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CASE REPORT
Peripheral Nerve

Inducing Ulnar Nerve Function while Eliminating Claw Hand and Reducing Chronic Neuropathic Pain

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Summary: Ulnar nerve injury induces chronic neuropathic pain and is frequently devastating due to loss of cupping the hand around objects (finger clawing) and diminished grip strength. There is little chance of restoring good function, eliminating finger clawing, or reducing the pain. A novel technique was tested for its efficacy in promoting ulnar nerve function and reducing finger clawing and chronic neuropathic pain. A 25-year-old subject presented 5.7 months after a wrist gunshot that created three nerve gaps proximal to the deep ulnar nerve branch. He sought restoration of function due to developing ulnar nerve injury-induced claw hand and increasingly severe chronic neuropathic pain. After resection of the scarred nerve tissue, each gap was 10 cm long. The gaps were bridged with two nonreversed sural nerve grafts within a PRP-filled NeuroMend collagen tube (Collagen Matrix, Oakland, N.J.). Some axons regenerated entirely across all three 10-cm-long repaired nerve gaps, restoring excellent topographically correct sensitivity of S4, including two-point discrimination of 4 mm, good M4 motor function, and full ROM. The ulnar nerve injury-induced finger clawing was eliminated, and the chronic neuropathic pain of 7 was reduced to 0 on a 0–10 validated scale and did not return over the following 3.75 years. Thus, this novel technique induces good sensory and motor function, despite repairing three 10-cm-long nerve gaps while eliminating ulnar nerve injury-induced hand clawing and chronic neuropathic pain. Further studies are required to determine whether the effects were due to PRP. *(Plast Reconstr Surg Glob Open 2023; 11:e4927; doi: 10.1097/GOX.0000000000004927; Published online 10 April 2023.)*

Ulnar nerve trauma is often devastating by causing loss of hand cupping around objects (claw hand) and diminished grip strength.[1] Clawing results from an imbalance between force generated by innervated strong extrinsic muscles and lack of force by uninnervated intrinsic muscles. Meta-analysis of ulnar nerve autograft repairs found 52% result in satisfactory motor (M4-5) and 43% in sensory recovery (S3+ to S4),[2] but none eliminate claw-hand defects.[1] Another challenge is that nerve traumas cause 50%–79% of individuals to develop chronic neuropathic pain,[3] and autografts do not induce pain reduction or elimination.[4]

Animal model[5] and clinical[6,7] studies show that applying PRP to nerves enhances axon regeneration and reduces pain. To test whether PRP within the gaps induced more extensive recoveries than autografts alone and eliminate hand clawing, three 10-cm-long ulnar nerve gaps were bridged with two autografts within a PRP-filled collagen tube.

METHODS

A 26-year-old man presented without primary nerve repair 5.7 months after a wrist gunshot wound to the deep ulnar nerve branch, distal to FDP ring, and small finger innervation. The ulnar-innervated intrinsic but not extrinsic digital flexor muscles were affected, resulting in a pronounced and characteristic ulnar-claw posture (Fig. 1A). Normal dorsal ulnar sensory branch sensation distribution indicated a lesion distal to the nerve branching point. The deep ulnar nerve branch injury was at the distal wrist crease, about 20 cm proximal to the targets. The subject

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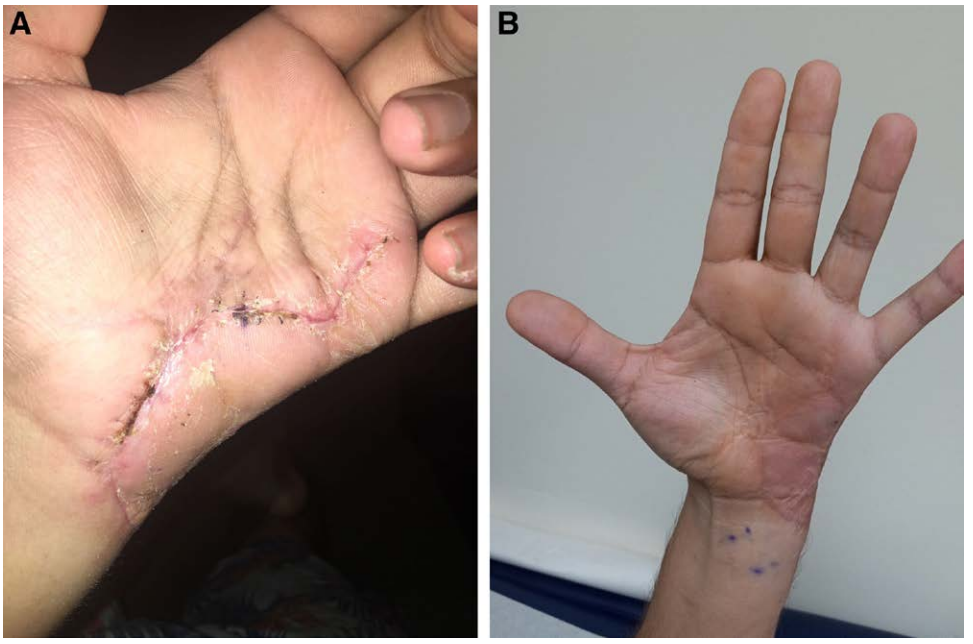

Fig. 1. Hand of subject before and after nerve repair surgery. A, The subject's hand after an initial exploratory surgery without nerve repair, showing the bent small and ring fingers (finger clawing). B, At 3.75 years after ulnar nerve repair, the fourth and fifth fingers could be completely extended and spread, with no signs of finger clawing.

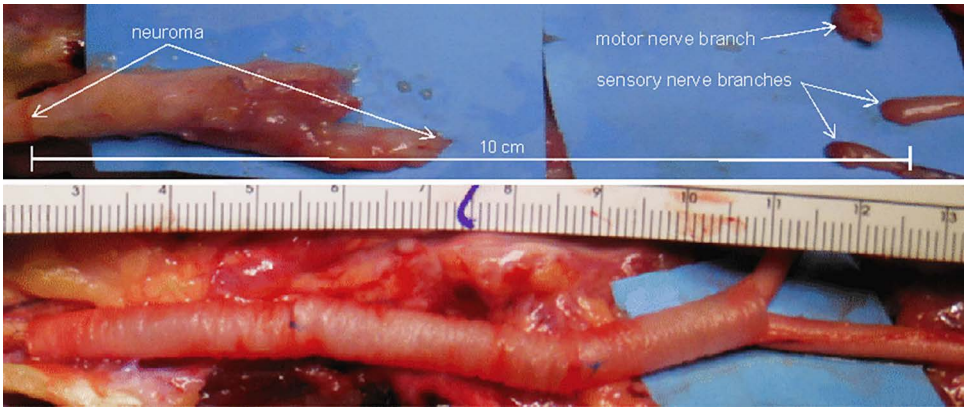

Fig. 2. Repairing the deep branch of the ulnar nerve in the wrist in the region where the nerve branches into one motor and two sensory branches. A, Exposed ulnar nerve injury site showing three nerve gaps and extensive trauma of the proximal and three distal nerve stumps. B, Repaired nerve. After removing the scarred ends of the nerve stumps, there were three 10-cm long gaps. The ends of two lengths of sural nerve graft were secured with sutures to the proximal nerve stump, and the other end of one was secured to the distal motor branch and the end of the other to the two distal sensory nerve branches. The ulnar nerve and grafts were slipped inside the collagen tube, which was then filled with PRP.

had chronic neuropathic pain score of 7 and presented seeking restoration of function and chronic neuropathic pain reduction/elimination (Fig. 1).

The exposed ulnar nerve had severe damage and gaps between the proximal and three distal nerve stumps (Fig. 2A). The damaged nerve stumps were refreshed under a microscope to where no scarring was visible, resulting in one 10-cm motor and two 10-cm sensory nerve branch gaps. The ends of two autografts were loosely secured with a single 9-0 suture to the motor and sensory axon fascicle regions of the proximal ulnar nerve stump and their opposite ends to the appropriate motor and sensory nerve stumps (Fig. 2).

A Y-shaped collagen tube (NeuroMend collagen tubes, Collagen Matrix, Oakland, N.J.) was created by inserting two smaller diameter tubes into the larger proximal one (Fig. 2). The longitudinal slit of each tube allowed them to be opened, the ulnar nerve and autografts slipped inside, and they then self-closed with a 25% overlap. PRP was prepared and injected, as previously reported.[7]

The final electrophysiological studies using needle electrodes and physical studies were performed 3.75 years post repair. This study was performed under a local IRB-approved protocol and in accordance with the World Medical Association Declaration of Helsinki (JBJS 79-A:1089-98,1997).

RESULTS

Nerve conduction studies found a small left ulnar motor evoked response (95% smaller than the uninvolved extremity) and an absent distal ulnar sensory response. Needle EMG studies revealed denervation potentials (fibrillations and positive sharp waves) in the left first dorsal interosseous and abductor digiti minimi muscles, with no voluntary recruitment. These findings are compatible with severe distal left ulnar nerve injury distal to the innervation of the flexor carpi ulnari and proximal to the deep ulnar branch, with evidence of axonal loss but conduction across the graft.

The recovered handgrip strength was 53 versus 75 pounds, and pinch strength 6 versus 12 pounds for the repaired versus intact hand, respectively. Based on the modified M0-M5 British Medical Research Council Score motor function scale, recovery was M4, which included complete range of motion, including good finger extension, flexion, and spreading, and elimination of fourth and fifth finger clawing (Fig. 1B).

The ring and little fingers developed normal topographically correct S4 sensitivity to heat, cold, pinprick, vibration, proprioception, 0.4-gm pressure (Semmes-Weinstein monofilaments), and 4-mm fingertip static two-point discrimination (Disk Criminator, Sensory Management Services LLC, Lutherville, Md.). By comparison, the little finger sensitivity was slightly diminished to all the sensory stimuli, with two-point discrimination more than 20 mm.

Before surgery, the subject had chronic neuropathic pain of 7 on a 0–10 linear scale. The pain began to decrease within 2 weeks of surgery, was zero within 2 months, and did not return during the following 3.75 years.

DISCUSSION

Stimulating the ulnar nerve at the wrist evoked small ADM and FDI motor responses, indicating axon continuity across the repaired motor nerve gap. Needle electromyography revealed denervation potentials (fibrillations and positive sharp waves) in the ADG and FDI muscles with no voluntary motor unit recruitment present. The small amplitude (95% asymmetry) responses compared with the right upper extremity response suggest axonal loss and incomplete restoration of motor function. No sensory response was obtainable, recording from the fifth digit suggesting sensory abnormalities due to minimal axon regeneration across the repaired sensory nerve gaps.

The physiological studies found sensitivity to all seven types of sensory stimuli, although the levels were less than the other reinnervated fingers. This recovery suggests few sensory axons regenerated across the repaired gap to the little finger. Nevertheless, the physiological studies found good restoration of motor (M4) and sensory (S4) functions.

The subject's recovery is consistent with two recent case studies using this technique.[6,7] Because autografts do not reliably induce good recovery of long gaps[2] or eliminate ulnar nerve injury-induced claw hand,[1] these results suggest that platelet-released factors induce recovery and eliminate finger clawing, effects that are limited by other techniques, especially across long nerve gaps.

Peripheral nerve trauma is associated with 50%–79% of patients developing chronic neuropathic pain.[3,8] Autografts may increase or slightly reduce chronic neuropathic pain but may not eliminate it.[4,9] While the autografts and the subject's relatively young age may have facilitated the recoveries and pain reduction, alternatively, the results are consistent with two recent case studies,[6,7] suggesting that PRP may underlie these changes, which can be tested by examining the efficacy of PRP alone.

CONCLUSIONS

Bridging three 10-cm-long nerve gaps with autografts within a PRP-filled collagen tube induced excellent sensory and motor function while eliminating ulnar hand clawing and chronic neuropathic pain. Autografts alone generally do not induce these effects. Further studies are required to determine whether PRP-released factors induced them.

Limitations

A limitation of this report is the lack of definitive evidence that PRP induced the outcomes.

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DISCLOSURE

The authors have no financial interest to declare in relation to the content of this article.

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