Percutaneous Ultrasound-Guided Hydrodissection of a Symptomatic Sural Neuroma

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Abstract

Symptomatic neuromas of the sural nerve are a rare but significant cause of pain and debilitation in athletes. Presentation is usually in the form of chronic pain and dysesthesias or paresthesias of the lateral foot and ankle. Treatment traditionally ranges from conservative measures, such as removing all external compressive forces, to administration of nonsteroidal anti-inflammatory drugs, vitamin B6, tricyclic antidepressants, antiepileptics, or topical anesthetics. This article reports a case of sural nerve entrapment in a 34-year-old male triathlete with a history of recurrent training-induced right-sided gastrocnemius strains. The patient presented with numbness in the right lateral foot and ankle that had persisted for 3 months, after he was treated unsuccessfully with extensive nonoperative measures, including anti-inflammatory drugs, activity modification, and a dedicated physical therapy program of stretching and strengthening. Orthopedic assessment showed worsening pain with forced passive dorsiflexion and manual pressure applied over the distal aspect of the gastrocnemius. Plain radiographs showed normal findings, but in-office ultrasound imaging showed evidence of sural nerve entrapment with edema and neuromatous scar formation in the absence of gastrocnemius or soleus pathology. Percutaneous ultrasound-guided hydrodissection of the sural nerve at the area of symptomatic neuroma and neural edema was performed the same day. The patient had complete relief of symptoms and full return to the preinjury level of participation in competitive sports. This case report shows that hydrodissection, when performed by an experienced physician, can be an effective, minimally invasive technique for neurolysis in the setting of sural nerve entrapment, resulting in improvement in clinical symptoms. [Orthopedics. 2015; 38(11):e1046-e1050.]
Sural nerve entrapment is an important but infrequent cause of pain and debilitation in athletes. Symptoms typically include chronic calf pain accompanied by paresthesia or dysesthesia along the lateral aspect of the foot and ankle. Classically, peripheral nerve entrapment is believed to arise from 2 contributory mechanisms: (1) space-occupying lesions occurring in a fibro-osseous tunnel and (2) fibrosis and thickening of an inextensible fascial opening. The latter is the predominant etiology implicated in sural nerve entrapment.

Conservative therapy for sural nerve entrapment involves removal of external compressive forces (eg, a tight-fitting shoe or boot) and administration of nonsteroidal anti-inflammatory drugs, vitamin B6, tricyclic antidepressants, antiepileptics, or topical anesthetics. Patients who continue to have pain are candidates for surgical decompression with neurolysis. Failure of internal and external neurolysis as a result of recurrent intraneural adhesions has been reported in a subset of cases. Under these circumstances, transection and burial of the nerve in adjacent muscle or fascia relieves symptoms but can lead to permanent loss of nerve function and potential pain.

Hydrodissection is a minimally invasive technique that involves high-pressure injection of fluid to dissect the anatomic planes and tissue spaces. Its utility has been shown in preservation of the neurovascular elements, expansion of tissue spaces to improve localization of injected anesthetics or corticosteroid, and lysis of fibrotic adhesions. Therefore, hydrodissection is a potentially useful treatment modality for peripheral nerve entrapment. A model of sonographically guided percutaneous hydrodissection was recently described for external neurolysis of the lateral femoral cutaneous nerve, with clinical improvement.

**CASE REPORT**

A 34-year-old male triathlete presented with right calf pain and a 3-month history of dysesthesia over the lateral aspect of the right foot and ankle that began after multiple strains of the gastrocnemius during running. The medical history was significant for multiple right-sided gastrocnemius strains that had occurred during training sessions involving ballistic jumping and long-distance running. He had undergone multiple conservative treatment measures, including rest, nonsteroidal anti-inflammatory drugs, compression and long-distance running. He had undergone multiple conservative treatment measures, including rest, nonsteroidal anti-inflammatory drugs, compression

Physical examination showed normal stance and gait, with full active range of motion of all joints bilaterally. Direct palpation and forced passive dorsiflexion of the ankle elicited pain along the distal aspect of the gastrocnemius. Neurologic examination showed decreased sensation to pinprick and light touch over the posterolateral foot and ankle. The results of strength and reflex testing were normal, as were the findings on examination of the contralateral leg.

Plain radiographs of the foot and ankle showed no abnormalities. Further imaging included ultrasound of the right lower limb that showed edema of the epineurium of the sural nerve at the distal aspect of the gastrocnemius. This finding corresponded to the site of pain and discomfort (Figure). Ultrasound examination showed no evidence of associated gastrocnemius, soleus, or Achilles tendon pathology.

Because conservative therapy was unsuccessful, more invasive treatment modalities were discussed with the patient. He preferred to avoid surgery and elected to proceed with ultrasound-guided hydrodissection of the sural nerve. An experienced sports medicine physician (J.H.) performed the hydrodissection and neurolysis as an outpatient procedure on the same day as testing and examination. Sural nerve imaging was performed with the patient in the prone position with a variable 8- to 12-MHz linear transducer set at 12 MHz and a depth of 2.5 cm to best visualize the tissue planes where the sural nerve was located. Images were obtained in short-axis (transverse) and long-axis (sagittal) views at the area of entrapment, proximal to and distal from the restriction. Comparative images were obtained on the contralateral leg. The area of point tenderness in the posterior mid-gastrocnemius correlated to a 3-cm region of the sural nerve that was hypoechoic, with fluid collecting in the epineurium that clearly delineated the neural bundles and was best seen on transverse images. This pattern suggested edema secondary to formation of the neuromatous scar. A pressure mark was made on the skin above the nerve, and mapping images were obtained to ensure that the needle would enter the epineurium.

Strict aseptic technique was observed during all procedures. The skin over the sural nerve was prepared with povidone-iodine antiseptic (Betadine; Purdue Pharma LP, Stamford, Connecticut) 3 times. The skin and subcutaneous tissues were infiltrated with 2 mL 1% lidocaine with 1:200,000 epinephrine and 2 mL 1% lidocaine with 1 mL betamethasone acetate/betamethasone sodium phosphate (Celestone Soluspan; Merck, Whitehouse Station, New Jersey), and 7 mL 5% dextrose was advanced through the skin and subcutaneous tissues and guided directly...
into the epineurium of the sural nerve at the point of entrapment. Location of the needle tip was confirmed with short- and long-axis real-time ultrasound imaging again with the 12-MHz linear transducer. The anesthetic solution was slowly injected along the nerve sheath, and hydrodissection was observed both proximally and distally as the anechoic fluid flowed along the sural nerve, indicating release of the nerve, as reported by Mulvaney. After the solution was injected, the needle was removed. The area was massaged both proximally and distally to enhance hydrodissection release. The patient tolerated the procedure well, with virtually no discomfort.

Telephone communication for follow-up and case monitoring was performed on days 1 and 14 after the hydrodissection procedure. The patient reported nearly complete relief of symptoms on day 1 and had complete relief, with return to participation in competitive sports without disability, by day 14. The patient reported sustained relief months after the procedure, but has not returned for formal re-evaluation and physical examination.

**Discussion**

The sural nerve is purely sensory, although rare variants that contribute sensorimotor innervations have been described. It comprises the union between the medial cutaneous sural nerve (a branch of the tibial nerve) and the lateral cutaneous sural nerve (a branch of the common peroneal nerve). Initially, the medial cutaneous sural nerve alone courses subfascially between the 2 bellies of the gastrocnemius until reaching the musculotendinous junction, where it re-emerges subcutaneously after penetrating a fibrous arcade. The medial cutaneous sural nerve subsequently anastomoses with the lateral cutaneous sural nerve to form the sural nerve, which travels in close proximity to the small saphenous vein over the midline of the posterior calf. Approximately 10 cm proximal to the calcaneus, the sural nerve crosses laterally over the edge of the Achilles tendon, descends immediately posterior to the lateral malleolus and superficial to the common peroneal tendinous sheath, and ramifies on reaching the base of the fifth metatarsal.

Although entrapment can occur anywhere along this course, the most common mechanism is fascial thickening of the fibrous arcade. Fabre et al reported thickening of the fibrous arcade in all athletes in their cohort who were diagnosed with sural nerve entrapment. Even when sural nerve entrapment occurs at sites other than the fibrous arcade, such as the posterior Achilles tendon and the deep fascia of the gastrocnemius nerve, compression and fixation commonly occur secondary to fibrotic adhesions. It is hypothesized that repetitive microtrauma stimulates a pronounced healing response that is characterized by excessive fibrosis that accounts for the increased incidence of sural nerve entrapment in athletes. This is consistent with the current patient, in whom fascial thickening of the fibrous arcade occurred secondary to noncontact injury associated with ballistic jumping and jogging.

Early diagnosis and treatment of sural nerve entrapment is imperative for the prevention of irreversible neuropathy. Acutely, nerve entrapment causes a reduction in intraneural microvascular flow within hours, and this precedes impaired axonal transport and Wallerian degeneration, which typically occurs within days. Continued compression of the nerve results in endoneural edema, segmental demyelination, and ultimately intraneural fibrosis that can occur within weeks. Resolution of nerve conduction block after treatment depends on the extent of demyelination and fibrosis, and severe cases are often irreversible.

In animal studies, surgical manipulation of nerves induced microvascular damage that culminated in a pathologic sequence similar to that of chronic nerve compression. This pathologic process is supported by the finding of recurrent intraneural adhesions in patients undergoing surgical neurolysis. In this regard, hydrodissection offers the advantages of a percutaneous approach and a blunt mode of dissection, minimizing the risk of nerve injury.

Ultrasound guidance is generally considered optional for use with hydrodissection, but the current authors believe that it is necessary. Ultrasound guidance precludes errant placement of the injection needle as well as incomplete hydrodissection. Further, ultrasound is particularly useful as a diagnostic modality for sural nerve entrapment. Up to 40% of patients may have sural nerve variants that lack mutual contributions from the medial cutaneous sural nerve and lateral cutaneous sural nerve or that deviate en route to the fifth metatarsal, complicating diagnosis based on normal anatomic considerations. Findings on physical examination may be unremarkable in patients with relatively minor sural nerve entrapment, or conversely, patients may show vague symptoms that are often mistaken for calcaneal tendinopathy.

Other diagnoses that must be excluded include herniation of the L4-L5 nucleus pulposus, peripheral venous thrombosis, and diabetic neuropathy. Electrophysiologic studies cannot distinguish these pathologies from sural nerve entrapment and show poor sensitivity at proximal anatomic sites. Thus, patients presenting with diffuse pain or altered sensation at or below the level of the calf should undergo ultrasound as a diagnostic modality for sural nerve entrapment. In the current patient, the site of sural nerve entrapment was confirmed by the observation of edema and neuromatous scarring on ultrasound imaging. Liu et al reported sensitivity of 90% for detection of sural nerve compression with measurement of diminished maximal thickness of the nerve fascicles on high-frequency ultrasound.

Potential limitations of the current study include a small sample size, short duration of follow-up, and the variable skill of the sonographer or physician performing the hydrodissection, which could alter results. Furthermore, the utility of ultrasound-
guided percutaneous hydrodissection may be decreased in cases of sural nerve entrapment resulting from osseous abnormalities, such as bony ganglion, osteochondroma, or myositis ossificans. In these rare circumstances, ultrasound visualization would be limited to the outer bony surface and hydrodissection would be inadequate to correct the structural abnormality. Further, relief of symptoms may have been the result of the anti-inflammatory properties of the betamethasone included in the injected mixture rather than the result of the dissection itself. Nonetheless, ultrasound-guided hydrodissection is a promising treatment modality in patients with sural nerve entrapment.

**Conclusion**

Sural nerve entrapment should be considered as a potential etiology in any patient presenting with paresthesia or dysesthesia along the posterolateral leg, foot, or ankle. Ultrasound imaging is helpful for the detection of peripheral entrapment neuropathies, especially when plain radiographic findings are normal. Ultrasound imaging also improves the precision of needle advancement during hydrodissection. In this case report, a 34-year-old male triathlete had right-sided sural nerve entrapment as a result of chronic fascial inflammation at the distal aspect of the gastrocnemius. After unsuccessful treatment with conservative measures, ultrasound-guided hydrodissection of the sural nerve was performed and resulted in complete resolution of symptoms at short-term follow-up. The patient resumed elite athletic training as early as 2 weeks after hydrodissection. Because hydrodissection is a minimally invasive procedure that can be performed in the outpatient setting, it offers an attractive treatment option for sural entrapment neuropathy.

**References**

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