Nerve Decompression for Complex Regional Pain Syndrome Type II Following Upper Extremity Surgery

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Purpose: To evaluate the results of nerve decompression for the symptoms of complex regional pain syndrome that developed after upper-extremity surgery.

Methods: Eight patients (5 men, 3 women) developed worsening severe pain, swelling, and loss of range of motion after an upper-extremity surgery. The diagnosis of complex regional pain syndrome was made at an average of 6 weeks (range, 1–10 weeks) after the surgical procedure. A clinical diagnosis of either median or combined median and ulnar nerve compression at the wrist was confirmed in all patients with electrophysiologic testing. Nerve decompression was performed at a mean of 13 weeks after the procedure. Subjective (Disabilities of the Arm, Shoulder, and Hand questionnaire; visual analog pain scale) and objective (forearm, wrist, and finger range of motion; grip strength) data from before and after nerve decompression were reviewed.

Results: The average score on the Disabilities of the Arm, Shoulder, and Hand questionnaire decreased from 71 to 30 ($p < .05$). The mean visual analog pain score decreased from 7.5 to 1.8. ($p < .05$) There was immediate and near-complete resolution of all somatic complaints including hypersensitivity to touch, hyperhydrosis, swelling, and cold sensitivity. Range of motion and grip strength improved.

Conclusions: Traditionally surgical treatment has been avoided in patients with complex regional pain syndrome; however, in the setting of clinical and electrophysiologic evidence of nerve compression surgical intervention may hasten recovery in these patients. (J Hand Surg 2005;30A: 69–74. Copyright © 2005 by the American Society for Surgery of the Hand.)

Key words: Carpal tunnel syndrome, complex regional pain syndrome, nerve decompression, reflex sympathetic dystrophy.

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The development of severe pain, excessive swelling, and stiffness after upper-extremity surgery should alert the surgeon to possible development of chronic regional pain syndrome. Complex regional pain syndrome (CRPS)—previously known as algodystrophy, reflex sympathetic dystrophy (RSD), causalgia, Sudeck's atrophy, sympathalgia, shoulder-hand syndrome, posttraumatic pain syndrome, and neurodystrophy—was defined at the American Pain Meeting in 1993[1] as (1) a syndrome that develops after an initial noxious event; (2) spontaneous pain and/or allodynia/hyperalgesia not limited to the territory of a single peripheral nerve and disproportionate to the inciting event; (3) evidence of edema, skin blood flow abnormality, or abnormal sudomotor activity; and (4) a diagnosis that is excluded by the existence of other conditions that would account for the degree of pain and dysfunction. Complex regional pain syndrome is a clinical diagnosis and may be subdivided into 2 types based on etiology: type I CPRS is precipitated by a noxious event whereas type II CRPS is related to a peripheral nerve injury.[1]

Several investigations have evaluated the relationship between nerve compression and CRPS.[2–5] Monsivais et al[4] showed that 30 of 35 patients presenting with RSD had compression of 1 or more peripheral nerves. Fifteen patients had a single nerve compression and the remainder had compression of 2 or more nerves. Of these patients 70% had median nerve compression, 47% had ulnar nerve compression at the elbow, 37% had posterior interosseous nerve compression at the elbow, 6% had ulnar nerve compression at the wrist, and 3% had superficial radial nerve entrapment at the wrist.

Grundberg and Reagan[2] showed that 29 of 93 cases of RSD that were resistant to conventional treatments had clinical and electrophysiologic evidence of peripheral nerve compression. After peripheral nerve decompression (22 at carpal tunnel, 5 at cubital tunnel, 1 at Guyon's canal, 1 herniated disk) all patients reported an improvement in pain, swelling, range of motion, and strength. Jupiter et al[3] reported on 9 patients with causalgia who were treated with surgical decompression, nerve repair, continuous sympathetic block, rotational muscle flap, or a combination of these procedures. The nerve lesions involved the median nerve at the wrist in 5 patients, the ulnar nerve at the elbow and the median nerve at the wrist in 1 patient, and the ulnar nerve at the elbow, the radial digital nerve of the index finger, and the posterior tibial nerve near the ankle in 1 patient each. All patients received sympathetic blocks for 24 to 72 hours after surgery. Subjectively, pain decreased in all patients within 72 hours after surgery and all patients showed functional improvement.

Despite these reports the importance of a thorough neurologic examination to evaluate for nerve compression in the setting of CRPS remains underappreciated.[6] There remains a continued hesitancy to recommend surgical decompression for CRPS.[7] The purpose of this investigation is to illustrate that the development of CRPS after upper-extremity surgery may be due to underlying peripheral nerve compression and to evaluate the subjective and objective results of our patients with postsurgical type 2 CRPS who were treated with nerve decompression.

Materials and Methods

A retrospective chart review was performed to identify all patients who developed CRPS after upper-extremity surgery. From January 2002 until December 2003, 14 patients were diagnosed with CRPS that developed after upper-extremity surgery (Table 1). Our criteria for diagnosis were similar to those proposed in 1993 at the American Pain Meeting[1] and included the development of pain after a noxious event (ie, upper-extremity surgery); pain out of proportion to the surgery when compared with patients having similar procedures in the past, particularly pain that was burning or throbbing in nature; increased temperature of the affected extremity; edema out of proportion to the surgical procedure; allodynia or dysesthesias of the affected upper extremity; profound loss of range of motion of the wrist and hand; and hyperhydrosis.

All 14 patients were diagnosed with CRPS by the attending surgeon (R.H.G., M.I.B., C.A.G.) based on the above criteria. All patients had marked pain that was out of proportion to the expected postsurgical course, had marked edema and dysesthesias, and had a profound loss of finger motion. Increased temperature was noted in all patients; hyperhydrosis was seen in only 4 patients. The diagnosis of CRPS was made at an average of 6 weeks (range, 1–10 weeks) after the index procedure.

Eight of these patients were diagnosed with a peripheral nerve compression based on the surgeon's clinical examination. The clinical diagnosis of peripheral nerve compression in the setting of CRPS was made at an average of 12 weeks after the index surgery and 6 weeks after the diagnosis of CRPS. This diagnosis of nerve compression was based on the identification of sensory and/or motor changes