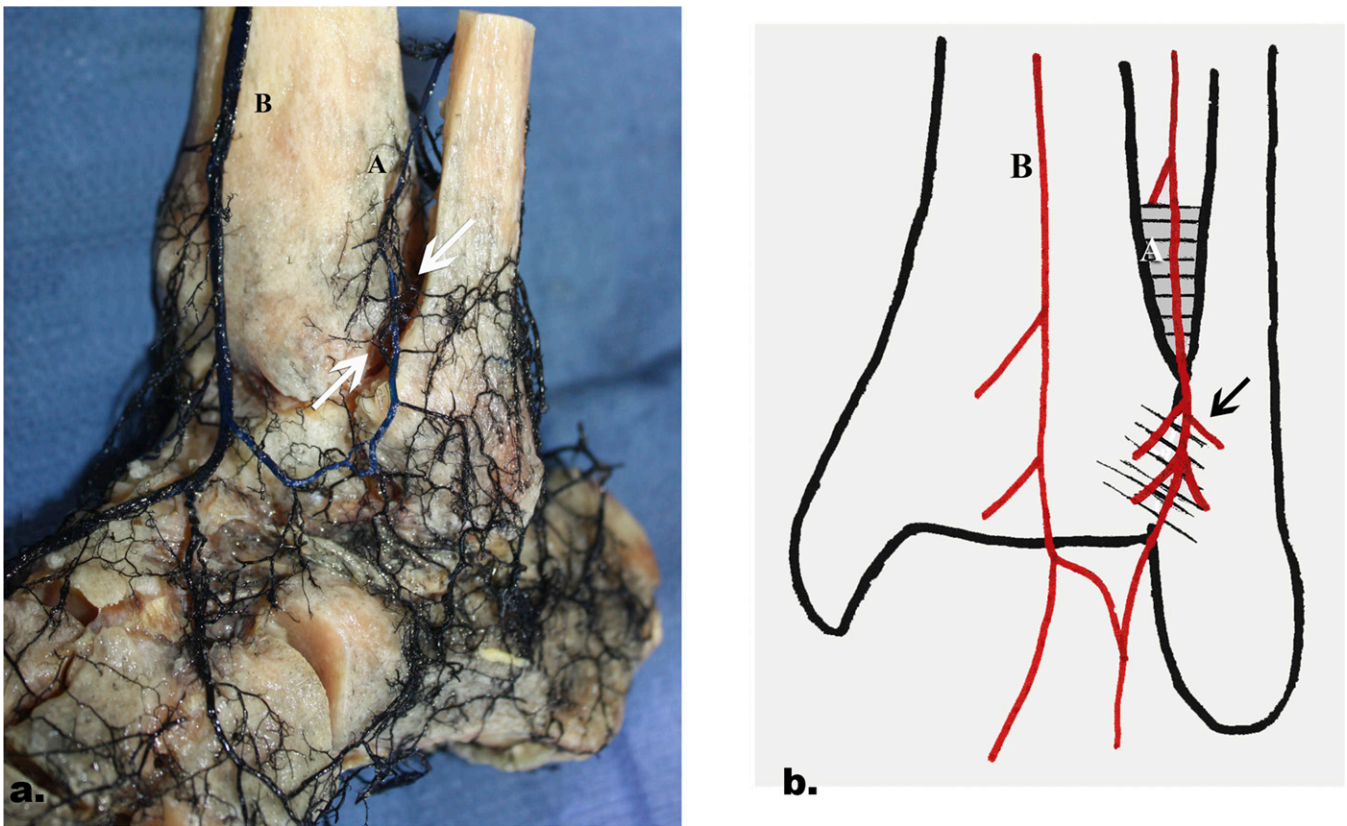


Fig. 4

Photograph (Fig. 4-A) and line drawing (Fig. 4-B) showing Anterior Type 1. The anterior syndesmosis is supplied by the perforating branch of the peroneal artery (A) with multiple small branches (arrows) at the level of the syndesmosis. There is no contribution of the anterior tibial artery (B) at the level of the syndesmosis.

Neither study showed specific branches from this artery running directly to the anteroinferior tibiofibular ligament or any other arteries that were also in proximity to the anterior syndesmosis.

The purpose of this study was to describe the vascular supply to the tibiofibular syndesmosis with use of a method of chemical debridement of cadaveric specimens.

Materials and Methods

Institutional review board exempt status was granted by our university Human Research Protection Office after formal review of the research protocol. Twenty-five pairs of legs (a total of fifty legs) from twenty-five fresh adult human cadavers were obtained from a university-associated body donation program. Only cadavers of individuals who had died within seventy-two hours and that had not been previously frozen or embalmed were accepted. Cadavers with evidence of prior foot or ankle trauma, surgery, deformity, or congenital abnormalities were excluded. A history of ankle sprain or syndesmotomic injury was unknown for all specimens.

The legs were amputated below the knee at the junction of the proximal and middle thirds of the tibial shaft. Skin incisions were made on the dorsal and plantar surfaces of each toe at the level of the proximal interphalangeal joint. The anterior tibial, peroneal, and posterior tibial arteries each were cannulated at the proximal aspect of the specimens by means of an 8-French triple lumen catheter and were manually injected with saline solution until the effluent from the toes was clear. Any obvious atherosclerosis of the vessels at the level of the amputation was noted. India ink was then injected into each artery under constant manual pressure until India ink staining was obvious on the cutaneous surface of the foot and India ink flowed from the skin incisions on the toes. Ward Blue Latex was then injected in a similar fashion.

After injection, the specimens were frozen for at least forty-eight hours. They were subsequently removed from the freezer and were allowed to thaw at room temperature for forty-eight hours. Once fully thawed, the specimens were amputated through the tibia 8 to 10 cm proximal to the ankle joint and the toes were amputated at the metatarsophalangeal joints. An axial pin was placed from the tibia through both the talus and the calcaneus to maintain the integrity of the ankle joint during chemical debridement. The skin and subcutaneous tissues were sharply dissected away, and the specimens were submerged in 6.0% sodium hypochlorite for four to six hours to complete the debridement of the soft tissues. The specimens were checked every thirty minutes and the sodium hypochlorite was refreshed as needed. The debridement process was stopped once the overlying soft tissues had been debrided adequately to allow examination of the vessels and ligaments of the tibiofibular syndesmosis. Although the vessel walls were debrided with the sodium hypochlorite, casts of the vessel lumens filled by Ward Blue Latex remained. At this point, the vascular supply to the syndesmosis was carefully examined, documented, and photographed.

Source of Funding

There was no external funding source for this investigation.

Results

The vascular supply to the anterior tibiofibular syndesmosis was clearly visualized in forty-three of the fifty specimens studied. Seven specimens were excluded from the analysis of the anterior arterial supply after injection and chemical debridement because of either vascular disease (two ankles) or poor injection results (five ankles). The two ankles that had

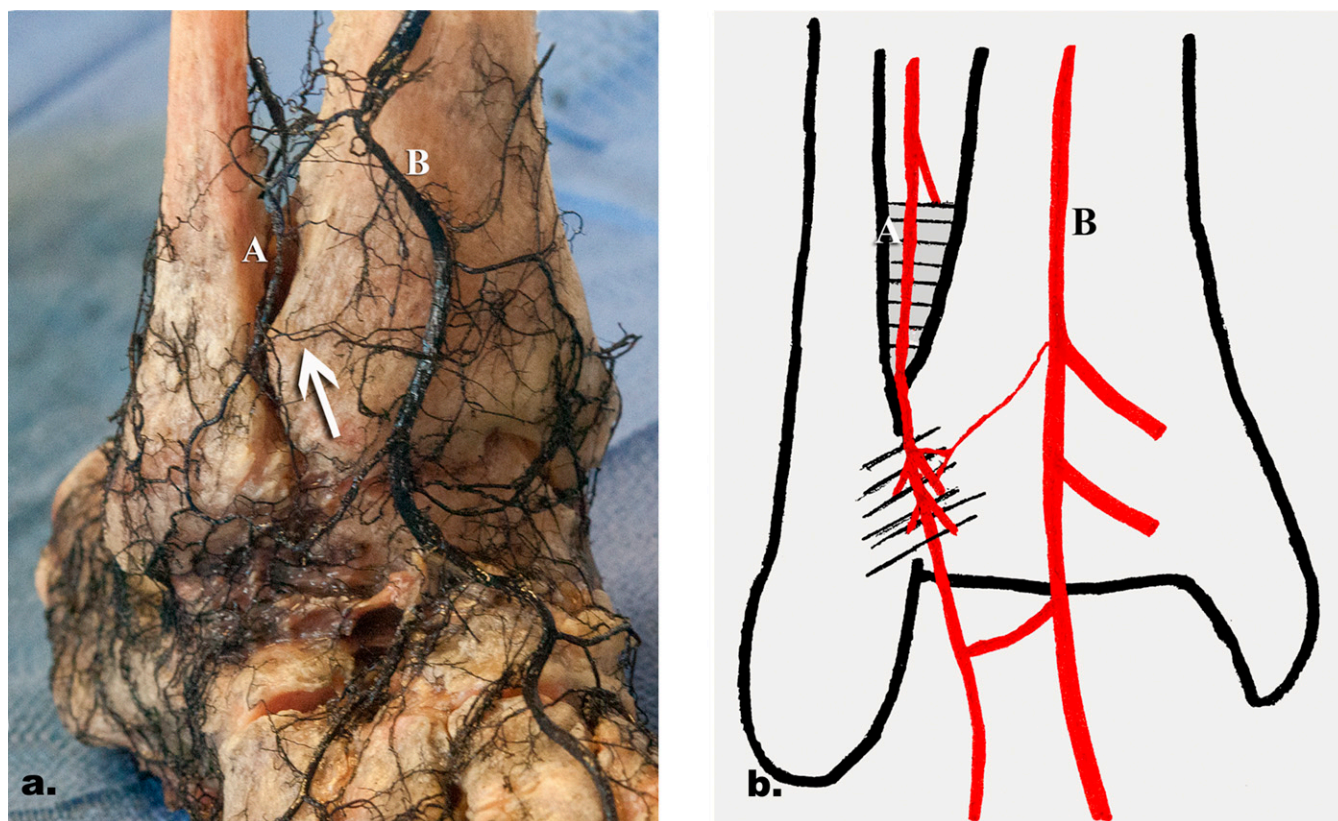


Fig. 5
Photograph (Fig. 5-A) and line drawing (Fig. 5-B) showing Anterior Type 2. The anterior syndesmosis is supplied by the perforating branch of the peroneal artery (A) with multiple small branches (arrow) at the level of the syndesmosis. The anterior tibial artery (B) also provides small branches to supplement the peroneal contribution to the anterior ligaments.

been excluded for vascular disease were a matched pair from the same cadaver with poor filling of the peroneal artery. These specimens had atherosclerotic plaques noted in the large vessels at the time of the injection. Poor filling due to the injection technique may also explain the results seen in these specimens. The specimens that had been determined to have poor injection results had obscuring of the vessels due to leakage of India ink or Ward Blue Latex into the surrounding tissues in one ankle and limited soft-tissue debridement in four ankles. The ankles in which the remaining soft tissues obscured the vessels had a limited response to the chemical debridement despite extending the time over which debridement was allowed.

In the forty-three specimens available for evaluation, there were three primary patterns of vascular supply to the anterior syndesmosis. In each of these patterns, the perforating branch of the peroneal artery contributed branches to the anterior syndesmosis (Fig. 3). These branches all arose distal to the point at which the vessel passed through the interosseous membrane, an average of 3 cm proximal to the ankle joint. Three distinct patterns of blood supply to the anterior ankle syndesmosis were seen.

The first and most common pattern, Anterior Type 1, was seen in twenty-seven (63%) of the forty-three specimens. In these specimens the perforating branch of the peroneal

artery was the only vessel to supply branches to the anterior syndesmosis ligaments. Occasional anastomotic vessels between branches of the perforating branch of the peroneal and the anterior tibial artery were seen distal to the anteroinferior tibiofibular ligament (Fig. 4).

The second pattern, Anterior Type 2, was seen in nine specimens (21%). In these specimens the peroneal artery supplied multiple branches to the anterior ligaments. The blood supply was supplemented by branches of a lesser caliber arising from the anterior tibial artery, although the predominant blood supply continued to arise from the perforating branch of the peroneal artery (Fig. 5).

The third and least common pattern, Anterior Type 3, was seen in seven specimens (16%). In these specimens the anterior tibial artery supplied branches of a caliber larger than that of the branches from the perforating branch of the peroneal artery. The perforating peroneal artery was noted in each of these specimens as a relatively small vessel with few branches (Fig. 6).

The vascular pattern was not always consistent in the matched cadaver pairs. The anterior blood supply was consistent in the right and left ankles in eleven matched ankle pairs consisting of twenty-two of the forty-three ankles evaluated. In nine matched ankle pairs (eighteen ankles) a difference was recorded in the vascular pattern in the right ankle compared

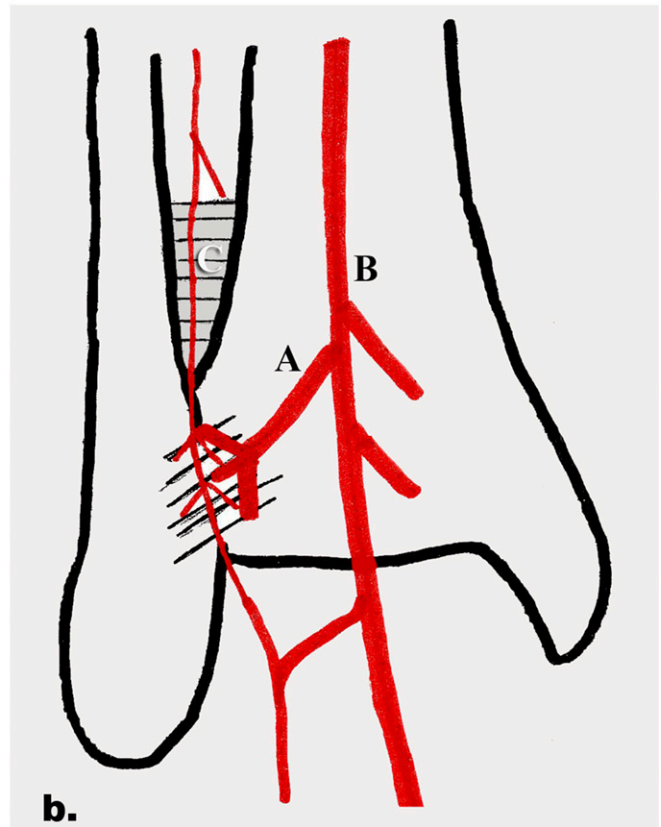


Fig. 6

Photograph (Fig. 6-A) and line drawing (Fig. 6-B) showing Anterior Type 3. The anterior syndesmosis is supplied primarily by larger branches (A) from the anterior tibial artery (B). The perforating branch of the peroneal artery (C) is a smaller vessel in these specimens.

with the left ankle. In three additional ankles the contralateral side was excluded for either vascular disease or poor injection results.

The vascular supply to the posterior tibiofibular syndesmosis was elicited in thirty-eight of the fifty specimens studied. Twelve specimens were excluded after injection and chemical debridement because of either vascular disease (three ankles) or poor injection results (nine ankles). Similar to the anterior syndesmosis, the ankles that had been excluded for vascular disease had poor filling of the posterior branch of the peroneal artery. The specimens that had been excluded for poor injection results had obscuring of the vessels due to leakage of India ink or Ward Blue Latex into the surrounding tissues in five ankles and limited soft-tissue debridement in four ankles. The vascular supply to the posterior syndesmosis arose completely from the peroneal artery in twenty-four (63%) of thirty-eight specimens (Fig. 7), termed the Posterior Type-1 circulation. In fourteen specimens (37%) the posterior tibial artery also provided small branches to supply the posterior syndesmosis (Fig. 8), termed the Posterior Type-2 circulation. There were no specimens in which the posterior tibial artery contribution was the dominant supply to the posterior ligaments. The anterior perforating branch of the peroneal artery did not contribute any branches to the poste-

rior syndesmosis, either proximal or distal to penetrating the interosseous membrane.

Similar to the anterior findings, not all matched pairs had the same posterior vascular pattern noted on the right ankle compared with the left ankle. In eleven matched pairs consisting of twenty-two of the thirty-eight ankles evaluated, the pattern was consistent in the left and right ankles. Seven matched pairs (fourteen ankles) had a difference in the vascularity of the posterior syndesmosis with one side supplied by the peroneal artery only and the other side supplied by both the posterior tibial artery and the peroneal artery. The contralateral side was excluded for vascular disease or poor injection results for two ankles.

Discussion

This descriptive, anatomic study confirms the previous descriptions of the perforating branch of the peroneal artery traveling through the interosseous membrane of the distal tibiofibular syndesmosis. Furthermore, in the majority of these cadaver specimens, this perforating branch provided the primary blood supply to the anterior ligamentous structures. Small branches supplied the anterior ligaments arising from the perforating branch after the artery penetrated the interosseous membrane.

The anterior syndesmosis ligaments are more commonly injured in ankle syndesmosis injuries^{12,13}. On the basis of the

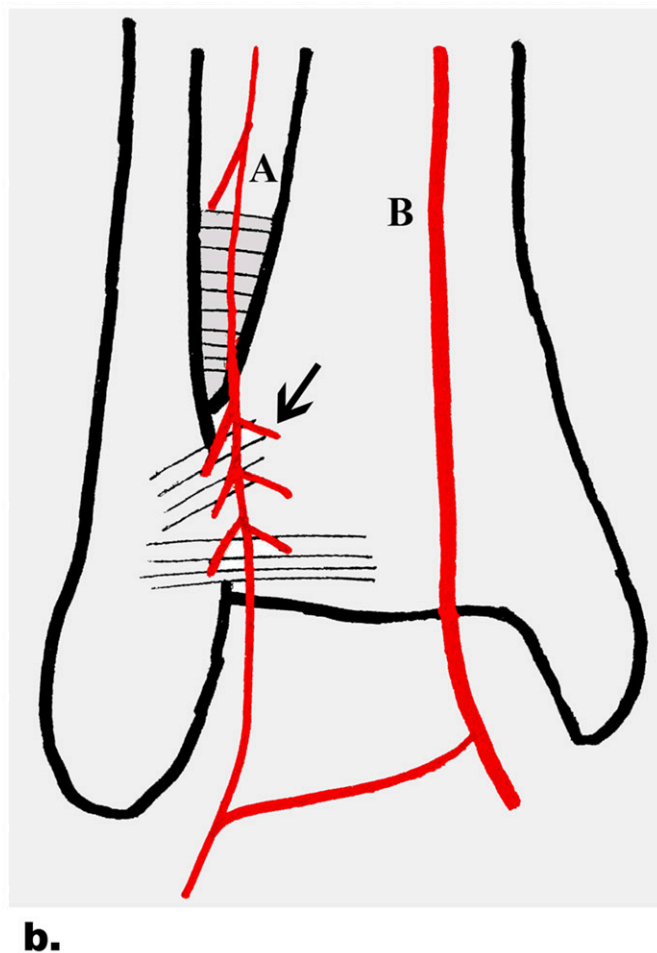
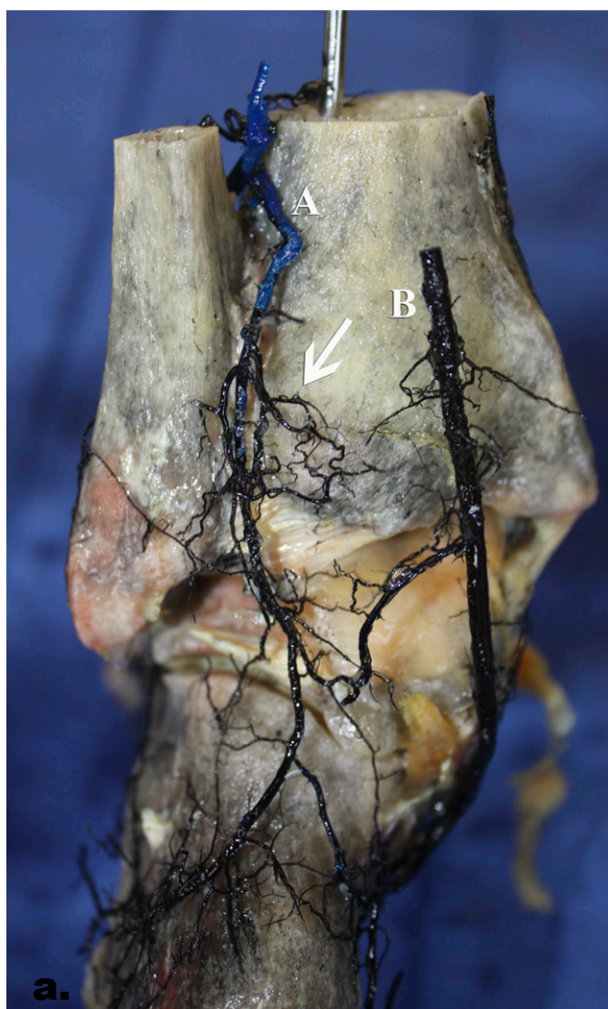


Fig. 7
Photograph (**Fig. 7-A**) and line drawing (**Fig. 7-B**) showing Posterior Type 1. The posterior syndesmosis is supplied by the posterior branch of the peroneal artery (A) with multiple small branches (arrows) at the level of the syndesmosis. There is no contribution of the posterior tibial artery (B) at the level of the syndesmosis.

findings in this study, the perforating branch of the peroneal artery is a primary contributor of the vascular supply to these ligaments. A disruption to this branch in an ankle injury is possible given its close proximity to the interosseous membrane. In syndesmotic disruptions that extend 3 cm proximal to the ankle joint, the perforating branch of the peroneal artery is in the zone of injury and vulnerable to rupture. The loss of this vessel may compromise the vascular supply to the anterior syndesmosis in 63% of ankles on the basis of this analysis and may cause a decrease in blood supply in another 21% of ankles. An injury to this arterial branch at the time of a syndesmotic injury may result in devascularization of the anterior syndesmotic ligaments and is a possible contributing factor to the prolonged healing that is seen clinically. The posterior ligaments have a similar singularity to their vascular supply; however, the primary arterial supply does not penetrate the ligament and may be less susceptible to injury.

Adequate vascular supply is a known prerequisite for tissue healing. The limited blood supply to a number of ana-

tomous structures has been proposed and investigated as a factor in delayed healing or importance when planning surgical exposure¹⁴⁻¹⁸. Surgical incisions in areas of skin with a known tendency for delayed wound-healing have been shown to have limitations in vascular supply. Borrelli and Lashgari described the vascular supply to the lateral hindfoot to offer an anatomic explanation for delayed healing of the corner of the flap with an extensile surgical approach to treat calcaneus fractures¹⁵. Fractures in watershed areas of bone necessitate increased time and are more likely to necessitate surgical intervention for healing to occur. The fifth metatarsal Jones fracture has been an ongoing area of study because of the known tendency for nonunion that is thought to stem from disruption of or limitations to the blood supply in the location of the fracture^{17,18}. Similarly, insufficient vascular supply to an area of ligamentous injury, specifically the tibiofibular syndesmosis, may lead to delayed healing and increased rates of complications.

Although it provided new insight into the anatomy of the tibiofibular syndesmosis, this study had several limitations.

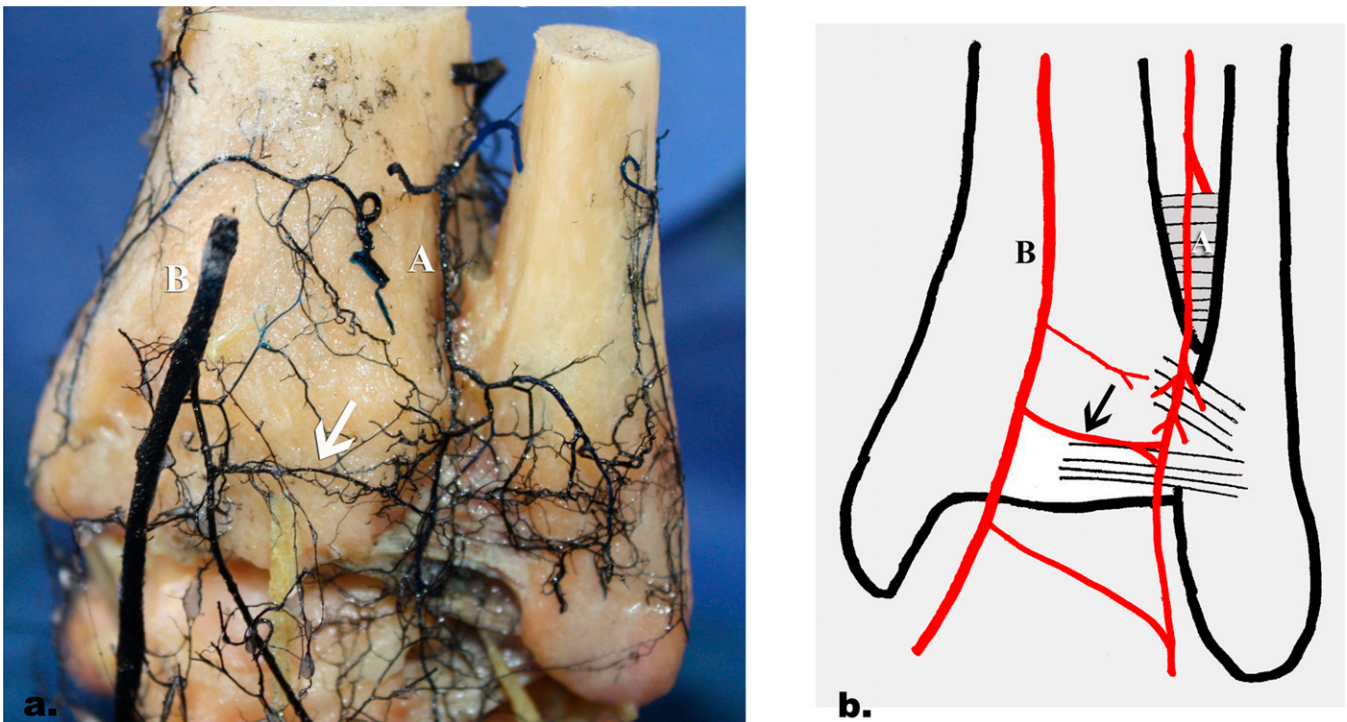


Fig. 8

Photograph (**Fig. 8-A**) and line drawing (**Fig. 8-B**) showing Posterior Type 2. The posterior syndesmosis is supplied by the posterior branch of the peroneal artery (A) with multiple small branches at the level of the syndesmosis. The posterior tibial artery (B) also provides small branches (arrow) to supplement the peroneal contribution to the posterior ligaments.

First, as with any anatomic study, this study was limited by the quality of the specimens. Any specimen with atherosclerotic disease or peripheral edema with dilated vessels could potentially have confused study results. Any of these specimens may have had a history of ankle sprain or syndesmotic injury that could have altered the observed vascular anatomy. There may have been other, less common patterns of vascularity that this study did not identify because of a limited sample size. However, to our knowledge, the present study represents the largest number of specimens for which the vascular supply to the tibiofibular syndesmosis has been described and the only study using injection methods with India ink followed by chemical debridement. Also, these methods resulted in purely qualitative data and lacked a way to quantify the vascular supply to the syndesmosis. The component of vascular supply to the syndesmotic ligaments directly from their osseous attachments was not elucidated with this method.

This descriptive study described the vascular anatomy of the tibiofibular syndesmosis. The location of the perforating branch of the peroneal artery places it at risk when injury to the

syndesmosis extends to the interosseous membrane 3 cm proximal to the ankle joint. Although there is no direct link to associate vascular injury with the clinical challenges of the healing of syndesmotic injuries or high ankle sprains, improved understanding of the anatomy of the syndesmosis may be an important factor in improving our treatment of these injuries. ■

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