Suprascapular Neuropathy

Rahul Singh[1], Ashish Babhulkar[1]

Abstract

Suprascapular neuropathy (SSN) is often missed or neglected. Although the occurrence is rare, it is for this reason that one needs to be aware of the correct clinical skill sets and investigations to nail the diagnosis. SSN pathologies are being diagnosed with increasing frequency in high probability groups like athletes, massive rotator cuff tear with fatty infiltration, labral tear and subsequent cystformation. With better understanding of the pathology, management of SSN has also improved in recent decades. With the advancement in electrodiagnostic modalities, SSN can be more reliably diagnosed now. The article highlights the aetiology and the points of compression, clinical picture to trigger the need for an EMG and the results of Arthroscopic decompression.

Keywords: Suprascapular neuropathy, paralabral cyst, transverse scapular notch, spinoglenoid notch, peripheralneuropathy.

Introduction

Suprascapular neuropathy was described first in 1957 in English literature[1]. In the past, it was chiefly considered as a pathology of athletic shoulder. Overhead sports such as baseball, volleyball, swimming, and tennis have been deemed high risk for suprascapular neuropathy [2, 3, 4, 5, 6, 7, 8, 9]. The prevalence of neuropathy varies between 12% and 33% in the athletic population but can reach as high as 52% in the high risk groups [8,10]. In recent decades, with the increased awareness and advancement in diagnostic modalities, suprascapular nerve (SSN) dysfunction has got increased attention. Ample research has been in done in the past two decades. Over the last decade, the association between massive rotator cuff tears and SSN has been studied extensively. Costouros et al. noted nearly 27% patient with massive rotator cuff tear (RCT) had concomitant SSN neuropathy [11]. Likewise, Shi et al. reported 39% patients of full thickness cuff tear also had nerve dysfunction [12]. However, this association had been argued lately and some authors suggest these could be mere associations [13]. Furthermore, authors had cited high degree of relationship between cuff fatty infiltrationand RC tear with nerve dysfunction [14, 15]. Beeler et al. noted distinct morphological feature in rotator cuff muscle after tear and neuropathy [16]. Clinical spectrum of SSN neuropathy varies from complete asymptomatic patient to shoulder impairment secondary to supraspinatus and infraspinatus muscle wasting and degeneration. SSN dysfunction could account for 1–2% of all shoulder pain [17]. With increasing awareness and careful examination, SSN pathologies are being diagnosed with increasing frequency in high probability groups such as athletes, massive rotator cuff tear with fatty infiltration, labral tear, and subsequent cyst formation [18]. With better understanding of the pathology, management of SSN has also improved in recent decades. Nevertheless, the etiology of neuropathy and indications for operative intervention remains elusive. Since SSN is rare it is seldom investigated and often missedhowever, with the advancement in electrodiagnostic modalities, SSN can be more reliably diagnosed now. Although, their sensitivity and specificity were concerning in the past, recent literature reports diagnostic accuracy of more than 90% [19].

Anatomy

A complete understanding of SSNanatomy is vital for shoulder surgeries. SSN arises from the upper trunk of brachial plexus and receives contribution from C5 and C6 nerve root in 76% (only C5=6%, C5+C6+C7=14%)[20]. After passing posterior to clavicle, the nerve travels obliquely to the suprascapular notch. Both Bigliani et al. and Greiner et al. had noted that nerve passes through suprascapular notch underneath the transverse scapular ligament [21,22].

[1]Department of Shoulder and Sports Injuries, Deenanath Mangeshkar Hospital, Pune, Maharashtra, India.

Address of Correspondence
Dr. Rahul Singh,
Department of Shoulder and Sports Injuries, Deenanath Mangeshkar Hospital, Pune - 411 004, Maharashtra, India.
E-mail: rahulortho85@gmail.com

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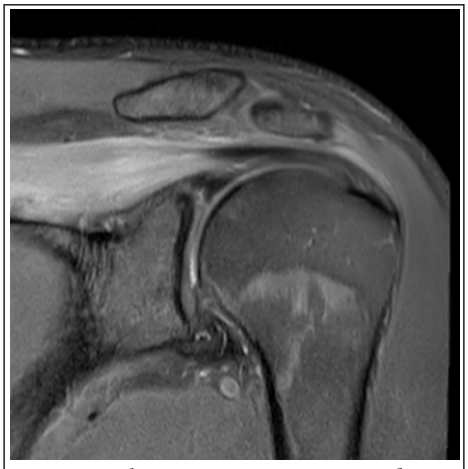

Figure 1: Classic parsonage turner syndrome MRI on coronal images.

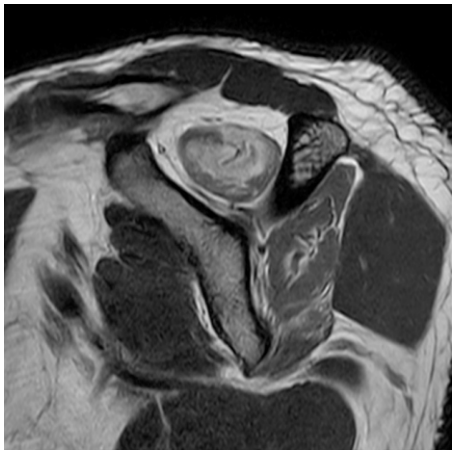

Figure 2: AClassic parsonage turner syndrome MRI T1 sag.

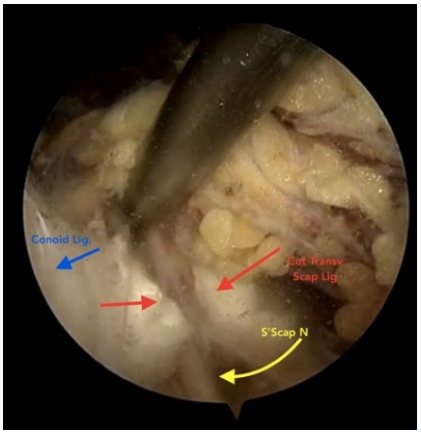

Figure 3: Arthroscopic SSN decompression at the transverse scapular notch.

Furthermore, the average distance of suprascapular notch from supraglenoid tubercle is 30 mm (range, 25–39mm) [21]. Rengachary et al. had described six different types of notches with increasing severity of notch narrowing, with type 5 and 6 with transverse ligament ossification. However, prevalence of type 5 and 6 lies between 10% and 20% [23,24]. Moreover, changes in notch morphology can be age related [24,25]. With advancing age,the notch becomes arrower and ossified [24,25]. In addition, three to six types of variations in superior transverse scapular ligament (STSL) shape have been described, but their clinical significance is yet to be ascertained [25,26]. Polguj et al. claim that the band like shape of the ligament significantly narrows the notch opening and hence, more likelihood for a neuropathy [27].

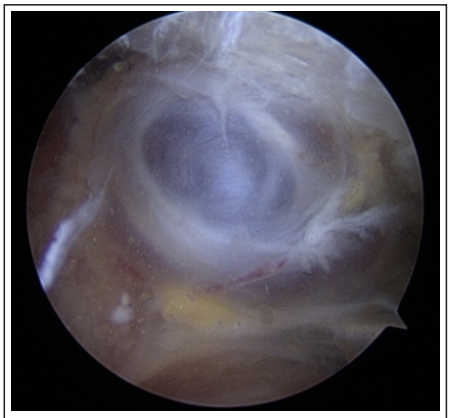

Figure 4: A large paralabral cyst exposed from Sub acromial side during arthroscopic decompression.

From suprascapular notch, the nerve runs obliquely toward the lateral side undersurface of supraspinatus muscle to the base of the scapular spine. Average distance of nerve at the scapular spine base is 25 mm (range,19–32 mm) from the supraglenoid tubercle and 18 mm(16–25 mm) from the posterior glenoid rim[22]. In 95% cases, motor branches within the supraspinatus fossa runs toward the medial side with nearly 8 mm freely underneath the muscle before intermingling [22]. From the base of the scapular spine, the nerve turns medially and within 10 mm, innervates the infraspinatus muscle by 2–4 branches[21]. Spinoglenoid ligament (inferior transverse scapular ligament) is an inconsistent structure and its true nature is debatable [28, 29, 30]. Recently, a large cadaveric study had proven its true ligament characteristics and its prevalence in all 58 specimens [30]. However, some authors reports its absence in 20% specimens [28]. Cummins et al. classified the spinoglenoid ligament as type 1 (thin fibrous band) and type 2 (true ligament)[28]. Another cadaveric study had highlighted its clinical significance and revealed its tightening with internal rotation at shoulder at all positions, making the nerve susceptible for compression [31].

SSN which was principally considered as motor nerve, based on recent literature, it seems to be a mixed nerve. It is Sensory branches supply the acromino-clavicular(AC) joint,glenohumeral (GH) joint, surrounding ligament, and skin [22]. Cadaveric studies have shown that even the first sensory branch can arise before entering into the notch in about 90% cases [22]. Evidently, nearly 70% of shoulder sensation are supplied by SSN which was further validated by clinical finding [32,33]. This also provides the rationale behind SSN block for pain relief after shoulder surgeries.

Aetio-pathogenesis

Numerous factors have been proposed for SSN neuropathy. In high demand athletes, micro-trauma from repetitive traction to a relatively tethered nerve has been blamed as a causal aetiology [2,7,9,22]. The peculiar anatomical course of the nerve makes it vulnerable to entrapment neuropathy. Suprascapular notch and spinoglenoid notch are two prime anatomical areas for nerve compression. To add, individuals with narrow notch,or the ossified transverse ligament, or band shape ligament are prone for compressive neuropathy [18,23]. Kim et al. recently reported one in every ten patient with RCT had ossified STSL[34]. Space occupying lesion from soft tissue or bone tumor or from cyst secondary to labral tear can cause neuropathy. Paralabral cyst is cited

as a major cause of compression at the at spinoglenoid notch but may extend to either side of scapular spine. Its proximity with posterior glenoid can be the contributing factor [1,35, 36]. Hosseini et al. had reported a concomitantSSN neuropathy and SLAP (superior-labrum anterior –posterior) lesion [37]. Spinoglenoid ligament may get stretched in internal rotation in any shoulder function [31].

SSN is also prone to traction injury after rotator cuff tear. One cadaveric study had revealed that retraction of supraspinatus tendon decreases the angle between the nerve and its motor branch and thus increases the tension on the nerve [38]. Shi et al. reported 18/46 patient with full thickness supraspinatus tear and 15/30 patient with complete infraspinatus tear had SSN neuropathy which was further validated with subsequent studies [10,11,12,39]. Ochiai et al. had studied a large sample size and concluded 13.4% (30/224) patient had SSN neuropathy with a large RCT, 25% patients (36/144) with massive cuff tear had nerve dysfunction, and 19.5% patients (16/82) with massive cuff tear with pseudo-paralysis had SSN involved [39]. More recently, researchers started arguing against this notion. Collins et al. failed to detect any association in massive RCT and SSN neuropathy [13]. They reported only 1 out of 49 patients, with retracted tear of both supraspinatus and infraspinatus, had SSN dysfunction.

Meyer et al. in anatomical study observed that retraction of muscle belly medial to the neural pedicle is potentially hazardous to the nerve [40]. Greiner et al. noted that 0.8–1 cm of medial motor branches of SSN are mobile [22]. This concept may possibly explain the disparity in literature. Rotator cuff mobilization for repair generally carries good prognosis. After average advancement of 2.5 cm (range, 2–3.5cm), Hoellrich et al. did not find any SSN dysfunction and concluded that 3.5 cm of cuff can be advanced without added threat to the nerve [41]. Warner et al. also noted that motor branches of nerve were under tension only after >3 cm of mobilization [42].

Suprascapular neuropathy also may occur autoimmune conditions such as rheumatoid arthritis, SLE as a part of systemic involvement [43]. Parsonage-Turner syndrome, a rare idiopathic neuritis, has also a definite predisposition for SSN[44,45][Figs. 1 & 2]. Gaskin et al. observed SSN involvement in 97% of patients with PTS with 50% patient with isolated suprascapular neuropathy [45].

SSN injuries have also been reported after traumatic events. GH dislocation, fracture around theshoulder and penetrating injuries can involve SSN[46, 47, 48, 49, 50, 51,52]. Visser et al. also reported good prognosis after traumatic injuries to SSN nerve indicating that most injuries are neuropraxia [51]. Recently, association between vascular anomalies around suprascapular and spinoglenoid notch and SSN compressive neuropathy have been reported[53,54,55]. Surgeon must be aware of these anomalies before undertaking surgical intervention.

SSN injury has also been described after a Latarjet procedure [56]. Laderman etal. illustrated increases screw angle for fixing coracoid graft may result in a nerve injury at the scapular spine[57]. Rarely, SSN can also get injured by anchor insertion while repairing SLAP lesions [58]. Sando et al. in their cadaveric study demonstrated that putting an anchor on posterior-superior glenoid through antero-lateral portal resulted in a high rate of glenoid perforation, risking the SSN[59]. Furthermore, placing inadvertent long screws in the posterior-superior quadrant of the base plate during reverse shoulder arthroplasty may injure the nerve[59].

Diagnostic Evaluation

It requires a high clinical suspicion to detect neuropathy in pure motor neuropathies. A SSN affection can frequently masquerade as a rotator cuff dysfunction. Gradual onset dull aching pain localized to posterior-lateral aspect of the shoulder is usually described for nerve dysfunction. Although, variable degree of night pain, fatigue, and muscle weakness has also been mentioned in association with overhead repetitive activity, history alone is a poor indicator for nerve dysfunction.

A thorough examination of cervical spine and bilateral shoulders is of paramount importance to rule out other possible pathologies such as rotator cuff tear and cervical spine lesions. Tenderness at the spinoglenoid notch by applying thumb pressure at back of the shoulder joint is indicative. Weakness of resisted external rotation and abduction along with wasting of supraspinatus and infraspinatus fossa can detected on physical examination [60]. A positive cross adduction test with the patient pointing to the back of the shoulder joint is more likely to be SSN compression. Pain on the AC joint (Finger sign) during the cross adduction test is classic for AC joint arthritis.

Since the SSN can be tethered at two different points the subsequent motor weakness is distinct. Lesion at the transverse scapular notch proximally will manifest with weakness of BOTH Supraspinatus and Infraspinatus. A distal lesion at the spinoglenoid notch will present with isolated wasting and weakness of Infraspinatus. In fact, isolated wasting of infraspinatus along with weak external rotation is diagnostic of neuropathy at spinoglenoid notch [31]. Patients with history and examination indicative of nerve dysfunction should undergo electromyography (EMG)nerve conduction velocity (NCV) and relevant investigations.

Radiographs are recommended as initial investigation to assess any fracture, osseous dysplasia, and bony tumors. Specialized suprascapular notch view can

delineate notch morphology and its variant. Computer tomographic (CT) scan has a limited value in majority of cases with nerve dysfunction. CT scan is best indicated when ossification of ligaments around nerve course is suspected and to define fracture geometry in a post-traumasituation.

Magnetic resonance imaging (MRI) is best suited imaging modality for certainconditions. An posterior abral tear with a paralabralcyst and rotator cuff wasting in addition to the nerve course can be assessed [61,62]. MRI accurately predicts the anatomical location and size of cyst which is imperative for surgical planning[63,64,65]. At times, MRI can depict the exact site of nerve entrapment by means of altered caliber of nerve and the extent of nerve injury [66,67]. Hour-glass appearance has been described for SSN on MRI for constrictive lesions [68].Furthermore, MRI greatly assists in diagnosing nerveinvolvement in inflammatory cause (parsonage turner syndrome) and concurrent brachial neuritis and cervical nerve pathology [67]. One can observe a focused demarcated high signal within the Supraspinatus and Infraspinatus on the T1 Sagittal images in parsonage turner syndrome.

Electrodiagnosticstudies, EMG, and NCVare considered as standard for diagnostic purpose. These studies are indicated in patients with persistent shoulder pain with unexplained muscle weakness. Furthermore, patients with fatty infiltration of supraspinatus infraspinatus muscle without evidence of tearare indications for EMG [69]. Overall sensitivity and specificity of electrodiagnostic studies can be variable and user dependent. However, Nardin et al. had reported 91% accuracy for EMG and NCV in SSN injury [19]. Moreover, in patient with massive RCT, preoperative electrodiagnostic studies may help to identify SSN dysfunction although, this remain debatable [70]. LeClere et al. reported four cases of suprascapular neuropathy with fatty infiltration on supraspinatus and infraspinatus muscles without associated tear on MRI and the EMG was positive in three cases[71]. With nerve conduction studies, nerve latency value from Erb point to supraspinatus and infraspinatus muscle is critical. Sensory velocity interpretation is less informative as the sensory area is not well defined. EMG demonstrates denervation as well as re-innervation potential for muscles with sharp waves and resultant fibrillation. Bilateral shoulder evaluation has also been proposed to compare both side and to improve diagnostic efficacy further.

Musculoskeletal ultrasonographyis a useful and convenient tool in the right hands[72]. Actual demonstration of atrophy (volume loss) along with the presence of fatty infiltration in a muscle or a group of muscles (Infraspinatus in isolation or Infraspinatus with Supraspinatus) is a reliable surrogate sign of nerve entrapment. Demonstration of a para-labral cyst is a corroborative finding reinforcing the diagnosis of SSN dysfunction and suggestive of a posterior labral tear. Absence of a rotator cuff tear with extremely poor muscle recruitment clinches the diagnosis if SSN compressive neuropathy.

To diagnose SSN neuropathy early one has to have a high clinical suspicion. Although it mimics a full thickness rotator cuff, one should watch for the fallacies. Patients with SSN neuropathy tend to be younger and their Subacromial (SA) space is uninvolved. Hence, Hawkins sign and impingement signs could be negative. The onset of wasting of Supraspinatus and Infraspinatus is rapid within a few weeks unlike the modest wasting associated with a full thickness rotator cuff tear. The occurrence of trauma may not be elicited and often the pain can be disproportionate to the radiographic and MR findings. As Burkhart clearly opined, approaching extra-articular transverse scapular notch in a genuine SSN neuropathy is easy as there is profound wasting of the said muscle. On the contrary, if there is no significant wasting, approaching the nerve under the clavicle can be difficult and may be a sign for the surgeon to verify his diagnosis.

Treatment

Conservative

Literature is sparse regarding non-surgical management of SSN neuropathy and restricted to small case series. Although, trial of physical therapy and activity modification can be offered to patients with mild, early stages without concurrent space occupying lesions or cuff tear [6,73]. Initially, scapular stabilization and muscle strengthening exercises must be promoted while avoidingvigorous activities and overhead exercises. Posture correction may have significant impact on reducing traction on the SSN. Lambardo et al. and Walsworth et al.,in their small case series reported improvement in 60%–100% patients with physical therapy [74,75]. Drez et al. noticed benefit with therapy in isolated suprascapular neuropathy in all four patients and recommend a trial of 6–8 months of conservative management [76]. Martin et al. with a relativelysizeable cohort of 15 patients, reported excellent result in five, good in seven and poor in three patients at nearly 4 years follow-up [73]. The exact causal pathway for improvement with physical therapy is not known. Finally, Callahan et al. had reported high failure rate with physical therapy at three to 6 months and advocated surgical release in all cases [77]. To add, Post et al. suggested to execute surgical release earliest to prevent muscle atrophy and assert physical therapy in setting of nerve compression in contraindicated [78]. In a proven SSN compressive neuropathya conservative line of treatment is likely to be suboptimal when compared to surgical treatment. However, a short trial of conservative treatment may be appropriate for SSN as a consequence of

overuse [73]. A delay ina genuine compressive SSN case may lead to irreversible muscle atrophy and weakness.

Operative treatment

Surgical treatment, in modern days, is entirely arthroscopy driven. The surgeon ought to plan the level of decompression and addressing any collateral lesions such as SLAP tear, posterior labral tear with or without paralabral cyst. When both supraspinatus and infraspinatus are involved, then it is mandatory to decompress both the suprascapular and spinoglenoid notch. If only the infraspinatus is involved clinically and on EMG and MRI shows focal infiltration restricted to the infraspinatus, then only decompression of spinoglenoid notch is obligatory. Surgical decompression is most beneficial in patients with actual compressionrather than a traction injury [79].

At suprascapular notch

Apart from formal compression at the suprascapular notch, extended indications would be in primary massive retracted cuff tear and anatomical variations such as arterial aneurysms, calcified transverse scapular ligament, and post scapular fracture malunions. Space occupying lesion is mainly accountable for compression at suprascapular notch. In majority of patient, treatment involves release of STSLwith decompression and removal of any mass if present. With arthroscopic techniques showing uniform success, open decompression is now limited to historical archives. Major disadvantage of open procedure was the recurrence of symptoms because of entrapment of nerve in fibrous tissue from the extensive dissection[75].

Arthroscopic technique was further illustrated by Lafosse et al. for ligament release[80] [Fig. 3]. Lafosse etal. recently publish their short term results and reported complete normalization in 7/10 patients, partial recovery in 2/10. Lafosse, in addition to clinical improvement also documented improvementon electrodiagnostic study with a meanfollow-up of 6 months, while, all patients resumed their work and sport activities within a mean of 3 weeks[80]. LeClere et al. had also reported complete resolution of symptom after SSN release at suprascapular notch in all patients(4/4) [71]. To add,Oziumi et al. further reported complete recovery of sensory disturbance around shoulder after arthroscopic decompression of the SSN nerve [81].

Tsikouris et al. had performed an interesting study to find out treatment options for SSN neuropathy occurring in association with other shoulder pathologies (rotator cuff tears, labral tear, and SA impingement) in professional athletes [82]. Their results indicated better mid-term results in terms of functional outcome and return to sport activity in patient in which an additional nerve release was done with shoulder arthroscopy, than without nerve decompression.

Recently, Viagsio et al. had reported poor outcome with arthroscopic/ minimal invasive notch decompression in hour glass like SSN constriction [83]. They suggested considering hour glass like constriction of nerve as for arthroscopic/minimal invasive surgeries as it might need an extensive neurolysis. Gerber et al. performed a randomized control trial and concluded no additional benefit of nerve release with cuff repair for massive tear. Instead, 3/9 patient with nerve release showed pathological EMG finding at 3 month [84].JP warner drew our attention to the fact that in massive rotator cuff tears, SSN can be under traction leading to neuropathy [42]. However, some authors recommended against prophylactic SSN release in patient with RCT [84].

At spinoglenoid notch

Nerve compression by a spinoglenoidcyst at spinoglenoid notch is the most common etiology for SSN neuropathy[85]. Spinoglenoid cyst secondary to labral tear and SLAP lesions are responsible for majority of nerve compression[63,86, 87]. Tirman et al. noticed all of their patients with spinoglenoid cyst were associated with a labral pathology on MRI [86]. Kimet al. reported 24/28 patients with spinoglenoid cyst had SLAP lesions[87]. Spinoglenoid notch decompression can be done arthroscopically either through GHorSAapproach.

The GH approach is simpler, involves only one viewing portal (the antero-superior portal to visualize the posterior labral repair and related decompression) and in terms of results is equal to the SA approach. The main drawback of the GH approach for spinoglenoid is an indirect decompression without actually visualizing the paralabral cyst. Seldom has the cyst failed to decompress but the closure of the posterior labral tear effectively decompresses the SSN. This would entail a standard arthroscopic posterior labrum or SLAP repair procedure.

As for the SA approach, the surgeon would have to switch the arthroscope between GH and SA joints. After an initial diagnostic round and confirming a posterior labral tear, the surgeon would then proceed to the SA joint and identify the raphe between the supraspinatus and infraspinatus with a blunt liberatorandseparate the two muscles at the raphe. Right underneath the rotator cuff is the large para-larbal cyst which can be identified and bluntly dissected and excised in-toto. The major advantage of the SA approach is direct visualization of the paralabral cyst [Fig. 4], complete excision and also superior visualization of the paralabral cystand also the supra scapula nerve directly. The major drawback of this procedure is the requirement to move the arthroscope to and frobetween the two joints and requires a little bit of experience to

identify the raphe and dissection. The SA approach is the preferred approach of the senior author.

Nolte et al. in their retrospective analysis with long term follow up of about 5 years, reported significant improvement in function and muscle strength without any complication or revision with arthroscopic nerve release [88]. Chen et al. observed resolution of SSN neuropathy in all their patients (3/3) after arthroscopic cyst decompression and SLAP repair[89]. Likewise, Lichtenberg et al. reported improvement in muscle strength and function in all patients managed arthroscopically for SSN neuropathy by cyst decompression and labral tear[90]. Other studies had also reported similar findings. Few authors suggested labrum repair alone without cyst decompression as adequate for SSN neuropathy secondary to a spinoglenoidcyst.Youmet al. repaired only the labrum inpatients with associated spinoglenoidcyst and SSN palsy andreinnervation of infraspiantus was recorded in EMG in all patients.The cystresolved on follow-up MRI after an average follow-up of 10 month[91]. Similarly debridement of SLAP lesion only resulted in cyst resolution on MRI and reinnervation on EMG[92]. However, Piatt et al. observed that significantly higher patient satisfaction with a combined cyst decompression and labrum repair then decompression alone[64].

Conclusion

Isolated suprascapular neuropathy is relatively rare and mainly occurs as a result of anatomical variation. This condition may easily be overlooked as it is largely a motor nerve and its sensory supply is restricted to the shoulder joint capsule. Overhead athletes are more prone for nerve dysfunction. MRI and electrodiagnostic studies are useful tools in diagnosis, in addition to documenting recovery. Paralabral cyst secondary to labral tear and rotator cuff tear are associated with isolated Infraspinatus dysfunction. Conservative treatment has a limited role and that too in early neuropraxia stage without overt muscle atrophy.Arthroscopic release remains the gold standard.

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

Conflict of Interest: NIL; **Source of Support:** NIL

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