Title Page

Suprascapular Neuropathy: A Review of 87 Cases

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Running Title: Suprascapular Neuropathy

"We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.''

Abstract:

Introduction: Suprascapular neuropathy (SSN) is rare, with an estimated prevalence of 4.3% in patients with shoulder pain.

Methods: This retrospective chart review included patients with SSN seen during a 16-year period. Demographics and clinical information were recorded. Descriptive statistics including percentages, means, and standard deviations were computed for the variables of interest for all patients.

Results: Of 87 patients included in this study, trauma (n=27) was the most common cause of SSN followed by neuralgic amyotrophy (n=21). Fifty-seven patients had isolated SSN. Other had SSN associated with axillary neuropathy (23 patients), brachial plexopathy (3), and long thoracic, radial, or spinal accessory neuropathy (1 each).

Discussion: SSN is commonly associated with axillary neuropathy. Trauma remains the most common cause of SSN. Electrodiagnostic findings aid in the initial diagnosis and may indicate the need for close clinical follow-up based on the severity of the axonal injury.

Keywords: Suprascapular neuropathy, shoulder pain, shoulder weakness

sports injury, proximal neuropathy, axillary neuropathy

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Introduction

Suprascapular neuropathy (SSN) is an uncommon source of shoulder pain. Weakness of abduction and external rotation, and the associated functional disturbance, can result from impaired innervation of the supraspinatus and infraspinatus muscles, respectively.[1] Severe denervation can lead to atrophy apparent on a physical exam as well as edema and fatty infiltration seen on imaging.[2]

The prevalence of SSN ranges from 1-2% of all shoulder pain to 4.3% in patients with shoulder complaints referred to an academic center.[3] Elite athletes engaging in overhead sports activities, such as volleyball, are at particularly high risk.[4]

While SSN may rarely be a result of neuralgic amyotrophy (Parsonage-Turner syndrome), the most common cause relates to mechanical compression.[5,6,7] Common locations for compression include entrapment at the suprascapular notch by the superior transverse scapular ligament and at the spinoglenoid notch by the spinoglenoid ligament.[8] Mass effect by a ganglion cyst may also occur.[9]

As the predominant clinical feature of SSN is shoulder pain, it is important to include similarly presenting conditions in the differential diagnosis such as other proximal mononeuropathies, arthropathy, tendinopathy and radiculopathy. [10] Electromyography (EMG) is the gold standard for the diagnosis of SSN[4] and imaging may be helpful.

The goal of this study was to better understand the clinical presentation, exam findings, etiology, and diagnostic studies in the evaluation of SSN and its association with other mononeuropathies. The secondary aim was to better understand the electrophysiology of SSN and its role in diagnosis and prognostication.

Methods:

Patients seen at the Henry Ford Health System between January 2000 and December 2016 with an EMG diagnostic code of SSN were identified via electronic medical records (EMR). The study was approved by the hospital's institutional review board. A retrospective chart review of patients with SSN was performed. Other mononeuropathies associated with SSN were recorded.

Patient age at onset of symptoms gender, ethnicity, side of neuropathy etiology, risk factors, and clinical features was recorded. Electrophysiological studies, diagnostic evaluation, treatment (pain management and physical therapy), follow-up, and recovery data were collected. A detailed history of clinical presentation including the presence of pain, medical research council (MRC) muscle scale functional impairment, atrophy of supraspinatus and infraspinatus muscles, and range of motion such as active forward flexion, abduction, and external rotation were noted. Recovery was defined as improvement in MRC motor scale functional impairment, pain, and range of motion during follow-up. Patients who had an initial clinic visit with no follow-up were classified as lost to follow-up. Potentially relevant risk factors for SSN were also recorded from the EMR which included shoulder trauma, falls, rotator cuff tear, rotator cuff surgeries, compression lesions, and repetitive overhead shoulder movements secondary to occupation or recreational activities.

Electrophysiologic data included compound muscle action potential (CMAP) amplitude of suprascapular nerve recorded using a surface electrode over the infraspinatus muscle while stimulating at Erb's point, and needle EMG data regarding fibrillation potentials and recruitment of motor unit action potentials (MUAPa) in the supraspinatus and/or infraspinatus muscles. Fibrillation potentials were graded as being either present or absent. Recruitment of MUAPs was classified as being normal or abnormal.

Magnetic resonance imaging (MRI) and ultrasound of the shoulders were reviewed where available to identify structural or mass lesions (paralabral cyst, desmoid tumor), rotator cuff tear, shoulder dislocation (Hill-Sachs deformity), joint capsule hypertrophy, and supraspinatus and infraspinatus muscle atrophy.

Statistical Analysis:

Descriptive statistics were used for patient variables as well as subgroups. For comparisons of the SSN subgroups, chi-squared and Fisher's exact tests were performed for categorical variables and a two-sample t-test was done for age at diagnosis. Wilcoxon two-sample tests were used to compare the SSN subgroups for the duration of symptoms and CMAP amplitude because these variables did not meet the assumption of normality using the Shapiro-Wilk test for normality. Wilcoxon two-sample tests also were used for the measurements of supraspinatus and infraspinatus motor strength because they were measured on the ordinal scale MRC scale. Similar analyses were done to assess the association of recovery with CMAP amplitude and EMG findings recorded from supraspinatus and infraspinatus muscles. Statistical significance was set at $p<0.05$. All analyses were performed using SAS 9.4 (SAS Institute Inc, Cary, NC, USA).

Results:

Of 87 patients included in this study, 66% were male; 72% were Caucasian and 22% were African American. The mean age at diagnosis was 47.4 years (range 14-81 years). The most common cause of SSN was trauma from a motor vehicle accident (MVA) or fall (31%) followed by idiopathic and weight-lifting (Table 1). Patients with idiopathic etiology were confirmed cases of neuralgic amyotrophy This diagnosis was established based on the patient's clinical presentation, in association with the exclusion of other potential causative factors by the examining physician.

Of all patients, 57 had isolated SSN followed by SSN associated with axillary neuropathy (SSN+ axillary, n=23). Additional patient characteristics can be found in Table 1. The median duration of symptoms (time between the onset of symptoms and EMG) was 4.6 months. Additional clinical and EMG information can be found in Table 2.

A total of 14 patients were lost to follow-up. Of the remaining 73 patients, the median follow-up time was 16.9 months (range 8 days to 13 years; interquartile range 3.7 to 52.4 months). Nine of the patients without recovery had follow-up time less than 3 months and were not included in the recovery analysis. Of the remaining 64 patients, 50 (78%) patients recovered with a median time to recovery of 8.2 months (interquartile range 4.8 to 13.5 months) and 45 of these recovered within 18 months. The median duration of follow-up was 13.4 months (interquartile range 4.6 to 41.7 months) for the 14 patients without recovery.

Details of patients by groups are shown in Supplementary Tables 1-3. Of the 87 patients, 42 (48%) had an abnormal shoulder MRI and 37 (43%) had an abnormal shoulder ultrasound (Supplementary Table 1). A comparison of patients with SSN+ axillary neuropathy versus isolated SSN showed a significant difference in etiology. Patients with isolated SSN were more likely to have sports-related and idiopathic etiologies while patients with SSN+ axillary neuropathy were more likely to have an etiology of trauma (MVA and falls). Patients with SSN + axillary neuropathy had a significantly higher rate of a limited range of motion compared to the isolated group. Significant differences were also found for supraspinatus weakness, with patients having no associated neuropathy demonstrating greater MRC values. (Supplementary Table 2.) The difference in CMAP amplitude between patients with and without recovery was not significant. The associations of recovery with EMG abnormalities (recruitment/fibrillation potentials) in supraspinatus and infraspinatus muscles were also not significant. (Supplementary Table 3.)

Only 17 of 87 patients had a repeat electrodiagnostic study. Of these, 11 recovered, 4 had no recovery, and 2 were lost to follow-up. Of the 11 with recovery, improvement of the needle examination was seen in 7 patients, 2 did not show improvement, and 1 did not have the needle examination performed.

Discussion

This study revealed that a majority of our cohort presented with shoulder pain, and that trauma and neuralgic amyotrophy were the most common causes of SSN. A 5-year retrospective study done in 65 patients with EMG-confirmed SSN also identified trauma and neuralgic amyotrophy as common etiologies.[1] Although we defined sports-related and weight-lifting as separate etiologies, because weight-lifting SSN is likely secondary to overuse, our group had a high rate of SSN from weight lifting/sports-related etiology whereas the Hills et al study[1] had few cases.

In our study, 48% of patients had reported abnormalities on shoulder MRI and 43% had abnormal shoulder ultrasound studies. On detailed electrodiagnostic data review, we identified that suprascapular neuropathy is commonly associated with axillary neuropathy. Our study results are supported by a shoulder MRI study done on patients with Parsonage-Turner syndrome in which 50% of patients had both suprascapular and axillary nerve involvement.[11]

Another interesting aspect of our study was that patients with isolated SSN were more likely to have sport-related and idiopathic etiologies whereas those with SSN+ axillary neuropathy was more likely to have a traumatic etiology (MVA and falls). Patients in the axillary neuropathy group had a higher rate of a limited range of motion because three of the four rotator cuff muscles (supraspinatus, infraspinatus and teres minor) were affected compared to only two affected (supraspinatus and infraspinatus) in the isolated SSN group.

The diagnosis of SSN requires electrodiagnostic testing, and the data gathered on CMAP amplitudes may be related to the severity of the disease. Although one study found a direct relationship between EMG changes and treatment response,[12] neither our study nor Hill et al study[1] found any significant relationship. The difference may lie in treatment. Antoniou et al[12] reported that 68% of patients with SSN were treated operatively for compressive lesions and trauma whereas only 35% had surgery in the Mayo Clinic study[1] and 36% in our study. Perhaps these findings can be related to the relationship between the etiologic lesion causing the SSN, degree of compression, and therefore the EMG changes, and the mode of treatment. If there is a lesser degree of compression on the suprascapular nerve, with mild EMG changes, then there will not be an as great response to a surgical correction, as Antoniou et al[12] found. However, when examining a cohort of patients in which a minority of patients undergo decompressive nerve surgery, the CMAP amplitudes may be less predictive, and recovery will be based on a variety of other factors.[1]

In our study, 19% of patients had repeat electrodiagnostic study, with 65% of these showing recoveries. A total of 63% showed improvement in needle examination and only 27% showed improvement in NCS. These findings support the results of other studies where CMAP amplitude was helpful for the initial evaluation and severity of SSN but had little prognostic implication.[1,12] All of our patients had needle examination of the infraspinatus muscle, which was a weakness of the Hill et al study where only a small portion (12%) of patients had infraspinatus muscle testing.

It is important that patients with Sunderland grade III-V injuries are given an early referral to a nerve surgeon. There is a finite window in which surgical management can be performed, whether this involves nerve transfer or nerve grafting. [13, 14, 15, 16] In contrast, tendon transfer (i.e., salvage procedure) to restore shoulder external rotation can be performed at any point after injury.

Limitations of our study include the retrospective design and lack of suprascapular nerve conduction studies (CMAPs) in one-fourth of patients. Another limitation is the lack of documentation of pain level and exact measurements of range of motion during the follow-up visits. A third limitation is that the studies were performed by several clinicians. However, we were able to determine that most of the clinicians followed the guidelines and protocols established in our EMG laboratory to evaluate SSN. All patients underwent infraspinatus needle examinations and 87% underwent needle examination of the supraspinatus muscle.

Conclusion

Our study demonstrates the association of SSN with axillary neuropathy. This information serves to guide electromyographers to carefully evaluate patients with SSN who might need additional needle testing of axillary nerve-innervated muscles.

Abbreviations:

Suprascapular neuropathy (SSN)

Electromyography (EMG)

Electronic medical records (EMR)

Compound muscle action potential (CMAP)

Magnetic resonance imaging (MRI)

Medical Research Council (MRC)

Motor vehicle accident (MVA)

Motor unit action potentials (MUAP)

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Table 1: Demographic and patient characteristics

Variable	Response	All Patients (N=87)	Isolated (N=57)	Axillary (N=23)	Brachial Plexopathy (N=3)	Long Thoracic (N=2)
			Associated Nerve Groups			
Age at diagnosis	Mean (SD)	47.4 (16.5)	44.9 (16.9)	49.6 (14.3)	60.3 (17.9)	53.5 (17.7)
Sex	Female	30 (34%)	19 (33%)	8 (35%)	1 (33%)	1 (50%)
	Male	57 (66%)	38 (67%)	15 (65%)	2 (67%)	1 (50%)
Etiology	Idiopathic	21 (24%)	13 (23%)	2 (9%)	2 (67%)	2 (100%)
	MVA	13 (15%)	5 (9%)	8 (35%)	0 (0%)	0 (0%)
	Fall	14 (16%)	5 (9%)	9 (39%)	0 (0%)	0 (0%)
	Sports related	10 (11%)	8 (14%)	2 (9%)	0 (0%)	0 (0%)
	Weight lifting	13 (15%)	12 (21%)	1 (4%)	0 (0%)	0 (0%)
	Other causes[1]	8 (9%)	8 (14%)	0 (0%)	0 (0%)	0 (0%)
	Rotator cuff tear	8 (9%)	6 (11%)	1 (4%)	1 (33%)	0 (0%)
Rotator cuff tear	No tear	49 (59%)	34 (62%)	10 (43%)	2 (100%)	2 (100%)
	Small/partial	13 (16%)	8 (15%)	5 (22%)	0 (0%)	0 (0%)
	Large/full	21 (25%)	13 (24%)	8 (35%)	0 (0%)	0 (0%)
Side	Right	32 (37%)	17 (30%)	10 (43%)	2 (67%)	2 (100%)
	Left	55 (63%)	40 (70%)	13 (57%)	1 (33%)	0 (0%)
Pain	Yes	76 (87%)	49 (86%)	22 (96%)	2 (67%)	2 (100%)
	No	11 (13%)	8 (14%)	1 (4%)	1 (33%)	0 (0%)

[1] For the other etiology, they were related to dislocations (n=2), "Pumping water" (n=1), Lipoma in the suprascapular fossa (n=1), seizure-related shoulder dislocation (n=1), desmoid tumor (n=1), laberal tear with palpebral cyst (n=2).

Table 2: Clinical and EMG findings

Variable	Response	All Patients (N=87)	Associated Nerve Groups: Isolated (N=57)	Axillary (N=23)	Brachial Plexopathy (N=3)	Long Thoracic (N=2)	Radial (N=1)	Spinal Accessory (N=1)
Duration (months)	Mean (SD)	8.8 (11.8)	9.8 (12.4)	8.3 (11.5)	1.7 (1.3)	2.6 (0.4)	0.8	0.2
	Median (IQR)	4.6 (2.0, 10.7)	5.8 (2.3,12.2)	2.7 (1.8, 10.1)	1.7 (0.8, 2.7)	2.6 (2.3, 2.8)		
Supraspinatus atrophy	Yes	23 (28%)	14 (26%)	8 (35%)	1 (50%)	0 (0%)	0 (0%)	0 (0%)
	No	59 (72%)	40 (74%)	15 (65%)	1 (50%)	2 (100%)	1 (100%)	0 (0%)
Infraspinatus atrophy	Yes	37 (45%)	26 (48%)	9 (39%)	2 (100%)	0 (0%)	0 (0%)	0 (0%)
	No	45 (55%)	28 (52%)	14 (61%)	0 (0%)	2 (100%)	1 (100%)	0 (0%)
CMAP amp (mv)	Mean (SD)	2.8 (2.8)	2.8 (2.9)	3.0 (2.5)	2.4 (4.0)			
	Median (IQR)	2.4 (0.6, 4.0)	2.4 (0.6, 3.6)	3.2 (0.6, 5)	0.2 (0, 7.1)			
Supraspinatus fibrillatio	Presence	26 (34%)	17 (34%)	6 (30%)	1 (50%)	0 (0%)	1 (100%)	1 (100%)

n potentials								
	None	50 (66%)	33 (66%)	14 (70%)	1 (50%)	2 (100%)	0 (0%)	0 (0%)
Supraspinatus recruitment	Abnormal	45 (59%)	26 (52%)	15 (75%)	1 (50%)	1 (50%)	1 (100%)	1 (100%)
	Normal	31 (41%)	24 (48%)	5 (25%)	1 (50%)	1 (50%)	0 (0%)	0 (0%)
Infraspinatus fibrillation potentials	Presence	57 (66%)	37 (65%)	15 (65%)	3 (100%)	1 (50%)	1 (100%)	0 (0%)
	None	30 (34%)	20 (35%)	8 (35%)	0 (0%)	1 (50%)	0 (0%)	1 (100%)
Infraspinatus recruitment	Abnormal	76 (87%)	50 (88%)	21 (91%)	3 (100%)	1 (50%)	1 (100%)	0 (0%)
	Normal	11 (13%)	7 (12%)	2 (9%)	0 (0%)	1 (50%)	0 (0%)	1 (100%)
Recovery	Yes	50 (78%)	31 (79%)	16 (80%)	1 (50%)	1 (50%)	1 (100%)	0 (0%)
	No	14(21%)	8 (21%)	4 (20%)	1 (50%)	1 (50%)	0 (0%)	0 (0%)