

Florid Suprascapular Neuropathy after Primary Rotator Cuff Repair Attributed to Suprascapular Notch Constriction in the Setting of Double Crush Syndrome

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Abstract

This report describes a patient who had an open repair of a small supraspinatus tendon tear performed 6 months after an arthroscopic acromioplasty with debridement had failed to provide pain relief. Three months prior to the tendon repair, he had a two-level cervical spine discectomy and fusion (C4–5, C5–6) that improved his neck pain. Florid suprascapular neuropathy was detected 10 weeks after the open rotator cuff repair. Evidence of some nerve recovery resulted in a long period of observation. But unsatisfactory improvement warranted decompression of the suprascapular notch, which was found to be very stenotic. At surgery, there was no evidence of neuroma, cyst, or other compressing lesion or tissue. Therefore, it was ultimately hypothesized that there was an exacerbation of a preexisting, but clinically unrecognized, entrapment of the suprascapular nerve in the suprascapular notch in the setting of cervical radiculopathy (primarily C5). Retrospectively it was also concluded that had this compressive etiology been recognized, it would have favored prompt decompression rather than the long observation period. Three years was required to achieve a good result following suprascapular notch decompression. The underlying C5 radiculopathy may have created a “double crush syndrome” that contributed to the propensity for injury and the prolonged recovery. There should be heightened awareness of this problem in patients who do not have satisfactory improvement in shoulder pain from previous shoulder and neck surgery.

Keywords

- ▶ suprascapular neuropathy
- ▶ rotator cuff tear
- ▶ shoulder pain
- ▶ suprascapular nerve
- ▶ supraspinatus
- ▶ double hit phenomenon
- ▶ double crush syndrome

Background

Several studies have shown that suprascapular neuropathy can occur in a significant percentage of patients with large-to-massive rotator cuff tears,^{1–5} but is a rare complication following primary rotator cuff repairs even when tears are massive.^{6,7} In a study of 26 patients with massive cuff tears who were evaluated with preoperative electrodiagnostic studies, 7 (38%) had isolated suprascapular injury.³ In six of

these patients, the neuropathy improved after repair (the seventh was irreparable). Costouros et al³ speculated that improvement occurred because the repair untethered the suprascapular nerve (SSN) from being compressed at the base of the scapular spine (→Fig. 1). When improvement does not occur, or when postoperative neuropathy occurs but is not detected preoperatively (as in this report), the differential diagnosis is broadened to include (1) direct

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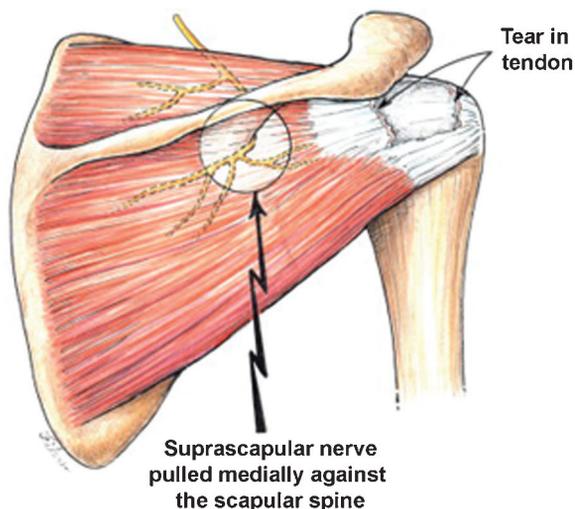


Fig. 1 Medial retraction of the suprascapular nerve (SSN) caused by a large-to-massive rotator cuff tear. Illustration of how the SSN can experience traction by being pulled medially at the base of the scapular spine following medial and inferior retraction of a large-to-massive tear. In some cases, repairing these tears can un-tether the nerve by mobilizing it laterally. Not only has this explanation been contested,⁸ but it is also not applicable in our patient because his cuff tear was small and significant suprascapular neuropathy occurred after the cuff repair. Reproduced from Costouros et al³ with permission of Elsevier Limited.

iatrogenic injury, (2) cervical radiculopathy, (3) brachial plexitis, (4) compression by an extrinsic mass such as a hematoma or ganglion cyst not detected preoperatively, and (5) indirect iatrogenic injury secondary to cuff-repair-related traction of the SSN that was constricted in the suprascapular notch.⁹ This latter possibility appears to have been an important factor in our unusual case, where repair of the rotator cuff exacerbated a preexisting, but preoperatively undetected, SSN dysfunction.

Case Presentation

A healthy 62-year-old (height: 1.83 m; weight: 98.4 kg; body mass index: 22 kg/m²) right-hand-dominant man, who was an accountant and avid golfer, was referred to our clinic with a chief complaint of left shoulder pain. There was no history of shoulder or neck trauma. Thirteen years ago, an arthroscopic acromioplasty was performed on his left shoulder, and 6 months prior to this first clinic visit a revision arthroscopic acromioplasty with cuff tendon debridement was performed for recurrent pain, but did not help. His diagnosis had been stage-two subacromial impingement syndrome.¹⁰ The pain was achy and occasionally sharp and was distributed over the anterior and superior-posterior aspects of the shoulder. There was a mildly positive left-side Spurling sign, but there was no winging of the scapula, and liftoff and glenohumeral instability tests were negative. Active range of motion was 150-degree flexion and 135-degree abduction, and manual muscle tests showed normal strength. Pain was moderate with impingement (Neer) and impingement reinforcement (Hawkins-Kennedy) maneuvers. His initial American Society of Shoulder and Elbow Surgeons (ASES) score

was 64 (best = 100) and other measures of general health and shoulder function are listed in ►Table 1.¹¹⁻¹⁶

Images from a magnetic resonance (MR) scan obtained 7 months prior are shown in ►Fig. 2A, B. An MR scan performed 6 weeks prior to his first visit showed a small tear of the supraspinatus tendon with mild muscle atrophy (►Fig. 2C-E). Both scans showed mild fatty degeneration of the infraspinatus (►Fig. 2B, C). A subsequent neck MR scan showed disk bulges/herniations at C2-3, C3-4, C4-5, and C5-6. A neurosurgeon then performed two-level disk fusions (C4-6), which relieved the neck pain but only mildly reduced the shoulder pain. Three months later (December 2006), the senior author (J. G. S.) performed arthroscopic debridement of anterior labral fraying and open repair of a small supraspinatus tear. The tear was retracted 15 mm and was easily mobilized without requiring tissue release beyond the glenoid labrum. Two suture anchors were used to affect a quasi-double-row repair.¹⁹ Active motion was not allowed for 8 weeks.

At 10 weeks postsurgery, there was substantial left shoulder weakness with attempts at active motion. Although the deltoid contracted well, there was (1) atrophy in the infraspinatus muscle, and strength of 3/5 in external rotation and 3 -/5 in elevation in the plane of the scapula, as well as (2) mildly positive left-side Spurling sign. The differential diagnosis included iatrogenic injury, extrinsic compression (e.g., hematoma, cyst, etc.), brachial plexopathy, and/or concomitant cervical nerve-root compression.^{20,21} A nerve conduction study and electromyography (NCS/EMG) showed findings consistent with SSN injury at the level of the suprascapular notch with absence of evidence of re-innervation. MR scans obtained of the left brachial plexus and shoulder showed humeral head elevation suggesting weakness of the superior rotator cuff (►Fig. 3) but no evidence of (1) a space-occupying lesion along the SSN, (2) injury of the brachial plexus and its nerve branches,²²⁻²⁴ or (3) unusual morphology of the suprascapular notch or transverse scapular ligament (retrospective examination showed that these scans were not sufficient to detect an anomaly of the notch).

At 105 days after shoulder surgery, the patient saw his neurosurgeon for a 7-month follow-up and the cervical fusion had healed. At 138 days after shoulder surgery, there was increased burning and aching that radiated across the posterior and lateral aspects of the shoulder and upper arm. Voluntary infraspinatus contractions were now more obvious. Active shoulder flexion and abduction were < 80 degrees each. The changing pain intensity and characteristics were presumed to reflect SSN recovery for which Lyrica (pregabalin) 75 mg twice/day was prescribed. At 162 days after surgery, the neuropathic pain was significantly decreased and usually did not require medication; however, there was no improvement in strength in flexion or external rotation.

At 213 days after cuff repair, results of another NCS/EMG revealed findings that could not be explained by an isolated suprascapular neuropathy: (1) denervation potentials in deltoid, rhomboids, and C5 cervical paraspinal muscles, as well as in the supraspinatus, suggesting C5 radiculopathy; (2) small amplitude fibrillation potentials in the abnormal

Table 1 The patient's shoulder function and general health

Six-year outcome data												
	First clinic visit	4 mo after RCR	SSN release	Months after SSN release								
				3	6	12	18	22	34	39	45	51
Active forward flexion	150°	70°		90°	105°	107°	105°	100°	120°	130°	130°	130°
10 cm VAS score on typical day (best equals 0)	2.3	-		-	3.9	2.9	3.3	4.4	1.7	1.2	1.5	2.7
ASES score (best equals 0)	64	-		-	51	59	54	45	73	-	85	77
WORC score (best equals 0 (100%))	-			1,118 (47%)	-	1,222 (42%)	926 (51%)	1,497 (29%)	672 (68%)	634 (70%)	564 (73%)	418 (80%)
Simple shoulder ^a (No. of yes responses)	-	-		8/12	-	7/12	10/12	5/12	10/12	10/12	12/12	12/12
DASH score (best equals 0)	-	-		32.5	-	42.5	31.0	62.1	26.7	15.0	15.8	12.9
Short Form-36 ^b												
Physical functioning	-	-		50	-	15	65	60	70	70	70	90
Role limitation due to physical health	-	-		0	-	0	0	0	0	25	25	100
Role limitation due to emotional problem	-	-		0	-	0	0	0	33.3	100	100	100
Energy/Fatigue	-	-		60	-	20	40	40	45	50	60	65
Emotional well-being	-	-		44	-	56	84	56	72	80	92	88
Social functioning	-	-		62.5	-	62.5	50	50	75	100	100	100
Pain	-	-		67.5	-	22.5	55	22.5	55	77.5	67.5	80
General health	-	-		45	-	30	85	75	65	70	62.5	75

Abbreviations: ASES, American Society of Shoulder and Elbow Surgeons; DASH, disabilities of arm, shoulder, and hand; deg., degrees; RCR, rotator cuff repair; SSN, suprascapular nerve; VAS, visual analog scale; WORC, Western Ontario rotator cuff.

^aNumber of yes responses/number of questions ("yes" responses correlate with better function than "no" responses).

^bAll questions are scored from 0 to 100 representing the highest level of function possible. Aggregate scores are compiled as a percentage of the total points, using the RAND scoring table.

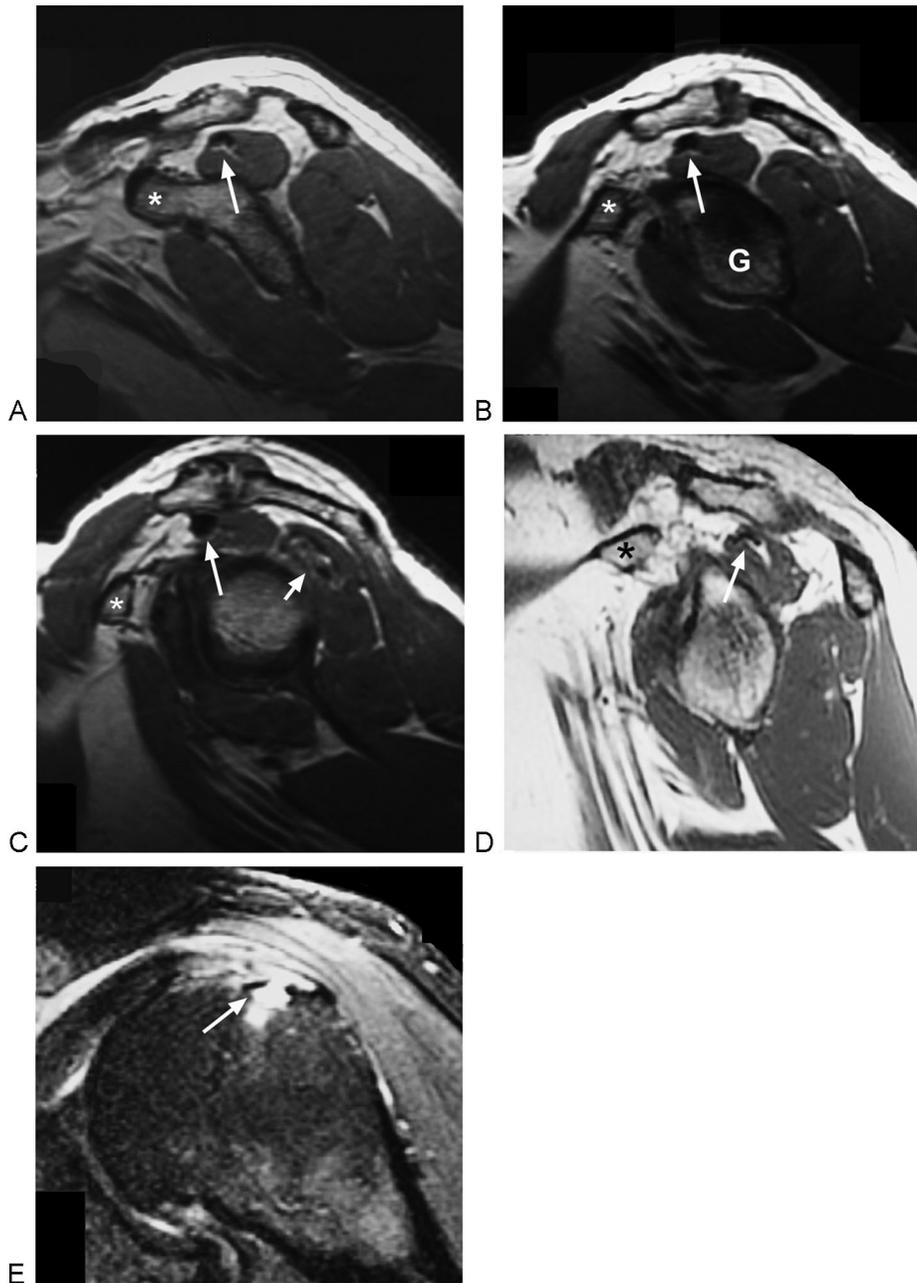


Fig. 2 MR images prior to rotator cuff repair. MR images 16 (A–C) and 10 (D, E) months prior to cuff repair; asterisk indicates the coracoid. (A, B) Evidence of scarring in the supraspinatus (white arrows) 16 months preoperation. (G = glenoid). (C) Mild fatty degeneration (short arrow)^{17,18} of the infraspinatus. Scarring in the supraspinatus (longer arrow). (D) Scar tissue is seen in (A) and (B) (arrow), and mild fatty degeneration^{17,18} of the supraspinatus is seen 10 months preoperation. (E) Small supraspinatus tear (arrow).

muscles, and complex repetitive discharges in the deltoid indicating chronic neuropathy; and (3) evidence of re-innervation in the abnormal muscles. There was also no evidence of conduction block along the suprascapular and axillary nerves. A diagnostic/therapeutic suprascapular notch injection with local anesthetic and a corticosteroid was recommended. However, the patient’s pain (but not strength) improved, causing him to cancel the injection. In view of evidence of early nerve recovery, and in the perspective that intraoperative trauma had somehow occurred to the SSN, a consulting surgeon specializing in peripheral neuropathies

recommended to continue nonoperative management. This recommendation reflected the general conclusions of Antoniou et al²⁵ who, in their analysis of the functional outcome of 23 patients, suggested that traumatic lesions of the SSN can have equivalent responses to operative and nonoperative treatment. (But, as discussed later, it was ultimately concluded that prompt scapular notch decompression would have been best for our patient.)

By 322 days after surgery, the pain recurred with greater intensity. A suprascapular notch injection with 0.25% bupivacaine was done using computed tomography (CT) guidance

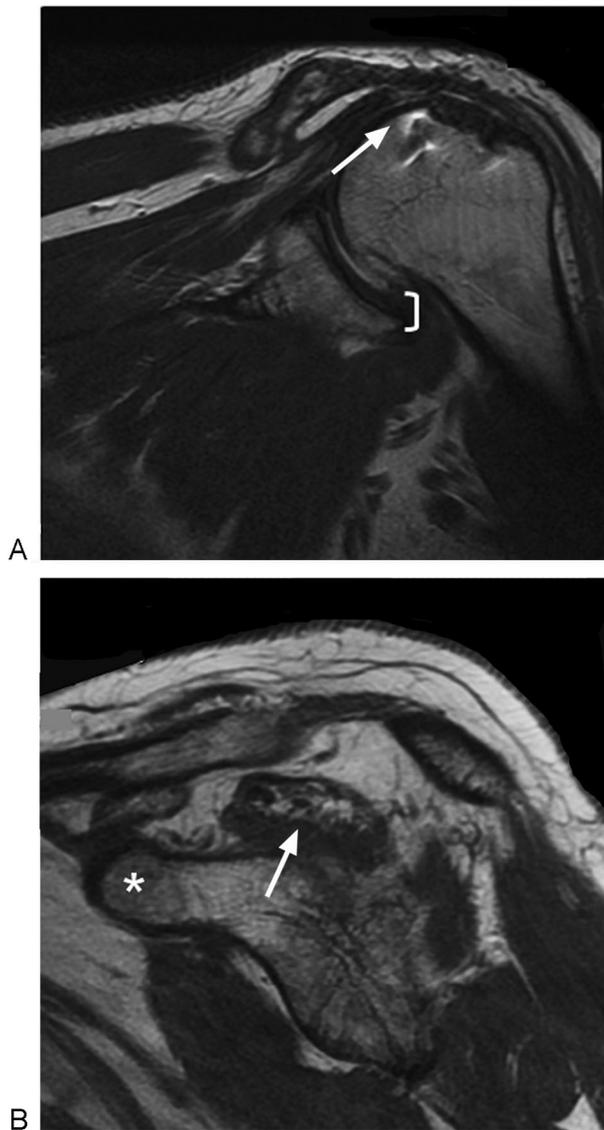


Fig. 3 MR images 4 months after rotator cuff repair. (A) Elevation of the humeral head (white bracket) and signal changes, and a possible defect in the supraspinatus insertion (arrow). (B) Moderate supraspinatus fatty degeneration (arrow). Asterisk indicates the coracoid.

and this yielded excellent, but temporary, improvement. An open surgical decompression of the suprascapular notch was performed (nearly 1 year after the cuff repair, ▶ **Table 1**) by the same consulting surgeon. The nerve was grossly compressed beneath a hypertrophic ligament in a narrowed suprascapular notch (▶ **Fig. 4**)²⁶ and it dramatically protruded upward after ligament transection. The first motor branch to the supraspinatus was distal to the notch²⁸ and the supraspinatus motor branches were intact. There was no evidence of neuroma or other gross neural, perineural, or vascular pathology, variation, or anomaly, and no compression caused by the deep fascia proximal or distal to the suprascapular notch or by the tissues forming the spinoglenoid notch.^{26,29} Consequently, further surgical exploration^{26,30} was not necessary. The transverse scapular ligament was excised and the notch was enlarged.^{25,31} During the following

6 months, the burning pain improved significantly but the weakness persisted.

At 22 months after notch decompression, the pain and function had worsened. A second postoperative MR scan showed increased fatty degeneration of the supraspinatus and infraspinatus (▶ **Fig. 5**). By contrast, substantial overall improvement occurred at 27 months—the pain had almost completely subsided and strength significantly improved. In fact, he was able to remodel his home (manual labor for several months) without significant discomfort. Active forward flexion was 120 degrees, but early fatigue with overhead motion was reported. At final follow-up at 4.5 years after notch decompression, he had reached a plateau in motion and strength (4 + /5) and the pain was minimal/tolerable except when golfing. An axillary lateral radiograph showed evidence of glenohumeral arthritis, which likely contributed to this activity related pain.

Conclusion

Among the various potential factors contributing to our patient's shoulder dysfunction, what is clear is that the SSN was constricted in a narrowed suprascapular notch. Because there are no data suggesting that a narrowed notch and hypertrophic transverse scapular ligament result from indirect iatrogenic injury during routine open repair of a small cuff tear,³² we conclude that the SSN was compressed in the notch prior to the repair. What likely occurred was that first the preexisting narrowed notch and hypertrophied superior transverse ligament made the nerve prone to injury even after typically inconsequential minor perturbation (e.g., nerve and other tissue motion during the rotator cuff repair).²⁸ Second, a vicious cycle ensued that produced focal swelling that interfered with axonal transport and caused vascular compromise, ultimately leading to more pronounced edema along the nerve with resulting distal muscle dysfunction. The anatomical bottleneck then became even more constrictive pronouncing the pathology, which in our patient's case resulted in a functionally (but not morphologically) complete lesion. While timely decompression of such a lesion can frequently produce good results, the delay in our patient's surgical decompression likely contributed to the prolonged delay in achieving a good result.^{25,33}

A significant amount of our patient's preoperative pain (i.e., before the cuff repair) was likely sensory dysfunction^{34,35} from notch constriction—consistent with descriptions of “tunnel syndrome of the SSN” or “carpal tunnel syndrome of the shoulder.”^{26,36} Suprascapular neuropathy has been postulated to occur from excess motion of the scapula because the nerve is tethered to at least two, and often three, anatomic locations: the suprascapular notch, the spinoglenoid notch, and Erb point in the neck.^{27,37,38} However, observations by Rengachary et al^{27,38} in fresh cadavers revealed no obvious movement of the SSN across the suprascapular notch with extreme shoulder motion, reflecting the fact that the neurovascular pedicle is fixed to the periosteum in the notch and to the supraspinous fossa. They speculated that SSN injury more likely results from kinking against the ligament (the “sling effect”) because of its close apposition to

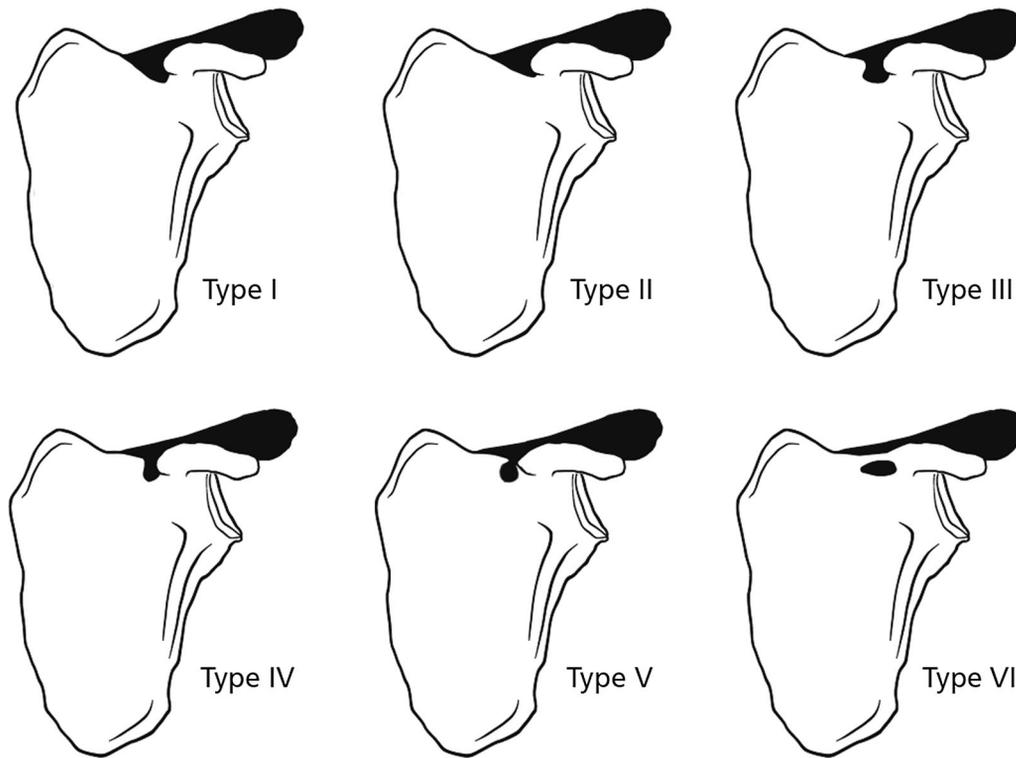


Fig. 4 Types of suprascapular notch morphologies. These original drawings are based on the photographs in Rengachary et al.²⁷ Type V (6% incidence) was found in our patient at the time of notch decompression.

its sharp inferior border with shoulder depression, retraction, and hyperabduction. This “sling effect” might become even more pronounced when the SSN is also constricted within a stenotic notch like that in our patient.

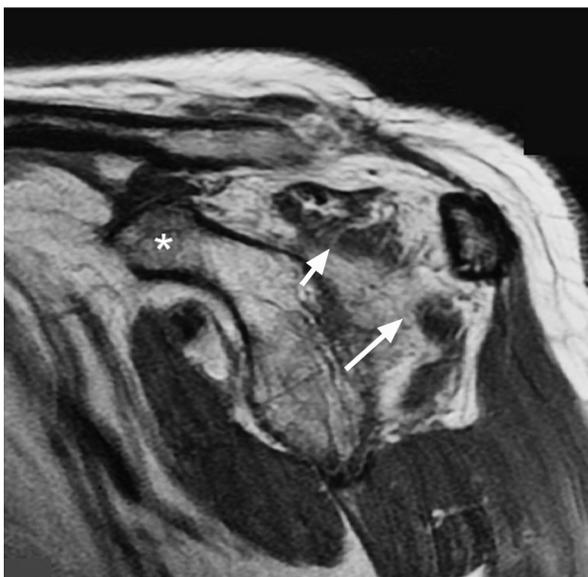


Fig. 5 MR image obtained 34 months after rotator cuff repair and 22 months after suprascapular notch decompression showed worsening of fatty degeneration of the supraspinatus (short arrow) and infraspinatus (long arrow). The coracoid is indicated with an asterisk.

Although a peripheral nerve can tolerate approximately 10% increase in its resting length before neurapraxia occurs,³⁹ altered conduction can occur when a nerve is stretched beyond 6% of its resting length.⁴⁰ Scarring of the neurovascular pedicle associated with some cuff tears can reduce the distance that the nerve can be stretched without injury.⁹ Support for the hypothesis that this is what happened in our patient includes: (1) his avid recreational activities (golf) causing repetitive trauma, potentially increasing constriction in his stenotic suprascapular notch,^{33,41,42} and (2) the scar-like tissue in the “intramuscular tendinous core” of the anterior supraspinatus⁴³ (► **Fig. 2**) can reduce the ability to safely mobilize the tendon during repair.^{44–46} These factors may have contributed to SSN injury in our patient even though tendon mobilization was within the safe range in normal cases.²⁸ The C5 axonal pathology is also potentially important because the SSN contains fibers primarily from C5 and C6. If the C5 radiculopathy caused impairment of SSN axonal transport, then it might have been more prone to neuropathy from peripheral constriction. This phenomenon is sometimes referred to as a “double crush syndrome” or the “double hit phenomenon.”^{4,47–49}

Because there were periods of improvement during his early postoperative course, a prolonged period of recovery was allowed. This opinion was based on the presumption that the SSN sustained intraoperative trauma and hence this opinion was consistent with the general conclusions of Antoniou et al.²⁵ The SSN lesions in their 23 patients that were caused by trauma (including traction and direction closed injuries) showed no statistical difference in the response to operative ($n = 12$) and nonoperative ($n = 11$) treatment. But the p -value for this

difference was not provided, leaving the reader with the impression that, based on the subsample means provided, the sample sizes were insufficient to discern clinically and statistically significant differences when all of these issues are considered: (1) trauma mechanism, (2) depth of nerve injury, (3) lesion level, (4) intraoperative electrophysiological evaluation result, and (5) microsurgical repair method chosen, which can have a significant effect on results obtainable (e.g., external neurolysis and/or nerve mobilization, vs. graft repair vs. end-to-end repair after neuroma excision). However, Antoniou et al²⁵ *did* show that, in contrast to “traumatic lesions,” “compressive lesions” attributable to suprascapular notch entrapment (which our patient had) had the best outcome with surgical decompression. Therefore, prompt scapular notch decompression would have been the best choice for our patient. This is supported by additional reports showing that when transverse scapular ligament release is done relatively soon after the diagnosis is made that milder muscle atrophy is more likely to subside along with shoulder pain.⁵⁰ But when ligament release was delayed and there was advanced atrophy, the pain improved but not the atrophy.

Recent studies show that a large percentage of massive rotator cuff tears with significant muscle atrophy can have significant clinical improvement after surgical repair without SSN decompression.⁵¹ This is one reason why current guidelines for the preoperative workup of most rotator cuff tears do not include electrodiagnostic testing.^{4,5,8,9,32} But when functional deficits that do not resolve, and there are signs of a possible nerve lesion, easy and cost-effective electrodiagnostic studies as well as nerve imaging should be included early in the differential diagnostic workup. SSN impairment can be diagnosed quite well electrophysiologically by NCS/EMG, and well differentiated from C5 injury. In conjunction with MR imaging of supraspinatus/infraspinatus muscle wasting and high signal due to denervation edema, a concomitant nerve lesion can be readily diagnosed. If this is the case, then exploration and macro-/microsurgical decompression or nerve repair should follow promptly. In less clear cases, neuroimaging in the form of MR neurography of the notch area (thin slice, 1 mm, in high resolution) or neurosonography, or both, can be used to enhance the ability to define the lesion.^{52,53} This includes the condition of the muscles (including wasting and fatty degeneration) and the nerve can be traced from its takeoff from the upper trunk to the notch area and beyond to reveal transection, neuroma, constriction, or edema.

In summary, our patient had an unusual postoperative course because of the exacerbation of preexisting entrapment of the SSN in the suprascapular notch. Three years was required to achieve a good result following suprascapular notch decompression, which was done nearly 1 year after the diagnosis was clearly established. There should be heightened awareness of this problem in patients who do not have satisfactory improvement in shoulder pain from previous shoulder and neck surgery, and this should prompt more immediate surgical decompression in some cases. The underlying C5 radiculopathy that our patient had may have created a “double crush syndrome” that contributed to the propensity for injury and prolonged recovery.

Consent

Written informed consent was obtained from the patient for written or electronic publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

J. G. S.: Largely contributed to the literature review and writing of the case report as well as performed or directly participated in all aspects of the clinical management of the case.

C. J. K.: Contributed to the literature review and writing of the case report.

B. B. H.: Contributed to the literature review and writing of the case report.

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