Suprascapular Neuropathy in a Shoulder Referral Practice

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ABSTRACT

Background:

Suprascapular neuropathy (SSN) is currently considered a rare condition that is usually made as a diagnosis of exclusion. Although there are papers examining the etiology and treatment of the problem, few studies have analyzed how commonly the condition is encountered in practice. In addition, while electrodiagnostic studies are considered the gold standard for diagnosis, the specific criteria for a diagnostic test are not well studied. The purpose of this study was to report a retrospective experience of electrodiagnostically proven SSN in a shoulder referral practice and highlight the specific electrodiagnostic findings that are currently being used to diagnose the condition.

Methods:

A retrospective review of a one year period was undertaken to identify all patients that were sent for electrodiagnostic studies to evaluate the suprascapular nerve. Clinical exam findings, subjective complaints, and associated shoulder pathology was documented. The diagnostic studies were reviewed and electromyography (EMG) and nerve conduction velocity (NCV) values were studied to compare specific parameters in patients whose studies were interpreted as diagnostic for SSN vs. those interpreted as non-diagnostic.

Results:

Ninety-two patients were sent for electrodiagnostic evaluation of the suprascapular nerve, of which 40 (42%) had a positive study. Patients with a massive rotator cuff tear (RCT) were more likely to have a diagnostic study than those without a tear. The majority of positive studies had motor unit action potential (MUAP) abnormalities (88%), while only 33% had EMG abnormalities. The average latency in diagnostic studies was 2.90 (+/- 0.08) for the supraspinatus and 3.78 (+/- 0.14) for the infraspinatus.

Conclusions:

Electrodiagnostically proven SSN is common in patients who present to a shoulder referral practice with a high clinical suspicion of the disease or massive RCT. Surgeons should consider electrophysiological evaluation in these patients and be cognizant of the parameters that constitute a diagnostic study.

LEVEL OF EVIDENCE: IV

Keywords: Suprascapular neuropathy, Electrodiagnostic testing, Massive Rotator Cuff Tear

INTRODUCTION

Suprascapular neuropathy (SSN) was first described by Thompson and Kopell in 1959[23,29]. It has since been assumed to be rare and can be overlooked as a source of shoulder pain. It is usually viewed as a diagnosis of exclusion and no studies to date have clearly elucidated its prevalence in patients with shoulder pain. SSN is thought to be caused by two main mechanisms: either traction or compression at the suprascapular or spinoglenoid notch. An injury to the nerve at the suprascapular notch will affect

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IRB approval
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Table 1: Previously reported occurrence and largest series of suprascapular neuropathy in the literature

Study	Prevalence or series
Holzgraefe, 1994	22/66 (33.3%) in high level male volleyball players
Feretti, 1998	12/69 (17.4%) in high level male volleyball players
Krivicas, 1998	14/169 (8.3%) in athletes undergoing EMG/NCVs for sports injuries
Brown, 2000	4/15 (26.7%) in patients with full thickness rotator cuff tears
Antoniou, 2001	Case series of 53 patients
Zehetgruber, 2002	Meta-analysis with 88 published cases (1959 -2001)
Vad, 2003	2/25 (8.0%) in patients with full thickness rotator cuff tears

Table 2: Electrophysiologic data in EMG/NCV's read as diagnostic vs. negative for SSN

	Diagnostic EMG/NCV	Normal EMG/NCV	p value
EMG Abnormaility (N)	13/40	1/52	N/A
MUAP Abnormality (N)	35/40	7/52	N/A
EMG and MUAP Abnormality (N)	13/40	0/52	N/A
Average latency to supraspinatus (ms) +/- SE	2.90 +/- 0.081	2.65+/- 0.062	0.018
Average latency to infraspinatus (ms) +/- SE	3.78 +/- 0.139	3.35+/- 0.069	0.004
Average latency between supra and infraspinatus (ms) +/- SE	0.86 +/-0.144	0.70+/-0.064	0.268

Table 3: Previously published mean normative distal latency standards (ms) for the suprascapular nerve

Study	Supraspinatus mean (SD)	Infraspinatus mean (SD)
Kraft (1972): Needle	2.7 (0.5)	3.3 (0.5)
Gassel (1964): Needle	2.6 (0.32)	3.4 (0.4)
Cassaza (1998):		
Concentric	2.76 (0.39)	3.41 (0.5)
Monopolar	2.54 (0.38)	3.06 (0.39)
Surface	2.57 (0.42)	3.07 (0.54)
Buschbacher (2009): Surface	3.2 (0.5)	3.6 (0.6)
Current Study: Needle	2.65 (0.062 SE)	3.35 (0.069 SE)

both the supraspinatus and infraspinatus, while an injury at the spinoglenoid notch will only affect nervous supply to the infraspinatus.

Irritation of the nerve may be secondary to traction from repetitive overhead activities, with recent data demonstrating a tightening of the spinoglenoid ligament over the nerve with the motion of overhead throwing[24]. Compression of the nerve may also occur from a cyst or space-occupying lesion. There are numerous studies demonstrating the connection between paralabral cysts and compression of the nerve at the spinoglenoid notch.[1,11,20,31,36]. Constrictive anatomical variants of the suprascapular notch and spinoglenoid ligament may also predispose to nerve injury[4,8-10,26,30]. Traction injury to the nerve from a massive RCT has been described[2,14,19,32,34], however, the actual incidence and prevalence of this association remains largely unknown. Vad demonstrated 8% SSN (2/25) in full thickness RCT with atrophy, while Mallon presented findings diagnostic for SSN in eight patients with a massive RCT[19,32]. A paper by Costouros studying patients with a massive RCT tear and fatty infiltration documented 27% (7/26) SSN[7].

Most data regarding the overall occurrence of SSN is extrapolated from case studies, many of which were in high level athletes. These papers estimated a prevalence of 20-33% in high level volleyball players[12,15], however analysis was limited to athletes only. The largest case series on SSN contains only 53 patients[3] (Table 1).

Historically, the suprascapular nerve has been considered primarily a motor nerve, however recent studies have shown that sensory branches are more common than once thought[33]. The nerve may provide up to 70% of the sensation of the shoulder including the superior aspect

of the joint and capsule with variable contribution to the overlying skin and soft tissues[16,27,28]. Clinically, patients with SSN may present with posterior/superior aching over the shoulder, weakness in overhead activities and/or weakness in abduction or external rotation, or in combination with other concomitant shoulder pathology. On physical exam, atrophy of the supraspinatus, infraspinatus or both may be noted in addition to tenderness at the suprascapular or spinoglenoid notch. The crossed body adduction test may be positive if the spinoglenoid notch is the area of pathology[24]. Magnetic resonance imaging (MRI) is often utilized in the evaluation of those with suspected SSN and helps assess the rotator cuff, muscle quality, space occupying lesions as well as other sources of pathology. EMG/NCV studies can be obtained to confirm a clinical suspicion of SSN. Electrodiagnostic criteria include prolonged motor latencies, denervation potentials, and a delay in time of conduction from Erb's point to the supraspinatus and/or infraspinatus. (Table 3)

SSN is usually treated with an initial trial of non-operative treatment as the indications for acute operative intervention remain unclear. Surgical treatment is dictated by the pathology and may consist of cyst decompression, repair of associated lesions including RCT or labral tear, and/or nerve decompression at the suprascapular or spinoglenoid notch[25]. Decompression of the suprascapular nerve may be accomplished through an open or arthroscopic approach[18,35,37]. This study attempts to describe the rate of positive electrodiagnostic studies in patients presenting to a shoulder referral practice with a suspicion of SSN. The secondary aim was to clarify the specific EMG/NCV findings that are currently being used to make the diagnosis.

METHODS

All study procedures were approved by the institutional review board. A retrospective review of a prospectively collected shoulder database from January 1, 2007 to January 1, 2008 was undertaken to identify all patients that were sent for electrodiagnostic studies to evaluate the suprascapular nerve. Patients of all races, ages, and sexes qualified for inclusion. A chart review was conducted to determine the clinical reasons for referral for an electrodiagnostic study including associated shoulder pathology on physical exam and/or imaging studies. All EMG/NCV studies were then reviewed to ascertain how many patients out of those referred had a positive study. The EMG/NCV values were studied to compare specific parameters in patients whose studies were interpreted as diagnostic for SSN vs. those interpreted as non-diagnostic. The number of patients with EMG abnormalities, MUAP abnormalities, or both was calculated for the infraspinatus and supraspinatus. An EMG abnormality was documented as any sharp wave or fibrillation. MUAP abnormalities were defined as any evidence of a mild, moderate, or large increase. The motor latencies were calculated from Erb's point to both the supraspinatus and infraspinatus; in addition latencies between the supraspinatus and infraspinatus were calculated. The latencies were compared between both groups by Student T-test with Microsoft Excel (Microsoft: Redmond, Washington). Statistical significance comprised $p < 0.05$. Exclusion criteria included a prior suprascapular nerve decompression, incomplete or inadequate electrodiagnostic studies, or incomplete medical records.

RESULTS

Review yielded 94 patients of whom 2 were excluded for incomplete EMG/NCV data. Ninety-two patients were identified as having either clinical or radiographic signs suggestive of SSN as ascertained by the senior author. These included patients who presented with: 1) chronic aching over the superior or posterior/superior aspect of the shoulder without concomitant cervical spine disease or other clear cause, 2) atrophy and/or weakness of the supraspinatus and/or infraspinatus on clinical examination or MRI, 3) RCT and fatty infiltration/atrophy of the supraspinatus and/or infraspinatus on magnetic resonance imaging and 4) massive RCT (greater than or equal to 5cm in maximum diameter in the coronal or sagittal plan). Overall there were 56 male and 36 female patients with a median age was 51.7 years (range 16-81). Of the patients identified, 38 had a massive RCT, 4 had a full thickness tear not defined as massive, 6 had a partial thickness tear, 1 had an isolated tear of the subscapularis, and 43 had no RCT. Twenty-eight patients had a previous failed rotator cuff repair and all of these patients had a massive RCT on MRI. All included patients had undergone standard EMG and NCV studies to evaluate the suprascapular nerve, most of which (88%) had been performed at the same center by one examiner. All of the electrophysiologic studies had been independently performed and interpreted by a neuromuscular specialist.

Overall 43% (40/92) studies were read as diagnostic for SSN by a neuromuscular specialist. Among the 40 patients with an electrophysiologic diagnosis of SSN, 13 (32.5%) had a mixed pattern around the shoulder including cervical spine, axillary nerve, or long thoracic nerve pathology in addition to SSN. Of the patients with an electrodiagnosis of SSN, 23 patients had a massive RTC tear, 2 had a full thickness tear not considered massive, and 15 did not have RTC pathology (there were no patients with a partial RTC tear). The likelihood of SSN was greater among patients with a massive RCT (60.5% (23/38)) than patients without massive RCT (31.5% (17/54), $p < 0.05$ chi-square test). The mechanism of injury in this group was thought to be traction secondary to retraction of the cuff musculature, as the injury to the nerve was at the suprascapular notch in all patients. In evaluating those with massive RCT, the presence of SSN was lower (54% (15/28); $p = 0.25$) among the 28 patients with prior attempted RCT repair as compared to the 14 patients without prior RCT repair (71% (10/14)), however this was not statistically significant. Fifteen individuals were found to have an electrophysiologic diagnosis of SSN without RCT. Among these 15, 10 (66.7%) had nerve injury at the suprascapular notch and 5 (33.3%) at the spinoglenoid notch. Among the 40 patients with a confirmed diagnosis of

SSN, 70% (28/40) underwent subsequent arthroscopic shoulder surgery including arthroscopic surgical decompression of the nerve and 30% (12/40) opted for non-surgical treatment.

In depth analysis of the electrodiagnostic data revealed that 13/40 (33%) positive studies had EMG abnormalities and 35/40 (88%) had MUAP abnormalities, with 13/40 (33%) demonstrating both. Five studies (12.5%) had isolated prolongation of motor latencies without EMG or MUAP abnormalities. The average motor latency to the supraspinatus in the non-diagnostic studies was 2.65 (+/- 0.06) as compared to 2.90 (+/- 0.08) for the diagnostic studies, while data for the infraspinatus yielded an average latency of 3.35 (+/- 0.07) for the non-diagnostic studies as compared to 3.78 (+/- 0.14) for the diagnostic studies (Table 2). P values were < 0.05 for both groups.

DISCUSSION

SSN has historically been presumed to be relatively rare and a diagnosis of exclusion. This is partly because there are few papers on the topic, with a meta-analysis identifying only 88 reports between 1959-2001[37]. The number of reports has increased in recent years with some studies suggesting the association of SSN with RCT[2,7,19,34]. In addition, although previously thought to be solely a motor nerve, it is becoming clear that suprascapular nerve injury can be a major contributor to shoulder pain, implying a sensory component. Studies in the orthopaedic and anesthesia literature reveal that suprascapular nerve blocks can significantly decrease pain after shoulder surgery[16,33]. The present study describes the diagnosis of suprascapular neuropathy presenting to a shoulder practice.

Since all patients presenting to the practice over a year were not tested, this study cannot calculate an actual prevalence. However, we did note that patients with electrodiagnostically proven SSN represented 4.3% of all new patients seen over a one year time period. In addition, surgical procedures for SSN comprised 5.8% of the total operations for the year. The true prevalence of SSN is likely much higher as we only tested a select group of patients and required EMG/NCV confirmation to make this diagnosis. In this select population of patients, a high number were found to have SSN on electrodiagnostic studies (43%) with those patients with a massive rotator cuff more likely to have a positive study than those with a full thickness tear, partial thickness tear, or no RTC injury. In addition nearly one third of patients with a diagnostic study had other signs of neurologic injury in the cervical spine or other nerves in the shoulder, raising the possibility of a "double hit" phenomenon or the previously unrecognized associations.

EMG/NCV studies have been shown to be 91% accurate in detecting muscle denervation, however these tests do not detect damage to the unmyelinated and thinly myelinated pain fibers that have been recently recognized as present within this nerve and thus may underestimate actual SSN prevalence[21,22]. This may explain some instances of chronic shoulder pain despite negative electrodiagnostic testing. In addition, although a number of studies have reported normal electrodiagnostic values for the suprascapular nerve, variability remains[5,6,13,17]. The distal latency values found on electrodiagnostic testing read as negative for SSN in this study are similar to those reported in normal subjects by Kraft, Gassel, and Casazza[6,13,17] however are lower than those found in a recent report by Buschbacher[5]. The noted difference may be secondary to technique as the Buschbacher study utilized surface electrodes while our EMG/NCV's were recorded with needle electrodes. (Table 3)

Our data are limited by the fact that they reflect the practice of a single shoulder surgeon at a single tertiary care center and may not be applicable to a general orthopedic practice. We also acknowledge that electrodiagnostic studies can be dependent on the center or individual performing the examination, however the majority of our studies (88%) were performed at the same center, limiting inter-observer variability.

The treatment for electrodiagnostically confirmed SSN is highly debated. Increased interest in the subject may result in increasing operative procedures. Surgical outcomes, in addition to whether RCT repair or decompression of a labral cyst in the presence of SSN are adequate without additional decompression of the nerve will only be answered in comparative trials. Nevertheless, a greater awareness of SSN and the electrodiagnostic criteria for diagnosis is useful to the practicing orthopaedic surgeon in identifying the condition and initiating treatment whether it may include activity modification, therapy, or surgical intervention.

CONCLUSION

SSN is commonly seen in a shoulder referral practice. Based on these findings, orthopaedists should consider the diagnosis and electrophysiological evaluation in patients presenting with chronic pain, weakness, aching over the superior aspect of the shoulder, fatty muscle infiltration or atrophy on MRI, and/or massive RCT. In addition, surgeons should be cognizant of the specific EMG/NCV parameters which constitute a positive study.

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