

Point-of-Care Ultrasonography in the Diagnosis and Management of Superficial Peroneal Nerve Entrapment: Case Series

Foot & Ankle International

1-5

© The Author(s) 2014

Reprints and permissions:

sagepub.com/journalsPermissions.nav

DOI: 10.1177/1071100714548198

fai.sagepub.com

Jennifer Luz, MD¹, A. Holly Johnson, MD², and Minna J. Kohler, MD³

Level of Evidence: Level V, expert opinion.

Keywords: sports, nerve entrapment, chronic lower extremity pain, musculoskeletal ultrasound

Superficial peroneal nerve (SPN) entrapment at the crural fascia is a rare but perhaps underdiagnosed entity. Over the past few years, advances in ultrasound technology have allowed point-of-care ultrasound to accurately visualize peripheral nerves in the lower extremity, and may be the imaging modality of choice for peripheral nerves due to its higher resolution and ability to track the course of the nerve, even when compared to MRI.^{3,4,17,20} To our knowledge, there have been no reports of utilizing dynamic ultrasound imaging with real-time clinical correlation to identify SPN entrapment. We present 2 cases of SPN entrapment which were successfully diagnosed and treated by point-of-care ultrasonography.

SPN entrapment was first reported by Henry in 1945. He called it “mononeuralgia in the superficial peroneal nerve.”¹¹ There have been few cases reported since its discovery.^{1,5,9,13,14,16,19,25} Among the elusive nerve entrapment syndromes of the lower extremity, SPN entrapment is a difficult diagnosis. First, it is a rare occurrence with early studies citing only 3.5% of cases of lower leg pain due to SPN entrapment.²² Classically, this diagnosis has been based on the physical exam producing sensory abnormalities over the lateral leg and dorsal foot, especially after exercise provocation. Electrophysiology studies and MRI are typically ordered in an attempt to confirm the diagnosis, but these studies can often be normal despite symptoms.⁷ The diagnosis of SPN entrapment almost always comes after a workup for chronic lateral exertional compartment syndrome as these 2 entities may present with an identical clinical history and physical exam.

Case Reports

Case 1

A 33-year-old man presented with 1.5 years of constant dull, aching, and intermittent burning pain of the lateral

right lower leg, without a history of trauma or prior injury. Pain was exacerbated with prolonged activity and resolved with rest. Elevating the painful leg and applying ice reportedly assuaged symptoms temporarily. Previously, he was very active, running 40 minutes, 3 to 4 times per week. At the time of presentation, he was unable to run secondary to pain. Multiple imaging studies (X-rays, EMG, and MRI) were performed previously and were unremarkable. Previous treatment included cast immobilization for 4 weeks, with transition to crutches for a short period of time. He reported minimal relief from lidocaine patch application at the site of pain or from custom orthotics.

The extremity exam revealed tenderness to palpation over the midlateral lower leg with a positive Tinel’s sign. Subtle swelling was noted at the site of pain, approximately 7 cm proximal to the lateral malleolus. The neurovascular exam was otherwise normal. The initial differential diagnosis included lateral chronic exertional compartment syndrome. Prior to compartment pressure testing, he underwent point-of-care ultrasound evaluation to examine local anatomy in correlation with his symptoms. Diagnostic ultrasound visualized the SPN with surrounding hyperechoic fat and a small amount of anechoic fluid at the point of emergence in the subcutaneous tissue between the peroneus longus and brevis

¹Spaulding Rehabilitation Hospital, Harvard Medical School, Department of Physical Medicine & Rehabilitation, Boston, MA, USA

²Massachusetts General Hospital, Harvard Medical School, Department of Orthopedics, Boston, MA, USA

³Massachusetts General Hospital, Harvard Medical School, Division of Rheumatology, Allergy, and Immunology, Yawkey Center for Outpatient Care, Boston, MA, USA

Corresponding Author:

Minna J. Kohler, MD, Massachusetts General Hospital, Harvard Medical School, 55 Fruit St, Yawkey 2C, Boston, MA 02114, USA.

Email: MKohler@partners.org

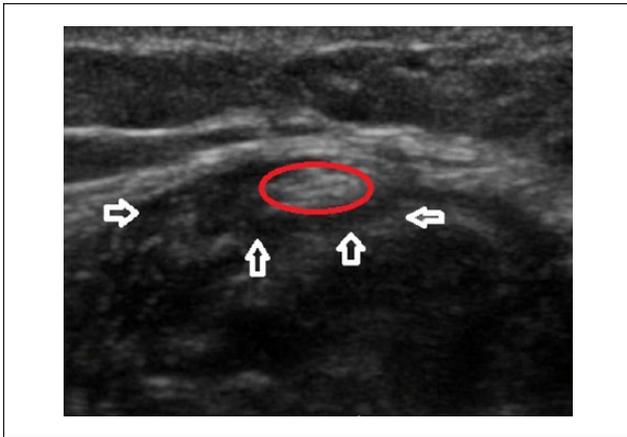


Figure 1. Ultrasound image demonstrating a thickened superficial peroneal nerve (outlined) and surrounding hypoechoic edema (arrows).

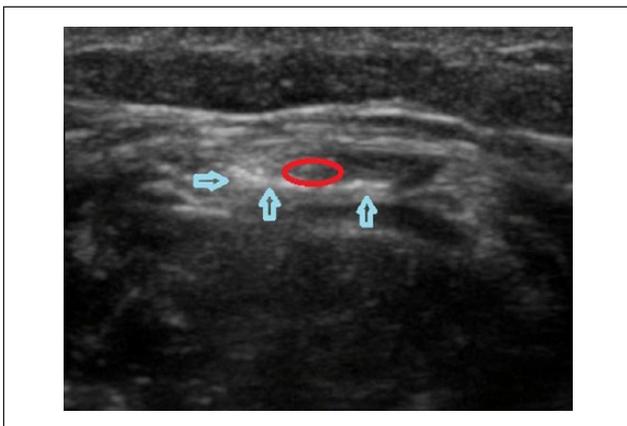


Figure 2. Ultrasound image demonstrating the superficial peroneal nerve (outlined) with surrounding hyperechoic corticosteroid (arrows) after ultrasound-guided injection.

muscles (Figure 1). Ultrasound with clinical correlation confirmed Tinel's sign over the nerve. Dynamic imaging of the nerve during active ankle dorsiflexion with eversion and passive plantarflexion with inversion reproduced his pain. There was normal muscle architecture without fascial herniation or defect.

The patient was diagnosed with SPN entrapment at the crural fascia by dynamic ultrasound imaging in the clinic. Subsequently, he underwent 2 ultrasound-guided perineural corticosteroid injections at the point of entrapment (Figure 2). As neither injection gave any sustained relief, he decided to pursue operative release. A right lateral compartment release and SPN neurolysis was performed. A 10-cm incision was made over the lateral aspect of the right leg in the area where the patient had identified his pain and a positive Tinel's sign. The nerve was first identified directly underneath the fascia



Figure 3. Lateral leg dissection demonstrating the anatomical position of the superficial peroneal nerve at its typical point of exit from the lateral compartment into the superficial soft tissues via the crural tunnel, approximately 7-10 cm proximal to the lateral malleolus.

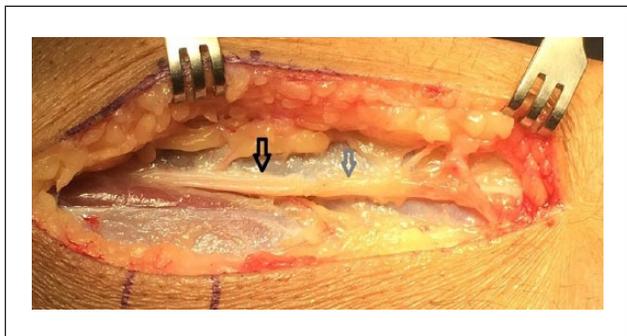


Figure 4. After release of the superficial peroneal nerve from surrounding fascia, there are anatomical changes in the exiting nerve (large arrow) due to strangulation in the tissues including discoloration and thickening (small arrow).

and traced distally to the point of exit into the subcutaneous tissue (Figure 3). The nerve was notably entrapped at its exit point as demonstrated by obviously reduced surrounding free space in the crural tunnel. Meticulous dissection was performed to separate the nerve from the entrapment point. Examination of the nerve revealed some peripheral fatty infiltrate and discoloration, both proximally and distally to the area of entrapment (Figure 4). The lateral fascia was then opened up along the length of the initial incision. The nerve was thoroughly dissected proximally and distally. The nerve rested without tension along the peroneal muscle bed. With ankle range of motion, the nerve glided smoothly along the crural tunnel.

The patient tolerated the procedure well without adverse events. At 2-week follow-up, he was able to partially bear weight on crutches and pain was well controlled with

minimal oxycodone as needed. At 1-month follow-up, he had started a course of physical therapy. He noted 40% overall improvement and described gains in strength training and decreased intensity of tenderness and swelling. At 5-month follow-up, he noted 85% improvement and was walking several hours at a time. He had returned to the gym, was riding a bike, and intended to return to running with further rehabilitation.

Case 2

A 54-year-old man presented with 6 months of worsening pain over the distal one-third of the lateral right leg, just proximal to the lateral malleolus. He described an aching, throbbing pain occurring with every step and persisting at rest, and intermittent paresthesias, which traveled to his foot. Elevation improved the pain, while weight-bearing exacerbated it.

Exam revealed a slight prominence over the lateral right leg approximately 10 cm proximal to the lateral malleolus. There was no tenderness to palpation or pain with range of motion. Neurovascular exam was normal with full strength of the foot and ankle and no gait abnormalities. Plain radiographs of the right leg were unrevealing. Diagnostic ultrasound performed in the clinic revealed normal muscle architecture without a fascial defect in the lateral leg compartment at rest. During dynamic movements, herniation of hyperechoic fat surrounding the SPN as it emerged from the subcutaneous tissues was directly observed. Tinel's sign over the nerve at this location, and dynamic ultrasound exam with active ankle dorsiflexion with eversion and passive plantarflexion with inversion elicited pain arising from the location of the nerve emerging from the subcutaneous tissue.

The patient was diagnosed with SPN entrapment via fat herniation in the crural tunnel. This anatomical situation was causing intermittent SPN entrapment without evidence of nerve edema or strangulation on ultrasound imaging. Subsequently he underwent ultrasound-guided perineural corticosteroid injection with adequate relief. He tolerated the injection procedure well without adverse events. At 1-month follow-up, he reported 90% overall improvement and had returned to running.

Discussion

Lower extremity nerve entrapment syndromes are a difficult entity to diagnose. The peripheral nerves of the lower extremity have a very variable anatomic course and therefore may present with vague, poorly localized symptoms. Physical exam alone may not be able to clearly localize pain and may vary greatly between cases. Most neuropathies of the lower extremity are either caused by mechanical entrapment within their anatomical tunnels through soft tissue or

by irritation in aggravating limb positions.² There are 3 described clinical phases of nerve entrapment that correlate with pathologic changes in the nerve ranging from temporary disruptions in axonal blood supply to more serious segmental demyelination and Wallerian degeneration.⁸ Clinical symptoms range from mild, intermittent paresthesias occurring primarily at night to constant pain which is unrelieved despite pressure relief on the nerve.

This is the first case series of SPN entrapment diagnosed by point-of-care ultrasound. On initial presentation of both cases, the primary differential diagnosis was lateral chronic exertional compartment syndrome (CECS). CECS is a condition of exercise-induced reversible ischemia usually affecting the young, competitive athlete population.⁶ It typically presents with a cramping, squeezing, or aching pain over a muscle compartment which is induced by exercise and relieved with rest.¹⁰ Nerve entrapment syndromes can present with an identical clinical picture to CECS. CECS causes pain due to muscle ischemia, and while there is a similar mechanism of action, nerve entrapment is usually caused by strangulation at a localized area.^{23,24}

In our cases, it was unclear whether symptoms in the lower leg were due to CECS or SPN nerve entrapment, as both conditions may present with a similar clinical picture. Dynamic ultrasound is the process of performing provocative maneuvers in real time under direct ultrasound visualization in an attempt to reproduce symptoms. Dynamic ultrasound was able to clearly identify the location of the SPN and reproduced symptoms as the SPN was directly observed to be entrapped. Diagnostic ultrasound confirmation spared the need for further compartment pressure testing.

Depending on the location of the entrapment, symptoms may vary. The anatomic course of the SPN begins at the bifurcation of the common peroneal nerve. It passes between the peroneal muscles, while supplying motor innervation to them, and the lateral side of extensor digitorum longus. It then travels through the lateral leg, pierces the deep crural fascia, and finally divides into terminal branches, the medial dorsal cutaneous nerve, and the intermediate dorsal cutaneous nerve. If the SPN becomes entrapped in a proximal location within the lateral compartment, exam may reveal weak ankle eversion and dorsiflexion due to affected innervation of the peroneus longus and brevis muscles. In most cases, the SPN becomes entrapped as it emerges through the deep fascia via a short fibrous tunnel to enter the subcutaneous tissue.^{21,22} It then travels distally through the lateral lower leg and divides into medial and intermediate branches which supply the dorsal foot. Entrapment in the fibrous tunnel typically causes localized pain at the tunnel or radiation into the distal one-third of the anterolateral leg. There is often diminished sensation or paresthesias over the dorsal foot, sparing the first web space.⁸ Symptoms are usually aggravated by exercise and relieved with rest. Exam may

reveal tenderness to palpation at the crural tunnel, or a Tinel's sign. Styf described 3 physical examination maneuvers during which the nerve is pulled taut against the crural fascia opening, thereby reproducing the patient's symptoms.²¹ These maneuvers are the following: pressure applied at the fibrous tunnel, while the patient actively dorsiflexes and everts the foot against resistance; passive plantarflexion and inversion without local pressure over the nerve; and while maintaining this position of stretch, gentle percussion is applied over the course of the nerve (Tinel sign). These maneuvers replicate the process by which the nerve is repetitively damaged at the crural tunnel, such as during athletic activities, explaining the etiology of their symptoms.

In several earlier studies, Styf et al described SPN nerve entrapment and noted only 10% of patients had a concurrent lateral compartment syndrome.²¹ There are many associated conditions which may trigger nerve entrapment. Direct causes may include local trauma, or iatrogenic causes such as anterior compartment fasciotomy. Most conditions place repetitive stretch on the nerve and thereby cause chronic damage and resultant symptoms. Such triggers include prolonged kneeling or squatting, fascial defects with muscle or fat herniation, and recurrent ankle sprains or instability.²¹ SPN entrapment has been described in dancers due to peroneal muscle hypertrophy or ballet slippers laced too tightly around the midcalf, both situations causing compression on the nerve.¹²

Commonly, SPN entrapment is diagnosed clinically based on history and physical examination. EMG and NCS may be helpful in some cases, but a normal result does not rule out SPN compression.^{8,18} MRI has been a primary diagnostic imaging modality in the past⁷ but with varying degrees of diagnostic accuracy. Local anesthetic injection has been proposed as both a diagnostic and treatment consideration.

In these cases, dynamic ultrasound imaging was used to identify the SPN as it traversed the lateral compartment during provocative physical exam maneuvers which confirmed the patients' symptoms. In the past, ultrasound had only been suggested helpful in the diagnosis to identify lesions which may be directly compressing the nerve.^{7,8} A study by Canella et al demonstrated the ability of high-resolution ultrasound to follow the course of the SPN. The study confirmed its anatomic variability as it is located in the anterior compartment in 26.7% of cases and divides into its terminal branches before exiting the crural fascia in 6.7% of cases.³ The nerve is easily localized by ultrasound superficial to the fibula between the peroneus brevis and the extensor digitorum longus. It is visualized at the intramuscular septum between the anterior and lateral compartments. It is especially well-depicted traversing the crural tunnel moving the transducer distally to follow its subcutaneous path.^{4,17}

To perform dynamic ultrasound imaging, the ultrasound probe is placed directly over the suspected area of nerve

pathology and real-time imaging of the anatomy in motion is visualized while simultaneously performing provocative maneuvers. This allows for direct observation of tethering of the SPN in the crural tunnel. Both patients confirmed the reproduction of their characteristic pain during the dynamic examination. The normal sonographic appearance of peripheral nerves demonstrates small rounded neurofascicles in a honeycomb pattern with a thin surrounding perineurium, without surrounding fluid or edema. Previous literature has demonstrated the use of ultrasound in visualizing peripheral nerve compression, sometimes better than MR images, and has been most commonly used for identification of carpal tunnel syndrome as well as lower extremity peripheral nerve entrapments. Ultrasound identified anechoic fluid surrounding the SPN in case 1 which provides evidence for perineural edema. Ultrasound also identified fat herniation through the crural tunnel in case 2, which was only visualized during dynamic movements. These observations provided evidence for an activity-induced mechanical nerve entrapment.

Several conservative treatment options exist. Lateral shoe wedge orthotics may decrease stretch on the nerve by placing the ankle in a more valgus position.⁸ Nonsteroidal anti-inflammatory drugs and a relative rest from provocative athletics may ease painful symptoms. Eradicating possible etiologies should be attempted such as removing compressive footwear or correcting biomechanical factors. If ankle instability is present, physical therapy may be helpful to strengthen the joint and for proprioceptive training. Local injection with corticosteroid or local anesthetic may be both diagnostic and therapeutic.¹²

Although conservative measures are useful in many lower extremity neuropathies, the SPN may require operative intervention.^{2,8} To date, there are no randomized controlled trials comparing conservative and operative treatment. Symptoms allowed to persist without operative intervention may result in permanent nerve damage and muscle atrophy.⁷ The most successful operative technique entails simple release of the SPN from the surrounding fascia, particularly within the crural tunnel.^{7,8,12,21,22} At times, a lateral compartment release may be necessary if lateral compartment syndrome is the cause, however this is not usually the case, and a local release is often successful with a less extensive procedure.⁸

Postoperative success depends on clearly identifying the point of compression prior to surgery due to the highly variable course of the SPN.¹⁵ The most common site of compression is usually at the crural tunnel. Styf reported 82% of patients had relief of symptoms after SPN decompression via complete fascial opening at the crural tunnel near the anterior intermuscular septum.²² Malavolta and Malavolta reported immediate pain relief in 12 cases status post an anterolateral longitudinal incision and partial fasciotomy to free the nerve at the site of entrapment. However, 4 of these 12 patients suffered residual permanent dysesthesia with

activity. There were no operative complications and all patients eventually returned to prior activities. All cases had previously failed prior conservative management.¹⁵

Conclusions

SPN entrapment at the crural fascia is rare and clinical presentation can be similar and often difficult to distinguish from CECS. Other diagnostic imaging modalities, such as EMG and MRI, are often unrevealing. Ultrasonography has the ability to visualize and track the course of peripheral nerves to assist with localizing lower extremity symptoms. Our cases demonstrate the ability of dynamic ultrasound imaging of the SPN correlating with provocative physical exam maneuvers to diagnose nerve entrapment syndromes at the point of care. Ultrasound-guided perineural injections may be an effective treatment consideration prior to operative intervention.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

References

- Banerjee T, Koons D. Superficial peroneal nerve entrapment. Report of two cases. *J Neurosurg*. 1981;55:991-992.
- Brewer RB, Gregory AJ. Chronic lower leg pain in athletes: a guide for the differential diagnosis, evaluation, and treatment. *Sports Health*. 2012;4(2):121-127.
- Canella C, Demondion X, Guillin R, et al. Anatomic study of the superficial peroneal nerve using sonography. *AJR Am J Roentgenol*. 2009;193(1):174-179.
- Chin KJ. Ultrasound visualization of the superficial peroneal nerve in the mid-calf. *Anesthesiology*. 2013;118(4):956-965.
- Daghino W, Pasquali M, Faletti C. Superficial peroneal nerve entrapment in a young athlete: the diagnostic contribution of magnetic resonance imaging. *J Foot Ankle Surg*. 1997;36(3):170-172.
- Davis DE, Raikin S, Garras DN, et al. Characteristics of patients with chronic exertional compartment syndrome. *Foot Ankle Int*. 2013;34(10):1349-1354.
- Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. *Radiographics*. 2010;30(4):1001-1019.
- Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle, and foot. *Foot Ankle Clin*. 2011;16(2):255-274.
- Garfin S, Mubarak SJ, Owen CA. Exertional anterolateral-compartment syndrome. Case report with fascial defect, muscle herniation, and superficial peroneal-nerve entrapment. *J Bone Joint Surg Am*. 1977;59(3):404-405.
- George CA, Hutchinson MR. Chronic exertional compartment syndrome. *Clin Sports Med*. 2012;31(2):307-319.
- Henry AK. *Extensile Exposure*. Edinburgh, UK: E&S Livingstone; 1945.
- Kennedy JG, Baxter DE. Nerve disorders in dancers. *Clin Sports Med*. 2008;27(2):329-334.
- Kernohan J, Levack B, Wilson JN. Entrapment of the superficial peroneal nerve. Three case reports. *J Bone Joint Surg Br*. 1985;67(1):60-61.
- Lowdon IM. Superficial peroneal nerve entrapment. A case report. *J Bone Joint Surg Br*. 1985;67(1):58-59.
- Malavolta M, Malavolta L. [Surgery for superficial peroneal nerve entrapment syndrome]. *Oper Orthop Traumatol*. 2007;19(5-6):502-510.
- McAuliffe TB, Fiddian NJ, Browett JP. Entrapment neuropathy of the superficial peroneal nerve. A bilateral case. *J Bone Joint Surg Br*. 1985;67(1):62-63.
- Peer S, Kovacs P, Harpf C, Bodner G. High-resolution sonography of lower extremity peripheral nerves. *J Ultrasound Med*. 2002;21(3):315-322.
- Rosenow DE. Superficial peroneal nerve. *J Neurosurg*. 2007;106(3):520-522.
- Sevinc TT, Kalaci A, Dogramaci Y, Yanat AN. Bilateral superficial peroneal nerve entrapment secondary to anorexia nervosa: a case report. *J Brachial Plex Peripher Nerve Inj*. 2008;3:12.
- Snaith R, Dolan J. Ultrasound-guided superficial peroneal nerve block for foot surgery. *AJR Am J Roentgenol*. 2010;194(6):W538; author reply W542.
- Styf J. Entrapment of the superficial peroneal nerve. Diagnosis and results of decompression. *J Bone Joint Surg Br*. 1989;71(1):131-135.
- Styf J, Morberg P. The superficial peroneal tunnel syndrome. Results of treatment by decompression. *J Bone Joint Surg Br*. 1997;79(5):801-803.
- Tucker AK. Chronic exertional compartment syndrome of the leg. *Curr Rev Musculoskelet Med*. 2010;3(1-4):32-37.
- Wilder RP, Magrum E. Exertional compartment syndrome. *Clin Sports Med*. 2010;29(3):429-435.
- Yang LJ, Gala VC, McGillicuddy JE. Superficial peroneal nerve syndrome: an unusual nerve entrapment. *J Neurosurg*. 2006;104(5):820-823.