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ORIGINAL ARTICLE

Peroneal nerve entrapment at the fibular head: Outcomes of neurolysis



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KEYWORDS

Nerve entrapment
syndrome;
Common peroneal
nerve;
Compression;
Neurolysis

Summary

Background: Common peroneal nerve (CPN) entrapment at the fibular head is the most common nerve entrapment syndrome at the lower limbs. Motor deficits predominate and the risk of persistent functional impairment is the main concern. The objective was to evaluate outcomes of neurolysis and to evaluate the benefits of performing surgery early.

Materials and methods: We retrospectively reviewed the medical charts of 15 patients (mean age, 32 years) treated with neurolysis. The diagnosis was idiopathic CPN entrapment in ten patients, indirect nerve injury with CPN paralysis due to an ankle injury in three patients, and postural CPN compression in two patients. Mean time to management was 7 months (range, 2–18 months).

Results: Mean follow-up after neurolysis was 42 months (range, 25 to 62 months). The outcome was considered excellent in seven cases, good in five cases, and fair in three cases. Mean time to functional recovery was 2.5 months (range, 2 weeks to 6 months). Of the ten patients with idiopathic CPN entrapment syndrome, nine had excellent or good outcomes. The three patients with fair outcomes had ankle injuries or polyneuropathy.

Discussion: Spontaneous recovery can take time and remain incomplete. We prefer to perform surgery between the third and fourth months in patients with persistent symptoms or incomplete recovery, even in forms confined to sensory dysfunction documented by electrophysiological testing. Time to recovery is shorter after surgical decompression than with rehabilitation therapy.

Level of evidence: Level IV, retrospective study

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Introduction

The most common nerve entrapment syndrome at the lower limbs is entrapment of the common peroneal nerve (CPN) at the head of the fibula. A motor deficit is the main manifes-

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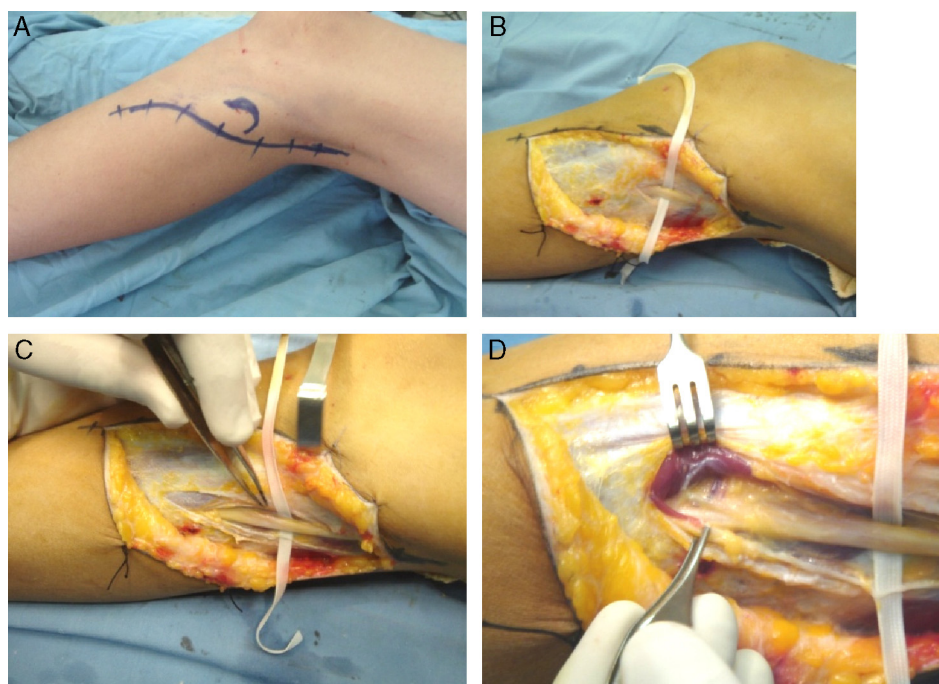


Figure 1 A. Skin incision. B. Identification of the common fibular nerve proximally. C. Incision of the fibular tunnel at the site of compression. D. Release of the nerve as distally as possible, down to the bifurcation into a superficial branch and deep branch.

tation and the risk of permanent functional impairment is the predominant concern. Below the knee, the CPN courses around the lateral aspect of the fibular neck, where it is highly vulnerable to injury. Apart from laceration or stretching of the nerve during fractures or dislocations of the proximal fibula or ankle, idiopathic entrapment syndrome is the most common cause of loss of CPN sensory and motor function.

The objective of this study was to evaluate our results in order to document the usefulness of early surgical neurolysis.

Material and methods

We retrospectively reviewed the medical charts of 15 patients (14 men and one woman) with CPN entrapment syndrome. We did not include patients followed up for less than 12 months or those having direct injuries to the CPN (laceration, contusion, or division by a bone shard after a fibular neck fracture).

Mean patient age was 32 years (range, 17–48 years). A history of low back pain and sciatica was reported by three patients. In addition, three patients had diabetes mellitus and two had chronic alcohol abuse.

Investigations identified a cause in five patients. Forced inversion of the foot with stretching of the CPN was the cause in three patients, including one with a fracture of the medial malleolus and two with lateral ankle sprains. In the remaining two patients, compression of the CPN occurred during prolonged squatting.

The motor deficit was evaluated chiefly by testing the tibialis anterior muscle and fibularis muscles. Subjective sensory symptoms, such as pain and paresthesia were

recorded. Sensory and motor function before and after surgery was evaluated according to the British Medical Research Council (MRC) classification system. Of the 15 patients, seven had isolated sensory dysfunction and eight had both sensory and motor dysfunction.

Electrophysiological testing was performed routinely. Sensory potential amplitudes were diminished in 12 cases and motor conduction velocities in seven cases. The electrophysiological findings established that the site of compression was the neck of the fibula.

Time from symptom onset to management ranged from two to 18 months. When the patients were seen within the first months, repeat electrophysiological testing was performed after 1 month to evaluate the extent of recovery. When no evidence of recovery was found, surgery was performed. The nine patients seen more than 4 months after symptom onset underwent immediate surgery based on the clinical and electrophysiological findings.

Surgery consisted in open release of the CPN. A curvilinear incision was made (Fig. 1) along the biceps femoris tendon proximally then, across the fibular neck distally. The CPN was placed in a noose proximally at the posteromedial edge of the biceps femoris muscle. Then, the CPN was released in the proximal-to-distal direction down to the fibular tunnel, which was opened as distally as possible. Exoneurolysis no more than 5 cm in length was performed in the ten patients with severe CPN compression. A spica cast extending to the foot was worn for 1 week. In patients with motor deficits, exercises to strengthen the tibialis anterior and fibularis muscles and to improve joint range of motion were started at the first visit and continued for a mean of 2 months after surgery.

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