CASE REPORT

Entrapment of the lateral cutaneous nerve of the calf

Nofal M Khalil,¹ Alessia Nicotra,¹ Charles Kaplan,² Kevin S O'Neill³

¹Department of Clinical Neurophysiology, Imperial College Healthcare NHS Trust, London, UK

²Department of Neurology, Charing Cross Hospital, London, UK

³Department of Neurosurgery, Imperial College Healthcare NHS Trust, London, UK

Correspondence to

Dr Nofal M Khalil,
n.khalil@imperial.ac.uk

SUMMARY

Isolated lesion of lateral cutaneous nerve of the calf (LCNC), particularly due to entrapment, is rarely reported in the literature. Patients usually present with sensory symptoms in the lateral aspect of the calf. Treatment is usually by local applications or local steroid/anaesthetic injection. We report the first case of LCNC entrapment in a 35-year-old man which is documented by nerve conduction studies. The patient had a temporary improvement following a local anaesthetic/steroid injection. Owing to the recurrence of symptoms, the patient opted for surgery. About 1 year after surgery, the symptoms disappeared completely.

BACKGROUND

Sensory symptoms in the lateral aspect of the calf are common. The most common cause is L5 radiculopathy. However, if the symptoms are purely sensory, one should think of pure sensory lesion involving the branches which arise from the common fibular (peroneal) nerve at the popliteal fossa. The largest series of 48 patients were reported by Haimovici¹ in 1972. Following that, there are only five single case reports^{2–6} in the literature. We believe that the condition is much more common than that reported in the literature. Thus, mono-neuropathy of LCNC should be considered in patients with sensory symptoms in the lateral aspect of the calf, when lumbar spine MRI and neurophysiology have excluded an L5 radiculopathy, and if conservative measures fail surgery is a viable option.

CASE PRESENTATION

A 35-year-old man presented with a 2-year history of sensory symptoms in the lateral aspect of the left calf. He described his symptoms as severe burning pain with numbness, tingling, itching and hypersensitivity to the touch. The area sometimes felt freezing cold and pale. The symptoms were present during the day and night, but worse at night when he tried to relax. Otherwise, he is a healthy young man with no history of trauma, diabetes or neurological disease.

His symptoms started when he was working as a driver of forklift truck in a warehouse of a water bottling company. His work involved loading and unloading water bottle containers working at least 8 h a day for about 2 years. His left knee was kept in a semiflexed position throughout the day.

Neurological examination including straight leg raising test, muscle tone, power and reflexes were normal in the arms and legs. Sensory testing revealed an area of diminished sensation to pin

prick and touch over the posterolateral aspect of the left calf extending from the knee to about the middle of the leg, measuring 18×11 cm. There was hyperalgesia, but no hyperpathia. There was hyperaesthesia and dynamic allodynia, but no static allodynia. There were no colour and temperature changes. The area of altered touch sensation was greater than the area of hyperalgesia, which is highly suggestive of a peripheral nerve lesion.

INVESTIGATIONS

Routine blood tests including full blood count, urea, electrolytes, C reactive protein and erythrocyte sedimentation rate were normal and so was the MRI of the lumbosacral spine. Detailed nerve conduction studies and needle electrode examination were performed. Needle electromyography (EMG) showed no evidence of denervation in L4, L5 and S1 myotomes. Standard nerve conduction studies including conduction in the proximal segment (as evidenced by F-responses and soleus H-reflex) were normal. In particular, there was no conduction delay/block in the common fibular nerve across fibular neck and the sural sensory nerve action potential (SNAP) was normal and symmetrical in both legs. The main finding of note was absent SNAP from the left LCNC (figure 1A), but a normal SNAP with an amplitude of 3.1 µV; a conduction velocity of 60 m/s was obtained from the right asymptomatic side (figure 1B), suggesting a local lesion in the LCNC on the left side. Ultrasound and MRI of the left calf area, including his popliteal fossa, and X-ray of the left knee were performed. Ultrasound and MRI showed no structural pathology or any significant abnormality of the neurovascular bundle and the X-ray of the left knee showed no local bony pathology that may be impinging on the nerve.

DIFFERENTIAL DIAGNOSIS

The main differential is from L5 radiculopathy which is the most common cause of sensory symptoms on the lateral aspect of the calf. This can be achieved by clinical examination; weakness in the distal (tibialis anterior, tibialis posterior, peroneus longus and extensor hallucis longus) and proximal (tensor fascia latae and gluteus medius) L5-innervated muscles, lumbar MRI and EMG evidence of denervation in at least two L5 innervated muscles supplied by two different peripheral nerves.

TREATMENT

The patient was offered local anaesthetic/steroid injection. The site of injection was determined by nerve stimulation and two-stage injection. The nerve was

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Figure 1 Two superimposed trials of 20 averaged traces from the lateral cutaneous nerve of the calf. No SNAP was recorded from the left LCNC (A), but SNAP (amp 3.1 µV, CV 60 m/s) was obtained from the right LCNC (B).

stimulated at the popliteal fossa with supramaximal stimuli and the point of maximum spread of impulses was marked. Then, 0.5 mL of 2% Lignocaine is injected and when the area of the nerve supply became numb, 2% Lignocaine (1 mL) and steroid (Methylprednisolone 40 mg/mL) were infiltrated. The LCNC was infiltrated twice, about 3 months in between the two. After each infiltration, the symptoms disappeared (almost completely) for 2 weeks, but then returned. Owing to the recurrence of symptoms, the patient opted for surgery.

At surgery, an incision was made in the popliteal fossa through the skin and subcutaneous tissue then the deep fascia. The common fibular nerve was exposed and a fibrous band was found around the LCNC (figure 2A), which appears to be part of the deep fascia extending into the origin of the peroneus longus and gastrocnemius. This band was sectioned at two points close to LCNC and common fibular nerves (figure 2B), then the LCNC was mobilised (figure 2C).

OUTCOME AND FOLLOW-UP

The postoperative period was unremarkable. The patient was discharged home the following day and was followed up

regularly after surgery. He reported a gradual disappearance of pain, itching, hypersensitivity and burning sensation. One year after surgery, the patient reported the total disappearance of symptoms apart from a small area of numbness at the outer aspect of the left calf, less than 10% of the original area of impaired sensation, which he only became aware of when touching it.

DISCUSSION

Mononeuropathy of the LCNC is rarely reported in the literature. This nerve is a cutaneous branch, which leaves the common fibular nerve at the popliteal fossa proximal to the fibular head. According to Gray's anatomy,⁷ there are two cutaneous nerves arising from the common fibular nerve at the popliteal fossa above the fibular head namely the sural communicating nerve and the LCNC. They may arise separately but more commonly they arise as one trunk known as the lateral sural cutaneous nerve as in figure 3A, which gives the LCNC that courses laterally and innervates the lateral aspect of the calf (figure 3B), and then continues medially as the sural communicating nerve which unites with the medial sural nerve from the tibial nerve to form the

Figure 2 Dissection in the popliteal fossa showing fibrous band around the lateral cutaneous nerve of the calf extending underneath the common fibular nerve (A). The fibrous band was lifted up and sectioned at two points (B). The lateral cutaneous nerve and the common fibular nerves after sectioning of the fibrous band (C).

sural nerve proper. This explains why there is no reduction in amplitude of the sural nerve action potential in common fibular nerve lesion at the fibular neck.

It is difficult to diagnose LCNC entrapment clinically as the area of altered sensation usually raises the possibility of L5 radiculopathy which is much more common than local nerve entrapment. However, the lack of weakness, the normal lumbar MRI and the detailed sensory examination of the area in particular the area of altered light touch sensation is greater than the area of altered pain sensation are highly suggestive of a peripheral/cutaneous nerve lesion.

This nerve is not routinely tested and the technique is somewhat difficult to perform.

Campagnolo *et al* (2000)⁸ are the first to publish the technique of antidromic and orthodromic recording from this nerve. It is easier to obtain an SNAP with antidromic (which was used in this case) than orthodromic recording. If an SNAP was obtained from one limb, there is more than 80% chance of obtaining an SNAP from the other side unless it is lost due to pathological reasons. The nerve is stimulated antidromically 2 cm medial to the fibular head and 4 cm proximally. The recording electrode is placed 12 cm distal to the stimulation point along a line joining the stimulation point and the calcaneus. Using this technique, no SNAP was obtained from the left LCNC (figure 1A), while the sural potential was normal. It appears that the fibrous band crosses the LCNC (figure 2A,B) as it courses laterally distal to the lateral sural communicating nerve which would explain the selective involvement of this nerve and preservation of the sural potential, similar to a fibular nerve lesion at the fibular neck. The combination of the clinical scenario, the absent SNAP from the left LCNC, the intraoperative findings and the patient's response to the treatments prove that a lesion of this nerve explained the patient's symptoms and findings.

It is Haimovici¹ who first described the entrapment of the cutaneous branches which arise from the common popliteal nerve at the popliteal fossa. He described a large series of 48 patients (60 extremities) with sensory symptoms on the lateral side of the popliteal fossa, radiating distally along the outer surface of the leg and occasionally proximally towards the posterior surface of the thigh. All cases were diagnosed clinically and most of them were treated by local anaesthetic injection. Fifty-two extremities were treated; complete relief was obtained in 43 (87.2%), partial relief in 6 (11.5%) and no response in 3 (5.8%).

Apart from Haimovici paper, there are only five single case reports²⁻⁶ in the literature of isolated mononeuropathy involving this nerve. Two^{2,3} are inpatients with diabetes as diabetes makes them more vulnerable for compression-ischemic changes with various postures of the lower limbs. The first case² was diagnosed clinically in a 61-year-old housewife and disappeared a year later by strict control of diabetes. The second case was³ of a 54-year-old man with underlying peripheral neuropathy, but his symptoms were mainly in the distribution of LCNC. The patient was treated conservatively: instructed not to fold or cross his legs and in a 10-day period regained 90% of sensation.

The third and fourth cases^{4,5} are due to a structural lesion, but only the third case⁵ (due to a congenital anomaly of the biceps femoris tendon) was treated/cured by surgery. The tendon was transected and repaired with Z-plasty and on follow-up all symptoms disappeared completely.

The fifth and most recent case⁶ appears to be entrapment neuropathy, documented by absent SNAP of the LCNC on the affected side. The patient was treated conservatively with topical non-steroidal gel with no response to treatment and there was no follow-up.

Our case is the first case of LCNC entrapment in a patient without diabetes and with no structural lesion visible on imaging which was successfully treated by surgery.

We postulate that the nerve is vulnerable for compression as it pierces the deep fascia near the fibular head and enters the subcutaneous space coupled with congenitally thickened deep fascial bands triggered by the prolonged flexion of the knee.

Unexpectedly, in our patient, the SNAP could not be obtained on two occasions during follow-up even 1 year after the operation with either antidromic or orthodromic techniques. This is probably due to severe axonal degeneration before surgery.

The symptoms improved postoperatively due to a combination of axonal regeneration and reinnervation from the nearby cutaneous nerves.⁹ However, the SNAP remained unobtainable possibly because there were not enough viable axons to generate a detectable waveform. In all cases of entrapment neuropathy, there are usually residuals even after successful decompression and full clinical recovery, that is, the amplitude and conduction velocity may never return to normal following, for example, successful decompression for carpal tunnel syndrome. And after neurectomy, one of the surgical treatment options of meralgia paraesthetica, the area of numbness reduces in size over time.¹⁰

Figure 3 The origin of the lateral cutaneous nerve of the calf in the popliteal fossa from the common fibular nerve (A) and the area of its cutaneous innervation (B).

To our knowledge, this is the first case of LCNC entrapment, which is successfully treated by surgery.

Learning points

- ▶ Mononeuropathy of the lateral cutaneous nerve of the calf may be more common than it has been reported in the literature.
- ▶ The possibility of a local lesion involving this nerve should be considered when a patient presents with pure sensory symptoms in the lateral aspect of the calf and L5 radiculopathy is excluded by clinical examination, electrodiagnostic testing and neuroimaging studies.
- ▶ The condition can be confirmed by nerve conduction studies comparing affected versus normal limbs.
- ▶ Surgery is a viable option when conservative measures including local anaesthetic/steroid injection fail and the symptoms remain intolerable with excellent results.

Contributors NMK did the NCS and drafted the manuscript and followed up the patient; AN saw the patient with NMK, reviewed the literature and the manuscript;

Dr CK referred the patient and started the investigations. KSO did the operation and followed up the patient.

Competing interests None.

Patient consent Obtained.

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REFERENCES

- 1 Haimovici H. Peroneal sensory neuropathy entrapment syndrome. *Arch Surg* 1972;105:586–90.
- 2 Finelli PF, DiBenedetto M. Bilateral involvement of the lateral cutaneous nerve of the calf in a diabetic. *Ann Neurol* 1978;45:480–1.
- 3 Gessini L, Jandolo B, Pascucci P, *et al*. Diabetic neuropathy of the lateral cutaneous nerve of the calf. A case report. *Ital J Neurol Sci* 1985;6:107–8.
- 4 Hackam DG, Zwimpfer TJ. Congenital entrapment of the lateral cutaneous nerve of the calf presenting as a personal sensory neuropathy. *Can J Neurol Sci* 1998;25:68–70.
- 5 Ginanneschi F, Rossi A. Lateral cutaneous nerve of calf neuropathy due to peri-popliteal cystic bursitis. *Muscle Nerve* 2006;34:503–4.
- 6 Schneider DS, Reddy S. The lateral cutaneous nerve of the calf revisited. *PMR* 2010;2:579–80.
- 7 Williams P, Warwick R. eds. *Gray's anatomy*. 36th edn. Edinburgh, London, Melbourne and New York: Churchill Livingstone, 1980:1115.
- 8 Campagnolo DI, Romello MA, Park YI, *et al*. Technique for studying conduction in the lateral cutaneous nerve of calf. *Muscle Nerve* 2000;23:1277–9.
- 9 Weddell G, Guttmann L, Guttmann E. The local extension of nerve fibres into denervated areas of skin. *J Neurol Psychiatry* 1941;4:206–5.
- 10 Williams PH, Trzil KP. Management of meralgia paraesthetica. *J Neurosurg* 1991;74:76–80.

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