Suprascapular neuropathy related to rotator cuff tears: a short review

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Abstract

Introduction

Suprascapular nerve neuropathy related to rotator cuff tears has become a topic of discussion. However, there are a few studies to support suprascapular neuropathy in cases of (massive) rotator cuff tears. There are also a few studies showing the indication for concomitant suprascapular nerve release; however, several clinical and basic studies provide some evidence that suprascapular nerve injury may be a part of the disease spectrum of rotator cuff pathology. In this short review article, suprascapular neuropathy related to rotator cuff tears is discussed.

Conclusion

Till date, there are a few studies to support suprascapular nerve neuropathy in cases of (massive) rotator cuff tears. Future studies will hopefully serve to elucidate these issues.

Introduction

Suprascapular nerve (SSN) entrapment neuropathy occurring at the transverse scapular notch was first described by Thomas in 1936. Although isolated SSN neuropathy is a relatively rare phenomenon, its aetiology is thought to be related to traction or compression of the nerve. Commonly referred to as a ‘sling effect’, this mechanism is well reported in the literature. Patients with SSN entrapment often present with pain, weakness and numbness of the shoulder and a history of symptoms provoked by dominant upper extremity movement. The condition may also be associated with atrophy of the supraspinatus or infraspinatus muscle. Therefore, this manifestation closely resembles that of rotator cuff tears.

Recently, SSN neuropathy related to rotator cuff tears has become a topic of discussion. Rotator cuff tears medialise muscle fibres and tether the SSN at the suprascapular notch, thus resulting in suprascapular neuropathy. When advancement of the rotator cuff or interval slides (anterior or posterior) is indicated in cases of massive cuff tears, the SSN needs to be retracted further, thus increasing the risk of damage.

In this short review article, we discuss suprascapular neuropathy related to rotator cuff tears.

Discussion

SSN anatomy

The SSN is a mixed peripheral nerve that contains motor and sensory fibres and originates at the ventral rami of the spinal nerves C-4, C-5 and C-6 or at the upper trunk of the brachial plexus. The SSN provides motor innervation to the supraspinatus and infraspinatus muscles. In addition, it branches to the coraco-humeral and coraco-acromial ligaments, subacromial bursa and acromioclavicular joint.

After approaching the shoulder joint, the nerve passes through the suprascapular notch before supplying motor branches to the supraspinatus muscle and sensory branches to the acromioclavicular and glenohumeral joints (Figure 1). The nerve continues past the spinoglenoid notch and supplies motor branches to the infraspinatus muscle.

Historically, the SSN has been thought to have no cutaneous branches; however, Horiguchi described the existence of sensory cutaneous branches in 1968. In a report by Ajmani, cutaneous branches of the SSN were observed in 14.7% of the cases. On the other hand, Vorster et al. observed in 87.1% of the cases. In addition, Habbaugh et al. reported a single case of SSN entrapment characterised by an area of numbness involving the upper lateral shoulder region. Anatomical studies have shown that the morphology of the suprascapular notch is highly variable in terms of width and shape. Rengachary et al. examined adult cadaveric scapulae and categorised the suprascapular notch shape into six types. Their categorisation of notch shape is regarded as a de facto standard. A U-shaped notch (Type 3) was the most common type and was identified in 48% of the cadavers examined. The morphology of the transverse scapular ligament (TSL), including width, thickness and rate of ossification, was also highly variable. The TSL is a unique ligament that connects to different parts of the scapula. The biomechanical role of the ligament is unclear; however, Moriggl et al. described the fibrocartilage at the enthesis of the TSL, suggesting that the ligament responds to both compressive and tensile loading. There is a complimentary relationship between the width of the suprascapular notch and the thickness of the TSL. Anterior to the TSL, an accessory ligament named as the anterior coracocapsular ligament is present in 60% of the specimens. Although its impact on

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SSN entrapment neuropathy remains unclear; the anterior coracocapsular ligament substantially narrows the suprascapular foramen. A narrow notch shape together with a thick TSL has been hypothesised to be associated with nerve entrapment; however, no direct correlation has been demonstrated.

Rotator cuff tears and SSN neuropathy
Several clinical and anatomical studies suggest a relationship between rotator cuff tears and SSN neuropathy. Moreover, measures to relieve pain after failed cuff repair have been explained in the context of SSN decompression. Retracted rotator cuff tears medialise muscle fibres and tether the SSN, which is relatively fixed at the point where it passes through the suprascapular notch. More extensive retraction (such as that in the case of massive rotator cuff tears) causes increased nerve traction. During retraction, the nerve is vulnerable to compression. This is more of a concern when advancement of the rotator cuff or interval slides (anterior or posterior) is indicated. Several authors have confirmed that lateral advancement of the supraspinatus muscle causes compression of the nerve and should be limited to 1 cm, otherwise the branches of the SSN may be damaged. Warner et al. examined 31 cadaver shoulders and found that the SSN was relatively fixed on the floor of the fossa underneath the TSL. The motor branches to the supraspinatus muscle were significantly shorter than those to the infraspinatus muscle. In 84% of the shoulders examined the first motor branch originated underneath the TSL or just distal to it. In one shoulder (3%), the first motor branch passed over the ligament. The standard anterosuperior approach allowed only 1 cm of lateral advancement of either tendon. Greiner et al. evaluated 24 cadaver shoulders to assess SSN damage during advancement of the supraspinatus muscle for rotator cuff repair. They identified four different patterns of branching and courses of the SSN: (1) branches located medial to the scapular notch (95.8%), (2) branches running in a posterior direction and crossing the bottom of the muscle on an extramuscular course (54.2%), (3) branches running in a posterior direction on an intramuscular course (12.5%) and (4) branches located lateral to the scapular notch, whereby they either remained inside the supraspinatus fossa (25%) or ran toward the infraspinatus fossa (16.6%). They found that the entire neurovascular pedicle was tethered after advancement; however, the subgroup with the branches located medial to the notch was at a greater risk of tethering and tension during advancement of the muscle 1 cm laterally.

Vad et al. retrospectively defined the prevalence of peripheral nerve injury associated with full-thickness rotator cuff tears presenting with shoulder muscle atrophy. The prevalence of associated suprascapular neuropathy was observed in 8% (2/25) of the patients examined by electrophysiological testing, including nerve conduction studies and needle examination. Mallon et al. reported two of four patients with massive retracted rotator cuff tears and suprascapular neuropathy who demonstrated reinnervation potentials after partial arthroscopic rotator cuff repair. Costouros et al. reported that all six patients with preoperative electrophysiologically confirmed suprascapular neuropathy showed nerve recovery after partial

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or complete rotator cuff repair. Asami et al. reported a case of bilateral SSN entrapment associated with rotator cuff tears. In this case, decompression of the SSN by releasing the TSL brought diminished pain, weakness and atrophy of the supraspinatus and infraspinatus muscles. Kaplan and Kernahan also reported six cases of the rotator cuff tears concomitant with SSN neuropathy diagnosed by electromyography (EMG). However, Zanotti et al. retrospectively reviewed the incidence of cuff repair and injury to the SSN after mobilisation and repair of a massive rotator cuff tear. After a mean follow-up period of 2.5 years, electromyographic examination confirmed that one of 10 patients had an iatrogenic SSN injury. They concluded that operative injury to the SSN during rotator cuff mobilisation can occur; but other factors such as inadequate cuff muscle function are more frequently responsible for the poor functional outcomes observed after successful repair of massive rotator cuff tears.

Other researchers proposed that severe fatty degeneration in patients with massive rotator cuff tears are caused by SSN damage. Rowshan et al. examined a rabbit subscapularis tenotomised model and concluded that fatty infiltration of the muscle occurs with chronic rotator cuff tears and may be explained by nerve injury. Liu et al. demonstrated that significant and consistent muscle atrophy and fatty infiltration were observed in the rotator cuff muscles after rotator cuff tendon transaction. They found that denervation significantly increases the amount of muscle atrophy and fatty infiltration after a rotator cuff tear. However, using a rabbit model, Gayton et al. showed that rotator cuff tears do not affect the motor endplate or innervation status of the supraspinatus, suggesting that fatty degeneration occurs independent of denervation of the supraspinatus.

**Diagnostic studies**

EMG of the SSN is thought to be a useful diagnostic tool in cases of primary SSN neuropathy. However, the sensitivity and specificity of EMG and Nerve Conduction Velocity (NCV) varies from 74% to 91%, thus a negative result does not necessarily preclude SSN neuropathy. Furthermore, because SSN entrapment related to rotator cuff tears is thought to be a dynamic phenomenon, the co-existence of SSN neuropathy and rotator cuff tears may not always be demonstrable on EMG.

Magnetic resonance imaging is also helpful in identifying changes in the supraspinatus and infraspinatus muscles secondary to denervation, such as decreased muscle bulk, fatty infiltration and homogeneous high-signal intensity on T2-weighted images. Beeler et al. observed the appearance of fatty infiltration and muscle atrophy on magnetic resonance imaging when comparing cases of rotator cuff tears and suprascapular neuropathy. They found that chronic rotator cuff tendon tears and suprascapular neuropathy were both associated with fatty infiltration and muscle atrophy; however, the pattern of fatty infiltration is markedly different in the two situations. These findings may have diagnostic potential.

Lafosse et al. described the ‘SSN stretch test’, which was judged to be positive (related to SSN neuropathy) if it reproduced or exacerbated pain in the posterior aspect of the shoulder. In this test, the clinician stands behind the patient and performs lateral rotation of the patient’s head away from the affected shoulder, holding it gently in position while using the other hand to gently retract the shoulder. However, they did not present clinical data in this study, and the clinical value of this technique is still under evaluation.

**Indication for SSN decompression**

Whether to release the SSN in cases of massive rotator cuff tears remains an unanswered question. The open technique for decompression via a superior incision requires extended dissection and is technically difficult. With advances in arthroscopic technique, studies have reported the advantages of all-arthroscopic decompression of the SSN over the open technique. All, but one, of the currently performed arthroscopic decompression techniques are through the subacromial space, although Reineck and Krishnan reported a technique for viewing and approaching from the anterior to the coracoid. They used a portal created medially to the coracoid to explore and dissect the TSL. All of these procedures could be performed continuously or antecubital with arthroscopic rotator cuff repair. It is regarded as a difficult procedure and is associated with a steep learning curve, though Lafosse et al. described that release of the nerve is a safe and simple technique with little risk of additional complications. Till date, there has been no compelling study showing a clear relationship between rotator cuff tears and SSN neuropathy, suggesting that there is no clear indication for SSN decompression concomitant with rotator cuff repair, even if technically possible. Lafosse et al. proposed the following indications for SSN release: patients presenting with weakness of the infraspinatus with or without wasting of the supraspinatus, with or without pain, and with or without positive EMG findings; patients with a thickened or ossified ligament on assessment during arthroscopic rotator cuff repair and patients who present with posterior shoulder pain with a positive SSN stretch test. However, large numbers of massive cuff tear cases are with muscle atrophy and no direct correlation has been demonstrated between SSN neuropathy and morphology of the suprascapular notch or TSL. Further research will be needed to determine the indications and optimal technique for decompression of the SSN.
be necessary to fully understand the role of decompression as well as the long-term outcomes of arthroscopic decompression.

Conclusion
Till date, there are a few studies to support SSN neuropathy in cases of (massive) rotator cuff tears. There are also a few studies showing the indication for concomitant SSN release; however, several clinical and basic studies provide some evidence that SSN injury may be a part of the disease spectrum of rotator cuff pathology. Future studies will hopefully serve to elucidate these issues.

Acknowledgement
The author would like to thank Enago (www.enago.jp) for the English language review.

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