

Early Surgical Treatment for Severe Idiopathic Compression of the Common Peroneal Nerve: A Case Series

Mariano O. Abrego, Victoria Barbaglia, Fernando Holc, Pedro Bronenberg Victorica, Ignacio Rellán, Agustín G. Donndorff, Gerardo L. Gallucci, Pablo De Carli, Jorge G. Boretto

Centro de Ortopedia y Traumatología "Carlos E. Ottolenghi", Hospital Italiano de Buenos Aires, Autonomous City of Buenos Aires, Argentina.

ABSTRACT

Introduction: Compression of the common peroneal nerve (CPN) is a common condition in the lower limb and can be either idiopathic or secondary. While secondary compressions have been extensively studied and show good outcomes with microsurgical decompression, evidence regarding idiopathic compressions remains limited. This study aims to report cases of severe idiopathic CPN compression treated surgically, evaluate clinical outcomes, and assess the need for a standardized treatment protocol. **Materials and Methods:** A retrospective review was conducted on patients diagnosed with idiopathic CPN palsy over the past 10 years. Inclusion criteria comprised cases with a positive electromyogram, no history of trauma, negative MRI findings, and normal intraoperative findings. Patients with secondary nerve entrapment, spinal pathology, psychiatric disorders, or pregnancy were excluded. Severe cases were defined as those presenting with a dorsiflexion motor deficit of $\leq 2/5$. Preoperative, intraoperative, and postoperative variables were analyzed. **Results:** Eight patients met the inclusion criteria (2 women, 6 men). The mean time from diagnosis to surgery was 30 days, with an average follow-up of 959 days. All patients regained at least 4/5 dorsiflexion strength. Six patients achieved full recovery of both strength and sensation. No complications were reported. **Conclusions:** Early decompression of the CPN is a safe and effective procedure for severe idiopathic compression. The establishment of a standardized treatment protocol is recommended.

Keywords: Common peroneal nerve; club foot; peripheral nerve compression.

Level of Evidence: IV

Tratamiento quirúrgico precoz para la compresión severa idiopática del nervio peroneo común. Serie de casos

RESUMEN

Introducción: La compresión del nervio peroneo común es frecuente en el miembro inferior y puede ser idiopática o secundaria. Las compresiones secundarias se han estudiado ampliamente y la descompresión microquirúrgica logra buenos resultados. Sin embargo, la evidencia sobre las compresiones idiopáticas es limitada. Este estudio busca comunicar casos de compresión idiopática severa del nervio peroneo común tratada con cirugía, evaluar los resultados y analizar la necesidad de un protocolo terapéutico estandarizado. **Materiales y Métodos:** Se realizó una revisión retrospectiva de pacientes con diagnóstico de parálisis idiopática del nervio peroneo común en los últimos 10 años. Se incluyeron casos con electromiograma positivo y sin antecedentes de trauma, con resonancia magnética negativa y hallazgos intraoperatorios normales. Se excluyó a pacientes con atrapamiento secundario, problemas raquídeos, alteraciones psiquiátricas o embarazo. Se definió como caso severo cuando el déficit motor de dorsiflexión era $\leq 2/5$. Se evaluaron variables preoperatorias, intraoperatorias y posoperatorias. **Resultados:** Ocho pacientes cumplieron los criterios de inclusión (2 mujeres y 6 hombres). El tiempo medio desde el diagnóstico hasta la cirugía fue de 30 días, el seguimiento promedio fue de 959 días. Todos recuperaron, al menos, 4/5 de fuerza en dorsiflexión. Seis pacientes recuperaron la fuerza y la sensibilidad completamente. No se observaron complicaciones. **Conclusiones:** La descompresión precoz del nervio peroneo común es un procedimiento seguro para la compresión idiopática severa. Se sugiere la creación de un protocolo estandarizado para su tratamiento.

Palabras clave: Nervio peroneo común; pie equino; compresión neurológica periférica.

Nivel de Evidencia: IV

Received on December 15th, 2024. Accepted after evaluation on January 28th, 2025 • Dr. MARIANO O. ABREGO • mariano.abrego@hiba.org.ar  <https://orcid.org/0000-0001-9783-7373>

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INTRODUCTION

Compression of the common peroneal nerve (CPN) is the most frequent focal neuropathy of the lower limb.^{1,2} It is the third most common compressive neuropathy overall, surpassed only by compression of the median and ulnar nerves.³ It is also the leading cause of numbness associated with pain and muscle weakness. CPN compression may present with sensory symptoms or motor deficits, including impairment of dorsiflexion and eversion of the foot. This condition is broadly classified into two groups: idiopathic compressions and secondary lesions.⁴ Secondary lesions of the CPN encompass a wide variety of causes, most of which have been reported as isolated cases or small series.^{5,6} Idiopathic compressions may affect individuals across all age groups.⁷

The clinical outcomes of microsurgical decompression of the CPN in cases of secondary compression (post-traumatic, iatrogenic, tumor-related) have been studied, with mixed results in most series.^{1,8,9} However, the literature is limited to heterogeneous case series from various specialties. There are reports from orthopedic surgeons, plastic surgeons, neurosurgeons, and sports medicine specialists.^{4,10} Treatment guidelines are inconsistent and poorly standardized.¹¹ Moreover, there is no consensus on how to evaluate the outcomes of either conservative or surgical treatment.^{12,13}

Available data on idiopathic CPN compression are scarce. It is a poorly defined entity in terms of severity and timing of treatment. Therefore, the aim of this article is to present a series of patients with severe idiopathic CPN compression treated surgically, evaluate the outcomes, and propose a standardized therapeutic protocol for the orthopedic surgeon.

MATERIALS AND METHODS

A retrospective review was conducted over a 10-year period on patients diagnosed with idiopathic CPN palsy at our institution. Idiopathic was defined as the absence of trauma or external compression, with negative MRI findings, no intraoperative pathology, and a positive electromyogram. Significant weight loss was also assessed as a potential contributing factor.

We excluded patients with secondary CPN entrapment—i.e., those with imaging or preoperative findings consistent with nerve compression—as well as those with CPN neuropraxia following knee surgery or symptoms of CPN compression secondary to spinal disease. Patients with a history of psychiatric disorders or who were pregnant at the time of symptom onset were also excluded.

Severe was defined as a case presenting with an initial dorsiflexion motor deficit $\leq 2/5$ on the Medical Research Council (MRC) Scale for muscle strength.

Preoperative Protocol

At the first medical consultation, patients were evaluated by a fellow trained in peripheral nerve disease and subsequently re-evaluated by the surgeon responsible for the procedure. The standardized anamnesis for patients with clubfoot included questions about abrupt changes in body mass index (notably marked weight loss), history of bariatric surgery, history of direct trauma to the knee or upper third of the affected leg (including the use of immobilizers at that level), history of knee surgery (arthroscopic or open), engagement in contact sports or postures that may favor nerve compression, history of metabolic disorders, and occupational postures (e.g., rural work, repetitive bending while bearing weight).

Motor function was evaluated by assessing the muscles innervated by the common peroneal nerve (CPN): tibialis anterior, extensor digitorum longus, fibularis tertius, extensor hallucis longus, extensor digitorum brevis, fibularis longus, and fibularis brevis. It is important to note that the fibular muscles are innervated by the superficial branch of the CPN, whereas the remaining muscles are innervated by the deep branch.

Muscle strength was assessed using the Medical Research Council (MRC) scale: 0, no contraction; 1, minimal muscle contraction; 2, active movement in the absence of gravity; 3, active movement against gravity; 4, active movement against gravity and resistance; and 5, normal strength.¹⁴ The CPN is not associated with any reflex, and there is no specific provocation maneuver linked to it.

Sensory function was assessed through manual stimulation of the dermal regions innervated by the nerve. The CPN proper innervates the proximal lateral aspect of the leg. The superficial branch innervates the dorsum of the foot and the distal anterolateral third of the leg, while the deep branch provides sensation to the first dorsal web space. Tinell's sign was routinely assessed along the nerve pathway.

As for complementary studies, patients with clinical symptoms suggestive of CPN compression were systematically referred for electromyography, including motor and sensory conduction studies of the lower limb. They were referred to the Neurology Department for this evaluation. Additionally, standard anteroposterior and lateral knee radiographs were obtained, as well as a dedicated MRI to detect peripheral nerve lesions (high-resolution MR neurography with intravenous contrast).

Patients presenting with severe clinical compression (clubfoot and $MRC \leq 2$) and a positive electromyogram, but without imaging findings, were considered to have severe idiopathic CPN compression and were indicated for surgical exploration and possible decompression. All patients completed the American Orthopaedic Foot and Ankle Society (AOFAS) questionnaire prior to surgery and at the latest available postoperative follow-up.

Surgical Protocol

The patient was positioned supine with the lower limb slightly flexed. A 5 cm oblique incision was made just below the head of the fibula, following the course of the CPN. The subcutaneous tissue was dissected to expose the superficial fascia overlying the nerve. The nerve is typically identified distally in its course medial and posterior to the fibular head. The fascia was incised parallel to the nerve, which was then decompressed using microsurgical techniques by releasing the surrounding ligaments and fascia until it was completely freed.

Dissection was extended to the nerve trifurcation, with particular attention to the articular branches to rule out the presence of intraneural ganglion cysts. The motor branches often perforate the intermuscular septa. It is essential to release the posterior crural intermuscular septum, located deep to the anterior border of the fibularis longus muscle. The anterior crural intermuscular septum and the surrounding fascia enveloping the CPN over the deep fascia were also identified and systematically released to ensure decompression at the fibular head.

The nerve is protected by perineural fat, which should be preserved as much as possible because it provides vascular support and facilitates gliding motion during joint movement. Hemostasis is critical to prevent hematomas that could compromise the decompressed nerve. The wound was closed with absorbable sutures for the subcutaneous tissue, while the skin was closed with either biological glue (tissue adhesive) or an intradermal nylon suture (Figure).

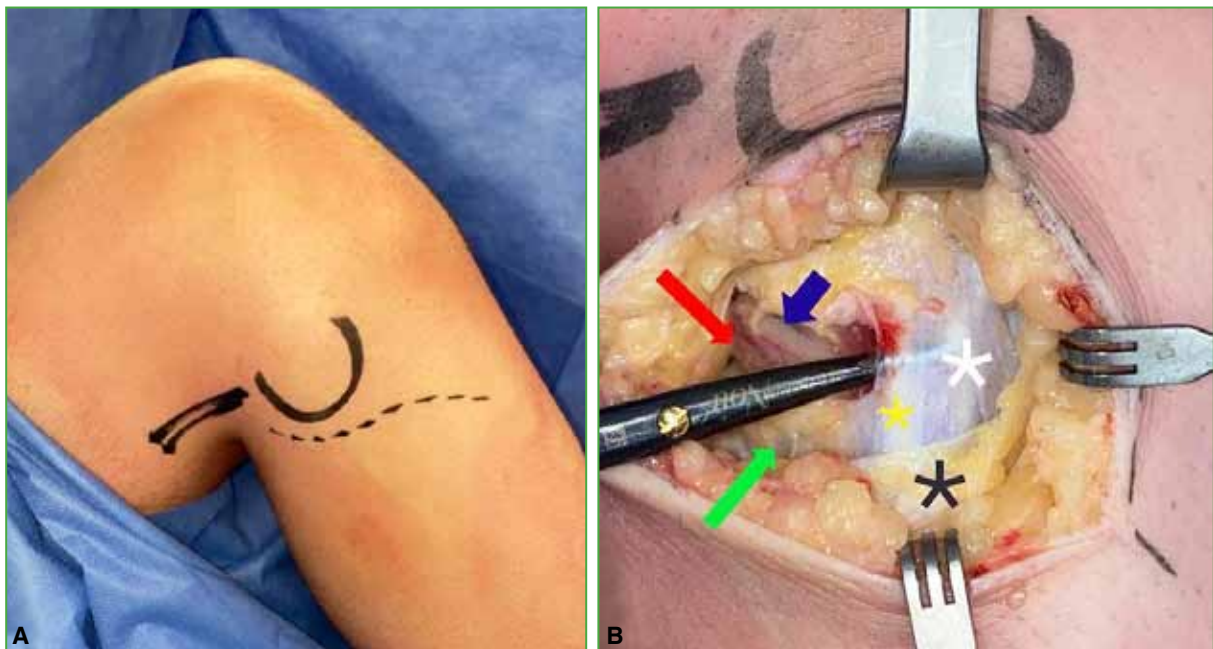


Figure. A. Preoperative skin marking of the surgical approach using the fibular head and nerve orientation as landmarks. B. Soleus muscle (red arrow), common peroneal nerve (blue arrow), anterior septum (green arrow), extensor digitorum longus (yellow asterisk), fibular muscles (white asterisk), anterior fascia (black asterisk).

Postoperative Protocol

Postoperatively, patients were discharged with the ankle and foot positioned at 90° in a protective orthosis (Walker boot). Due to the severity of the condition, many patients had already been advised to use the boot preoperatively to maintain appropriate ankle positioning. Although weight-bearing was not contraindicated, patients were instructed to keep the limb elevated during the initial postoperative days to reduce edema and promote healing. If edema was present, compression stockings were recommended. Physical therapy began five days post-surgery and followed a neuromuscular rehabilitation protocol focused on gait training, management of residual edema and scar tissue, maintenance of ankle range of motion, and strength recovery.

Motor and sensory recovery was evaluated monthly, beginning 30 days after surgery, and continued until full recovery or until the maximum available follow-up was reached. The AOFAS score was used to assess outcomes at the final follow-up.

All patients provided informed consent and agreed to participate in the study. This research was conducted in accordance with the Declaration of Helsinki.

RESULTS

Over a 10-year period (2013–2023), 16 decompression procedures of the CPN were performed. Eight patients met the inclusion criteria (2 women and 6 men). The mean time from diagnosis to surgery was 30 days. The average follow-up duration was 959 days. All patients recovered at least grade 4/5 dorsiflexion motor strength of the affected foot. Six of the eight patients recovered full motor strength and normal sensation. None experienced abrupt weight loss. No relevant medical history associated with the condition under study was reported, and none of the patients were smokers. Demographic data are detailed in [Table 1](#).

Table 1. Demographic and preoperative data.

Pa-tient	Age	Sex	Laterality	Smoker	Time from diagnosis to surgery (days)	Elec-tromyogram	MRI	Sensitivity	TA	EHL	EDL	Fibu-laris	AOFAS Score
1	16	M	R	No	30	Positive	No findings	Total paresthesia	M0	M0	M0	M0	38
2	78	M	R	No	20	Positive	No findings	Hyperesthesia in the lateral aspect of the leg plus hypoesthesia in the dorsum of the foot.	M2	M0	M2	M2	24
3	78	F	L	No	15	Positive	No findings	Hypoesthesia in the dorsum of the foot	M0	M1	M2	M2	44
4	63	F	L	No	40	Positive	No findings	Hypoesthesia in the dorsum of the foot	M0	M0	M0	M0	24
5	60	M	R	No	35	Positive	No findings	Hypoesthesia in the dorsum of the foot	M2	M2	M1	M1	40
6	47	M	R	No	40	Positive	No findings	Hypoesthesia in the lateral aspect of the leg plus hypoesthesia in the dorsum of the foot.	M0	M2	M0	M2	24
7	41	M	R	No	40	Positive	No findings	Hypoesthesia in the lateral aspect of the leg plus hypoesthesia in the dorsum of the foot.	M1	M2	M1	M2	24
8	16	M	L	No	20	Positive	No findings	Hypoesthesia in the lateral aspect of the leg plus hypoesthesia in the dorsum of the foot.	M0	M0	M1	M1	24

M = male; F = female; R = right; L = left; MRI = magnetic resonance imaging; TA = tibialis anterior; EHL = extensor hallucis longus; EDL = extensor digitorum longus; AOFAS = American Orthopaedic Foot and Ankle Society.

There were no intraoperative or postoperative complications. In all cases, the nerve injury was classified as neurapraxia according to Seddon's classification,¹⁵ with no evidence of axonotmesis or neurotmesis; all cases corresponded to Sunderland type I lesions.¹⁶ All electromyographic studies demonstrated acute neurogenic compromise, with ongoing denervation activity, absence of reinnervation at the time of evaluation, and topography consistent with a lesion at the level of the CPN in the knee, showing conduction block. The mean AOFAS score was 30 prior to surgery (range, 24–44) and 97 at the end of follow-up (range, 93–100). Table 2 summarizes the results at the final follow-up.

Table 2. Postoperative evaluation

Patient	Intraoperative findings	Follow-up (days)	TA	EHL	EDL	Fibularis	AOFAS score
1	Negative	465	M5	M5	M5	M5	93
2	Negative	755	M4	M4	M4	M5	95
3	Negative	2550	M4	M5	M5	M5	93
4	Negative	435	M5	M5	M5	M5	100
5	Negative	395	M4	M4	M4	M4	100
6	Negative	1850	M5	M5	M5	M5	95
7	Negative	730	M5	M5	M5	M5	100
8	Negative	495	M5	M5	M5	M5	100

TA = tibialis anterior; EHL = extensor hallucis longus; EDL = extensor digitorum longus; AOFAS = American Orthopaedic Foot and Ankle Society.

DISCUSSION

We present a case series of early decompression of the CPN in patients with idiopathic and severe involvement. The mean time from diagnosis to surgical intervention was 30 days. Functional outcomes were favorable in all patients following CPN decompression. Our findings are comparable to those of the most significant published series to date, which included 14 patients diagnosed with severe idiopathic compression. Notably, that study was conducted and published in the field of Neurosurgery rather than Orthopedics.¹³ Although it was a prospective study, one patient underwent surgery more than 100 days after diagnosis; in contrast, all patients in our series were operated on within the first month. That study concluded that earlier decompression is associated with better outcomes. Although the precise time frame for defining “early” decompression remains unclear, we believe that the success observed in our series is partly attributable to the short interval between diagnosis and surgery. There is currently no consensus on the ideal timing, and it would be ethically unfeasible to conduct a prospective trial involving a watch-and-wait strategy in patients with severe presentations to evaluate the differential response to decompression. Similarly, a control group was not feasible in our study, as we believe—based on theoretical and clinical grounds—that severe cases warrant prompt surgical intervention.

The fibers of the CPN originate from the L4–S1 spinal nerve roots and descend as part of the sciatic nerve before diverging into the fibular division. The nerve is particularly vulnerable to compression as it winds superficially around the fibular neck on the lateral aspect of the knee, where it is protected only by skin and subcutaneous adipose tissue.¹⁷ The anatomical course of the CPN exhibits considerable variability. Although certain “safe zones” have been described, the high interindividual variation precludes the recommendation of standardized decompression approaches.

The most frequent sites of CPN compression include the intermuscular septum, the convergence of the proximal insertions of the soleus and fibularis longus muscles, the entrance of the fibrous tunnel, the fibrous band of the deep head of the fibularis longus, and the fascia of the fibular muscles.^{18,19} Currently, PCN compression is considered a dynamic condition. Intraoperative pressure measurements have shown that nerve pressure progressively decreases as the most common sites of compression are sequentially released.²⁰

The diagnosis of idiopathic compression of the CPN is not straightforward, and several aspects must be taken into account. The clinical presentation may initially resemble that of other conditions, such as chronic compartment syndrome. The etiology of clubfoot can be highly diverse. A thorough clinical examination and a detailed patient history are essential to determine the cause and, most importantly, to assess the likelihood of recovery without surgical decompression.

Patients presenting with acute, rapidly progressive CPN palsy and no early signs of motor recovery are candidates for nerve exploration and possible decompression.²¹ In general, surgical decompression of lower limb nerves significantly improves patients' quality of life.²¹ However, there are currently no prospective randomized studies available to establish standardized recommendations on the timing of nerve decompression.¹¹

Once compression has been diagnosed, the tendency to prescribe a series of poorly standardized conservative treatments may hinder the patient's recovery. These may include activity modification, physiotherapy, stretching exercises, massage, nerve blocks, or iontophoresis. In severe cases, however, recovery is often incomplete.²⁰

In 2023, Oosterbos et al.²² conducted a survey and concluded that there are not only substantial differences in therapeutic approaches among physicians within the same specialty but also between different specialties trained to manage this condition and potentially perform surgery. Furthermore, no studies have evaluated the cost-effectiveness of non-invasive treatment compared to surgical management.

Another issue is the lack of standardization in the evaluation of outcomes after both surgical and conservative treatment. A systematic review of 31 articles published in 2023 found that only 83.9% reported motor strength outcomes, 38.7% reported sensory findings, 25.8% assessed pain, 12.9% used validated foot and ankle functional scores, 9.7% used electrodiagnostic studies, and only 3.2% included imaging results. In total, 29 different outcome measures were used.²³

As described in our preoperative protocol, all patients underwent electromyography (EMG) as part of the diagnostic process. The sensitivity and specificity of EMG are generally high. However, as previously reported in the literature, the absence of abnormal findings in patients with severe symptoms—as in this series—is usually due to technical limitations, given that EMG is operator-dependent. For this reason, our protocol mandates that all studies be performed in our institution by the same experienced team of neurologists. Additionally, the absence of sensory findings on EMG should not preclude the decision to proceed with early decompression. In this patient group, early surgical intervention has been shown to yield better outcomes than conservative treatment.¹⁹

In 2013, Maalla et al.¹⁹ reported that clinical outcomes were worse when surgery was delayed by more than 12 months. Similar results were seen in patients who presented with sensory symptoms and underwent surgery after more than 6 months. The authors also emphasized that advanced age should not be considered a contraindication to decompression. In our series, two patients were 78 years old.

The use of MRI for preoperative evaluation of the CPN has also been explored. While MRI has high specificity (>90%), its sensitivity barely exceeds 50%.²⁴ Certain advanced sequences, such as high-resolution MR neurography with intravenous contrast, are believed to provide more detailed information. Although the absence of imaging abnormalities does not preclude surgery (it simply classifies the case as idiopathic), we believe that imaging is important to rule out more serious secondary conditions, such as neoplastic lesions. The use of complementary studies, such as compartment pressure measurements to exclude other pathologies—such as chronic compartment syndrome—has also been debated.¹⁰

This study has several limitations. First, its retrospective design. Second, the absence of a control group, which is not feasible due to the small number of patients and ethical considerations. Third, the overall sample size is limited. However, considering the existing literature and the narrow focus on severe idiopathic compression, we believe this series is consistent with the rarity and specificity of the condition.

CONCLUSIONS

Early decompression of the common peroneal nerve is a safe procedure that should be considered in cases of severe idiopathic compression. This is a rare condition, and conservative treatment may delay decision-making and compromise prognosis. Orthopedic surgeons trained in peripheral nerve surgery should be familiar with this disease and able to manage it without referral. We believe it is important to establish standardized preoperative, intraoperative, and postoperative protocols to optimize outcomes.

Conflict of interest: The authors declare no conflicts of interest.

V. Barbaglia ORCID ID: <https://orcid.org/0009-0009-3788-6718>

F. Holc ORCID ID: <https://orcid.org/0000-0002-1224-3312>

P. Bronenberg Victorica ORCID ID: <https://orcid.org/0000-0003-0131-3124>

I. Rellán ORCID ID: <https://orcid.org/0000-00034045-339X>

A. G. Donndorff ORCID ID: <https://orcid.org/0000-0002-6384-4820>

G. L. Gallucci ORCID ID: <https://orcid.org/0000-0002-0612-320X>

P. De Carli ORCID ID: <https://orcid.org/0000-0002-94748129>

J. G. Boretto ORCID ID: <https://orcid.org/0000-0001-7701-3852>

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