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CASE REPORT

Hand/Peripheral Nerve

Peroneal Nerve Repair of a 9 Year Old: Return of Motor Function after 2 Years

Oren Tessler, MD, MBA*
Lynn Bourn, BSN†
Jasmine Kudji†
Gabriel C. Tender, MD‡

Summary: Major factors that influence functional nerve recovery, postrepair, are length of the nerve defect, type of injury, operative technique, time until treatment, and age of the patient. We present a severe motor nerve defect in a complicated peroneal nerve injury in a 9-year-old that showed functional return after a delayed period of 23 months with sural nerve cable grafting. This case revealed the increased resiliency and regenerative capacity of motor end plates in young patients. In conclusion, autograft for a deep peroneal nerve repair, by means of sural nerve graft, proved to be an acceptable option in children. *(Plast Reconstr Surg Glob Open 2018;6:e1835; doi: 10.1097/GOX.0000000000001835; Published online 19 June 2018.)*

INTRODUCTION

When determining an approach to surgical repair of a peripheral nerve injury, 2 primary elements are to evaluate the mechanism of injury (blunt versus sharp) and length of the defect. Crush injury components are associated with increased inflammation that lasts for up to 3 weeks postinjury and can result in a nebulous zone of injury; thus, complex injuries are often tagged with definitive repair at 3 weeks postinjury to allow for greater clinical recovery.[9] If tension-free end-to-end coaptation cannot be achieved, nerve grafting via an autograft or allograft is performed to repair the gap.[4,11] For gaps less than 5 cm, Cho et al.[1] demonstrated similar outcomes for allograft and autograft repair for motor nerve injuries. Nerve graft length and time lapse before treatment are important factors affecting outcomes for reinnervation. Specifically, the common peroneal nerve repair will produce suboptimal results if surgery is performed more than 12 months after injury or with a graft of more than 12 cm.[2] Although there is no consensus on critical length or delay to repair deep peroneal nerve injuries, research shows a 75% motor recovery rate for nerve grafts < 6 cm and a significant decrease in reinnervation to 35–40% for 6–12 cm.[4,7] It has also been shown that significant muscle fiber atrophy and decreases in regeneration of distal nerves occurs as early as 3 months after injury resulting in limited reinnervation and recovery.[6] Additionally, age plays a role in nerve plasticity and regaining function. Younger age has been associated with increased recovery due to children's increased regenerative capabilities, with children having faster recovery times (mean of 18 months) compared with adults (average of 30–39 months) for peroneal nerve repairs..[4,5,11,12]

CASE DESCRIPTION

Our patient was a 9-year-old female who presented, following a jet-ski accident, with a posttraumatic avulsion and crush injury of her peroneal nerve from a right fibular fracture, ultimately, resulting in a deep peroneal motor loss with foot drop and loss of eversion. She initially underwent exploration and debridement of wound with the nerve ends tagged for later repair due to significant crush component. Subsequently, the patient remained with a 5-cm defect, repaired with an allograft nerve at 1 month postinjury. Physical therapy (PT) noted a decreased right leg girth 1.5 months after initial surgery with a 1 cm loss at ankle and 4 cm loss at calf. At 1 year, the patient showed minimal functional improvement but was able to minimize gait deformity with a compensatory ankle-foot orthosis splint. PT noted 0 degrees of dorsiflexion/eversion, and nerve conduction studies showed no evidence of reinnervation; therefore, further surgical exploration and repair with

*From the *Department of Surgery, Section of Plastic & Reconstructive Surgery, Louisiana State University Health Sciences Center, New Orleans, La.; †Louisiana State University Health Sciences Center, School of Medicine, New Orleans, La.; and ‡Department of Neurosurgery, Louisiana State University Health Sciences Center, New Orleans, La.*

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Video Graphic 1. See video, Supplemental Digital Content 1, which demonstrates return of motor function 2 years after injury, ***http://links.lww.com/PRSGO/A796***.

sural nerve cable autograft was performed. At 9 months postsecond operation, MRC 1/5 motor function became evident with minimal dorsiflexion and eversion. At 16 months postsecond operation and 28 months postinjury, motor function improved to 4/5 dorsiflexion of toes and ankle with 3/5 eversion (**see video, Supplemental Digital Content 1**, which demonstrates return of motor function two years after injury, ***http://links.lww.com/PRSGO/A796***). PT follow up recordings of active and passive range of motion revealed marked improvement at the ankle (Table 1).

Initial Repair with Nerve Allograft

Dissection was carried out under the microscope to trace previous Prolene tagging sutures to identify the distal and proximal portions of the deep peroneal nerve and the ends were cut back to healthy bleeding fascicles. The final gap was approximately 4 cm, and it was decided to use nerve allograft with a 2–3 mm diameter Avance nerve allograft of 5 cm. Epineurial repair was effected with 9-0 Nylon sutures and TISSEEL fibrin sealant and was supported with AxoGuard nerve protectors at the proximal and distal appositional repair sites (Fig. 1).

Table 1. PT Reports of AROM of Right Ankle following Sural Nerve Surgery

PT right ankle range of motion after second surgery	
Date	**AROM (° = degrees)**
2 mo	Inversion: 40° Eversion: 0° Plantar flex: 50° Dorsi-flex: 0°
1 y	Inversion: 38° Eversion: 5° Plantar flex: 50° Dorsi-flex: 13°
2 y	Inversion: 56° Eversion: 100° Plantar flex: 65° Dorsi-flex: 20°

AROM, active range of motion.

Nerve Repair with Sural Nerve Graft

The nerve and allograft were explored and identified. Repair sites were intact, but a large neuroma was identified proximal to the proximal anastomosis site. The pediatric neurophysiology department was present for intraoperative nerve stimulation, which revealed intact branches supplying anterior compartment musculature distal to the previous allograft. Neuroma and graft were then excised, and the nerve was bread loafed proximal and distal until encountering healthy punctate fascicular bleeding. The resultant nerve defect was close to 7 cm. The patient's ipsilateral sural nerve was harvested in standard fashion with staggered incisions and was fashioned to provide 2 cable grafts for repair. The cable grafts were anastomosed with 8-0 Nylon sutures and TISSEEL fibrin sealant. The repair was again supported with nerve conduit AxoGuard nerve protectors on each coaptation site (Fig. 2).

DISCUSSION

As discussed previously, the primary factors that influence postoperative outcomes are length of the nerve defect, type of injury, time until treatment, and age of the patient. Our initial gap was 4 cm and an appropriate allograft length, based on empirical evidence, of 5 cm was used. The traumatic insult was a complex nerve injury with a crush component. Our initial repair at 1 month may have been insufficient to adequately declare the zone of injury; evidence shows higher success with at least 2 months for resolution of inflammation following blunt trauma.[4] Thus, despite the gross appearance of healthy fascicular bleeding, further initial resection may have been required for improved outcome. Complex injuries can have intraneural scarring that extends beyond what is externally visible and can hinder nerve regeneration.[2] The time from initial injury to return of function was approximately 2 years. Despite the large defect and prolonged interruption in repair, recovery outcomes differ in children when compared with adults due to plasticity. It is possible that there were fascicles crossing the allograft that mitigated motor end plate atrophy, as confirmed with intraoperative nerve conduction studies. The volume of crossing nerve fibers was limited by the neuroma and inadequate for clinical function; however, motor end plate atrophy is likely delayed in the pediatric patient compared with adults. This case revealed the increased resiliency and regenerative capacity of motor end plates in young patients. In conclusion, autograft for a deep peroneal nerve repair, by means of sural nerve graft, proved to be an acceptable option in children.

Oren Tessler, MD, MBA
LSUHSC Department of Surgery
Division of Plastic & Reconstructive Surgery
1542 Tulane Ave, Rm 734
New Orleans, LA 70112
E-mail: otessl@lsuhsc.edu

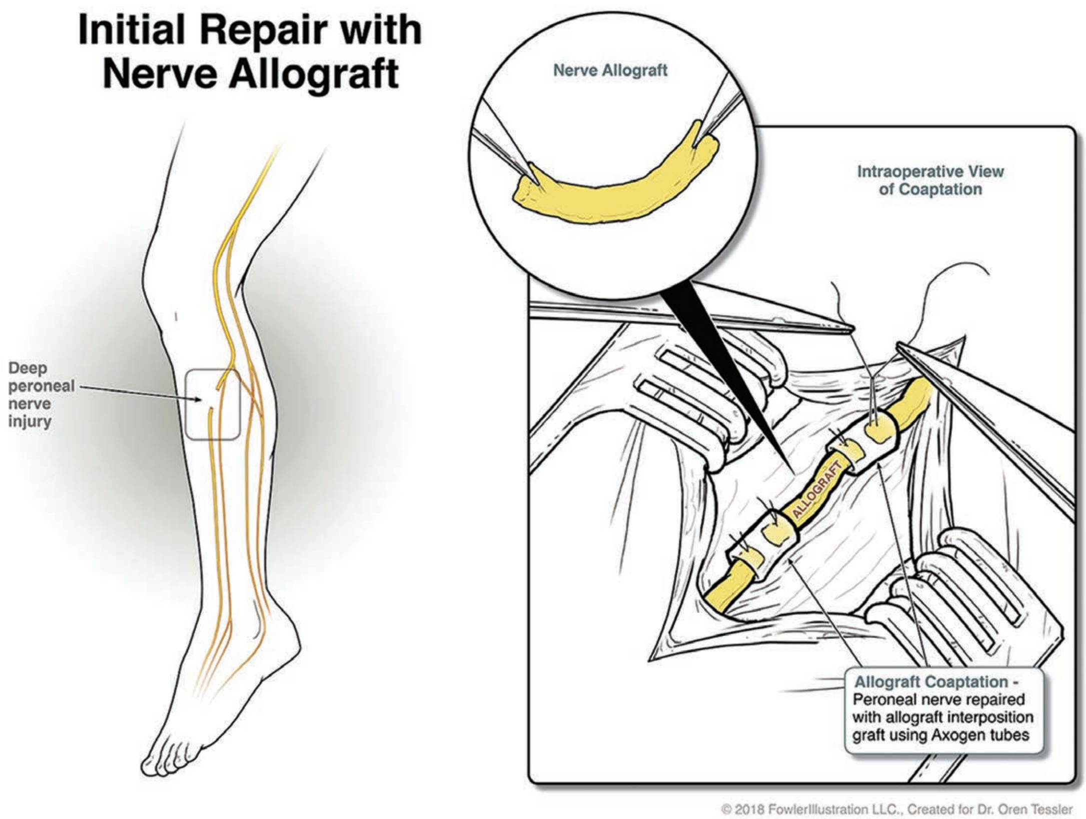

Fig. 1. Primary repair with nerve allograft 1 month after injury.

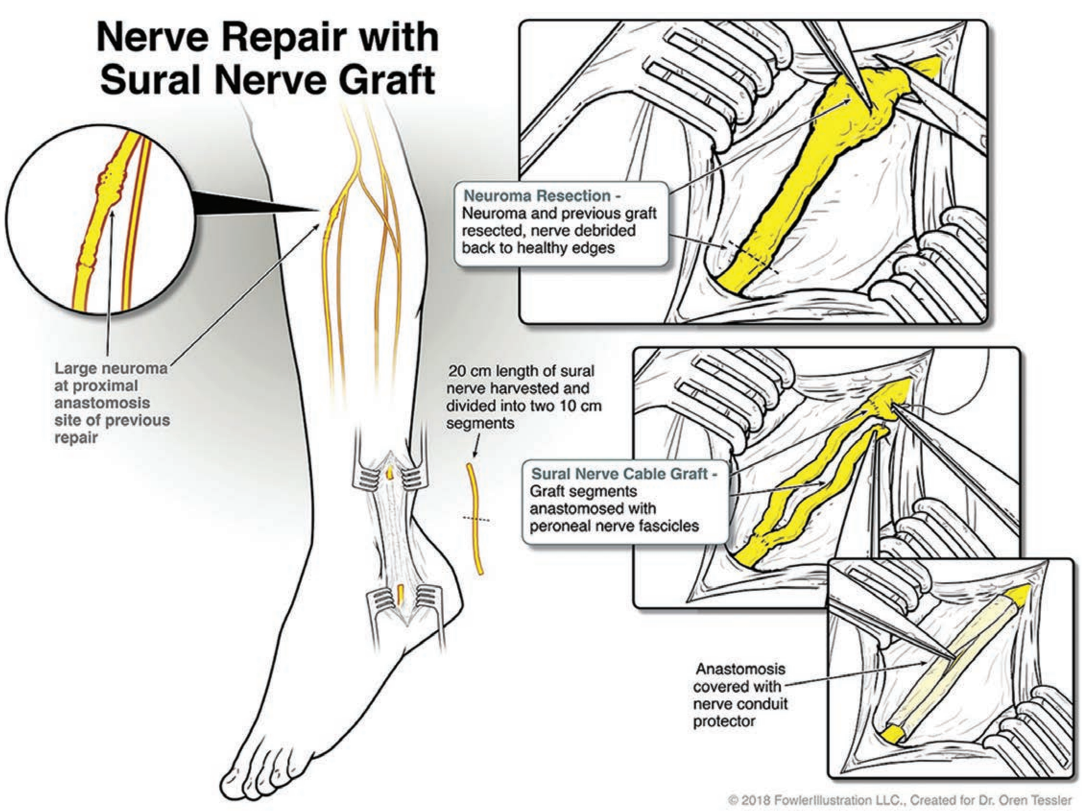

Fig. 2. Secondary repair with sural nerve autograft 1 year after injury.

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