

A Tale of Two Suprascapular Neuropathy Presentations: A Case Report

Dustin H Massel¹, Ronald M Swonger², Sagie Haziza¹, Julianne Muñoz¹

Learning Point of the Article:

SSN neuropathy is a rare condition. It is a frequently overlooked diagnosis in the setting of pain and dysfunction of the shoulder. Surgeons should be suspicious of the diagnosis in all patients with shoulder pathology of unclear etiology, especially in patients who participate in repetitive overhead activity.

Abstract

Introduction: Supra-scapular nerve (SSN) neuropathy is a rare condition that can cause shoulder dysfunction. Prior literature has identified mass effect, space occupying lesions, trauma, and repetitive overhead activities as possible etiologies.

Cases Presentations: We report two cases of SSN neuropathy; a 21-year-old competitive female volleyball player with traction associated neuropathy and a 45-year-old male with a large paralabral ganglion cyst causing compressive neuropathy.

Conclusion: The following report highlights two different etiologies of SSN neuropathy and reviews the treatment of these conditions. Clinicians should consider SSN neuropathy in patients with unexplained shoulder pain and dysfunction.

Keywords: Suprascapular nerve neuropathy, suprascapular nerve entrapment, dynamic nerve compression, paralabral ganglion cyst.

Introduction

Suprascapular nerve (SSN) neuropathy is a rare condition that is often forgotten in the setting of shoulder dysfunction [1, 2, 3]. Prior literature has identified mass effect, space occupying lesions, trauma including rotator cuff tears, repetitive overhead activities, and anatomical variants as possible primary or secondary etiologies [4, 5, 6, 7]. The prevalence of SSN neuropathy accounts for 1–2% of shoulder pain cases, with elite competitive athletes engaging in repetitive overhead activities at a higher risk [7, 8]. In this report, we examine two different etiologies of SSN neuropathy in patients who presented to an outpatient clinic and review the treatment of these conditions.

medical history presented to outpatient sports medicine clinic with chronic right shoulder pain and weakness. The patient is a highly active student athlete competing on a Division I collegiate volleyball team as the outside hitter. In her final season, athletic trainers identified visible atrophy of the infraspinatus muscle and subjective dominant shoulder external rotation weakness without pain or discomfort. A full musculoskeletal exam was performed significant for reduced 4/5 right shoulder external rotation strength with considerable atrophy of the right infraspinatus muscle. Plain radiographs demonstrated no acute fractures or dislocations, without degenerative changes or obvious bony abnormality or deformity (Fig. 1). Magnetic resonance imaging (MRI) without contrast of the right shoulder demonstrated nonspecific scarring, fatty infiltration, atrophy, and edema of the infraspinatus muscle belly reflecting chronic denervation changes (Fig. 2). The fat planes surrounding the SSN at the level of the spinoglenoid and suprascapular notches

Case Report

Patient 1

A 21-year-old, right hand dominant, female (85.7 kg) without

Author's Photo Gallery



Dr. Dustin H Massel



Mr. Ronald M Swonger



Dr. Sagie Haziza



Dr. Julianne Muñoz

¹Department of Orthopaedic Surgery, University of Miami Hospital, Miami, Florida,

²University of Miami, Miller School of Medicine, Miami, Florida.

Address of Correspondence:

Dr. Sagie Haziza,

Department of Orthopaedic Surgery, 1611 NW 12th Street, University of Miami Hospital, Miami 33136, Florida.

E-mail: sagiehziza@gmail.com

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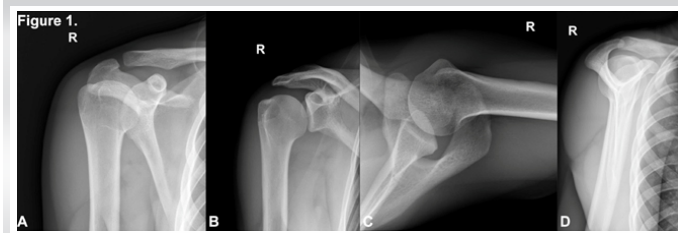


Figure 1: Normal (a) anterior posterior, (b) Grashey, (c) Axillary, and (d) Velpeau radiographs of the right shoulder in (Patient 1) a 21-year-old, right hand dominant, female without evidence of acute fracture or dislocation, bony abnormality, or deformity.

were preserved without evidence of a paralabral cyst, compressive or hypertrophic ligamentous pathology, labral tear, or bony abnormality. There was mild insertional infraspinatus tendinosis with low grade 0.5 × 0.5 cm articular surface partial-thickness tear.

The patient was diagnosed with dynamic SSN injury due to traction at the spinoglenoid notch. She was advised to refrain from overhead activity for 6 weeks and a prescription for rotator cuff and periscapular strength rehabilitation was provided. Nonsteroidal anti-inflammatory drugs (NSAIDs) were prescribed to help reduce inflammation associated with potential neuritis. At 6-week follow-up, the patient was compliant with rehabilitation 5 times per week and was progressing well without pain or discomfort when performing overhead activities. Despite subjective improvement in external rotation strength, the atrophy and objective weakness persisted. A strict rehabilitation and strength conditioning program were recommended to enhance external rotation strength and limit overhead hitting as much as possible. She is currently 8-months from her initial presentation and has completed her senior year season; her function and performance have not declined; however, she has not regained full strength or muscle mass.

Patient 2

A 45-year-old, right hand dominant, male (83.9 kg) presented to clinic for acute right shoulder pain without identifiable traumatic event. The patient reported an intermittent achy sensation over his right long head of the bicep tendon and posterior shoulder with radiation to the ipsilateral elbow worse with shoulder abduction and internal rotation. He had previously trialed conservative management with NSAIDs and

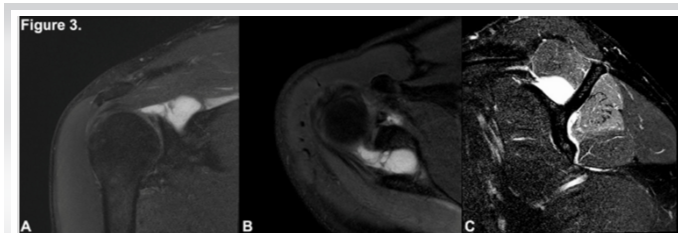


Figure 3: Abnormal magnetic resonance imaging of the right shoulder without contrast in (Patient 2) a 45-year-old, right hand dominant, male. (a) Coronal T2-weighted turbo spin echo fat saturated (TSE FS) image, (b) axial proton density (PD) TSE FS image, (c) sagittal T2-weighted TSE FS image demonstrating denervation edema of the supraspinatus and infraspinatus muscles associated with a 4.1 × 2.6 × 3.6 cm paralabral ganglion cyst and superior labrum anterior posterior tear.



Figure 2: Abnormal magnetic resonance imaging of the right shoulder without contrast in (Patient 1) a 21-year-old, right hand dominant, female. (a) Coronal proton density turbo spin echo (TSE) image, (b) axial T1-weighted image, (c) sagittal T1-weighted image, (d) sagittal T2-weighted TSE fat saturated image demonstrating scarring, fatty infiltration, and edema within the infraspinatus muscle reflecting chronic denervation changes.

a physical therapy program without improvement. On physical examination, he reported tenderness to palpation over the bicep tendon and posterior shoulder. He demonstrated reduced 4/5 right shoulder external rotation strength without evidence of muscular atrophy. Plain radiographs demonstrated no acute fractures or dislocations, no degenerative changes, or obvious bony deformities. MRI without contrast of the right shoulder demonstrated denervation edema of the supraspinatus and infraspinatus muscles with an associated paralabral ganglion cyst extending from the supraspinatus fossa displacing the muscle belly of the supraspinatus superiorly and tracking inferiorly around the spinoglenoid notch. It measured 4.1 × 2.6 × 3.6 cm and corresponded with an anterior-based superior labrum anterior posterior (SLAP) tear. The rotator cuff tendons were intact (Fig. 3).

Surgical decompression of the cyst and labral repair was recommended and performed without complications. The SLAP tear was anteriorly based from the 12 to 2 O'clock position. The posterior labrum and bicep tendon was intact without abnormality. The cyst was identified protruding from the posterior capsular space posterosuperior to the glenoid, which was somewhat unusual as the clear SLAP pathology was anterior. We attempted to decompress the cyst from the anterior labral pathology by reflecting it off the anterior glenoid neck. There was a small release of cystic appearing fluid with this maneuver, but it did not achieve release of the obvious bulging cyst in the posterosuperior capsular space. We then made a small rent in the capsular tissue directly over the bulging cyst and just off the posterosuperior labrum, which achieved a large egress of cystic fluid and decompression of the bulging cyst. The area was milked with an arthroscopic probe releasing more fluid intra-articularly. The SLAP tear was repaired with suture anchors at the 12:30 and 1:30 positions paying attention not to strangulate the biceps anchor. The capsular incision was repaired in side-to-side fashion with a knotless, all-suture anchor placed into the posterior glenoid neck at the site of the rent. Physical therapy was initiated at the 1-week follow-up visit. At his 6-weeks follow-up visit, he reported improvement in pain and range of motion, but was not compliant with his physical therapy protocol. At 3-month follow-up, he reported continued improvement with only mild intermittent discomfort in the



posterior shoulder, no tenderness to palpation, improved external rotation strength 5/5, and full range of motion. He was recommended to continue a home exercise program and all activity restrictions were removed. He is now 7-months post-operation and has had no complications or residual symptoms.

Discussion

SSN neuropathy is a rare pathology resulting from compression or traction injury anywhere along the SSN course from the upper trunk of the brachial plexus to its motor insertions at the supraspinatus and infraspinatus muscles [2, 9]. Injury can result in nerve ischemia, conduction impairment, and if left untreated, may result in irreversible damage [2]. Injury typically occurs as the nerve passes through the suprascapular and spinoglenoid notches. Suprascapular notch compression leads to impairment of supraspinatus and infraspinatus muscles [1, 2, 9]. In contrast, distal compression of SSN as it courses around the scapular spine at the spinoglenoid notch causes impairment of the infraspinatus muscle, exclusively [9]. Patients classically present with a gradual dull, achy pain in the posterior and lateral shoulder with weakness in abduction and external rotation, depending on location, and severity of injury [1, 9, 10]. On physical exam, supraspinatus and/or infraspinatus muscles atrophy along with shoulder abduction and external rotation deficits may be observed [11]. MRI is the preferred modality to assess for the pathologic cause of neuropathy [11]. Electromyography (EMG) and nerve conduction studies may be used as confirmatory studies [1, 11]. Identifying the causative etiology is necessary to determine the proper treatment approach [9].

Patient 1 was an elite competitive athlete performing repetitive overhead sport-specific activity resulting in traction associated SSN dysfunction. Conservative management is recommended for most patients with traction and dynamic overuse injuries with no focus of nerve compression [6, 9]. Treatment includes anti-inflammatory medications, activity modification, and physical therapy [1, 6, 9]. Many reports describe traction injury to SSN neuropathy in various overhead athletics including volleyball, golf, basketball, tennis, weight-lifting, and swimming [1, 12, 13, 14, 15, 16, 17, 18, 19, 20]. SSN neuropathy is more common in athletes playing volleyball with a prevalence of infraspinatus, supraspinatus muscle atrophy, and weakness in up to 20–45% of volleyball players compared to 12–33% across all athletes [12, 17, 21]. The exact mechanism of injury is contentious. Some authors describe compression of the SSN during terminal shoulder external rotation and abduction, or cocking phase and subsequent follow through [16]. Using EMG and nerve conduction study of 35 professional beach volleyball players, Lajtai et al. described infraspinatus atrophy in

34% of participants and significant external rotation and elevation strength deficits in 90% and 93% of their cohort's hitting shoulder, respectively [13]. EMG identified a greater latency in the hitting shoulder compared to the contralateral shoulder suggesting a demyelination mechanism [13]. The majority of prior literature evaluating SSN dysfunction in overhead athletes identifies isolated infraspinatus impairment from traction at the spinoglenoid notch as the most common cause. Patient 1 continues to respond well to non-surgical treatment. As she has now completed her volleyball career, we plan to monitor return of motor function and muscle mass.

Compression of the SSN from a paralabral cyst is the most common extrinsic or secondary cause of neuropathy. Compression neuropathy has been reported in patients with SLAP tears, ganglion cysts, Bennett lesion, ossified or hypertrophied superior transverse scapular and spinoglenoid ligaments [22, 23, 24]. In a 2018 systematic review, Memon et al. report the most common cause of compressive SSN neuropathy was a cyst at the spinoglenoid notch identified in 42% of patients [23]. Paralabral cysts are often associated with labral tears and can cause compression of the SSN at the spinoglenoid notch or suprascapular notches [22, 25]. Authors have described arthroscopic and open decompressions of the suprascapular and/or spinoglenoid notch, including possible transverse scapular ligament release [11, 14]. Prior literature supports surgical decompression with improvement in pain, strength, and function; however, some authors report variable return of muscle strength and mass [9]. In our second patient, an arthroscopic approach was used to decompress the paralabral cyst and repair the labral tear resulting in pain reduction, and improved strength and function.

Conclusion

While SSN neuropathy is an infrequent pathology in the setting of shoulder dysfunction, it is important to be aware of the possible etiologies. SSN neuropathy can be difficult to diagnose and may mimic more common shoulder pathologies [7]. SSN neuropathy should be considered in all patients with unexplained shoulder pain and dysfunction, especially in patients performing repetitive overhead activities.

Clinical Message

SSN neuropathy is a rare and overlooked condition in the setting of shoulder pain and dysfunction. This report describes two unique presentation of SSN neuropathy including the presenting signs, symptoms, and treatments, and discusses the clinical importance of suspecting the pathology.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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