Resolution of Idiopathic Dropfoot Following Multiple Nerve Decompression: A Case Report

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Statement of Purpose

Dysfunction of nerves and associated negative effects can have a dramatic impact on patient function and quality of life. The purpose of this report is to describe the case of a 53-year-old female patient who underwent decompression procedures for multiple nerves in the lower extremity to treat sudden onset of idiopathic dropfoot.

Introduction

Entrapment or compression of nerves as they course through the body can be attributed to a variety of events, including systemic disease, inflammatory states, adhesions, edema, or external pressure (1, 2, 3). Short-term compression with concurrent blood flow restrictions, secondary to limb positioning or external bracing, cause the quickly reversible parasthesias that are commonly described when one's limb "falls asleep." Of more clinical significance is chronic ongoing compression neuropathy, which involves longer-lasting sequelae and can generally be classified as a progression in three stages. Stage one consists of sporadic sensory changes with occasional parasthesias. Stage two develops after a longer period of compression, and manifests with more consistent symptoms than seen in stage one. During stage three, the morphology of the nerves begins to change with notable demyelination (1).

Pathology of the common peroneal nerve (CPN) is a relatively common condition due to the anatomic location of the structure (4, 5). The nerve is a branch of the sciatic nerve (L4,5, S1,2) which begins in the thigh, extends around the fibular neck and courses distally where it divides into the superficial and deep branches. It is thought that the fibrous arch formed from the aponeurosis of the peroneal muscles and soleal aponeurosis, creates a constricting region distal to the fibular neck (4, 5, 6). A number of papers have depicted dysfunction of the nerve secondary to ischemic injury, compression or idiopathic means (3, 7). Sidey (1969) described 23 cases of entrapment neuropathy of the CPN, with 8 of the 23 patients experiencing an insidious onset and gradual worsening. Treatment via decompression of the peripheral nerves has been well described by a number of authors (8, 9, 10).

Case Presentation

A 53-year-old female presented to our office with main complaint of slap-gate and tripping over her right foot as well as numbness and tingling for duration of three months. The patient denied any history of trauma to the right lower extremity or lower back.

Physical Examination:
- Weakness with dorsiflection and eversion against resistance;
- Positive Tinel's sign at the tarsal tunnel, CPN, SPN, and DPN;
- Absent protective sensation to the dorsum and plantar aspect of the foot;
- Inability to actively dorsiflex digits.

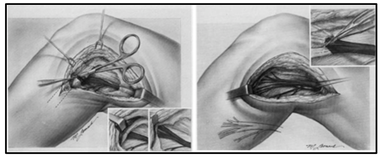
NCV's revealed absent right peroneal response and the EMG demonstrated positive waves in the anterior tibialis, extensor hallucis, extensor digitorum longus and peroneus longus, suggestive of nerve entrapment. At five months following the onset of symptoms, the risks, benefits and complications of conservative versus surgical treatment were discussed with patient in detail and it was decided that surgical intervention consisting of multiple nerve decompression in the lower extremity would be performed.

Study	Motor Latency	Motor NCV
Peroneal	No response	No response
Tibial	5.3	49.4

Study	Latency	% dropout
Peroneal F-wave	No response	-
Tibial F-wave	52.8	-
Superficial Peroneal	No response	-

E.M.G.

Muscle	Nerve	Waves
Tibialis Anteior	Peroneal	2-3+
Extensor Hallucis Longus	Peroneal	2-3+
Peroneaus Longus	Superficial Peroneal	2-3+



Operative technique for CPN release as described by Mackinnon and Dellon. Illustration from the text, *Surgery of the Peripheral Nerve*. 1988. Thieme Medical Publishers

Technique

An oblique 4 cm incision was placed over the fibular neck and it was deepened through the skin and subcutaneous tissue until the deep fascia was identified. The common peroneal nerve was identified beneath the deep fascia which was released and followed posteriorly. The deep fascia of the peroneal longus was identified and incised. The area was then flushed and closed with 4-0 monocryl.

A second incision was made at the distal third of the lower right lateral extremity over the superficial peroneal nerve and deepened until the deep fascia was incised and intermuscular bellies were identified. The superficial peroneal nerve was seen at the junction of the fascial level and neurolysis was performed distally and proximally. The area was then flushed and incision closed in the same manner as above.

A 3-4 cm incision was then made just proximal to the first interspace. It was deepened through the deep fascial layer. The extensor hallucis brevis muscle belly was identified and retracted. The deep peroneal nerve was identified and noted to be completely flattened against the osseous structures. Neurolysis of the nerve was performed proximally and distally and the incision was closed. Finally, an incision was made over the tarsal tunnel region. The flexor retinaculum was incised and the tibial nerve was identified. The tibial, medial and lateral plantar and the medial calcaneal nerves were then bluntly released from surrounding soft tissue structures. Incision was closed with 4-0 monocryl and nylon sutures.

Results

Postoperatively, the patient was allowed immediate weight-bearing in a CAM boot. Once the incisions healed, she was referred to physical therapy. The patient progressed to a full motor and sensory recovery at six months post surgical multiple nerve release.

Post-operative E.M.G.

Muscle	Nerve	Waves
Tibialis Anteior	Peroneal	0
Extensor Hallucis Longus	Peroneal	0
Peroneaus Longus	Superficial Peroneal	0

The CPN is identified and neurolysis is performed as part of the multiple nerve decompression.

Deep peroneal nerve release at the dorsum of the foot

Tarsal tunnel release on a cadaveric model

Discussion

The most common area of CPN compression is around the fibular neck within the fibro-osseous tunnel. It is also thought that the nerve becomes chronically irritated as it glides within the tunnel during flexion and extension of the knee (4, 5, 12). Patients may present with sensory, motor or sensory-motor deficits (4, 5, 12). Additionally, they may have partial or complete loss of sensation in the nerve distribution and complain of pain and symptoms of drop foot. Electrophysiological testing is an essential part of the preoperative work-up prior to surgical intervention and is also needed to confirm clinical diagnosis in order to proceed with surgery. Decrease in amplitude of sensory potential confirms sensory deficits and diagnosis of motor deficit is seen by decrease in nerve-conduction velocity (10, 11, 12).

Fabre et al performed common peroneal nerve decompression on 38 patients with drop foot deformity (5). Eighty-seven percent had good results after surgical intervention with average recovery time of 2.5 months. In another study by Ramanan and Chandran, 11 of 16 patients had resolution of their dropfoot, and patients showed improved motor function if they underwent surgery within 12 months of symptom onset (10). Vastamaki (12) and Humphreys et al (9) had similar results with improvement in motor response of dropfoot patients. Generally, most patients present months to years after the onset of symptoms. Spontaneous recovery from symptoms can occur up to 2 years after onset although conversely, those patients who do not recover are left with pain, sensory and motor dysfunction (11). Mont et al treated nine patients conservatively and only three of those showed motor and sensory improvement (13).

Multiple studies reported good outcomes after surgical decompression of CPN and suggest even better results when time between onset of symptoms and surgery is within a few months. Vastamaki recommends decompression to be considered after 2 month with no recovery or response to conservative measures, or at 4 months when patients show slow recovery (11).

References

1. Flanigan RM, DiGiovanni BF. Peripheral nerve entrapments of the lower leg, ankle and foot. Foot Ankle Clin NA. 16(2):255-274:2011.

2. Bell KM, Chalmers J. Recurrent common peroneal palsy in association with the Ehlers-Danlos syndrome: A case report. Acta Orthop Scand 62(6):612-613:1991.

3. Sprowson AP, Rankin K, Shand JE, Ferrier G. Common peroneal and posterior tibial ischemic nerve damage, a rare cause. Foot Ankle Surg. 16(e16-e17:2010.

4. Sidey JD. Weak ankles. A study of common peroneal entrapment neuropathy. BMJ 3(623-626:1969.

5. Fabre T, Piton C, Andre D, Lamour F, Durandeau A. Peroneal nerve entrapment. J Bone Joint Surg. 80(1):47-53:1998.

6. Ryan W, Mahony N, Delaney M, O'Brien M, Murray P. Relationship of the common peroneal nerve and its branches to the head and neck of fibula. Clin Anat (6):501-505:2003.

7. Reinders MF, Geertzen HB, Rietman JS. Neuropraxia of the common peroneal nerve- A rare complication resulting from wearing a KBM prosthesis: A case report. Prosther Orthot Int 29(3):197-198:1996.

8. Williams EH, Williams CG, Rosson GD, Dellon AL. Combined peroneal and proximal tibial nerve palsies. Microsurgery. 29:259-264:2009.

9. Humphreys DB, Novak CB, Mackinnon SE. Patient outcome after common peroneal nerve decompression. J Neurosurg 107(2):314-318:2007.

10. Ramanan M, Chandran KN. Common peroneal nerve decompression. ANZ J Surg 91(1):707-12, 2011.

11. Berry H, Richardson PM. Common peroneal nerve palsy: a clinical and electrophysiological review. J Neurol Neurosurg Psychiatry. 39(12): 1162-1171: 1976.

12. Vastamaki M. Decompression for peroneal nerve entrapment. Acta Orthop Scand 57(6): 551-554: 1986.

13. Mont MA, Dellon AL, Chen F, Hungerford MW, Krackow KA, Hungerford DS. The operative treatment of peroneal nerve palsy. J Bone Joint Surg 78(6): 863-869: 1996.