Case Report

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Steroid Infiltrations Can Alleviate Refractive Superficial Peroneal Nerve Neuropraxia after Ankle Sprain: A Case Series

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Introduction

Ankle sprains occur mainly in young patients with high sports activity levels.[4] Prolonged symptom risk after injury is high and around 40% report permanent symptoms.[4] Nerve injuries account for a small proportion of complications associated with ankle sprains, but the exact incidence remains unclear.[9] The superficial peroneal nerve (SPN) is mainly affected because its anatomical course makes it particularly susceptible to damage after inversion-supination injury. Nerve traction is suggested as the main mechanism of damage during ankle sprain,[5] where excessive traction can disrupt arterial microcirculation. This can cause perineural swelling with a local inflammatory reaction, scar formation, and ultimately, permanent nerve compression.[5]

Affected patients usually suffer from hyperalgesia in the SPN innervation area at the foot dorsum with radiating pain during inversion-supination movements.[5] Diagnosis is based on clinical criteria, yet Matsumoto et al[8] have shown neurophysiological examinations as unnecessary. The area of nerve pathology can, in fact, be reliably determined by localizing the Tinel sign without ultrasonographic guidance.[7] Common peroneal nerve injuries after ankle distortion are most often treated conservatively followed by surgical nerve release when initial therapy fails.[9] Conversely, very few isolated SPN injury cases have been reported.[3,5,6] In the absence of improvement 2 to 3 months after initial rest, ice, compression and elevation (RICE) with subsequent physiotherapy, surgical release has been described as a reliable treatment.[3] Corticosteroid injections also have an ameliorative effect on posttraumatic peripheral nerve issues.[10] Yet evidence is lacking as to what effects local corticosteroid injections have on trauma-related perineural inflammation causing SPN neuropraxia after ankle sprain. We evaluated the effect of perineural corticosteroid infiltrations on patients with SPN-related complaints.

Methods

This retrospective analysis evaluated the effect of perineural corticosteroid infiltration on SPN-related complaints of pain and complications. Patients provided informed consent to use their data for research.

We screened records of consecutive patients treated at our tertiary orthopaedic clinic between January and June 2022 for SPN neuropraxia after ankle sprain.

Included patients with persistent SPN neuropraxia had symptoms lasting at least 3 months after injury. Diagnosis was made by 2 attending foot and ankle surgeons based on a positive Tinel sign over the SPN—known to reliably indicate the affected area of nerve pathology—and 2 additional criteria of hyperalgesia in the innervation area and pain at rest localized to the dorsolateral aspect of the foot and ankle.

Nineteen patients were documented with SPN neuropraxia-associated symptoms. All patients underwent conservative therapy directly after their injury, that is, RICE

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Table 1. Overview of Persistent SPN Neuropraxia Cases.

Case	Age	Sex	Symptom Duration	Number of Infiltrations	Sensitive Zone	VAS (Baseline)	VAS (8 wk)	VAS (4 mo)	VAS (1-y FU)	Complications
1	32	F	3 y	2	Crural fascia	6-7	4	1	0	–
2	56	M	12 wk	2	Crural fascia	5	5	0	0	–
3	19	F	14 wk	1	Crural fascia	5	0	0	0	Depigmentation
4	40	F	1.5 y	1	Antero-lateral ankle	6	1	0	0	–
5	39	F	2 y	1	Crural fascia	4-5	1	1	1	–
6	48	F	6 y	2	Crural fascia	5	3	1	0	–
7	16	F	12 wk	1	Crural fascia	4	0	0	0	–
Mean	35		26 wk			5.1	2.8	0.4	0.1	
SD	12.6		20.05			0.78	1.85	0.49	0.34	

Abbreviations: SPN, superficial peroneal nerve; VAS, visual analog scale.

for 3 days followed by physiotherapy with tolerated weight-bearing. Twelve patients were excluded because symptoms improved after 3 months.

Eligible patients were informed about risks and side effects before consenting to treatment comprising 1 mL Diprophos (5 mg betamethasone dipropionate / 2 mg betamethasone sodium phosphate) with 2 mL mepivacaine (20 mg/mL) administered into the subcutaneous fat tissue perifocal to the SPN area where the Tinel sign could be most severely provoked.

Each patient attended a clinical follow-up 6-8 weeks post infiltration. When patients reported only temporary symptom improvement, a second injection was administered in the same manner as described above. These patients attended a further 6- to 8-week follow-up after this second infiltration. All patients attended a final clinical follow-up at least 4 months after the last infiltration. Each patient was also contacted via telephone and asked about their condition 1 year since the last infiltration.

Prior to treatment (ie, baseline) and at all follow-ups including the telephone survey, patients rated their local pain before and after each infiltration on a 0-10 visual analog scale (VAS), where 0 indicates none and 10, maximum pain. Any complications were documented at all follow-ups. Treatment was defined as complete when each patient achieved unrestricted mobility with low to no pain.

Because of the observational nature of this work, standard descriptive statistics were presented.

Results

Seven patients showed clinical signs of persistent SPN neuropraxia at least 12 weeks postinjury (Table 1).

For 6 patients, the most sensitive zone was located at the SPN's presumed passage through the crural fascia (Figure 1). For the last patient, Tinel sign was at the anterolateral area of the ankle joint approximately 6 cm distal to the presumed passage.

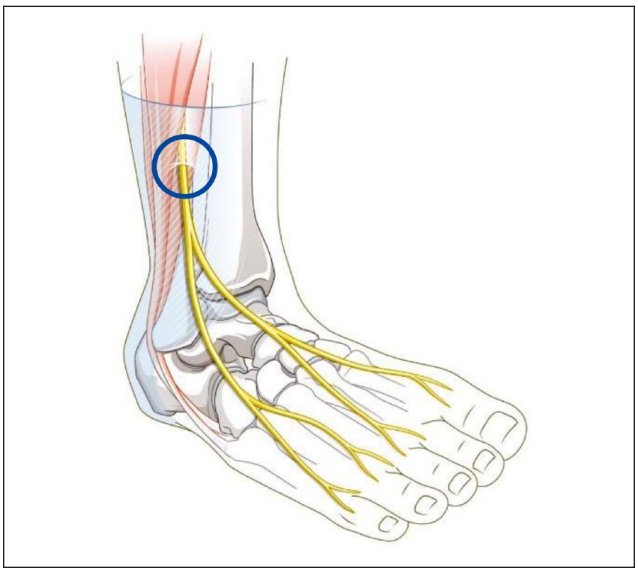

Figure 1. Diagrammatic representation of the superficial peroneal nerve (SPN) penetrating the crural fascia (blue circle).

Baseline VAS scores ranged from 4 to 7 with a mean of 5.1. After the primary infiltration, 4 patients experienced low to pain-free symptoms after 8 weeks. Three patients received a second infiltration. At a final follow-up 1 year after the last infiltration, all patients were completely or almost free of pain (ie, VAS score 0-1). Only 1 patient reported depigmentation around the infiltration site.

Discussion

We observed a relevant ameliorative effect on prolonged neuropraxia-associated symptoms after perineural corticosteroid infiltration of the SPN. Although surgery can be advantageous when conservative measures are exhausted,[3] perioperative risks exist.[2]

From our experience, SPN neuropraxia occurs either acutely following ankle distortions or frequently after a

previous injury with long-lasting, therapy-resistant diffuse residual complaints. Main complaints are hyper-/hypoesthesia, pain at rest, and electrifying pain during plantar flexion/inversion. Within a few weeks post onset, complaints often spontaneously regress under conservative treatment.

Johnston et al[5] highlighted a certain predisposition of the SPN to traction neuropraxia within the anatomical stricture at the passage through the crural fascia. Six of our patients exhibited particularly pronounced, clinically triggerable symptoms in this area, and all infiltrations applied here resulted in marked symptom relief.

A previous investigation showed only temporary improvement after perineural steroid infiltrations on peripheral nerves.[10] This is in contrast to our study that showed no recurrence of symptoms 1 year after the last infiltration.

Brinks et al[1] described the safe application of extra-articular corticosteroid injections, whereby relevant complications were depigmentation or perifocal skin atrophy. Our case of depigmentation led neither to complaints associated with the adverse event nor additional treatment.

For trauma-related SPN neuropraxia following ankle distortion, infiltrations resulted in improvements in pain. Although observed for a small patient number and VAS for routine pain documentation is a simple, nonvalidated tool without activity-dependent features, our evidence forms the basis for a larger systematic study employing the collection of an activity-related score.

Readers should note that interpretation of this study is limited by the relatively small number of patients, lack of controls, and lack of ultrasonographic confirmation of the presumed location of the passage of the nerve though the crural fascia.

Conclusion

In our series, all patients with recalcitrant symptoms from SPN neuropraxia after ankle sprain benefited from perineural infiltration around the SPN, both in relatively acute and long-lasting symptom cases. Corticosteroid infiltrations may be considered for trauma-related SPN neuropraxia after ankle sprain and represent a simple, surgery-sparing treatment without perioperative complication risks.

Ethical Approval

All patients gave consent for their data to be collected for research purposes. Ethical approval was not sought for the present study because this study was not based on a study protocol and did not define exact procedures used to identify and describe the cases. Instead, it describes 7 cases identified during routine clinical practice. Thus, it was not a "method-driven search for generalized knowledge," which is a precondition for studies. Therefore, it is not classified as research by law and does not require ethical approval (Swiss Federal Human Research Act, Art. 3).

Declaration of Conflicting Interests

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