Symptomatic Accessory Deep Peroneal Nerve Treated with Conduit-Assisted Neurorrhaphy to the Superficial Peroneal Nerve: A Case Report

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Statement of Purpose

The deep peroneal nerve (DPN) is responsible for innervation to the extensor digitorum brevis (EDB) muscle belly with sensory contribution to the first pedal interspace. An accessory variant can be present in up to 28% of individuals (1,3). If a variant goes unrecognized, there is risk of iatrogenic damage during surgery (1). This poster presents a case study of an accessory deep peroneal nerve (ADPN) with previous trauma which became symptomatic to the patient. Therefore, it was treated with resection and neurorrhaphy to the adjacent superficial peroneal nerve (SPN) by assistance of a bovine conduit.

Literature Review

After wrapping anteriorly around the head of the fibula, the common peroneal nerve (CPN) descends into the lower extremity and divides into the SPN and DPN (4). The DPN then continues into the anterior compartment while the SPN descends within the lateral compartment (4). Patients commonly experience CPN dysfunction due to compression at the fibular head. However, insult of either terminal branch of the nerve distally may create neurologic deficits and symptoms.

The ADPN contains both motor and sensory branches with the motor branches supplying the peroneal longus and EDB muscle bellies (3). One way to determine the presence of an ADPN is to monitor the response of the EDB muscle after peroneal nerve stimulation. The Compound Muscle Action Potential (CAMP) can then be measured at both the proximal and distal levels of the course of the nerve (3). This determines a ratio assessing the level of highest muscle response. A more distal response can confirm the presence of a true accessory nerve variant (3).

To treat LE neurogenic pain surgical options include neurectomies, nerve relocation, or ablative procedures (5). However, these interventions do not always address the underlying pathology. Transection of a nerve without repairing the severed end can create a neuromal stump formation eliciting increased pain to the patient (6). This process can occur from angiogenesis, scarring, or fiber regeneration of the nerve (6). Preventing the stump formation is the best method to halt its development (6) therefore, suturing the transected portion to either the end or side of a reciprocal nerve is recommended preventing the occurrence of regeneration distally.

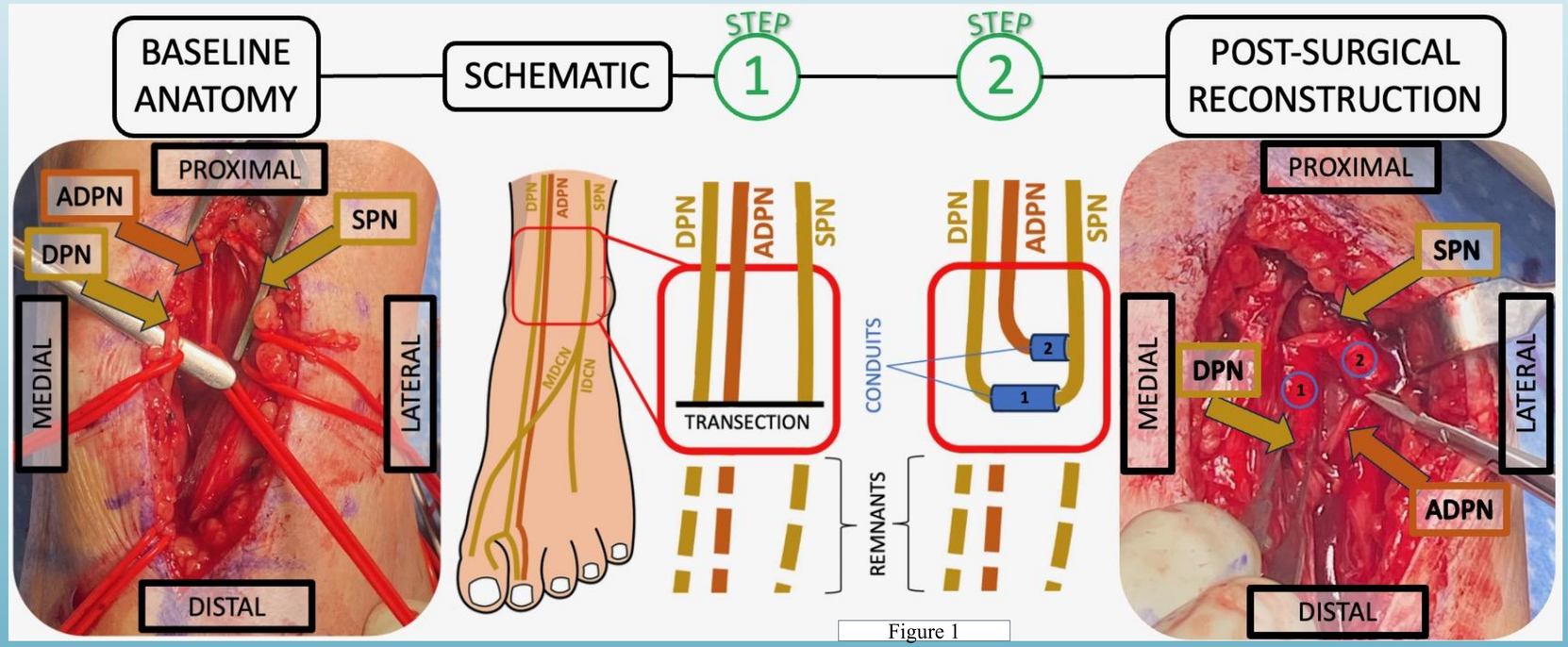

Figure 1

Case Report

The patient is a 65-year-old female with an *extensive surgical history* of the left lower extremity (LE) due to a previous ankle fracture and attempted excision of the DPN. The patient underwent *two previous nerve decompressions, including excisions of the DPN* at the level of the left first interspace and ankle as well as a *conduit repair with resect neurectomy of the primary DPN*. The patient experienced intermittent symptom relief, but ultamitely her pain persisted. Therefore, the patient underwent treatment for severe localized peripheral neuropathy secondary to entrapment of the DPN in the foot and ankle of the left LE.

A preoperative electromyogram study demonstrated findings consistent with DPN entrapment. Preoperative injections identified the zone of primary *pain to traverse the DPN course just proximal to the level of the ankle*. It was confirmed during the second attempt at conduit repair that an accessory peroneal variant was present within the anterior bundle sheath. Upon stimulation of that variant, with a check point stimulator, motor response of the hallux was elicited.

Repeat intervention consisting of end-to-end conduit repair of the primary DPN to SPN and an end-to-side repair of the ADPN to SPN was performed (Figure 1).

Results

After the first attempt of DPN conduit repair, the patient reported approximately 30% relief of symptoms. Trial and error of local injections to both the DPN and SPN were performed, identifying the areas of discomfort. Following the transection of each nerve at the level of the ankle joint, the sensory distribution to the distal aspect of the foot was compromised. Her pain however, was completely eliminated. After one year follow up, the patient reported no active symptoms or foot and ankle complaints.

Discussion

To the author's knowledge, the foot and ankle literature is limited on neurorrhaphy of the DPN to SPN with even greater paucity when an ADPN is present. As mentioned above, finding patients with a symptomatic ADPN is also finite. However, orthopedic case reports regarding traumatic nerve transections with end-to-end or end-to-side repair can be found. Our report demonstrates a highly innovative and restoring option for those suffering from chronic, ongoing, and recalcitrant neuromas and other localized neurogenic pathologies.

Level of Evidence: V

References:

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