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 inversion. 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 ankle bilaterally. Straight leg test was negative for lumbar sacral nerve root compression.

An MRI of her right knee demonstrates an intraneural 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 normal conduction velocity. 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 intraneural 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.

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 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]. While et al. (2012) found adequate axon counts in the motor branches to the lateral gastrocnemius, and ankle plantarflexion, 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 ankle bilaterally. Straight leg test was negative for lumbar sacral nerve root compression.

The purpose of this study is to describe a novel treatment of a common peroneal nerve palsy caused by an intraneural ganglion cyst. The purpose of this study is to describe a novel treatment of a common peroneal nerve palsy caused by an intraneural 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