

Shoulder Impingement Syndrome, A Common Affliction of the Shoulder: A Comprehensive Review

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ABSTRACT

The shoulder joint is the most mobile joint in the human body. It comprises a ball and socket structure that enables a series of functional movements to be carried out. These range from simple movements such as reaching for the top shelf to highly explosive overhead activities which involve throwing a baseball or serving with a tennis racquet as seen in athletes. Shoulder impingement syndrome (SIS) is one of the most common shoulder disorders seen in general practice. It is caused by a multitude of factors. Pathology can arise either from the rotator cuff tendon itself, structures external to this tendon or both in combination. The resultant shoulder pain with SIS not only causes distress but also limits the quality of life of patients. This article aims to provide a comprehensive review of the pathogenesis, aetiologies, clinical tests, investigations and management options for SIS based on current literature and research.

Keywords: Shoulder impingement syndrome, Subacromial impingement syndrome, Rotator cuff tendinopathy, Rotator cuff tendinitis, Shoulder pain

INTRODUCTION

Shoulder pain is a disabling symptom frequently encountered in primary care. The estimated prevalence of shoulder complaints is 7–34%¹ with about 14.7 new cases per 1000 patients per year seen in clinics². Of all the shoulder disorders, shoulder impingement syndrome (SIS) is the most commonly reported, accounting for 44–65% of all shoulder pain complaints². In simple terms, SIS can be defined as a collection of shoulder symptoms and signs caused by pathology within the rotator cuff tendon itself (intrinsic) or structures external to it (extrinsic), causing impingement in the narrowed space between the acromion and humeral head³. At times, both intrinsic and extrinsic pathologies may occur in combination.

The multi-factorial aetiology of SIS makes it difficult to clinically localise the lesion and formulate an effective treatment plan. Given the high prevalence and diagnostic challenges involved in this syndrome, the aim of this review is to evaluate the pathogenesis, aetiologies, diagnostic accuracy of clinical tests,

imaging modalities and various conservative and surgical treatment options relating to SIS.

PATHOGENESIS

In 1972, Neer described three stages of the extrinsic impingement process⁴. Stage-I impingement is characterised by the oedema and haemorrhage of the subacromial bursa and rotator cuff which is usually seen in patients less than 25 years old. Stage-II impingement demonstrates irreversible changes such as fibrosis and tendinitis of the rotator cuff seen in those aged 25–40 years old. Stage-III impingement is characterized by more chronic changes such as partial or complete-thickness rotator cuff tears, usually seen in patients aged more than 40 years old. From this three-stage impingement classification, it is evident that SIS is associated with rotator cuff tendinitis and subacromial bursitis. However, the issue of tendon/bursal inflammation is not without controversy as a casual relationship between the extrinsic impingement mechanism and the resultant inflammation has not yet been firmly established.

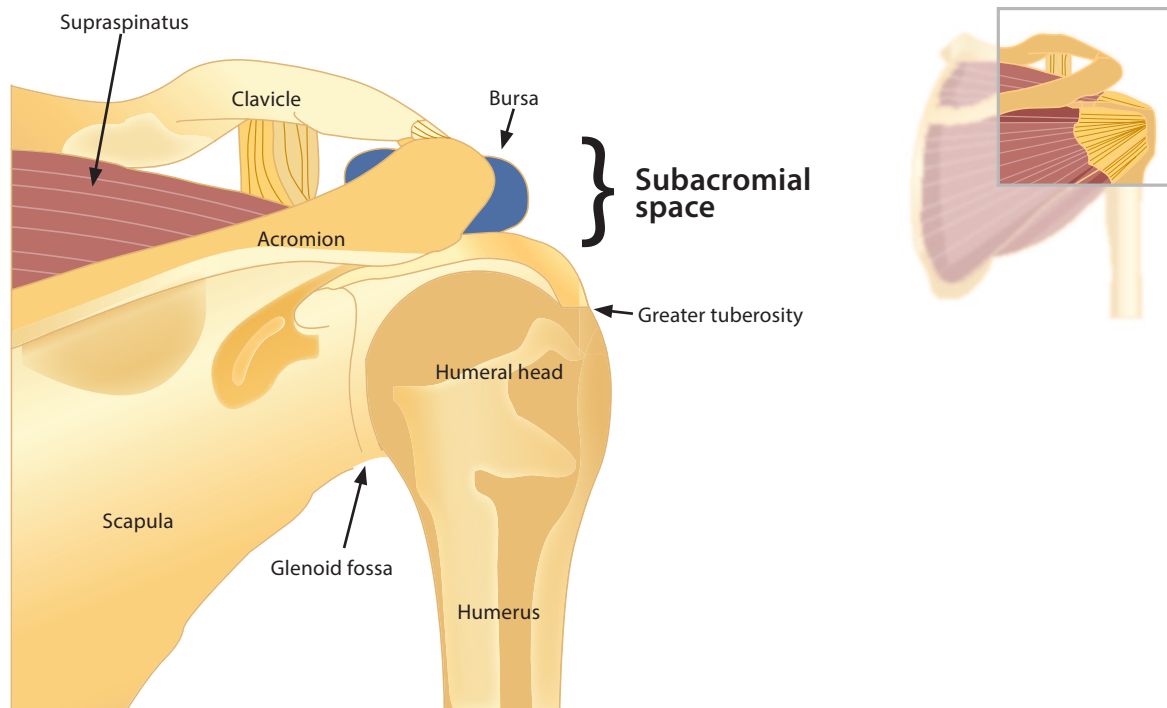


Fig. 1. The structures associated with the subacromial space.

AETIOLOGY

According to Neer, the subacromial space is defined inferiorly by the humeral head and superiorly by the coracoacromial arch which comprises three structures: the under surface of the anterior third of the acromion, coracoacromial ligament (CAL) and coracoid process⁵. The acromioclavicular joint (ACJ) is situated superior and posterior to the CAL⁵ (Figs. 1 and 2). As defined earlier, SIS can be caused by intrinsic (intratendinous) or extrinsic (extratendinous) factors or both³.

Intrinsic Factors

The "intrinsic impingement" theory postulates that degenerative changes due to age, overuse, trauma or tension overload result in partial or full thickness tears of the rotator cuff tendon, thereby causing SIS.⁶

Two prospective studies^{7,8} have demonstrated a statistically significant increase in prevalence of full-thickness rotator cuff tears with increasing age. Yamaguchi *et al.* also found that the average size of a symptomatic tear was 30% greater than an asymptomatic tear⁸. As the tear size was shown to be a factor in symptomatic development, they recommended yearly interval monitoring for tear size progression in patients undergoing non-operative treatment for symptomatic or asymptomatic full thickness rotator cuff tears.

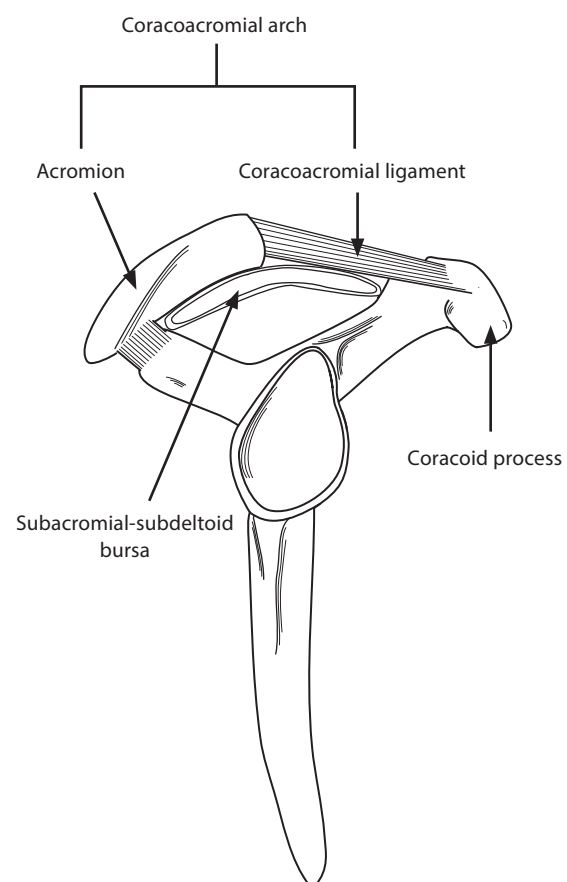


Fig. 2. The structures comprising the coracoacromial arch.

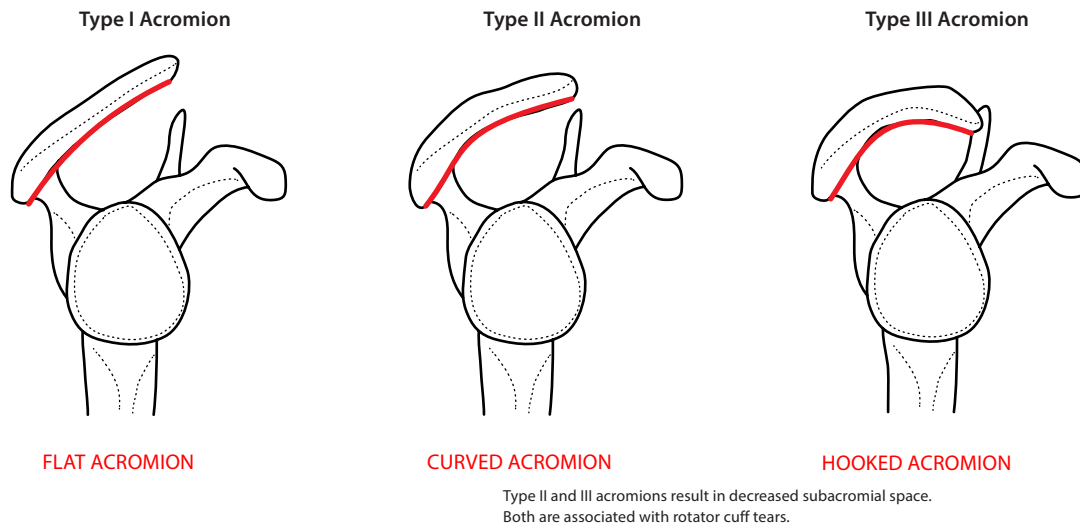


Fig. 3. The three different structural types of acromion.

In sports that require overhead motion such as swimming, throwing or racquet sports, athletes tend to overuse their rotator cuff tendons, causing inflammation and thickening to the tendons (tendinitis) and subacromial bursa (bursitis), this contributes to the impingement process. This overuse phenomenon can also be seen in jobs that require frequent heavy lifting of goods.

Extrinsic Factors

Neer's theory of "extrinsic impingement" described the mechanical compression of the supraspinatus tendon by the inferior under surface of the anterior third of the acromion, the CAL and ACJ⁵. This can occur as a result of anatomical factors, abnormal rotator cuff and scapular musculature or poor body posture⁶. Other causes include an os acromiale (unfused distal acromial epiphysis) and posterior capsule tightness⁶.

Anatomical factors include acromial morphology variations, and degenerative changes at the inferior surface of the acromion, ACJ or CAL. Bigliani *et al.* described three distinct acromial morphologies: Type I (flat), Type II (curved) and Type III (hooked) acromion⁹ (Fig. 3). He argued that due to the shape and resultant damage, the Type II and Type III acromions had a greater predisposition to a rotator cuff tear and hence SIS⁹. This classification has been widely criticised due to poor inter-observer reliability¹⁰. More recent studies^{11,12} have not shown a significant association between acromial morphology and rotator cuff pathology. Gill *et al.* found a significant correlation between age and rotator cuff pathology and argued that the Type III

acromion was more likely a result of a degenerative process (acquired) rather than a morphological variation (congenital)¹². According to Neer, arthritic changes to the ACJ can occur with age, causing joint space narrowing and osteophytes to form at the distal clavicle and acromion articulation⁵. The CAL can also cause stress-induced acromial spurs on the undersurface to form due to the higher tension on the acromial insertion of the CAL as compared to the coracoid side¹³. A thickened CAL caused by repeated strain overtime can also cause narrowing of the subacromial space.

Weak or dysfunctional rotator cuff musculature can cause superior translation of the humeral head¹⁴, thereby narrowing the subacromial space, resulting in SIS. Scapular musculature serves to stabilise and rotate the scapula during movements. During overhead arm movements, weak scapular muscles fail to elevate the scapula and acromion sufficiently, causing impingement of the underlying rotator cuff muscles¹⁵.

Furthermore, a slouched posture involves an increase in thoracic kyphosis, downwardly rotated, anteriorly tilted and protracted scapula, thereby decreasing glenohumeral joint flexion, elevation and abduction range¹⁶. In light of this, proper postural assessment and scapular and rotator cuff muscle strengthening through physiotherapy may help improve impingement symptoms.

With two different impingement theories, it is difficult to ascertain cause and effect. Did the intrinsic tendon degeneration or the extrinsic

structures cause the impingement process in the first place? Confusion regarding the impingement aetiology can occur due to overlap of factors. For example, athletes in addition to rotator cuff tendinitis caused by overuse may also have underlying glenohumeral instability. By the time they present with shoulder symptoms, it is clinically challenging for the physician to ascertain if the rotator cuff tendinitis was caused by overuse activity or due to subtle glenohumeral instability. In this case, glenohumeral instability as the primary cause should be ruled out first using imaging studies¹⁷ before diagnosing overuse.

External vs. Internal Impingement

Impingement syndrome is an umbrella term which can also be classified into external (outlet) versus internal (inlet). External impingement also known as subacromial/shoulder impingement refers to any pathology or structures encroaching in and hence narrowing the subacromial space. This forms the main focus of our article. However, this should not be confused with a more subtle internal impingement; which refers to any pathology affecting structures within the glenohumeral joint space itself. These structures include the under-surface (articular side) of the supraspinatus tendon, infraspinatus tendon and posterior-superior glenoid labrum. In 1992, Walch described the internal impingement process as a condition caused by repetitive contact of the posterior aspect of the greater tuberosity of the humeral head with the posterior-superior aspect of the glenoid labrum¹⁸. As a result, this impinges and damages both the articular side of the supraspinatus tendon and the glenoid labrum in the process¹⁸. The classic thrower's position typically seen in baseball players; with the arm in 90 degrees abduction and full external rotation causes this internal impingement¹⁸. This clinical entity is beyond the scope of this article.

CLINICAL EVALUATION AND DIAGNOSIS

History and Physical Examination

Shoulder impingement syndrome has a tendency to be overdiagnosed as the primary cause of shoulder pain. A careful and thorough history is essential in ruling out more subtle or sinister causes. Patients often complain of pain in the anterolateral aspect of the shoulder. This site corresponds to the insertion of the supraspinatus tendon at the greater tuberosity of the humerus. Pain is made worse with overhead movements such as reaching for the top shelf or in athletes involved in throwing,

racquet sports or swimming. The onset of the pain in SIS can be acute in a recent traumatic shoulder injury or more chronic if the impingement is due to osteophytes. Other associated symptoms include pain when lying on the affected shoulder, weakness and loss of arm function.

On physical examination, it is important to check for any signs of shoulder swelling, deformity, tenderness, muscle wasting or stiffness. Both active and passive range of shoulder motion should be tested. Abduction of the arm in the 60–120° angle range causes the greater tuberosity of the humerus to impinge on the under surface of the acromion, causing anterolateral shoulder pain. This is the painful arc sign which can indicate a minor rotator cuff injury or a supraspinatus tendinitis.

Special Clinical Tests

Two systematic reviews^{19,20} found low specificities each for Hawkins-Kennedy test, Neer's sign and empty can test for the diagnosis of SIS and did not recommend individual tests for pathognomonic diagnosis of SIS.

Park *et al.* evaluated eight well known clinical tests to determine their diagnostic values to help distinguish between bursitis, partial-thickness rotator cuff tears and full-thickness rotator cuff tears (n=913)²¹. For the diagnosis of SIS, the best combination of three tests was a positive: Hawkins-Kennedy test, painful arc sign and weakness in external rotation with arm at the side (best post-test probability 95%)²¹. To diagnose a full-thickness rotator cuff tear, the best combination of three positive tests was: painful arc sign, drop arm sign and weakness in external rotation (best post-test probability 91%)²¹. As shown above, using a combination of tests as opposed to tests in isolation tends to provide a more reliable result. This is because the rotator cuff unit is a dynamic structure where all the muscles and tendons work synergistically in a compound movement. Rotator cuff tendons do not function as separate entities²². In reality, it is difficult to truly isolate one muscle or tendon using each individual clinical test alone, although it is possible to provoke more pain at the specific muscle or tendon tested. Furthermore, concomitant pathology affecting other adjacent structures such as the subacromial bursa which contain nociceptors (innervations from lateral pectoral nerve, suprascapular nerve) can cause pain²³, thereby affecting the reliability of the tests.

Imaging Studies

The three main imaging modalities for the shoulder in SIS are plain film radiographs, ultrasonography (US) and magnetic resonance imaging (MRI) or arthrography (MRA).

Routine radiographs involve three standard views. These include the antero-posterior (AP) view, scapular Y view and axillary view. AP radiographs may demonstrate a narrowed subacromial space, subacromial osteophytes, sclerosis of the acromion undersurface ("sourcil" sign) and subchondral sclerosis or cyst formation in the greater tuberosity of the humerus^{3,14}.

Ultrasonography and MRI are both useful in excluding partial and full-thickness rotator cuff tears. This important information not only has prognostic value but also guides orthopaedic surgeons in their choice method of surgical repair (open or arthroscopic). Two prospective studies had demonstrated that US and MRI had comparable accuracy for identifying and measuring the size of partial and full-thickness rotator cuff tears^{24,25}. Compared to MRI, US tends to be more operator dependent but is not claustrophobic, is less costly and more easily accessible as it can be performed in a clinic or at the bedside. A study comparing patient satisfaction on both US and MRI showed that most patients prefer US as an imaging modality for shoulder pain²⁶. Iannotti *et al.* had demonstrated MRI to have great diagnostic value in distinguishing a normal tendon from one with tendinitis and impingement signs, sensitivity and specificity were 93% and 87% respectively²⁷.

However, another study evaluating the MRI results of 96 asymptomatic individuals revealed a high prevalence of rotator cuff tears in all age groups²⁸. The overall prevalence of tears was 34%, of which 15% were full-thickness tears and 20% were partial thickness tears²⁸. The study also found a significant correlation of rotator cuff tears with increasing age in individuals who had normal, painless shoulder function²⁸. These findings highlight the potential dangers of diagnosing rotator cuff tears and dictating surgery on the basis of MRI alone, without complementing the results with the overall clinical picture.

Magnetic resonance arthrography is useful for assessing the glenoid labrum and glenohumeral ligaments for ruling out bankart lesions and

glenohumeral instability respectively. The contrast (gadolinium) enhancement enables good visualisation of intra-articular anatomy to detect any damage. A full-thickness rotator cuff tear is diagnosed if the contrast dye leaves the glenohumeral joint and enters the subacromial space. For the detection of rotator cuff pathology, MRI is less sensitive than MRA due to its inability to detect partial-thickness tears and associated soft-tissue injuries²⁹. Although minimally invasive, arthroscopy is currently the only technique which enables direct visualisation of all the glenohumeral joint structures. Once a lesion is detected on a magnified screen, the surgeon can also subsequently choose to treat it.

MANAGEMENT PLAN

Conservative Methods

Patient Education

Patients should be educated on the potential early warning signs of impingement through advice and educational leaflets. Early intervention in the form of activity modification and adequate rest can prevent further deterioration of pain, strength and function. In the early stages of impingement, patients should generally avoid activities which involve raising the arm over the head such as reaching, lifting, cleaning, climbing or other activities which may aggravate the pain. The main message to convey to patients is a gradual return of shoulder function within the limits of pain.

Physiotherapy

Physiotherapy for SIS can involve multiple interventions ranging from simple advice to structured rehabilitative exercises, manual joint mobilisations, acupuncture and electrotherapy³⁰. The main goals of a structured shoulder exercise program are to relieve pain, restore joint range of motion, increase strength, improve proprioception and promote healing³⁰. A quantitative and qualitative systematic review and meta-analysis of 16 studies (n=1162) demonstrated a statistically significant benefit in terms of pain and function with multiple exercises involving, stretching, scapular stabilising and rotator cuff strengthening for the shoulder³⁰. These findings highlight the importance of a structured and varied exercise program being incorporated as part of shoulder rehabilitation in SIS patients.

Oral Non-Steroidal Anti-inflammatories (NSAIDs)

Oral NSAIDs include aspirin, ibuprofen and

naproxen. Although there are reports indicating there is little or no inflammation involved in rotator cuff tendinopathies, many studies³¹ have shown that oral NSAIDs seem to improve pain and clinical outcomes in the short term. This could be due to the decreased pain which allows the patient to carry out physiotherapy exercises which strengthen the rotator cuff muscles. A randomised double-blinded placebo-controlled trial carried out on 100 patients with a painful shoulder showed oral naproxen ($P=0.02$) was superior to a placebo in the treatment of painful shoulder³². We recommend a short course of NSAIDs for two weeks as there are long term gastrointestinal side effects associated with its use. If longer term use is required, coverage with proton-pump inhibitor drugs such as omeprazole can be instituted.

Corticosteroid VS NSAID Injections

When more conservative measures have failed, corticosteroid injections together with local anaesthetic can also provide symptomatic relief and improved function due to its anti-inflammatory mechanism. A randomised double-blinded control trial on 58 patients compared the subacromial injection of tenoxicam (NSAID) with methylprednisolone (Corticosteroid) in SIS patients³³. Both groups were injected with lignocaine. Outcome measures were assessed using the Constant-Murley Shoulder Score (CMSS), Disability of Arm, Shoulder and Hand (DASH) and the Oxford Shoulder Score (OSS). At six weeks post-injection, CMSS was significantly greater in the methylprednisolone group than the tenoxicam group ($p=0.003$)³³. Improvement in DASH score was also statistically significant at week 2 ($p<0.01$), week 4 ($p<0.01$) and week 6 ($p<0.02$) post injection³³. Oxford Shoulder Score improvement was also consistently greater in the corticosteroid group but not statistically significant at week 6 ($p=0.055$)³³.

These findings suggest that corticosteroid injections are more effective than NSAID injections in improving shoulder outcomes in the short term. However, there is a lack of evidence in the study to suggest the long term effectiveness of both injection groups. Another more recent randomised control trial on 32 SIS patients showed that single ketorolac (NSAID) injections demonstrated better outcomes than triamcinolone (Corticosteroid) injections at four weeks follow-up³⁴. Evidence from both studies may be conflicting but both do indeed show that NSAIDs and corticosteroids are effective.

Long-term use of corticosteroid injections can cause immunosuppression, spontaneous tendon rupture, localised osteoporosis, skin depigmentation around injection site and should thus be administered with caution^{33,34}. In patients who are concerned about the long term side effects of corticosteroids, NSAIDs may be a safer alternative.

Other Conservative Treatments

Extracorporeal shock-wave therapy for rotator cuff tendinitis has not shown to be of any additional benefit when compared to placebo in recent studies^{35,36}. Similarly, ultrasound therapy combined with exercise and NSAIDs was of little or no benefit when compared to placebo in another study³⁷. More large scale trials are needed to confirm the value of ultrasound therapy in improving pain and functional outcomes. As these treatments become commercialised in the private healthcare sector, patients have to approach these modalities with caution.

Surgical Methods

After a 3- to 6-month trial of failed conservative measures, surgical intervention is the next treatment option. To increase the subacromial space (subacromial decompression), the anterior inferior third of the acromion is resected (acromioplasty) together with either a release or removal of the CAL³. Sometimes, a posterior capsule release may also be performed. ACJ resection is only done if the joint is tender or if osteophytes are contributing to impingement. With the advent of shoulder arthroscopy, open acromioplasty is gradually becoming more obsolete.

Arthroscopic Subacromial Decompression

In 1985, Ellman devised this alternative technique. His study performed a 1–3 year follow up on the pre and post-operative outcomes of 50 consecutive arthroscopic subacromial decompression (ASD) cases³⁸. Eighty per cent of the cases had stage II impingement without rotator cuff tear and 20% had full-thickness rotator cuff tears. Patients were evaluated according to the UCLA shoulder rating scale which assesses pain, function, range of motion, strength and patient satisfaction³⁸. The majority of the cases (88%) were rated satisfactory and the remainder (12%) unsatisfactory³⁸. Arthroscopic subacromial decompression is now the gold standard as arthroscopy allows direct visualisation inside the glenohumeral joint to detect other pathologies and the option to treat on the spot³⁹.

For ASD to be successful, the diagnosis has to be a primary mechanical (extrinsic) impingement process. Arthroscopic subacromial decompression is usually indicated for young and active patients with stage II impingement syndrome who are eager to resume their daily or sporting activities. Older patients may also undergo ASD if a series of conservative measures fail to address the structural impingement. Complications of ASD include acromial fractures or insufficient acromion or osteophyte removal, necessitating a revision procedure³.

The operation can be carried out either in the lateral decubitus position or beach-chair position. The latter being favoured by surgeons as this not only reduces the risk of neurological injury³, but also enables easy manipulation of the humerus during surgery. In the procedure, three incisions or portals are created around the shoulder. The anterior portal is for saline inflow or outflow, posterior portal for camera viewing and lateral portal for instrumentation. Alternatively, two portals can also be created around the shoulder⁴⁰. The anterior portal is used as an instrumentation portal and the posterior portal for the arthroscope⁴⁰. The arthroscope is introduced in the glenohumeral joint which is filled with saline to allow visualisation. The subacromial space can also be viewed on screen and any inflamed subacromial bursa removed via a bursectomy. A high speed burr is deployed to trim the anterior inferior undersurface of the acromion to prevent impingement. The idea is not to shorten the acromion undersurface but to smoothen it by shaving off any bony spur irregularities. If necessary, the distal 1 cm of the clavicle together with any surrounding osteophytes can be resected with the surgical burr. Over time, the space between the acromion and clavicle fills with scar tissue which strengthens and stabilises the ACJ.

There has been much clinical debate regarding the routine release or resection of the CAL. Studies on cadaveric shoulders with deficient rotator cuffs showed post-operative proximal migration of the humeral head after CAL resection⁴¹. One clinical study of 10 patients who had revision surgery following failed symptomatic relief after ASD with partial resection of the CAL was observed to have complete regeneration of the CAL which exhibited histology indistinguishable from normal ligament⁴¹. The study raised the possibility of the CAL regeneration as being the cause of ASD failure

but concluded that this was part of the normal healing process and was unlikely. Nonetheless, the study did not recommend the routine excision of the CAL in SIS patients with rotator cuff disruption as this causes proximal migration of the humeral head thereby causing shoulder instability. In addition to an ASD, the rotator cuff tendons can also be inspected and repaired if necessary.

Surgical versus Conservative Methods

Although there have been reports documenting the favourable clinical outcomes of surgical intervention, much of the current literature evidence fails to show any significant differences between surgical and conservative treatment for SIS. A systematic review of four randomised controlled trials failed to demonstrate any differences in outcome between both surgical and conservative treatment groups with SIS⁴². However, several studies⁴³ have also documented favourable clinical outcomes for SIS patients undergoing surgical intervention who were non-responsive to conservative treatment. The take-home message is that surgery should only be considered after a trial of conservative measures has been fully exhausted. The mechanism of impingement, the clinical history and tests, imaging results, responsiveness to conservative treatment as well as patient's wishes should be considered before proceeding with surgical intervention for SIS.

CONCLUSION

As SIS can present with other shoulder pathologies, the clinician has to keep an open mind with a list of differential diagnoses. It is also important to identify the underlying pathology causing SIS as this determines the management plan. SIS has a good prognosis as non-operative and operative treatments are usually successful in addressing the impingement mechanism. This is made possible if early clinical evaluation, prompt diagnosis and treatment is undertaken.

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