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Clinical outcomes of suprascapular nerve decompression

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Background: While the incidence and prevalence of suprascapular neuropathy (SSN) remains largely unknown, the evaluation and treatment of SSN appears to be increasing. Despite multiple technique articles demonstrating nerve decompression, there has been no clinical evidence to support the efficacy of SSN decompression in the absence of rotator cuff disease.

Methods: Between October 2006 and February 2010, 27 patients underwent arthroscopic suprascapular nerve decompression at the suprascapular and/or spinoglenoid notch. Eighty-nine percent (24/27) of patients had preoperative positive electromyography and nerve conduction EMG/NCV studies documenting suprascapular nerve pathology. All patients had either a computed tomography (CT) arthrogram or magnetic resonance imaging (MRI) documenting rotator cuff integrity. All patients were evaluated with pre and postoperative subjective shoulder values (SSV) and American Shoulder and Elbow Society (ASES) self-assessment scores. Additionally, patients were questioned whether they would have the procedure again and approximately at what week they experienced noticeable pain relief.

Results: The 27 patients were followed for an average of 22.5 months (range, 3-44). Three patients were lost to follow-up. Seventy-one percent (17/24) of patients reported pain relief (VAS [Visual Analogue Scales] pain scale) that was statistically significant ($P = .0001$) at an average of 9.4 weeks from surgery. Seventy-five percent (18/24) and 71% (17/24) had statistically significant improvement in ASES ($P = .0001$) and SSV scores ($P = .0014$), respectively. Seventy-one percent (17/24) would have the surgery again.

Conclusion: The present study demonstrates a large series of patients treated for SSN without rotator cuff pathology. Our results show statistically significant improvement in VAS, ASES, and SSV.

Level of evidence: Level IV, Case Series, Treatment Study.

Keywords: Suprascapular nerve decompression; arthroscopic; outcomes

Recently, the evaluation and treatment of suprascapular neuropathy (SSN) have become a topic of increased research. Originally described in the French literature in 1936,[37] the clinical entity of SSN was further defined by Thompson and Kopell in 1959.[38] Prior to 2000, SSN was viewed as a diagnosis of exclusion. However, numerous studies have revealed the multifactorial causes of suprascapular neuropathy including mass effect often from a ganglion,[23,35,45] trauma and subsequent traction on the nerve,[36,40,42,46] repetitive overhead activities,[11,17,22,32,44] and rotator cuff tears.[25] Moreover, anomalous anatomic variants of the transverse scapular ligament and/or suprascapular artery

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have been described as well in the setting of SSN. The diagnosis of suprascapular neuropathy is usually made when an individual presents with subjective symptoms of posterior superior pain about the shoulder, isolated atrophy of the infraspinatus and/or combined atrophy of the supraspinatus, weakness of the supraspinatus and/or infraspinatus, and no other findings consistent with rotator cuff disease. Magnetic resonance imaging (MRI) findings of muscle edema and atrophy of the supraspinatus and/or infraspinatus, as well as abnormal electromyography and nerve conduction (EMG/NCV) studies, may be confirmatory.[15]

The prevalence and incidence of SSN remains largely unknown. A meta-analysis by Zehetgruber et al showed only 88 reports of this condition from 1959 to 2001.[47] However, with an increasing incidence in the literature of this condition during this past decade, the prevalence has been suggested to range from 12% to 33% in the athletic population and 8-100% in patients with massive rotator cuff tears.[7,11,17,22,25,41] The senior author reported a 4% (92/937) incidence in his exclusive shoulder practice.[4]

Lafosse et al were the first to report on the results of arthroscopic suprascapular nerve release at the suprascapular notch in the absence of rotator cuff tear.[21] He reported improved Constant scores as well as normalization of voluntary motor action potentials of the supraspinatus and infraspinatus at 6 months after surgery. Prior to this study, several studies evaluated the results of open ligament release showing improvement in subjective and objective outcome measures with excellent and good results ranging from 69% to 97%.[2,6,9,28] Since Lafosse et al's study, many other technique papers have been published that detail methods of decompressing the suprascapular nerve at the suprascapular and/or spinoglenoid notch,[12,33] as well as 1 case report that shows improvement in a Constant score from 51 to 84.[18] However, there have been no additional clinical evidence to support the efficacy of arthroscopic SSN when performed in the absence of significant rotator cuff disease.

The purpose of our study was to report our outcomes in patients who had an arthroscopic suprascapular nerve release in the absence of a rotator cuff tear. We hypothesized that release of the suprascapular nerve at the suprascapular and/or spinoglenoid notch would result in improved subjective and objective outcomes.

Materials and methods

Between October 2006 and February 2010, 27 patients underwent arthroscopic suprascapular nerve decompression at the suprascapular and/or spinoglenoid notch (secondary to cyst formation). Preoperatively, patients had a computer tomography (CT) arthrogram or MRI to document the absence of rotator cuff tear. This was further confirmed at surgery. Twenty-four of 27 patients had preoperative positive EMG/NCV studies documenting suprascapular nerve pathology. Three patients had negative EMG/NCV tests and were sent to for fluoroscopic guided steroid injection into the suprascapular notch, with a diagnostic response being complete temporary relief of pain after the injection. All patients were able to participate in a supervised rehabilitation protocol formulated by the senior author. Exclusion criteria included patients with rotator cuff tear and labral pathology resulting in anterior or posterior instability. Concomitant shoulder pathology such as acromioclavicular joint arthritis, superior labral anterior posterior (SLAP) tears, biceps tendonitis/tear, impingement syndrome, and adhesive capsulitis were included in the study.

Clinical presentation and examination

Patients in the current study uniformly presented with dull, deep, posterior shoulder pain insidious in its onset. Most patients complained of night pain and weakness and usually had advanced imaging documenting structural integrity of the rotator cuff. Inspection of the shoulder revealed some degree of atrophy of the supra and/or infraspinatus muscles. With testing, patients often had difficulty initiating abduction and had some degree of weakness with external rotation in adduction. Patients had features of other pathology as described above, but it was the deep, posterior aching in the shoulder which led us to consider the diagnosis of SSN.

Surgical technique

All patients were examined under anesthesia to document range of motion. Patients were positioned in a beach chair position using the Tenet and Spider arm holder (SPIDER Limb Positioner; Tenet Medical Engineering, Calgary, Alberta, Canada) for arm positioning.

A standard posterior portal and diagnostic shoulder arthroscopy was performed. Other intraarticular pathology was addressed as appropriate, including SLAP repair and capsular release as described below. The arthroscope was then placed in the subacromial space and a bursectomy with or without acromioplasty was performed based on clinical symptoms and radiographic findings of impingement. An arthroscopic distal clavicle excision was performed in 1 patient who had findings consistent with AC joint disease. Additional portals, primarily lateral (approximately 2-3 cm distal to the lateral edge of the acromion in line with the posterior aspect of the clavicle) and anterolateral (2 cm off the anterolateral edge of the acromion) were created for the decompression and subsequent nerve release. Our technique for suprascapular nerve decompression is similar to that as described by Lafosse et al.[21]

SSN decompression at the scapular notch

Utilizing the lateral portal for viewing and the anterolateral portal as a working portal, the coracoacromial ligament was identified and traced to its insertion on the coracoid. The lateral aspect and undersurface of the coracoid were skeletonized with a radiofrequency device to its base. A superior portal was made with needle localization in the trapezius approximately 7 cm from the lateral edge of the acromion, and a blunt instrument (scope trocar) was used to bluntly dissect and retract adipose tissue medially from the coracoid base (Fig. 1). The arthroscope was advanced in the plane between the coracoid base and the anterior edge of the supraspinatus muscle. The transverse scapular ligament (TSL) was identified along with the suprascapular nerve and artery, below