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CASE REPORT **OPEN ACCESS**

Allodynia Relief Post Ultrasonography-guided Dextrose 5% Perineural Hydrodissection for Superficial Peroneal Nerve Entrapment: A Case Report

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Abstract:

Background: Superficial peroneal nerve entrapment is a rare neuropathy caused by mechanical compression of the nerve, usually at its exit from the crural fascia, and may be induced by forced foot inversion and plantar flexion. Conservative initial treatment, including steroid injections, physical rehabilitation, appropriate footwear, and surgical decompression, may be indicated in cases refractory to nonoperative options. Ultrasonography-guided dextrose 5% (D5W) perineural hydrodissection is an interventional therapeutic option for peripheral neuropathy caused by nerve entrapment. We present a case demonstrating the efficacy of dextrose 5% hydrodissection for superficial peroneal nerve entrapment.

Case Presentation: A 43-year-old female flight attendant with no history of trauma presented with progressive burning, tingling, and pain upon touching the inferolateral third of the leg and dorsum of the foot for 2 months, which was refractory to conservative treatments. Tinel's sign and allodynia were found approximately 10 cm above the right lateral malleolus, confirming the diagnosis of peroneal nerve entrapment. Ultrasonography-guided neural hydrodissection of the superficial peroneal nerve using 10 mL D5W was performed 3 times at 1-week intervals. Pain and allodynia were rapidly reduced and progressively disappeared after the third injection and 6-month post-procedure follow-up.

Conclusion: Ultrasonography-guided D5W perineural hydrodissection may be a therapeutic option for superficial peroneal nerve entrapment, as three injections performed at 1-week intervals showed quick recovery at the 6-month follow-up with no adverse events reported.

Keywords: Hydrodissection, Superficial peroneal nerve, Pain management, Neuropathy, Ultrasonography-guided dextrose 5%, DPN, Electromyography.

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1. INTRODUCTION

Entrapment neuropathy, or nerve compression syndrome, refers to a peripheral nerve lesion presenting without an evident external cause and is localized in a narrow path where the nerve passes [1]. The etiology of entrapment neuropathy remains largely unknown. Genetic predisposition is one of the strongest risk factors for entrapment neuropathy [2, 3]. Other risk factors include an increased body mass index, occupational or physical factors, and predisposing systemic diseases, such as diabetes or hypothyroidism.

Entrapment neuropathies of the lower limbs are misunderstood and underdiagnosed. They are marked by pain, dysesthesia, and weakness, more pronounced by specific provoking movements on physical examination. Proximal tibial neuropathy, sural nerve neuropathy, deep gluteal syndrome, and sciatic nerve entrapment are the most frequently occurring neuropathies [4]. The most prevalent are neuropathies of the common peroneal nerve (CPN) or popliteal sciatic nerve in the fibular head [5]. However, entrapment has also been reported in the branches of the CPN, including the deep peroneal nerve (DPN) and superficial peroneal nerve (SPN).

The SPN descends into the leg within the fascial plane between the peroneus longus and extensor digitorum longus muscles before piercing the lateral deep fascia in the distal third of the leg [6]. The sensory function of this nerve pertains to the anterolateral leg and dorsum of the foot, except for the skin between the first and second digits, where the DPN supplies the sensation [7]. In the distal leg, approximately 10 cm proximal to the lateral malleolus, the SPN runs through the lateral compartment of the leg until it becomes superficial at the level of the distal third. The SPN then crosses the deep crural fascia and passes to the anterior compartment, which is the most frequent location of entrapment [8]. Bifurcation of the SPN occurs superficially on the lateral aspect of the ankle and foot, turning the SPN into the medial dorsal and intermediate dorsal cutaneous branches. This superficial location in the lower extremities makes them particularly susceptible to injuries caused by direct damage mechanisms or, in rare cases, compression by the fascia [7]. Ankle sprains and twisting can stretch and compress the SPN while it exits the deep fascia of the legs [9].

SPN entrapment is an uncommon neuropathy that may occur because of mechanical nerve compression. Such neuropathy usually occurs at the exit, where the nerve pierces the crural fascia and travels within the subcutaneous tissue [7, 10]. SPN entrapment is commonly observed in dancers and athletes, where forced foot inversion and plantar flexion overstretch and injure the nerve at the exit point. Other causes include recurrent stretching, ankle sprains, or masses, such as ganglia [11]. SPN involvement is marked by hypesthesia or dysesthesia in the lower lateral leg and dorsum of the foot, burning pain in the anterolateral leg and dorsum of the foot, and retrograde pain upon pressure application at the entrapment point [12].

Conservative initial treatment includes physical therapy, appropriate footwear, injections, and rehabilitation for ankle instability. Enhancing the power of the dorsiflexors and evertors is recommended for injuries involving muscle weakness [11]. Ultrasonography (USG)-guided perineural hydrodissection using a nonirritating solution is an alternative treatment that should be considered prior to surgical intervention [13]. Recently, USG-guided peripheral nerve hydrodissection has been widely used by medical personnel, especially in pain management and musculoskeletal medicine. The perineural hydrodissection technique uses a fluid injection to separate the nerve from the surrounding or adjacent structures, such as the fascia, tendons, or ligaments, which are believed to cause narrowing of the area around the nerve, irritating the nerve during movement and at rest [14].

Perineural hydrodissection procedures involving steroids, platelet-rich plasma, and dextrose are widely used in various solutions. Steroids combined with platelet-rich plasma (PRP) or 10 mL 5% dextrose (D5W) are widely used for nerve entrapment. Here, we report on a case of SPN entrapment and explore the efficacy of treatment with D5W alone.

2. CASE REPORT

A 43-year-old female flight attendant presented with a burning and tingling sensation in the inferolateral third of the leg and dorsum of the foot that had been present for 2 months. The pain was initially mild but progressively became severe and sensitive to touch despite the consumption of anti-inflammatory and neuropathic pain medications and physical therapy for a few weeks, with a Numeric Rating Scale (NRS) of 9. The pain persisted and interfered with her work activities. She had worked as a flight attendant for 14 years and claimed to have never experienced trauma or sprains or actively exercised. One month before the hospital visit, the tingling sensation decreased and became intermittent. The patient denied having lower back or radicular pain.

During physical and neurological examinations, applying pressure to the lower lateral leg produced a radiating tingling pain that extended to the dorsum of the left foot. Tinel's sign and allodynia were observed approximately 10 cm above the right lateral malleolus. The painful area did not exhibit hypoesthesia; however, pain presented when the patient stood up and resolved when she rested, with an NRS of 8. No skin, hair, sweat, or trophic changes were observed. The patient had an intact motor function. Electromyography (EMG) and nerve conduction studies of the lower extremities during this first visit to the hospital were within normal limits (Table **1** and Fig. **1**).

Table 1. Nerve conduction studies.

ANTI SENSORY SUMMARY											
Stim Site	**NR**	**Peak (ms)**	**Norm Onset (ms)**	**O-P Amp (μV)**	**Norm O-P Amp**	**Site1**	**Site2**	**Delta-0 (ms)**	**Dist (cm)**	**Vel (m/s)**	**Norm Vel (m/s)**
Left Peroneal Sup Anti Sensory (Ant Lat Mall)											
14 cm		2,8		24,6	>5,0	14 cm	Ant Lat Mall	2,2	14,0	64	
Right Peroneal Sup Anti Sensory (Ant Lat Mall)											
14 cm		2,8		17,3	>5,0	14 cm	Ant Lat Mall	2,1	14,0	67	
Left Sural Anti Sensory (Lat Mall)											
Calf		2,7		12,4	>5,0	Calf	Lat Mall	2,0	14,0	70	
Right Sural Anti Sensory (Lat Mall)											
Calf		2,6		17,3	>5,0	Calf	Lat Mall	1,9	14,0	74	
MOTOR SUMMARY											
Stim Site	**NR**	**Peak (ms)**	**Norm Onset (ms)**	**O-P Amp (μV)**	**Norm O-P Amp**	**Site1**	**Site2**	**Delta-0 (ms)**	**Dist (cm)**	**Vel (m/s)**	**Norm Vel (m/s)**
Left Peroneal Motor (Ext Dig Brev)											
Maleolus medial		2,1	<6,1	6,7	>2,5	B fib	Malleolus medial	7,7	14,0		>38
B Fib		9,8		7,0							
Right Peroneal Motor (Ext Dig Brev)											
Maleolus medial		2,6	<6,1	8,7	>2,5	B Fib	Maleolus medial	7,1	32,0	45	>38
B Fib		9,7		7,3							
Left Tibial Motor (Abd Hall Brev)											
Ankle		4,0	<6,1	16,1	>3,0	Knee	Ankle	7,0	34,0	49	>35
Knee		11,0		16,1							
Right Tibial Motor (Abd Hall Brev)											
Ankle		4,3	<6,1	17,8	>3,0	Knee	Ankle	7,4	32,0	43	>35
Knee		11,7		12,8							
F WAVE STUDIES											
Min-F	**Max-F**	**Dispersion**	**Persistence**		**Mean-F**	**F-Norm**		**L-R Mean-F Norm**		**F/M Ratio**	**F-M Lat (ms)**
Right Tibial (Curs) (Abd Hallucis)											
46,88	52,19	5,31	100,00		48,23	<61		<5,7		1,08	43,13
ANTI SENSORY LEFT/RIGHT COMPARISONS											
Stim Site	**L lat (ms)**	**R lat (ms)**	**L-R Lat (ms)**	**L Amp (μV)**	**R Amp (μV)**	**L-R Amp (%)**	**Site1**	**Site2**	**L Vel (m/s)**	**R Vel (m/s)**	**L-R Vel (m/s)**
Peroneal Sup Anti Sensory (Ant Lat Mall)											
14 cm	2,2	2,1	0,1	24,6	17,3	29,7	14 cm	Ant Lat Mall	64	67	3
Sural Anti Sensory (Lat Mall)											
Calf	2,0	1,9	0,1	12,4	17,3	28,3	Calf	Lat Mall	70	74	4
MOTOR LEFT/RIGHT COMPARISON											
Stim Site	**L lat (ms)**	**R lat (ms)**	**L-R Lat (ms)**	**L Amp (μV)**	**R Amp (μV)**	**L-R Amp (%)**	**Site1**	**Site2**	**L Vel (m/s)**	**R Vel (m/s)**	**L-R Vel (m/s)**
Peroneal Motor (Ext Dig Brev)											
Maleolus medial	2,1	2,6	0,5	6,7	8,7	23,0	B Fib	Maleolus medial		45	
B Fib	9,8	9,7	0,1	7,0	7,3	4,1					
Tibial Motor (Abd Hall Brev)											
Ankle	4,0	4,3	0,3	16,1	17,8	9,6	Knee	Ankle	49	43	6
Knee	11,0	11,7	0,7	16,1	12,8	20,5					

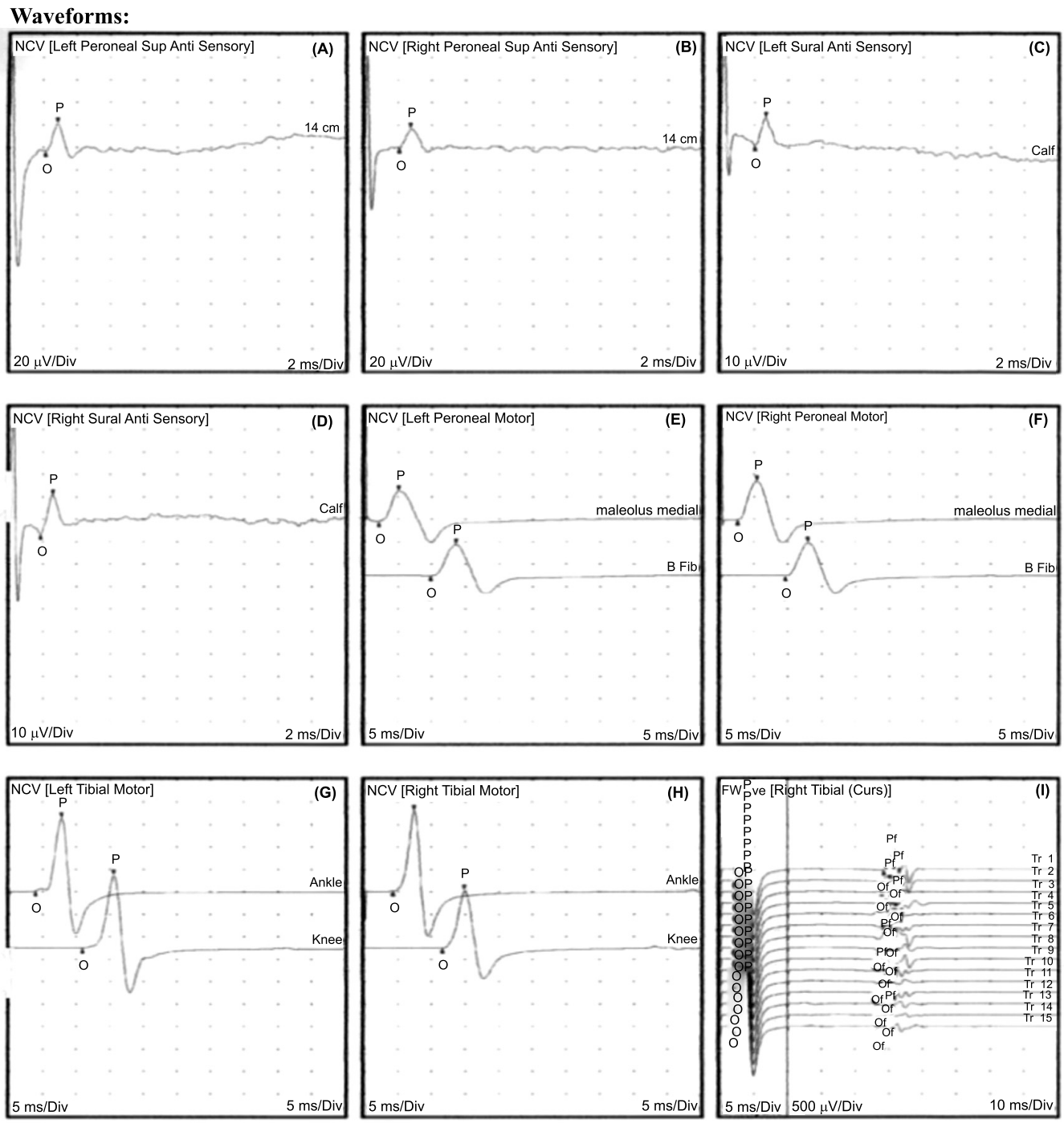

Fig. (1). Electroneurography findings showing (**A**) Left superficial peroneal sensory nerve, (**B**) Right superficial peroneal sensory nerve, (**C**) Left sural sensory nerve, (**D**) Right sural sensory nerve, (**E**) Left peroneal motor nerve, (**F**) Right peroneal motor nerve, (**G**) Left tibial motor nerve, (**H**) Right tibial motor nerve; and (**I**) F-wave of right tibial nerve.

A diagnostic block and hydrodissection of the SPN were performed under USG guidance. A linear high-frequency probe (Wisonic Navi) was used, and a 21G 5 cm needle was inserted from posterior to anterior, targeting the SPN below the crural fascia (Fig. **2**). The nerve was hydrodissected from the fascia using 10 mL D5W. Neural hydrodissection was performed three times with a 1-week interval between each procedure. After the first

procedure, the patient immediately felt a decrease in pain, especially with weighted walking on the affected leg, with an NRS of 4. The pain scale score at the second procedure (1 week after the first procedure) was 3 on the NRS and further reduced to 1 at the third procedure. At the 2-week and 6-month follow-up appointments, no allodynia, pain while walking, or Tinel's sign were reported. During all procedures, no adverse event was reported. Post-procedural electromyography (EMG) and nerve conduction studies of the lower extremities were not done due to the resolution of clinical symptoms. The complete timeline is presented in Fig. (**3**).

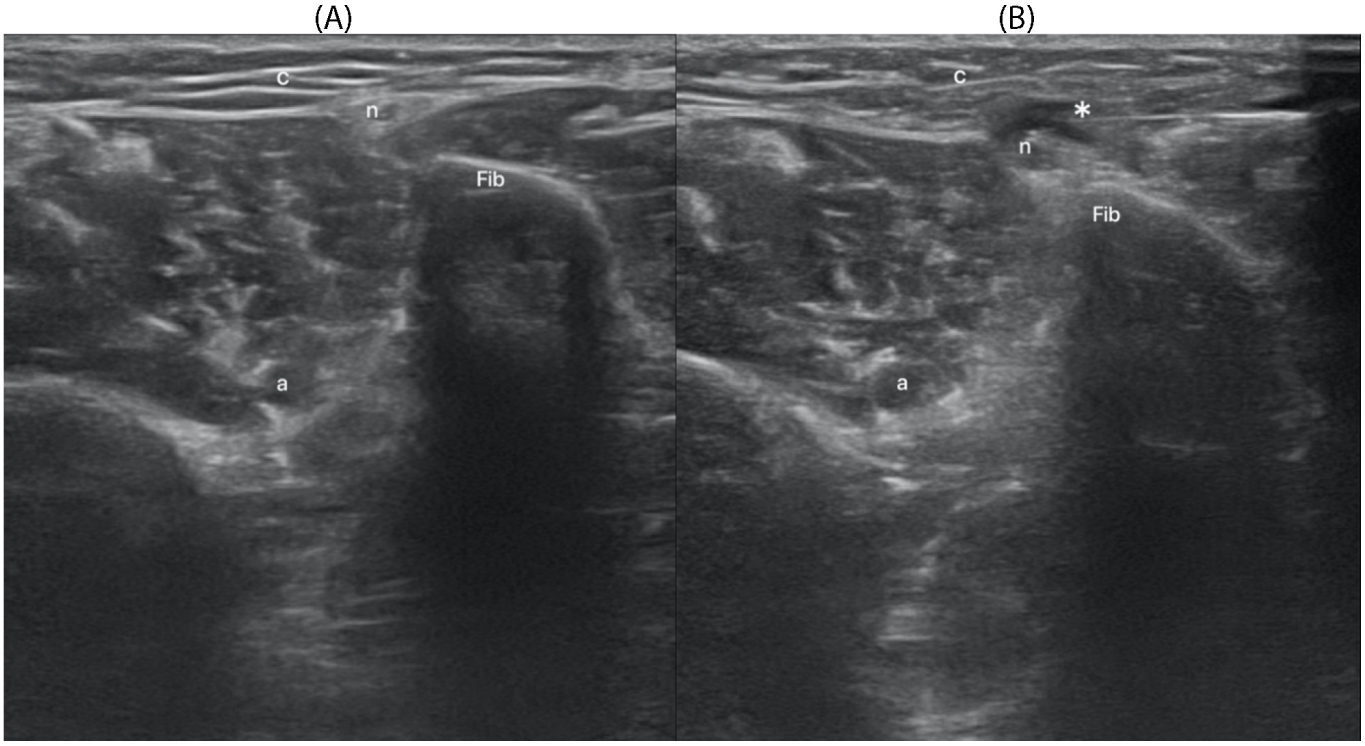

Fig. (2). Superficial peroneal nerve (SPN) entrapment and hydrodissection (**A**) Anatomical variation of the SPN. (**B**) Hydrodissection targeting the SPN. Fib = fibula, a = artery, c = crural fascia, n = SPN, * = needle tip

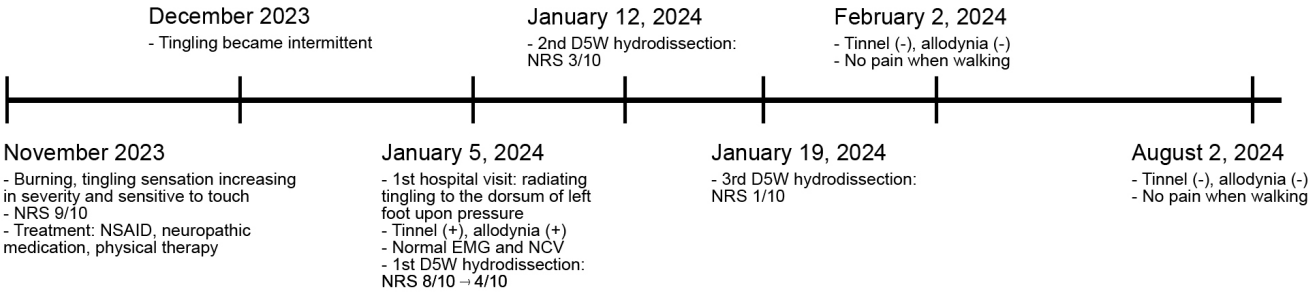

Fig. (3). Timeline of the case presentation.

3. DISCUSSION

We presented the case of SPN entrapment and the effectiveness of hydrodissection using D5W. SPN entrapment is often diagnosed based only on clinical manifestations. In certain cases, electrodiagnostic examination can detect abnormalities in the SPN's conduction velocity, latency, and amplitude but not the side or location of nerve compression. Neuromuscular USG can indicate the compressed side of the SPN. In cases of nerve entrapment, a focal area of nerve enlargement can be observed proximal to the compression site [15]. EMG examination did not show any signs of small-fiber neuropathy, and the USG did not show any enlargement of the nerve proximal to the compression area; this indicated that the diagnosis was based only on clinical findings.

In this case report, the patient did not experience direct trauma to the lower leg or engage in athletic activity demanding intense use of the lower leg. She worked as a flight attendant and wore high heels at work; frequent and prolonged use of high-heeled shoes can cause excessive strain and pressure on the fascial tissues of the feet and calves. Furthermore, these shoes can change the position of the foot and affect weight distribution, increasing pressure on the tissues around the foot and potentially causing inflammation, strain, and adhesion in the fascia. Changed foot position can also affect the calf muscles when walking. The calf muscles work harder than usual to balance the body and support its weight, increasing pressure on the muscles and fascia. When the fascia develops abnormal adhesions, it exerts pressure on the nearby nerves [16, 17].

Although there is no evidence connecting prolonged wearing of high heels to pain due to pressure on the SPN, our patient had no history of trauma or other activities that could cause fascial adhesions or nerve pressure. The presenting symptom in this patient was allodynia in the area where the SPN pierced the fascia, approximately 10 cm above the calcaneus, without focal swelling, pain, or point tenderness.

As in other cases of entrapment, SPN entrapment therapy starts with conservative treatments, including a combination of non-steroidal anti-inflammatory drugs with rest and muscle strengthening through physical rehabilitation in cases of weakness or recurrent ankle sprains and removal of risk factors and triggers. The use of antineuropathic medications can also help reduce or eliminate symptoms, especially in complex regional pain syndrome (CRPS) [18]. Diagnostic block using an injection of steroids with lidocaine around the targeted site in the lower leg can confirm the area of nerve compression and also reduce symptoms. In our reported case, combined treatment using medication and physical therapy had already been administered for a short duration without a significant reduction in pain; therefore, other treatment options were explored.

For cases of SPN entrapment refractory to conservative treatment, surgical decompression with partial or full fasciotomy is indicated to release the compressed nerve at the lateral leg. Styf and Morberg reported that 80% of their patients were symptom-free or satisfied with the results after decompression of the SPN [19]. Sridhara and Izzo performed surgical decompression and reported complete symptom relief. Johnston and Howell reported dramatic pain relief following release and anterior transposition of the nerve in neuralgia patients due to inversion ankle sprain [18].

A novel perineural injection technique used recently in cases of nerve entrapment is USG-guided neural hydrodissection, which has been proven to be effective and safe by multiple randomized controlled trials [20, 21]. Perineural hydrodissection utilizes an anesthetic or solution to create a separation between the nerve and the surrounding tissue, fascia, or nearby structures that compress or irritate the nerve, whether during movement or at rest [20]. The fluids used in hydrodissection include normal saline (NS), corticosteroids, D5W, and PRP [22].

Nuno et al. and Abdelkader-Azizar et al. suggested that consecutive USG-guided hydrodissection techniques using a combination of local anesthesia and corticosteroids may be valuable options to treat SPN entrapment neuropathy due to trauma. Nuno et al. reported a case of chronic neuropathic pain due to compression of SPN by peroneus brevis, where under USG guidance, in-plane hydrodissection was performed using a combination of 10 mL local anesthesia and corticosteroids until the epineurium was completely separated from the surrounding tissue. The results were satisfactory, with complete resolution of the clinical symptoms after injection [5, 23]. Song et al. presented a similar case of SPN entrapment as proven by enlargement of the peroneal nerve on USG, where hydrodissection using NS and corticosteroids resolved the clinical symptoms completely [13]. Another case series reported long-term clinical improvement of superior cluneal nerve entrapment following ultrasound-guided perineural D5W injection [24]. In this case series, neural hydrodissection was performed twice at two-week intervals, with patients receiving oral acetaminophen for pain relief. In contrast, our case involved three hydrodissection procedures without the use of oral analgesics.

In our case, neural hydrodissection was performed using D5W without corticosteroids or local anesthesia. D5W is frequently utilized in clinical practice due to its lack of significant adverse effects. Nevertheless, the use of D5W hydrodissection for treating SPN entrapment has yet to be reported. The 6-month follow-up data of our patient treated with USG-guided D5W hydrodissection demonstrated significant clinical improvement, as seen through the reduction in NRS. Perineural hydrodissection with D5W was performed under ultrasound guidance to separate the SPN from the fascia and cover it to provide a decompression effect. Ultrasound guidance improves injection accuracy by allowing real-time visualization of soft tissue planes and facilitating precise hydrodissection, ensuring that the injectate surrounds the nerve and separates it from adjacent fascia [25].

Several hypotheses regarding D5W's role in pain relief have been made. D5W is thought to downregulate transient receptor potential vanilloid-type 1 (TRPV-1), affecting the sensorineural mechanism. Another proposed hypothesis suggests that glucose alleviates neurogenic inflammation and that correcting hypoglycemia can reduce C-fiber activation. The rapid normalization of high C-fiber activity following glucose administration suggests that D5W injection may stabilize neural activity, regulate neurogenic metabolism, and reduce neurogenic inflammation, hence decreasing neuropathic pain [14]. Our patient's pain and allodynia reduced rapidly after the first week, and she progressively recovered until the symptoms disappeared after the third injection and at the 6-month post-procedure follow-up. The use of D5W without anti-inflammatory therapy in this case report is a rare approach for SPN entrapment and demonstrates the effectiveness of D5W in reducing pain, especially non-chronic pain without neurological deficits. Further research could extend the follow-up period to evaluate the long-term effectiveness of D5W hydrodissection or demonstrate efficacy through case series studies.

CONCLUSION

This case underscores the potential of D5W as a safe, non-pharmacologic, and minimally invasive treatment option for entrapment neuropathies, particularly in patients without structural nerve damage or chronic deficits. From a clinical standpoint, this approach may be especially valuable in patients who are contraindicated for steroid use or prefer steroid-free interventions. Moreover, it supports the integration of musculoskeletal ultrasonography into the diagnostic and therapeutic algorithm for peripheral nerve entrapment syndromes, enhancing diagnostic accuracy and procedural safety. USG-guided D5W hydrodissection could serve as a therapeutic option for SPN entrapment refractory to conservative treatments.

AUTHORS' CONTRIBUTIONS

It is hereby acknowledged that all authors have accepted responsibility for the manuscript's content and consented to its submission. They have meticulously reviewed all results and unanimously approved the final version of the manuscript.

LIST OF ABBREVIATIONS

CPN	=	Common Peroneal Nerve
DPN	=	Deep Peroneal Nerve
SPN	=	Superficial Peroneal Nerve
USG	=	Ultrasonography

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

HUMAN AND ANIMAL RIGHTS

Not applicable.

CONSENT FOR PUBLICATION

Informed consent was obtained from the participant.

STANDARDS OF REPORTING

CARE guidelines were followed.

AVAILABILITY OF DATA AND MATERIALS

The data and supportive information are available within the article.

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None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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