

Double Axonal Crush: Resection and Implantation of Deep Peroneal Nerve for Intractable Pain

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Statement of Purpose

Intractable pain following calcaneal fracture fixation is a rare, but debilitating complication for a patient.¹ The present study assesses whether our double axonal crush technique with distal deep peroneal nerve (DPN) neuro-osteodesis is effective in alleviating residual intractable pain after treatment for Sanders III and IV calcaneal fracture.

Methodology

Eleven patients (10 male) with a history of intra-articular calcaneal fractures (Sanders III and IV) with evolving pain to the level of incapacitation despite previous treatment for pain symptoms were included in this study. Each patient's chart was reviewed in detail and all were noted to have received various conservative treatments. EMG and NCV studies for all 11 patients noted decreased DPN signal 1.5 cm proximal to the ankle joint extending to the sinus tarsi.



Image 1: Preoperative imaging



Image 2: Anterior Neurovascular bundle

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Procedure

A linear mid-lateral compartment incision was made to avoid all vital neurovascular structures. The neurovascular bundle of the anterior compartment was identified at the floor of the anterior compartment and dissected in order to identify the Deep Peroneal Nerve(Figure 1). The Deep Peroneal Nerve was dissected out in the distal one third of the lower extremity. An external neurolysis was performed on the deep peroneal nerve. The nerve was transected distally and a double axonal crush (Figure 2) was performed proximally. The tip of the proximal end of the nerve was cauterized with bipolar cautery. The tibia was then exposed and a hole was drilled into the tibia. The proximal end of the deep peroneal nerve was then buried into the drill hole and secured with a periosteal epineural stitch using 11.0 suture(Figure3). The knee was then ran through flexion and extension to ensure the nerve was properly secured.



Image 3: "Double Axonal Crush"



Image 4: DPN Tibial Implantation

Numerous studies have shown that the sequelae of intractable pain can include physical, psychological, social, and economical debilitation.¹⁻⁵ These patients often fail a number of conservative measures. Traditionally, decompression of the nerve with or without grafting is done for entrapment neuropathies with subsequent disruption of nerve signal.⁶ The diameter of the deep peroneal nerve in the distal 1/3rd of the leg is often 1-3 mm, making it too small to graft.^{7,8} Double axonal crush is a technique which disrupts the internal fascicles of the axon while preserving the myelin, causing an axonotmesis.. This decreases the incidence of neuroma formation. Although not common, nerve transection, double axonal crush, and subsequent re-implantation has been recorded in the literature for the treatment of causalgia. McKinnon et al. reported on a 19 year old patient with CRPS II that resulted from an ankle sprain without fracture. The patient failed numerous treatment modalities, and eventually underwent successful nerve resection, axonal crush, and implantation.⁹

Results						
Patient	Age	Gender	Comorbidities	Sanders	Pre-op VAS	Post-op VAS
1	17	М		4	10	2
2	26	М		3	10	3
3	27	М	smoker	4	9	3
4	29	М		3	10	3
5	33	М		4	9	1
6	34	М		3	8	1
7	39	М	Smoker, htn	3	8	1
8	41	М	DM	4	7	2
9	48	М		3	9	1
10	56	F	htn	4	7	2
11	66	М	DMII	4	8	2

Literature Review

 Table 1: 11 Patients were followed for an average of 26 months (14mo-41mo).
 The VAS pain improvement range

was 4-8 points, alleviating a significant amount of pain in all patients. The VAS pain scale decreased, on average, by 6.6 (8.6 to 2) points. These results, summarized in Table 1, were found to be statistically significant(p value < 0.0001) Patient's also completed pre and post operative Quality of Life Questionnaires which also demonstrated subjective decrease in pain and increase in quality of life with emphasis on ADL's after undergoing the procedure.

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We describe here a series of patients with long standing intractable pain after Sanders Type III and IV calcaneal fractures relieved by a surgical procedure to the deep peroneal nerve. The fact that neuropathic pain did not return or was minimally residual after the procedure appears to be due to the lack of stump neuroma formation secondary to performing a double axonal crush of the deep peroneal nerve proximal to resection site resulting in axonotmesis, with implantation. This suggests that the neuropathic pain syndrome was dependent on neuronal activity originating distal to the resection. Such a result challenges the concept that central neuroplastic changes become permanent and independent of a peripheral afferent drive.¹⁰⁻¹² In these cases, the deep peroneal nerve was resected distal to motor branches to leg musculature. The extensor digitorum brevis was deemed to be worth sacrificing due to its limited biomechanical advantage.¹³ The option of peripheral nerve surgery should be heavily considered for long standing intractable pain that has failed conservative management. The risk that this type of surgery may fail or even exacerbate pain is unknown, and the true outcomes of this procedure can only be known from careful, quantitative follow-up of many other cases.



Analysis & Discussion

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