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

Table 1. Summary of Patient Clinical Data

	Affected Side	Dominant Side	Age (y)	Gender	Index Procedure	Time to Diagnosis of CRPS	Nerve Compressed (m/s)	Time to Decompression From Index Procedure (wk)
1	L	R	67	M	Scapholunate ligament repair	8 wk	Median DML: 4.5 DSL: 3.7	12
2	R	R	64	F	ORIF distal humerus fracture	6 wk	Median DML: 4.3 DSL: 4.5	10
3	R	R	58	F	Dorsal ORIF distal radius	8 wk	Median DML: 3.7	16
4	R	R	59	M	Flexor digitorum superficialis opponensplasty	8 d	Ulnar Motor latency L: 4.2, R: 9.9 F wave latency L: 24.4, R: 28.4 Median DML: 6.9	20
5	L	R	46	M	Dorsal ORIF distal radius	4 wk	Median DML: 4.0 DSL: 2.7	13
6	L	R	55	M	Dupuytren's–partial selective fasciectomy	2 wk	Median DML: 4.3 DSL: 2.8 Ulnar DSL: 1.9	11
7	R	R	68	M	Volar and dorsal ORIF distal radius	10 wk	Median DML: 5.9 DSL: nd	14
8	R	R	68	F	Volar ORIF distal radius	6 wk	Median DML: 3.9 DSL: 3.1 EMG: amplitude loss and denervation potentials	10

EMG, electromyograph; ORIF, open reduction internal fixation.

together with positive provocative signs. In all 8 patients the nerve compression was confirmed with electrophysiologic testing (Table 1). All patients had slowing of either sensory or motor nerve conduction (distal motor latency [DML] $>$ 4.0 m/s, distal sensory latency [DSL] $>$ 3.2 m/s). Despite the presence of allodynia all patients were able to complete the electrophysiologic testing.

Nerve decompression[8] was performed at an average of 13 weeks after the index procedure (Table 1). Nerve decompression was performed under intravenous regional (5 patients), general (1 patient), or axillary block (2 patients) anesthesia. No patient was treated with endoscopic or mini open carpal tunnel release. At the time of decompression all nerves had hyperemia and mild synovitis. These changes were similar to the findings noted during routine carpal tunnel releases. No abnormal fibrosis was identified.

Subjective and objective data were obtained before surgery, after surgery, and at the final follow-up examination. Immediate postsurgical data were obtained an average of 8 days after surgery and final follow-up data were obtained an average of 60 weeks (range, 36–90 weeks) after surgery. Subjective data included the Disabilities of the Arm, Shoulder, and Hand (DASH) questionnaire, a 30-question test that has proven valid, reliable, and responsive for disorders of the upper extremity.[9] A visual analog pain scale was used to compare presurgical and postsurgical pain levels.[10] Six patients did not have a complete presurgical subjective analysis available at the time of final follow-up evaluation; in these the pain

and DASH assessments were obtained retrospectively (average, 24 weeks).

The same occupational hand therapist performed the range-of-motion and strength assessments[11] of the affected upper extremities. Wrist flexion and extension were measured with a goniometer along the ulnar border of the forearm and hand. Finger range of motion was assessed by measuring in centimeters the distance from the distal palmar crease to the fingertip.

Data were analyzed using a repeated-measures analysis of variance and statistical software (SPSS version 11.5; SPSS, Inc, Chicago, IL). Significance was set at an alpha level of .05.

Results

Presurgical, postsurgical, and follow-up data are provided and are listed with their respective standard error and 95% confidence intervals (standard error, upper-limit 95% confidence interval/lower-limit 95% confidence interval). Pain improved immediately with the pain score decreasing from 7.5 (1.2, 4.7/10.4) before surgery to 3.5 (1.0, 1.2/5.9) immediately after surgery ($p < .05$). The pain score continued to decrease to a level of 1.8 (0.9, −0.3/3.9) at follow-up evaluation. Similarly, function was improved immediately after surgery; the DASH score decreased from 71 (7.3, 53.2/87.7) before surgery to 53 (6.4, 38.7/69.1) after surgery and continued to decrease to 30 (7.5, 12.4/48.1) at follow-up evaluation ($p < .05$).

Wrist range of motion and grip strength and finger range of motion (distal palmar crease measure) all were improved immediately after surgery and continued to show improvement at follow-up evaluation. Because of our small sample size, however, these clinical improvements were not statistically significant. Wrist flexion improved from 17° (3.4°, 1.2°/22.8°) before surgery to 28° (5.2°, 12.3°/45.2°) after surgery to 45° (4.1°, 23.7°/49.9°) at follow-up evaluation. Similarly, wrist extension improved from 26° (8.9°, −4.8°/52.3°) before surgery to 36° (10.1°, 3.5°/68.0°) after surgery to 50° (5.3°, 22.4°/56.1°) at follow-up evaluation. Grip strength improved from 5.4 kg (3.5 kg, 5.8/16.7 kg) before surgery to 9 kg (4.1 kg, −4.2/22.1 kg) after surgery to 20.4 kg (8.8 kg, −7.7/48.7 kg) at follow-up evaluation. Finger range of motion increased notably: the distal palmar crease improved from 65 mm before surgery to 37 mm after surgery to 13 mm at follow-up evaluation. All patients reported immediate relief of somatic complaints including swelling, hypoesthesias, hyperhydrosis, and cold extremity at their first visit after surgery.

Case 1

A 67-year-old man (Table 1, patient 1) developed diffuse swelling and pain 8 weeks after scapholunate ligament repair. A positive Tinel's sign was present over the median nerve at the wrist although 2-point discrimination remained normal. Electrodiagnostic studies at the wrist were normal; a diagnosis of CRPS type I was made. One month later, however, the patient noted worsening pain and tingling in the thumb and index and middle fingers. Repeat electrodiagnostic studies showed objective signs of median nerve compression (Table 1). The diagnosis was changed to type II CRPS and median nerve decompression was performed. The DASH scores improved from 32 before surgery to 25 immediately after surgery to 4 at final follow-up evaluation.

Case 2

This patient (Table 1, patient 4) noted severe hand pain and swelling 8 days after a flexor digitorum superficialis opposition transfer. The operating surgeon believed that the level of pain was out of proportion to the surgery. The patient was diagnosed with CRPS and was treated with multiple stellate ganglion blocks but had minimal relief. Two weeks after the surgery the patient complained of numbness in the ring and little fingers. No other diagnostic tests or intervention were performed. The patient was referred to our institution 18 weeks after the index procedure. There was weakness and atrophy of ulnar and median innervated hand muscles, a Tinel's sign was present over the median and ulnar nerves at the wrist, and the patient's 2-point discrimination was increased to 7 mm in the ring finger and 10 mm in the small finger. Electrodiagnostic testing showed compression of both the ulnar and median nerves at the wrist (Table 1). Twenty weeks after the opposition transfer the patient was treated with median and ulnar nerve decompression at the wrist and the flexor digitorum superficialis transfer was found to be compressing directly the ulnar nerve. Recovery was not complete and improvement took longer than with some patients; the DASH scores improved from 47 before surgery to 42 immediately after surgery to 18 at final follow-up evaluation. The patient's chief complaint limiting his function was continued range of motion loss despite extensive therapy.

Discussion

There have been multiple taxonomy changes for CRPS; however, the difference between type I and type II CRPS has remained constant: type I is initiated by a noxious event and type II is related to peripheral nerve pathology. The successful treatment of CRPS is based on its timely diagnosis. The treatment for type I CRPS is nonsurgical; additionally, the patient is managed rarely by the hand surgeon. In patients with type II CRPS, who have a defined peripheral nerve dysfunction, intervention must address the underlying nerve abnormality. The first difficulty in the treatment of type II CRPS is its accurate diagnosis. In a case series Thimineur and Saberski[12] describe 3 patients with type II CRPS that was misdiagnosed as type I CRPS. The researchers note, "The clinical entity of CRPS quite apparently encompasses symptomatology caused by peripheral nerve entrapment, irritative lesions, and neuroma. As such, its use as a diagnostic end point may overlook these treatable conditions." The 2 patients discussed specifically in the results section illustrate the difficulty in distinguishing types I and II CRPS. In our patient 4 there was a clear anatomic explanation for the patient's severe pain: nerve compression. Nonetheless the severity of the pain led to a diagnosis of CRPS and the initiation of a nonspecific treatment path appropriate for type I CRPS. Delayed nerve decompression provided good pain relief but the patient had little improvement in his finger joint contractures. These findings show the importance of evaluating patients with CRPS for signs of nerve compression.

The case of patient 1 further shows the diagnostic difficulty with type II CRPS in the perioperative period. Type II CRPS was suspected but because the initial electrophysiologic study results were normal a diagnosis of type I CRPS was made and treatment was begun. When the subsequent study results were positive the diagnosis of carpal tunnel syndrome was made and the nerve decompression was performed (albeit in a delayed fashion). We believe immediate evaluation for peripheral nerve compression should be sought in all postsurgical cases of CRPS. Clinical examination provides the most important data in making an accurate diagnosis. Electrodiagnostic testing is also helpful; however, we have become more dependent on our clinical findings in making an accurate diagnosis because electrical change on nerve studies may be delayed.

The first clinical sign alerting us to the possible development of CRPS in all patients was severe swelling that was greater and persisted for a prolonged period after the index procedure. The first symptom alerting us to the early development of CRPS was pain out of proportion to the surgical procedure. The combination of excessive postsurgical pain and swelling even in the absence of positive neurologic findings should alert the clinician to the possibility of nerve compression.

If the first challenge for the clinician is to diagnose correctly type II CRPS the next challenge is implementing the most efficacious treatment. Although the coexistence of peripheral nerve compression and CRPS has been noted in several previous reports[2–5,13–17] surgical nerve decompression has been recommended only rarely.[2–5,12] In a recent review describing evidence-based treatment options for CRPS only 1 of the 90 articles cited addresses surgical decompression for CRPS type II; the cited article highlights a brief report of 3 cases.[6,12] Historically there has been hesitation to recommend surgery of any kind in the setting of CRPS.

There have been several previous reports of nerve decompression in the setting of CRPS. Grundberg and Reagan[2] treated 29 cases of recalcitrant CRPS (those patients with a delayed diagnosis of type II CRPS) with nerve decompression and reported good outcomes. Similarly, Jupiter et al[3] reported satisfactory outcomes in 9 patients treated for type II CRPS with nerve decompression. Our investigation confirms that surgical decompression is very effective in relieving the pain and other somatic complaints associated with CRPS. The results of Jupiter et al were similar to ours in several respects. First, both studies report nearly immediate decreases in the severe pain after decompression. These findings are shown most impressively with our DASH and visual analog scale findings. In contrast to Jupiter's treatment we did not use an indwelling stellate ganglion block after nerve decompression. Based on our findings, as documented by the rapid improvement in visual analog pain scores and DASH scores, we do not believe that the stellate ganglion block is necessary for rapid symptomatic improvement. The key to the rapid improvement in the symptoms of type II CRPS is nerve decompression.

Second, despite improvements shown by the significant decrease in the DASH scores both groups of patients had residual functional limitations. We believe that is due to the delay in nerve decompression in both groups: 13 weeks in our patients and 17 weeks in Jupiter's report.[3] An increased awareness of

the relationship between CRPS and nerve compression may allow more rapid referral of these patients for further evaluation. Early identification of patients with type II CRPS will allow earlier surgical intervention and potentially improved outcomes.

There are several limitations in the evaluation of these data. The small sample size limits the statistical power of these data. We believe that the rarity of this syndrome and the confusion surrounding its accurate diagnosis make these numbers acceptable. Furthermore, distinguishing the diagnoses of types I and II CRPS is difficult and thus patients with a misdiagnosis of type I CRPS instead of type II CRPS may have gone undetected. We obtain currently electrophysiologic testing in all postsurgical patients with CRPS to identify patients with type II CRPS. This investigation used retrospectively obtained DASH scores; this may be less ideal than DASH scores obtained before surgery. Although no specific data exist regarding the validity of retrospectively obtained DASH forms, however, other health-related outcome studies have found that retrospective measures may be more sensitive to change and correlated more strongly with patient satisfaction.[18] Furthermore, reasonably high agreement has been shown between prospective and retrospective evaluation of function over a 6-month period.[19] Finally, this investigation addresses only patients with type II CRPS who were evaluated and treated after upper-extremity surgery; these results may not be applicable to other patients.

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