



NOVEL NERVE TRANSFER FOR THE TREATMENT OF PERONEAL NERVE PALSY SECONDARY TO AN INTRANEURAL GANGLION: CASE REPORT AND REVIEW

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CASE REPORT

An otherwise healthy 74 year-old female presents with a 5 month history of progressive pain in the superficial and deep peroneal nerve distribution and a dense foot drop requiring an ankle foot orthosis. She is currently on gabapentin and pregabalin.

Clinical exam reveals a foot drop with a high step gait. A palpable mass posterior to the right fibular head is present. The patient demonstrates BMRC (British Medical Research Council) motor (M) grade 0/5 ankle dorsiflexion, toe extension and eversion. She has M5 ankle plantar flexion, toe flexion and inversion. Sensory deficits are appreciated in both the superficial and deep peroneal nerve distributions (2-point discrimination >8mm). A Tinel's sign is absent at the fibular head. Reflexes are symmetric (2+) in the knees and ankles bilaterally. Straight leg test was negative for lumbosacral nerve root compression.

An MRI of her right knee demonstrates an intraneuronal cyst (6 mm AP x 4 mm transverse x 28 mm longitudinal) within the common peroneal nerve (Figure 1). There were no elicitable motor units on electromyography (EMG) in the peroneal nerve distribution, while nerve conduction studies (NCS) demonstrated nerve continuity. CT and MRI of the lumbar spine were performed and were grossly unremarkable.

Foot drop correction is obtained by ligating the articular branch of the peroneal nerve, decompressing the intraneuronal cyst, and performing a nerve transfer in which the tibial motor nerve branch to flexor hallucis longus (FHL) is transferred into the deep peroneal motor nerve branch of the anterior tibialis muscle. At final follow-up (>1year), the patient demonstrated M5 ankle dorsiflexion, normal gait, no need for orthotics, and no evidence of cyst recurrence. There were no tibial nerve deficits and no peri-operative complications.

PURPOSE

The purpose of this study is to describe a novel treatment of a common peroneal nerve palsy caused by an intraneuronal cyst: by ligating the articular branch of the peroneal nerve, decompressing the intraneuronal cyst and performing a nerve transfer in which the tibial motor nerve branch to flexor hallucis longus (FHL) is transferred into the deep peroneal motor nerve branch of anterior tibialis muscle.



Figure 1. MRI image of right knee. Multiple internal cysts observed within the common peroneal nerve just proximal to the fibular head extending 2.8 cm (longitudinal) by 6 mm (anteroposterior) by 4 mm (transverse). T1-weighted axial (A) and sagittal (B) views of the knee. (C) T2-weighted axial image demonstrating multiple intraneuronal cysts. White arrows indicate the location of the common peroneal nerve in images (A) to (C).

INTRODUCTION

Foot drop results in a debilitating gait abnormality owing to a loss of ankle and toe dorsiflexion [1]. Unfortunately, current treatment modalities offer limited restoration of function. Ankle foot orthotics (AFO's) remain the mainstay of treatment; however, patients are often unsatisfied due to discomfort, hygiene, and mobility issues [2]. Tendon transfers commonly divert the posterior tibialis tendon to the dorsum of the foot and are a reasonable surgical option. Yet, only limited ankle dorsiflexion has been achieved with this strategy. Tendon transfers may also result in undesirable hindfoot valgus deformity, flatfoot deformity, or arthritis [2]. More recently, autologous nerve transfers have been proposed as a new strategy for deep peroneal neuropathies. The principle is based on using a functional but less important nerve as a source of donor axons for distal, denervated nerves [2]. White *et al.* (2012) found adequate axon counts in the motor branches to the lateral gastrocnemius, extensor hallucis longus, and flexor hallucis longus to restore ankle dorsiflexion following nerve transfer [3]. The motor branch to the soleus muscle has also been postulated as a donor nerve but was found to confer poor (M2 or less) ankle dorsiflexion [1].

Intraneuronal ganglia are benign mucinous cysts that can occur within the epineurium of nerves and are a rare cause of foot drop [4,5]. The diagnosis is made on MRI, wherein tubulocystic structures are seen within the affected nerve that appear hyperintense on T2-weighted images and hypointense on T1-weighted images [4]. Some uncertainty surrounds the mechanism explaining intraneuronal ganglion cyst formation. The most accepted synovial (articular) theory proposed by Spinner *et al.* (2009) suggests that a pedicle connects the synovial joint to the nerve by way of cystic fluid infiltration along an articular nerve branch [5,6]. In the lower extremity, the common peroneal nerve is the most common site of occurrence. Here, the pedicle is thought to originate from the proximal tibiofibular joint. Surgical outcomes depend largely on the duration and extent of cystic destruction, early recognition, and timely intervention [5]. Extraneuronal decompression and epineurotomy remain the mainstay of treatment. Disruption of the pedicle stalk is thought to be essential in preventing recurrence [7].

SURGICAL PROCEDURE

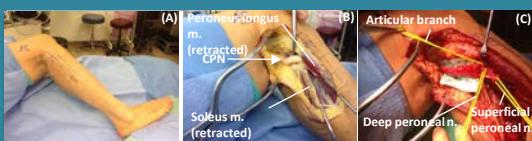


Figure 2. Surgical treatment of a common peroneal nerve palsy secondary to an intraneuronal cyst. (A) Pre-operative incision markings. (B) Identification of an enlarged common peroneal nerve (CPN) due to an intraneuronal ganglion cyst. (C) The CPN trifurcates into the articular, deep and superficial branches. The articular branch is ligated and an interfascicular nerve dissection is performed to decompress the cyst.

References: [1] Flores, L.P., *et al.* 2013. *Neurosurg.* 73(4):609-616. [2] Giuffre, J.L., *et al.* 2012. *Ann Plast Surg.* 69:48-53. [3] White, C.P. *et al.* 2012. *Can J Plast Surg.* 20(1):24-27. [4] Van den Bergh, F.R.A., *et al.* 2013. *Insights Imaging.* 4:287-289. [5] Colbert, S.H. and Le, M.H. 2011. *Hand.* 6:317-320. [6] Spinner, R.J., *et al.* 2009. *Neurosurg.* 65(4suppl): A115-24. [7] Muramatsu, K., *et al.* 2013. *Acta Neurochir.* 155:1757-1761.



Figure 2 (continued). Nerve transfer of the tibial motor fascicle to FHL to the tibialis anterior nerve branch for correction of footdrop. (D) Interfascicular dissection of the deep peroneal nerve identifies the motor branch to tibialis anterior. (E) Interfascicular dissection of tibial nerve identifies the motor branch to FHL. (F) Tibial nerve branch to FHL (below) adjacent to the peroneal motor nerve branch to anterior tibialis (above). A tension-free neurorrhaphy is performed with 9-0 Ethilon sutures.

RESULTS



Figure 3. Post-surgical restoration of ankle dorsiflexion at most recent follow-up (1 year). The patient demonstrated complete recovery (BMRC grade M5) of ankle dorsiflexion and toe extension, normal gait, and no need for orthotics. Post-operative pain and dysesthesias resolved by 6 months post-operation. There were no tibial nerve deficits and no peri-operative complications. (A) Ankle plantarflexion. (B) Ankle dorsiflexion. (C) Great toe and D2-D5 flexion. (D) Great toe and D2-D5 extension.

DISCUSSION

We present a case report of an elderly patient with a 5-month history of a dense common peroneal nerve palsy secondary to an intraneuronal ganglion. The accepted synovial (articular) theory [6] recommends ligating the articular branch in a timely fashion to obtain optimal recovery. It is commonly known that motor nerve recovery is ideal if the nerve reaches the motor endplates of the muscle by one year. Given that this elderly patient was presenting with a proximal peroneal nerve lesion and a 5 month history of a dense foot drop with no elicitable motor units on EMG, we decided to augment the ligation of the common peroneal articular nerve branch with a distal tibial to peroneal nerve transfer to improve her chances of recovery. We were able to successfully restore her foot function without any donor deficits. Given the paucity of clinical data detailing treatment of proximal intraneuronal ganglia, the combination of ligating the articular branch and performing a distal nerve transfer may improve the chances of a full recovery.