

Hardware-Associated Superficial Peroneal Nerve Entrapment Following Syndesmotic Stabilization: A Case Report

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Learning Point of the Article:

Persistent neuropathic pain following syndesmotic stabilization should prompt evaluation for superficial peroneal nerve entrapment, and ultrasound-guided diagnostic injection can establish the diagnosis even when metallic hardware limits direct nerve visualization

Abstract

Introduction: Persistent neuropathic pain following syndesmotic stabilization should prompt consideration of superficial peroneal nerve (SPN) entrapment. The authors report a rare case of hardware-associated SPN entrapment after syndesmotic stabilization for chronic ankle instability following a triplane ankle fracture and highlight the diagnostic value of ultrasound-guided injection when metallic hardware limits direct nerve visualization.

Case Report: A 13-year-old female underwent arthroscopic debridement, syndesmotic stabilization, and lateral ligament reconstruction for persistent pain and instability following a triplane ankle fracture. Her pre-operative mechanical symptoms resolved; however, she subsequently developed dorsal foot paresthesias and electric shock-like pain in the distribution of the SPN. Ultrasound evaluation was limited by acoustic shadowing from syndesmotic hardware, but ultrasound-guided diagnostic injection and hydrodissection resulted in complete but transient symptom relief, supporting the diagnosis of hardware-associated SPN entrapment. Surgical exploration demonstrated the SPN immediately adjacent to the syndesmotic fixation construct. SPN decompression and hardware removal resulted in complete resolution of neuropathic symptoms.

Conclusion: Patients who develop new dorsal foot paresthesias, allodynia, or electric shock-like pain following syndesmotic fixation should be evaluated for SPN entrapment. When metallic hardware limits direct nerve visualization, ultrasound-guided diagnostic injection can provide valuable functional information to establish the diagnosis and guide definitive surgical management.

Keywords: Superficial peroneal nerve, syndesmotic stabilization, suture-button fixation, hydrodissection, ultrasound, peripheral nerve entrapment.

Introduction

Triplane fractures are transitional injuries of the distal tibia that occur during asymmetric physeal closure in skeletally immature adolescents [1,2]. The mechanism of injury involves supination with external rotation and axial compression, producing fracture lines that extend through the epiphysis, physis, and metaphysis in

three planes [1,3]. Triplane fractures account for approximately 5–10% of pediatric intra-articular ankle fractures [2].

Because plain radiographs do not consistently show all fracture fragments and given the intra-articular nature of the fracture, computed tomography (CT) is routinely recommended for accurate fragment assessment and articular displacement

Author's Photo Gallery



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Figure 1: Initial computed tomography scan of the left ankle. Axial (left), coronal (center), and sagittal (right) images demonstrate a triplane fracture of the distal tibia with intra-articular displacement.

[1,4,5]. The accepted threshold for surgical intervention is generally >2 mm of articular displacement, as residual incongruity has been associated with worse long-term functional outcomes and increased risk of post-traumatic arthritis [6,7].

Although most patients achieve favorable outcomes following appropriate treatment, persistent pain after fracture healing should prompt evaluation for occult intra-articular pathology, instability, impingement, or less commonly, peripheral nerve injury [8,9,10].

Superficial peroneal nerve (SPN) injury is an uncommon but recognized complication of ankle trauma [10,11]. Due to its variable anatomy and superficial course across the anterolateral ankle, the SPN and its branches are susceptible to traction, scar entrapment, and iatrogenic injury [10,12,13]. To our knowledge, symptomatic hardware-associated SPN entrapment following syndesmotic stabilization after a triplane fracture has not previously been reported. We present the case of an adolescent athlete who developed persistent neuropathic pain secondary to SPN entrapment following surgical treatment of post-traumatic ankle instability after a triplane fracture.

Case Report

A 13-year-old female soccer player sustained a left triplane ankle fracture after falling from a golf cart. Initial radiographs and CT (Fig. 1) were obtained in the Emergency Department and were interpreted at an outside institution as demonstrating a non-displaced fracture. She was treated non-operatively with cast

immobilization followed by progressive rehabilitation and return-to-sport protocols.

Approximately 8 months after injury, she developed persistent anterior and anterolateral ankle pain upon returning to full athletic activity. Symptoms failed to improve despite physical therapy, activity modification, and sport-specific rehabilitation. Radiographs demonstrated a healed fracture with maintained alignment; however, continued pain prompted referral to our institution.

Physical examination demonstrated focal tenderness over the anterolateral ankle without instability. Review of the original CT scan revealed a comminuted intra-articular triplane fracture with approximately 5 mm of articular

displacement. Magnetic resonance imaging (MRI) demonstrated residual post-traumatic changes within the distal tibial epiphysis, focal chondral irregularity, and signal abnormalities involving the anterior inferior tibiofibular ligament and anterior talofibular ligament (Fig. 2).

To further evaluate the source of her symptoms, the patient underwent a diagnostic and therapeutic intra-articular injection consisting of 40 mg triamcinolone acetonide (Kenalog) and lidocaine. The injection provided temporary relief, suggesting a component of post-traumatic synovitis; however, the patient continued to report ankle instability and apprehension during soccer participation. After failure of prolonged non-operative management, she elected to undergo

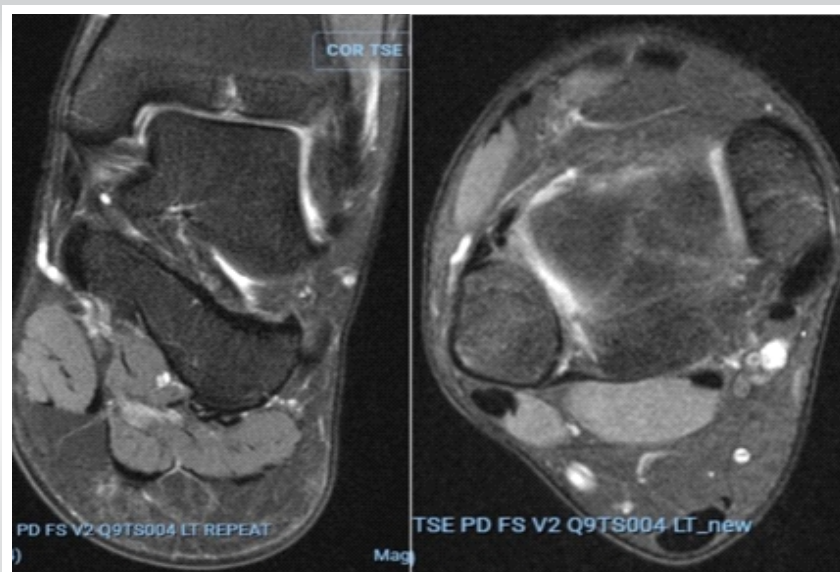


Figure 2: Magnetic resonance imaging of the left ankle obtained for persistent pain following triplane fracture healing. Coronal (left) and axial (right) images demonstrate residual linear signal abnormality in the distal tibial epiphysis, focal chondral irregularity, and signal abnormalities involving the anterior inferior tibiofibular ligament and anterior talofibular ligament.

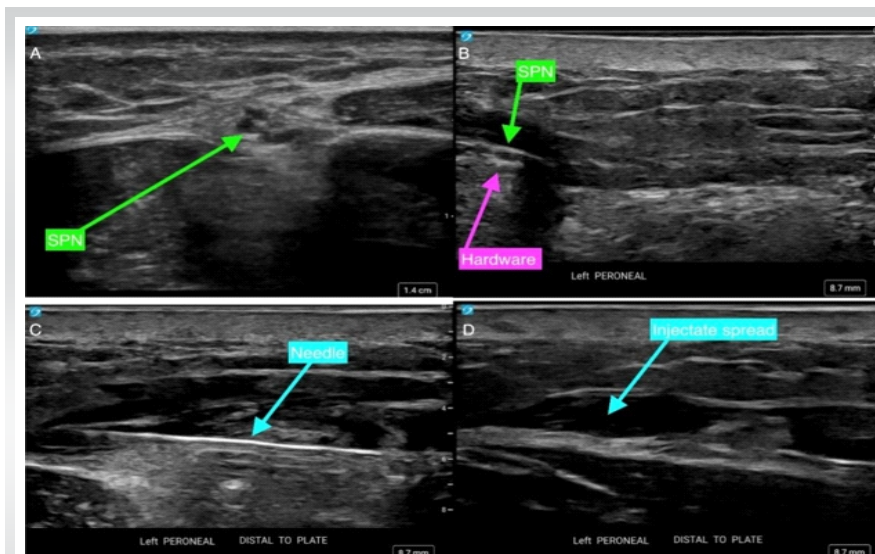


Figure 3: Ultrasound evaluation of the superficial peroneal nerve (SPN) in the setting of suspected SPN entrapment. (a) Ultrasound image demonstrating identification of the SPN with preservation of normal fascicular architecture. (b) Localization of the SPN in the region of syndesmotic fixation. (c) Ultrasound-guided diagnostic injection and hydrodissection. (d) Post-procedural image following hydrodissection demonstrating spread of injectate surrounding the nerve. Despite limited visualization caused by metallic artifact, ultrasound facilitated nerve localization and guided diagnostic intervention.

left ankle arthroscopy, syndesmotic debridement and stabilization, and lateral ligament reconstruction.

Diagnostic arthroscopy demonstrated a prominent anterolateral distal tibial exostosis without significant chondral injury. Open inspection revealed impingement of the syndesmotic ligaments against the distal tibial exostosis, as well as a torn Bassett's ligament, which was debrided. The distal tibia exostosis was excised, after which persistent syndesmotic instability was confirmed under direct visualization. Syndesmotic stabilization was performed using a lateral fibular plate with two suture-button fixation devices (TightRope®, Arthrex) placed under fluoroscopic guidance. The constructs were sequentially tensioned with the ankle held in dorsiflexion, resulting in anatomic reduction and restoration of syndesmotic stability on direct visualization and external rotation stress testing. Concomitant lateral ankle instability was addressed with a Broström-Gould lateral ligament reconstruction using knotless all-suture anchors and suture tape augmentation.

Following surgery, the patient's pre-operative anterior ankle

pain completely resolved. However, she subsequently developed new dorsal foot pain associated with paresthesias, allodynia, and intermittent electric shock-like pain that worsened with athletic activity. Examination demonstrated altered sensation in the distribution of the medial dorsal cutaneous branch of the SPN without recurrent ankle instability.

Given concern for peripheral nerve entrapment, ultrasound evaluation was performed. Visualization of the SPN was significantly limited by acoustic shadowing from the syndesmotic hardware, preventing reliable assessment of the nerve's relationship to the implant (Fig. 3). The nerve could not be continuously traced. Despite these limitations, ultrasound-guided diagnostic injection and hydrodissection were performed. Real-time visualization demonstrated distal spread of the injectate along the course of the SPN, supporting nerve continuity. The injection

resulted in complete but transient resolution of symptoms. Although hydrodissection temporarily alleviated the patient's neuropathic pain, symptoms recurred, suggesting persistent mechanical compression or irritation of the SPN. Pre-operative radiographs obtained before surgery demonstrated maintained syndesmotic reduction with the retained lateral fibular plate and dual suture-button fixation construct (Fig. 4). These findings supported the diagnosis of hardware-associated SPN entrapment and prompted surgical exploration.

Three years after the index procedure, the patient's neuropathic symptoms persisted despite non-operative management. Pre-



Figure 4: Pre-operative radiographs obtained before superficial peroneal nerve (SPN) exploration and hardware removal. (a) Bilateral standing anteroposterior ankle radiographs demonstrating maintained syndesmotic reduction. (b) Left ankle mortise radiograph demonstrating the retained lateral fibular plate and dual suture-button syndesmotic fixation construct. (c) Left ankle lateral radiograph demonstrating maintained alignment of the ankle mortise and retained syndesmotic fixation construct.



Figure 5: Pre-operative localization of the superficial peroneal nerve (SPN). Clinical skin marking (left) and corresponding ultrasound image (right) were used to identify the projected course of the SPN relative to the syndesmotomic hardware before surgical exploration. The longitudinal scar represents the previous lateral surgical approach for syndesmotomic stabilization and is separate from the ultrasound skin marking.

ultrasound (Fig. 6), and was decompressed throughout its course. The nerve appeared structurally intact, and the suture-button fixation construct was removed (Fig. 7).

At the most recent follow-up, approximately 3 weeks after SPN exploration and hardware removal, the patient's neuropathic pain had completely resolved. She had resumed competitive soccer with only 1 day of post-operative downtime. Although these early clinical outcomes are encouraging, longer-term follow-up is necessary to confirm maintenance of syndesmotomic stability and evaluate for the development of post-traumatic degenerative changes.

Discussion

Triplane fractures generally demonstrate favorable outcomes when anatomic reduction is achieved and residual articular displacement is minimized [1,6,14]. Nevertheless, persistent pain following apparent fracture healing should prompt careful evaluation for occult pathology. In the present case, residual articular incongruity, anterolateral impingement, syndesmotomic pathology, and lateral ankle instability contributed to ongoing symptoms and ultimately required operative intervention. Although these mechanical symptoms were successfully addressed, the patient subsequently developed dorsal foot paresthesias, allodynia, and electric shock-like pain that worsened with athletic activity.

Unlike her initial mechanical symptoms, the patient's post-operative complaints were highly suggestive of neuropathic pain. The SPN, particularly its medial dorsal cutaneous branch, is vulnerable to injury because of its superficial course and substantial anatomic variability. Previous studies have demonstrated considerable variation in the location and branching pattern of the SPN, increasing its susceptibility to traction injury, scar entrapment, and iatrogenic injury during ankle surgery [11,13,15,16]. Although SPN injury has been described following ankle trauma, arthroscopy, Broström procedures, and syndesmotomic fixation, symptomatic hardware-associated entrapment remains uncommon and may be difficult to recognize [11,17,18,19]. To the author's knowledge, symptomatic SPN entrapment caused by syndesmotomic stabilization following treatment of a triplane fracture has not previously been reported. Although the SPN was identified and protected during the index procedure, its variable course and the close proximity of the suture-button construct to the nerve likely contributed to delayed post-operative entrapment. This

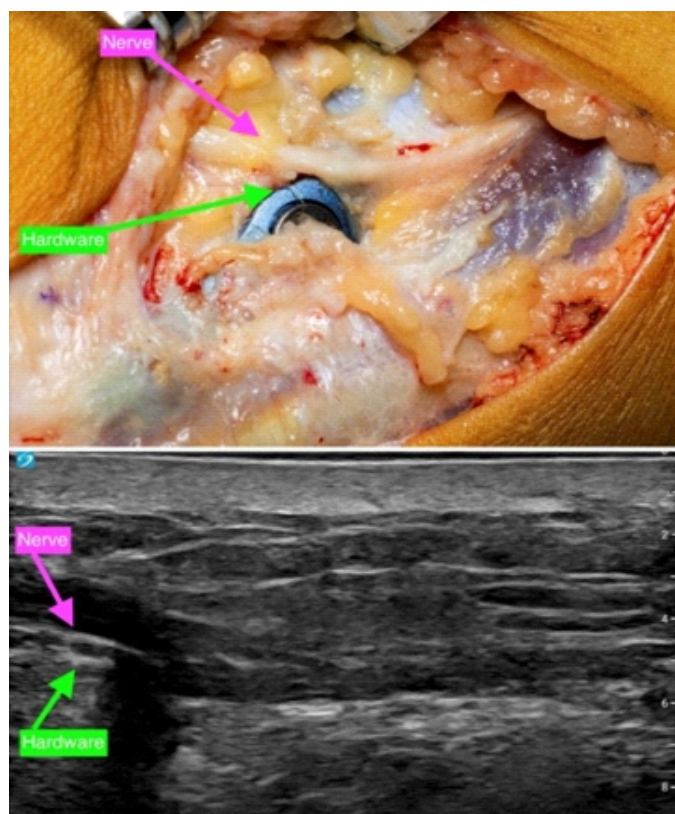


Figure 6: Correlation between pre-operative ultrasound and intraoperative findings. Ultrasound (bottom) demonstrates the superficial peroneal nerve (SPN) immediately adjacent to the syndesmotomic hardware. Surgical exploration (top) confirmed close proximity of the SPN to the hardware, supporting the diagnosis of hardware-associated nerve entrapment.



Figure 7: Intraoperative photographs demonstrating the superficial peroneal nerve (SPN) in close proximity to the syndesmotic hardware. Sequential images show identification of the nerve, neurolysis and decompression at the level of the implant, exposure of the suture-button fixation device, and hardware removal. The SPN remained structurally intact throughout its course.

case illustrates that meticulous intraoperative nerve protection does not completely eliminate the risk of subsequent delayed post-operative hardware-associated nerve irritation.

In the present case, SPN exploration and removal of the suture-button syndesmotic fixation were performed 3 years after the index procedure. At the time of hardware removal, the syndesmosis had healed, and clinical and radiographic assessment demonstrated maintained reduction without evidence of recurrent instability. Therefore, removal of the suture-button construct to facilitate nerve decompression was not expected to compromise syndesmotic stability [20].

A notable feature of this case was the diagnostic complexity. The patient had multiple potential symptom generators throughout her course, including residual articular incongruity, post-traumatic synovitis, syndesmotic instability, and anterolateral impingement. Consequently, her post-operative neuropathic symptoms could have been attributed to persistent instability or incomplete rehabilitation. Instead, the development of dorsal foot paresthesias, allodynia, and exertional neuropathic pain raised suspicion for peripheral nerve pathology. Recognition of the change in symptom quality from mechanical ankle pain to neuropathic dorsal foot pain proved critical in establishing the diagnosis.

A particularly unique aspect of this case was the role of ultrasound in the diagnostic workup. Visualization of the SPN was significantly limited by acoustic shadowing from the metallic syndesmotic hardware, preventing continuous visualization of the nerve and obscuring its relationship to the implant. Consequently, the site of compression could not be directly identified on imaging. Despite these limitations, ultrasound remained clinically valuable by facilitating targeted diagnostic injection and hydrodissection. Complete temporary symptom resolution following injection strongly supported the diagnosis of SPN entrapment despite the inability to directly visualize the site of compression. This finding is particularly relevant in post-operative patients, where metallic hardware may limit the diagnostic utility of conventional imaging modalities. Although pre-operative ultrasound successfully localized the SPN before surgery in this case, this single case does not support routine ultrasound mapping before primary syndesmotic stabilization. Rather, ultrasound may be a useful adjunct in patients with suspected post-operative neuropathic symptoms to aid diagnosis and facilitate pre-operative planning for SPN exploration and hardware removal.

Ultrasound-guided nerve injections have demonstrated excellent outcomes in the evaluation and treatment of foot and ankle neuropathies [21]. Furthermore, ultrasound-guided hydrodissection has emerged as a safe and effective technique for separating entrapped nerves from surrounding scar tissue, with particular utility in surgical entrapment neuropathies [22]. In the present case, hydrodissection resulted in complete but temporary symptom relief, supporting a dynamic compressive neuropathy rather than intrinsic nerve injury. However, because the SPN remained immediately adjacent to the syndesmotic fixation construct, hydrodissection alone did not eliminate the underlying mechanical source of irritation. Symptom recurrence therefore prompted definitive surgical decompression and hardware removal.

This case underscores the diagnostic challenges of evaluating post-operative neuropathic pain after syndesmotic stabilization. Although metallic hardware limited direct visualization of the SPN on ultrasound, ultrasound-guided diagnostic injection provided critical functional information to support the diagnosis, which was confirmed intraoperatively and successfully treated with SPN decompression and hardware removal.

Conclusion

Hardware-associated SPN entrapment is an uncommon but important cause of post-operative neuropathic pain following syndesmotic stabilization. In patients who develop dorsal foot

paresthesias, allodynia, or exertional neuropathic pain following syndesmot fixation, SPN entrapment should be included in the differential diagnosis. Even when metallic artifact limits direct visualization, ultrasound-guided diagnostic injection can provide valuable functional information to support the diagnosis and guide definitive surgical management.

Clinical Message

Persistent neuropathic pain following syndesmot fixation should prompt consideration of SPN entrapment, even when routine imaging is inconclusive. Ultrasound-guided diagnostic injection can establish the diagnosis despite metallic artifact limiting nerve visualization and guide definitive surgical management.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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