Suprascapular nerve entrapment

A summary of seven cases

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The suprascapular nerve, formed from the upper trunk of the brachial plexus, can be entrapped at the suprascapular notch and result in significant patient morbidity. Seven patients with suprascapular nerve palsy are presented, and their evaluation, treatment, and outcome over a mean follow-up period of 24 months are described. Six of these patients were treated surgically and one medically; all experienced good results. In a review of the relevant literature, this entity is distinguished from other causes of shoulder pain, the typical presenting signs and symptoms are outlined, and the appropriate management of these patients is addressed.

KEY WORDS • shoulder pain • suprascapular nerve • entrapment neuropathy

PAIN and discomfort in the shoulder are a frequent cause of patients seeking medical attention. While suprascapular nerve entrapment is a relatively uncommon etiology of shoulder discomfort, awareness of its clinical presentation (referred shoulder pain with associated weakness and atrophy of the spinatus musculature) can lead to a correct early diagnosis. The common differential diagnoses are primarily orthopedic, and include rotator cuff injuries, supraspinatus syndrome, rupture of the long head of the biceps, bicipital tenosynovitis, arthritis (either traumatic or rheumatoid), infraspinatus tendonitis, calcific abscess, and adhesive capsulitis. A significant number of these patients will have been seen by orthopedic surgeons prior to seeking neurosurgical evaluation. Shoulder pain can also be the presenting symptom in cervical radiculopathy, primarily of the C-5 root. A detailed history and physical examination aided by electromyography (EMG) and nerve conduction velocity (NCV) studies should result in the correct diagnosis.

We present our experience with seven patients with suprascapular nerve palsy. The treatment of this entity, including both medical and surgical approaches, is discussed.

Anatomical Considerations

The suprascapular nerve has a long course and originates from the upper brachial plexus where the C-5 and C-6 roots join at Erb's point. In up to 50% of cases, the suprascapular nerve will receive fibers from the C-4 root, but it will rarely arise solely from the distal end of the C-5 root.13,29 The nerve extends laterally and deep to the trapezius and omohyoid muscles as it courses with the suprascapular artery on its way to the suprascapular notch (Fig. 1). It is separated from the artery at the notch by the superior transverse scapular ligament. The nerve passes under the ligament through the notch, providing an anatomical predisposition for entrapment, while the artery courses above the ligament. Rengachary and associates23 discovered significant anatomical variability of the notch and overlying superior transverse scapular ligament. The nerve passes under the ligament through the notch, providing an anatomical predisposition for entrapment, while the artery courses above the ligament.

Having entered the supraspinatus fossa, the nerve extends deep to the supraspinatus muscle and gives off motor branches to that muscle and sensory branches to the shoulder and acromioclavicular joints. It curves around the lateral border of the spine of the scapula and continues in company with the suprascapular artery under the inferior transverse scapular ligament. The nerve then enters the infraspinatus fossa at which point it typically supplies two motor branches to the infraspinatus muscle and sensory filaments to the shoulder joint and scapula. The inferior transverse scapular ligament represents another potential site for distal suprascapular nerve entrapment.1,10,33

Summary of Cases

Seven patients, all men, presented to us for evaluation of shoulder pain and weakness. The age, etiology, clin-
ical presentation, and duration of symptoms for these seven patients are depicted in Table 1. The symptom that brought these patients to medical attention was shoulder pain often aggravated by shoulder motion and shoulder weakness, particularly related to upper-extremity abduction or external rotation of the upper arm. In all cases there was marked atrophy of the supraspinatus and infraspinatus musculature.

Trauma was directly related to the shoulder complaints in four patients and indirectly related in one case. All seven patients had positive EMG findings with respect to the spinatus musculature but had no EMG evidence of deltoid weakness. Although there may be clinical evidence of deltoid weakness due to disuse atrophy, deltoid EMG studies remained normal. The NCV studies uniformly demonstrated delayed conduction along the suprascapular nerve, further supporting the diagnosis of a localized suprascapular nerve palsy.13

Six of the seven patients warranted surgical decompression of the nerve (Table 2). Five of these patients improved. One patient (Case 3) had a ganglion cyst which caused permanent suprascapular nerve injury resulting in persistent symptoms. One patient (Case 5) was treated conservatively with suprascapular nerve blocks, which gave lasting benefit. Conservative measures consist primarily of shoulder exercises or local injection of steroids and analgesics, or both.

Illustrative Cases

Case 3

This 28-year-old right-handed man had some mild discomfort of the right shoulder for several years prior to his injury. On December 1, 1981, he slipped on ice, striking his right shoulder posteriorly. He continued working but noticed increased pain and weakness in the right shoulder. On February 11, 1982, EMG demonstrated denervation potentials in the infraspinatus muscle and decreased motor function in both the supraspinatus and infraspinatus musculature. Nerve conduction velocity studies revealed increased latency in the suprascapular nerve.

Physical examination on February 22 revealed a 3/5 weakness of the supraspinatus muscle and a 4/5 weakness of the infraspinatus muscle on the right side, accompanied by significant atrophy of both muscles. The remainder of the examination was normal. On March 2, the patient underwent surgical release of the suprascapular nerve. At the time of surgery, a large mass containing clear gelatinous material was discovered under the transverse scapular ligament. This mass, which appeared to extend from the shoulder capsule, was removed. The transverse scapular ligament was sectioned. There was significant expansion and erosion of the suprascapular notch. The nerve was never visualized. Pathological analysis of the mass revealed it to be a ganglion cyst.

In late March, 1982, the patient had normal strength in both the supraspinatus and infraspinatus muscles. His pain resolved; however, the atrophy remained unchanged. In May, he again noted weakness of the supraspinatus and infraspinatus muscles. Repeat EMG was unchanged from the preoperative study performed 3 months earlier. A third EMG, performed on September 28, showed increased fibrillations and further motor loss when compared to the prior two studies. A CT scan of the right shoulder on October 14 was unremarkable except for postoperative changes. There was no evidence of a mass or mass effect. On October 26, the patient underwent exploration of the right suprascapular fossa and shoulder joint. A large recurrent ganglion cyst was discovered occupying the entire suprascapular fossa under the supraspinatus muscle. It followed the spine of the scapula and extended into the infraspinatus fossa. The cyst appeared to originate from the shoulder joint itself and was removed in toto. The architecture of the suprascapular notch had been obliterated, the nerve presumably destroyed by the cyst. On March 9, 1983, the patient had a 3/5 weakness and marked atrophy of both the supraspinatus and infraspinatus muscles. An arthrogram of the right shoulder was normal. The patient was unchanged at reevaluation in July, 1984, with persistent shoulder discomfort.

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**Table 1**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Etiology of Entrapment</th>
<th>Presentation*</th>
<th>Duration of Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27</td>
<td>motorcycle accident</td>
<td>• • •</td>
<td>13 mos</td>
</tr>
<tr>
<td>2</td>
<td>60</td>
<td>work</td>
<td>• • •</td>
<td>15 mos</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>unknown</td>
<td>• • •</td>
<td>years</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>unknown</td>
<td>— — •</td>
<td>6 mos</td>
</tr>
<tr>
<td>5</td>
<td>33</td>
<td>spontaneous</td>
<td>• • •</td>
<td>acute</td>
</tr>
<tr>
<td>6</td>
<td>53</td>
<td>fall</td>
<td>• • • • • •</td>
<td>18 mos</td>
</tr>
<tr>
<td>7</td>
<td>35</td>
<td>backpacking</td>
<td>• • •</td>
<td>6 mos</td>
</tr>
</tbody>
</table>

*Symptoms were graded from — (not present) to • • • (severe).

**Table 2**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Treatment</th>
<th>Outcome &amp; Follow-Up Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>transection of ligament</td>
<td>increased bulk &amp; strength; no pain</td>
</tr>
<tr>
<td>2</td>
<td>transection of ligament</td>
<td>normal bulk &amp; strength; 3 yrs</td>
</tr>
<tr>
<td>3</td>
<td>excision of ganglion cyst</td>
<td>persistent weakness &amp; atrophy of spinatus muscles; 2 yrs</td>
</tr>
<tr>
<td>4</td>
<td>transection of ligament</td>
<td>normal bulk &amp; strength; 4 yrs</td>
</tr>
<tr>
<td>5</td>
<td>steroid/lidocaine injec-</td>
<td>increased bulk &amp; strength; no pain; 2 yrs</td>
</tr>
<tr>
<td>6</td>
<td>transection of ligament</td>
<td>slightly improved strength; no pain; 6 mos</td>
</tr>
<tr>
<td>7</td>
<td>transection of ligament</td>
<td>normal strength; no pain; 2 mos</td>
</tr>
</tbody>
</table>
Suprascapular nerve entrapment

Case 5

This 33-year-old right-handed policeman awoke at 4 a.m., 5 weeks before admission, with pain in the left shoulder of sufficient severity that he immediately went to an emergency room for an analgesic injection. The pain involved the left scapula and the muscles of the top of the shoulder and radiated down to involve the entire left upper extremity. The patient slept in a recliner chair for the entire 5 weeks prior to admission because pressure on the scapula while lying supine was painful, precluding sleep. Two years previously the patient had experienced pain in the left shoulder, which cleared spontaneously after 1½ weeks.

Examination revealed marked atrophy of the left supra- and infraspinatus muscles and virtually no ability to externally rotate the left arm with the arm adducted against the trunk. No other muscles were weak or atrophic, and tendon reflexes and sensation were intact. There was considerable tenderness over the left scapula. Electromyography revealed increased insertional activity and denervation potentials isolated to the left supra- and infraspinatus muscles, suggesting a suprascapular nerve abnormality. An x-ray film of the left shoulder showed small areas of calcification, consistent with calcific peritendonitis.

Four blocks of the suprascapular nerve at the suprascapular notch were carried out, with 10- to 22-cc injections of 2% lidocaine and 40 mg of Depo-Medrol (methylprednisolone acetate) over a 15-day period. The patient received considerable temporary benefit from the first block with reduction of tenderness so that he was able to sleep for about 3 hours lying flat, a position he had not used for sleeping during the entire 5 weeks of his present illness. The patient returned to regular work 1 week after his fourth injection. Eighteen months later he was still pain-free and had nearly normal shoulder strength and spinatus muscle bulk.

Case 7

This 35-year-old man went backpacking in August, 1984, for several days. After the trip he experienced left shoulder pain for approximately 10 days. The discomfort decreased somewhat but was still present in February, 1985. He also noted weakness of the left shoulder. Electromyography and NCV studies were abnormal, demonstrating decreased motor function in both the spinatus muscles and delayed suprascapular nerve conduction, respectively. Physical examination revealed atrophy of the left supra- and infraspinatus muscles with a prominent scapular spine. Motor power was rated 4/5 for the supraspinatus muscle and 3/5 for the infraspinatus muscle. The remainder of the motor and neurological examination was normal.

On February 15, 1985, the patient underwent decompression of the suprascapular nerve, with transection of the transverse scapular ligament. Postoperatively, he has had no further pain and his strength has returned to normal. Muscle bulk, although improved, remains reduced relative to the contralateral shoulder.

Surgical Technique

For surgery, the patient should be placed in the lateral decubitus position with the arm of the involved shoulder elevated and supported on a Mayo stand. This allows motion and manipulation of the affected upper extremity at the time of surgery. A transverse incision is made parallel and approximately 2 cm above the spine of the scapula (Fig. 2). The trapezius muscle is
separated along its fibers. The atrophied supraspinatus muscle is identified and the suprascapular notch found by either following the suprascapular artery or palpating the transverse ligament (which may be calcified). The transverse scapular ligament is dissected and excised after the suprascapular nerve below it has been identified.5,18,24,25,33

It is not always possible to identify the nerve at the time of decompression. Extended efforts to locate the nerve are unwarranted and may result in iatrogenic nerve injury. With transection of the transverse scapular ligament, the nerve is usually but not always pushed outward as a result of the decompression. An alternative surgical technique is to enlarge the notch rather than to section the transverse ligament.21 We do not believe that notch enlargement is as efficacious a surgical technique as transection and excision of the transverse scapular ligament; therefore, we did not employ it in our cases.

Discussion

The first report in the English literature defining the syndrome of suprascapular nerve entrapment was that by Kopell and Thompson in 1959.16 Since that time, many authors have published their clinical experience with palsies of the suprascapular nerve.1-12,14,16-36 Our seven cases, the largest series reported thus far, increases the total number of reported cases to 75. Despite these reports in the literature, suprascapular nerve entrapment is a rare clinical diagnosis, which we believe is due in large part to examiner inexperience with the reports in the literature, suprascapular nerve entrapment was that syndrome manifests itself so infrequently. Increased awareness by the physician of its clinical presentation, diagnostic characteristics, and treatment may lead to more frequent correct diagnoses, earlier therapy, and reduced patient morbidity.

The most common cause of suprascapular nerve entrapment is trauma to the shoulder, usually direct trauma with injury to the nerve and/or damage to the transverse scapular ligament or surrounding tissues (which may cause subsequent scarring and compression of the nerve).17,18,33 A number of cases of suprascapular nerve palsy have been associated with fractures of the scapula,8,26,27 and proximal humerus,33 and shoulder dislocation.32,34 In patients without a history of direct trauma, Sunderland29 hypothesized that the suprascapular nerve (which is relatively fixed at the suprascapular notch) is subject to friction with motion of the scapula and shoulder joint. Forced cross-body adduction of the arm, with either persistent or repetitive forced adduction, may result in traction injury to the suprascapular nerve, compromising it against the rigid edge of the suprascapular notch.16,26 Other causes of suprascapular nerve palsies are lipomas, ganglion cysts10,14,32 (as in our Case 3), penetrating injury,33 arthritic inflammation, systemic lupus erythematosus,25 and surgical dissection.31

The diagnosis of suprascapular nerve entrapment is made from the patient’s history, clinical examination, x-ray film evaluation, and neurophysiological electrodiagnostic studies. These factors will help differentiate a specific suprascapular nerve palsy from other causes of shoulder pain and weakness; namely rotator cuff injuries, arthritic shoulder syndromes, rupture of the long head of the biceps, tendonitis (of the biceps, supraspinatus, or infraspinatus muscles), degenerative cervical spine disease, and C-5 radiculopathy.

The most common and consistent presenting symptom of entrapment of the suprascapular nerve is shoulder pain, usually described as a deep, dull ache located posterolaterally in the shoulder. Patients often suffer for months or years before seeking medical attention (the mean duration of symptoms among our seven cases was 12 months). The pain may be exacerbated by movement of the shoulder girdle, particularly with cross-body adduction of the extended arm.17,18,33 While usually well-localized, the ache may extend down the arm to the elbow or up into the neck. Occasionally, the discomfort can become so severe that it disturbs sleep, with the patient unable to lie upon the involved shoulder (as in our Case 5). Apparently, pressure upon the scapula forces it forward, further compromising the irritated nerve.33

Weakness of the shoulder and arm is a common secondary complaint. Strength in the supraspinatus musculature can be tested by examining the patient’s ability to abduct the arm both passively and against resistance (the first 15° of abduction isolates the supraspinatus muscle). The infraspinatus muscle can be tested by external rotation of the arm against resistance, with the elbow held in 90° flexion. There is almost always wasting and atrophy of the supr and infraspinatus musculature associated with a deltoid muscle of normal bulk and power.

Radiography of the shoulder and neck should be performed. Anteroposterior and lateral roentgenograms may reveal a scapular fracture as the cause of suprascapular nerve embarrassment, or another non-entrapment entity may be identified as the etiology of the shoulder discomfort. Donovan and Kraft7 have described arthrography of the shoulder as a useful tool to help distinguish rotator cuff injuries from suprascapular nerve palsy.

A suprascapular nerve block with lidocaine (with or without cortisone) may relieve the shoulder discomfort and serve as a useful diagnostic maneuver.5-12,14,15,19,33 The single most helpful diagnostic study is EMG, which should demonstrate motor loss in the spinatus musculature, a reduced recruitment pattern, and denervation potentials.5-7,15,19,33 Nerve conduction velocity determinations are more difficult to obtain but if performed should reveal delayed conduction along the suprascapular nerve.5,7,15,33

Treatment of entrapment of the suprascapular nerve
Suprascapular nerve entrapment

<table>
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<th>TABLE 3</th>
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**Typical profile of a patient with suprascapular nerve entrapment**

<table>
<thead>
<tr>
<th>Patient Profile</th>
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<tbody>
<tr>
<td>male</td>
</tr>
<tr>
<td>age 25–55 yrs</td>
</tr>
<tr>
<td>history of trauma to shoulder</td>
</tr>
<tr>
<td>presents with pain, weakness, &amp; atrophy</td>
</tr>
<tr>
<td>positive electromyogram</td>
</tr>
<tr>
<td>surgical/medical treatment</td>
</tr>
<tr>
<td>good prognosis</td>
</tr>
</tbody>
</table>

depends on the duration of symptoms, the location of the entrapment, and the etiology of the nerve insult. Our initial treatment in patients with recent onset of symptoms and minimal weakness is conservative and consists of elevation and exercise of the shoulder, combined with local supportive therapy (such as massage, heat packs, or anti-inflammatory medications). A series of three to four suprascapular nerve blocks should be considered, with injection of a combination of steroid and local anesthetic agents into the region of the superior transverse scapular ligament. We advocate surgical therapy for patients with fractures of the scapula; patients with long-standing disease and pronounced weakness and atrophy; patients who have failed the aforementioned more conservative therapies; and those with suprascapular nerve palsy due to structural lesions (such as ganglion cyst, callus, lipoma, or nerve transection). The surgical technique for releasing the suprascapular nerve consists of sectioning the superior transverse scapular ligament. We prefer this approach rather than attempting to enlarge the scapular notch as described by Rask. Operative results are uniformly good unless the nerve has been severely damaged, as noted in Case 3. Alterations of the surgical approach are required for isolated infraspinatus muscle involvement as described by Aiello, et al., or Thompson, et al.

Conclusions

The syndrome of suprascapular nerve entrapment exists much more frequently than is reported. It is an entity that should be considered in a patient with shoulder pain, particularly those with supra- and infraspinatus muscle weakness or atrophy (Table 3). A careful clinical examination, radiography, and neurophysiological electroadiagnostic evaluation by EMG and NCV studies will differentiate this entity from other causes of muscle and joint dysfunction. Surgical intervention is usually warranted, although conservative treatment in the less involved cases should be tried.

References


Manuscript received August 6, 1985. Accepted in final form November 4, 1985.
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