

Clinical Features and Surgical Treatment of Superficial Peroneal Nerve Entrapment Neuropathy

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Abstract

Superficial peroneal nerve (S-PN) entrapment neuropathy (S-PNEN) is comparatively rare and may be an elusive clinical entity. There is yet no established surgical procedure to treat idiopathic S-PNEN. We report our surgical treatment and clinical outcomes. We surgically treated 5 patients (6 sites) with S-PNEN. The 2 men and 3 women ranged in age from 67 to 91 years; one patient presented with bilateral leg involvement. Mean post-operative follow-up was 25.3 months. We recorded their symptoms before- and at the latest follow-up visit after surgery using a Numerical Rating Scale and the Japan Orthopedic Association score to evaluate the affected area. We microsurgically decompressed the affected S-PN under local anesthesia without a proximal tourniquet. We made a linear skin incision along the S-PN and performed wide S-PN decompression from its insertion point at the peroneal tunnel to the peroneus longus muscle (PLM) to the point where the S-PN penetrated the deep fascia. One patient who had undergone decompression in the area of a Tinel-like sign at the initial surgery suffered symptom recurrence and required re-operation 4 months later. We performed additional extensive decompression to address several sites with a Tinel-like sign. All 5 operated patients reported symptom improvement. In patients with idiopathic S-PNEN, neurolysis under local anesthesia may be curative. Decompression involving only the Tinel area may not be sufficient and it may be necessary to include the area from the PLM to the peroneal nerve exit point along the S-PN.

Key words: superficial peroneal nerve, decompression, nerve entrapment

Introduction

Common peroneal nerve (C-PN) entrapment (C-PNE) is the most common neuropathy of the leg. When the PN is entrapped around the peroneal head, the symptoms are motor weakness, numbness, and pain. While C-PNE is a known pathology, symptomatic compression of the superficial PN (S-PN) may be a more elusive clinical entity whose treatment has not been established. The S-PN can be entrapped around the peroneal tunnel after it branches from the C-PN. This results in pain and paresthesia in the area affected by the S-PN at the lateral aspect of the lower shin and the dorsum of the foot.

Superficial peroneal nerve entrapment (S-PNE) was first described by Henry¹⁾ who observed that

the pain could be reproduced by compressing the nerve at the point where it emerged from the deep fascia. Styf²⁾ subsequently reported diagnosing S-PNE in 17 of 480 (3.5%) patients with chronic leg pain; S-PN decompression surgery was successful in 80% of patients treated by Styf and Morberg.³⁾ Others^{4–9)} also obtained good decompression results. Surgical procedures to address S-PNE have been reported; in patients with idiopathic S-PNE, the type of surgery depended on the site requiring decompression.^{3,4,6)}

Superficial peroneal nerve entrapment and C-PNE may be concurrent. Franco et al.⁸⁾ reported that 78% of patients who underwent S-PNE surgery had previously been treated by C-PN decompression surgery, suggesting the undetected presence of S-PNE in some patients with recurrent or persistent symptoms after C-PNE surgery. We report the surgical treatment and clinical outcomes in 5 patients with S-PNE; all had previously undergone C-PNE surgery on the ipsilateral side.

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Patients and Methods

Between September 2014 and June 2016, we diagnosed and surgically treated 5 S-PNE patients at our institutions (Table 1). Informed consent was obtained from all patients included in this study.

The patients were 2 men and 3 women ranging in age from 67 to 91 years (average 74 years). In one patient (case 2) both legs were involved. All 5 patients had previously undergone surgery for ipsilateral C-PNE. Pre-treatment, all reported pain and/or paresthesia of the lateral aspect of the shin and the dorsum of the foot without severe paresis. All exhibited a Tinel-like sign at the PN at the neck of the fibula. All but one patient (case 5) suffered intermittent claudication. After C-PNE surgery, one patient (case 2) continued to experience symptoms in a narrow area of the S-PN. In the other 4 patients the symptoms disappeared transiently after C-PNE surgery, however, symptoms in the S-PN area reappeared 2–12 months postoperatively. Although the Tinel-like sign from the C-PN had disappeared and the symptom involved an area narrower than the symptoms of C-PNE and was confined to the periphery, it affected one-third of the shin to the peripheral side.

Our diagnosis of S-PNE was primarily based on clinical symptoms. All patients suffered pain and/or paresthesia of the lateral aspect of the shin and the dorsum of the foot, and in all we observed a Tinel-like sign. None of the patients obtained pain relief by conservative treatment.

After C-PN surgery, the symptom duration from onset to treatment averaged 5.5 months (range 2–15 months); the mean post-operative follow-up period was 25.3 months (range 18–39 months). One patient had Parkinson's disease, one had suffered ipsilateral Achilles tendon rupture approximately 30 years earlier, and one had been operated for a herniated lumbar disc. In 3 patients magnetic resonance imaging revealed lumbar spinal canal stenosis.

Surgical method

With the patient in the lateral position with the lesion site on top, we microsurgically decompressed the affected S-PN under local anesthesia. No proximal tourniquet was applied. Anatomically, the S-PN begins at the bifurcation of the C-PN, runs deep to the peroneus longus muscle (PLM), and then passes anteroinferiorly between the peroneus longus/brevis muscle and the extensor digitorum longus muscle. It penetrates the deep fascia of the lower leg and divides into medial and lateral branches. In most patients, the area requiring decompression was located

posterior to the site where the nerve penetrated the PLM before it penetrated the deep fascia.

Our long linear skin incision along the S-PN included the site(s) with a Tinel-like sign (Fig. 1). We exposed the superficial fascia, identified the S-PN at its distal portion (Fig. 2A), cut the fascia, and performed S-PN decompression in a distal-to-proximal direction. The S-PN tended to bulge out of the deep fascia opening (Fig. 2B). We then decompressed the nerve at a site distal to where it penetrated the deep fascia. In the proximal area, it was decompressed to the site of insertion into the PLM (Figs. 2C and 2D). We confirmed disappearance of the Tinel-like sign. To ascertain the disappearance of dynamic compression we also confirmed sufficient decompression and symptom improvement, evidenced by painless intraoperative ankle movement.

Outcome evaluation

We recorded symptom severity before and at the latest follow-up visit after surgery based on a Numerical Rating Scale (NRS) of the affected area and the Japan Orthopedic Association (JOA) score.¹⁰⁾ Data were expressed as the mean \pm standard deviation.

Results

The pre-treatment NRS for the affected area was 7.8 ± 1.3 , the JOA score was 10.7 ± 5.0 ; 4 patients experienced painful intermittent claudication due to S-PNE neuropathy (S-PNEN).

At surgery, we made an 8–21-cm skin incision that included the area exhibiting the Tinel-like sign(s); the S-PN was decompressed as described above. In 2 limbs, we noted obvious entrapment at the site where the S-PN penetrated the deep fascia; in 4 of the 6 involved limbs, we observed no- or only mild local nerve strangulation. In the patient with bilateral limb involvement who required re-operation (case 2), the left leg had been treated by limited decompression around the entrapment point; in the other 4 patients (5 limbs), we completely decompressed the peroneal tunnel, including the site with the Tinel-like sign.

All patients reported symptom abatement just after the operation; their Tinel-like sign(s) had disappeared. There were no intraoperative complications. The clinical course of 4 patients who underwent a single operation was good and they experienced no recurrence of symptoms.

In the left leg of patient 2, we noted a Tinel-like sign at a point where the nerve penetrated the deep fascia; S-PN decompression led to transient symptom improvement. However, 4 months after the

Table 1 Case summary of 5 patients with superficial peroneal nerve syndrome after surgery for common peroneal nerve entrapment

Case	Age	Gender	Side	Underlying disease		Intermittent claudication (m)	Symptom duration (months)	Onset after CPN (months)	Operative findings	Linear skin incision (cm)	Tinel sites	NRS pre/post	JOA pre/post	Last follow-up (months)
				History of trauma and surgery	Lumbar disease									
1	75	F	Lt	Achilles tendon rupture (Lt)	L3-5 LCS, Vertebral compression fracture	2	2	2	No entrapment	8	One	8/1	5/23	39
2	68	M	Lt	Post operation for LDH	Post operation L4/5LDH	100	3	0	Strong entrapment at piercing site *No entrapment	7 *21	One *Multiple	6/2	12/18	27
3	67	F	Lt	Same as above	Same as above	100	3	0	Mild entrapment on proximal side (fibrous tissue)	11	Multiple	6/2	12/18	26
4	91	F	Rt	Tarsal tunnel syndrome, bil	L4/5 mild LCS L2/3,4/5,5/S1 LCS	100	15	12	No entrapment Mild entrapment at piercing site	8 13	Multiple Multiple	10/5 8/4	6/15 9/20	23 19
5	69	M	Rt	Tarsal tunnel syndrome, bil	None	None	7	6	Entrapment at piercing site	18	Multiple	9/0	19/20	18

bil: bilateral, CPN: common peroneal nerve neurolysis, F: female, JOA: Japanese Orthopedic Association score, LDH: lumbar disc herniation, Lt: Left, LCS: lumbar canal stenosis, M: male, m: meter, NRS: numeric rating scale, PD: Parkinson disease, Rt: right, *: reoperation.

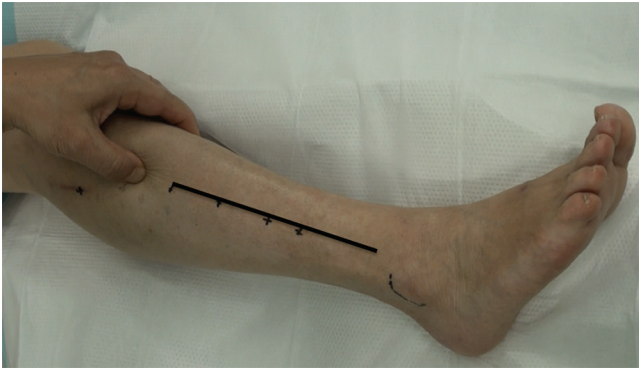


Fig. 1 Photograph of the skin incision on the right leg.

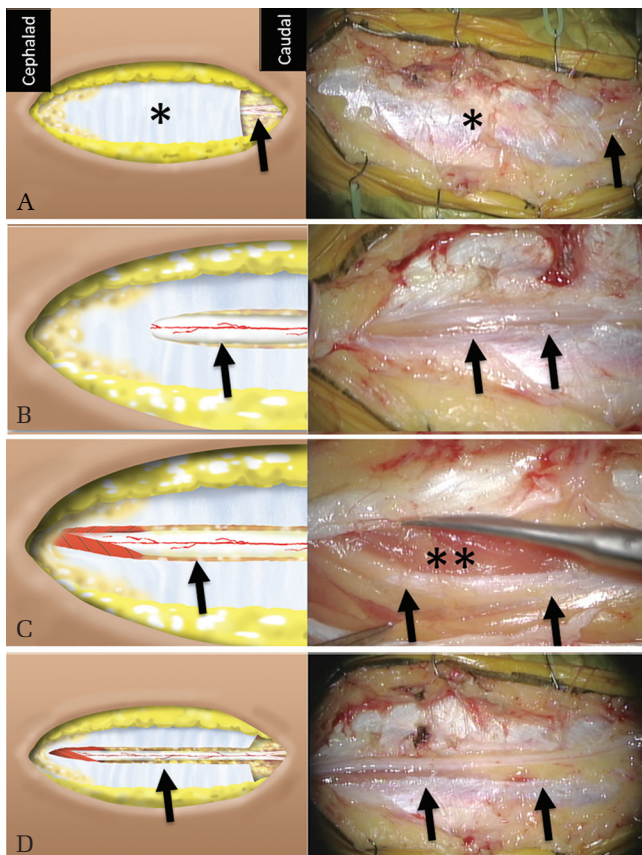


Fig. 2 Surgical methods to address right-sided superficial peroneal nerve (S-PN) entrapment neuropathy. (A) Exposure of the superficial fascia (*) and identification of the S-PN (arrow) at the distal portion. (B) S-PN (arrow) bulging upon surgical decompression. (C) In the proximal area, the S-PN (arrow) is decompressed up to the point of insertion into the peroneus longus muscle (**). (D) Intraoperative view after S-PN (arrow) decompression.

first operation he suffered symptom recurrence. We detected several sites with Tinel-like signs proximal to the site addressed in the first operation. After additional complete nerve decompression in the

peroneal tunnel this patient reported symptom improvement and no further recurrence.

Post-operatively, the NRS for the affected area and the JOA score improved in all patients (average NRS from 7.8 ± 1.3 to 2.3 ± 1.9 , JOA score from 10.7 ± 5.0 to 19 ± 2.6).

Discussion

Superficial peroneal nerve entrapment is relatively rare; it is diagnosed based on the symptomatology because it is difficult to diagnose with nerve conduction studies.^{2,3,6-8} The symptoms are pain and paresthesia of the affected area, e.g. the lateral aspect of the lower shin and the dorsum of the foot; these symptoms tend to be exacerbated by walking and exercise.^{2,3,5,6,7,9} The differentiation of S-PNE from other diseases such as lumbar disease is important. The detection of Tinel-like signs is diagnostically useful,²⁻⁷ as is transient improvement elicited by lidocaine-blocking of the S-PN.^{4,6} Our patients were diagnosed based on their symptoms and all reported improvement after S-PNE decompression surgery. As 4 of our 5 patients also reported intermittent claudication, dynamic compression may be involved in S-PNE.

According to Franco et al.,⁸ 78% of patients who underwent S-PNE surgery had previously been treated by C-PN decompression. They hypothesized that an area proximal to the compression site may render areas further downstream more susceptible to compression. This is called the “double-crush” phenomenon.¹¹ All of our patients had first undergone C-PNE decompression surgery and 4 had co-existing lumbar degenerative- or Parkinson’s disease, or had suffered ipsilateral Achilles tendon rupture. Although the etiology of their S-PNEN is unknown, not only double-crush but also their abnormal posture due to their clinical condition may have overloaded their lower limbs in the presence of chronic myotonia, resulting in increased pressure on muscle division.

Other factors thought to be involved in S-PNE are entrapment due to muscle herniation,^{2,3,5,7,9} trauma,² compression by mass lesions, e.g. varicose veins,² lipoma,⁶ and idiopathic entrapment.^{2-4,6} As none of our patients manifested PNE at a fascial defect, had experienced trauma or surgical complications,² or presented with mass lesions, we made a diagnosis of idiopathic S-PNEN. In such cases, decompression in a sufficiently large area or at the site of nerve penetration of the deep fascia^{4,6} or complete opening of the peroneal tunnel³ has been suggested. One of our patients (case 2) suffered symptom recurrence within 4 months after S-PNEN surgery that had involved restricted decompression around the

Tinel-like sign. The recurrent symptom was addressed by an additional decompression procedure comprised of the complete opening of the peroneal tunnel. We think that the decompression range at the first operation had been too small and that pressure around the S-PN increased upon postoperative lower-limb loading. Styf and Morberg³⁾ stressed that the nerve must be decompressed by complete opening of the peroneal tunnel to minimize the risk of residual irritation from the edge of the fascia and the muscle tissue, and to avoid muscle herniation. We agree that in patients with idiopathic S-PNEN, extensive decompression from the PLM to the S-PN penetration site is necessary even when the Tinel sign suggests a small area of involvement and symptoms disappear intraoperatively.

Styf and Morberg³⁾ reported that most patients presented with multiple points of compression along the PN. We also noted Tinel-like signs at several sites. Macroscopically we observed severe strangulation of the S-PN in only two legs (patients 2 and 5). Deep fascia cutting and opening revealed strong bulging of the S-PN involving adjacent fat and muscle in all patients but no macroscopic nerve strangulation. These observations suggest that S-PNEN may be elicited not only by nerve entrapment at certain sites but also by entrapment along the peroneal tunnel.

Others^{2,3)} performed S-PNEN neurolysis under general anesthesia and with placement of a tourniquet, all 15 of their patients presented with a fascial defect and only 2 with idiopathic S-PNEN. In all 5 of our patients S-PNEN was idiopathic. We used local anesthesia because it permitted confirmation of the disappearance of dynamic compression and of symptoms by the intentional intraoperative ankle movement. Local anesthesia contributes to reducing the burden on patients and it permits intraoperative confirmation of the disappearance of the Tinel-like sign reflecting abatement of nerve compression. Based on our experience we recommend S-PN neurolysis under local anesthesia in patients with idiopathic S-PNEN.

Our study has some limitations. The study population was small and the follow-up periods were relatively short. We will continue to monitor the patients reported here to evaluate their long-term treatment outcomes.

Conclusion

To treat S-PNE successfully, we performed neurolysis under local anesthesia. In patients with idiopathic S-PNEN, the nerve may be entrapped not only where

it penetrates the fascia but also along the peroneal tunnel. In such patients, extensive decompression from the PLM to the S-PN penetration site is required.

Conflicts of Interest Disclosure

None.

Ethical Approval

All procedures were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments and comparable ethical standards.

Informed Consent

Informed consent was obtained from all patients included in this study.

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