The Painful Shoulder: Shoulder Impingement Syndrome

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Abstract: Rotator cuff disorders are considered to be among the most common causes of shoulder pain and disability encountered in both primary and secondary care.

The general pathology of subacromial impingement generally relates to a chronic repetitive process in which the conjoint tendon of the rotator cuff undergoes repetitive compression and micro trauma as it passes under the coraco-acromial arch. However acute traumatic injuries may also lead to this condition.

Diagnosis remains a clinical one, however advances in imaging modalities have enabled clinicians to have an increased understanding of the pathological process. Ultrasound scanning appears to be a justifiable and cost effective assessment tool following plain radiographs in the assessment of shoulder impingment, with MRI scans being reserved for more complex cases.

A period of observed conservative management including the use of NSAIDs, physiotherapy with or without the use of subacromial steroid injections is a well-established and accepted practice. However, in young patients or following any traumatic injury to the rotator cuff, surgery should be considered early. If surgery is to be performed this should be done arthroscopically and in the case of complete rotator cuff rupture the tendon should be repaired where possible.

Keywords: Shoulder impingement syndrome, rotator cuff, arthroscopic subacromial decompression (ASAD).

INTRODUCTION

Rotator cuff disorders are considered to be among the most common causes of shoulder pain and disability encountered in both primary and secondary care, with subacromial impingement syndrome in particular being the most common disorder, resulting in functional loss and disability, of the shoulder [1].

The concept of shoulder impingement syndrome is attributed to Charles Neer following his paper published in 1972 [2]. The term shoulder impingement itself however now belongs to a group of terms that essentially describes pain in the shoulder region as a result of mechanical ‘impingement’ of the rotator cuff as it passes under the coraco-acromial ligament. If left untreated rotator cuff impingement may proceed to partial or complete rotator cuff tendon rupture.

Diagnosis of this condition remains a clinical one and an initial careful assessment is crucial in identifying shoulder impingement as the particular cause of shoulder pain from the list of differentials. Early recognition and subsequent management is important also as this can help reduce the risk of impingement progressing and causing increased further morbidity to patients in the form of pain, reduced activity or subsequent partial or even complete rotator cuff tears.

Advances in imaging modalities have enabled an increased understanding of the pathological process and specific causes of shoulder impingement. Plain x-rays are useful as an initial shoulder evaluation tool in the majority of shoulder pathologies however in the case of rotator cuff disorders, including shoulder impingement, they are often normal initially and require supplementation with other imaging modalities such as ultrasound or MRI scanning.

Depending on both the stage of the condition and individual patient factors, there are a variety of treatment options available; however a patient specific treatment plan should always be implemented. In the majority of cases an initial conservative approach may be completely adequate in leading to a resolution of symptoms, however in certain cases, various surgical options are available which look to address the cause of the impingement symptoms i.e. the acromion, rotator cuff or both.

This review article aims to look holistically at ‘Shoulder Impingement’ and specifically review the evidence base for diagnosing and treating this commonly encountered condition.

PATHOLOGY

The shoulder girdle is made up of 3 bones, the scapula, clavicle and humerus. The shoulder also consists of 3 main joints, the sternoclavicular, acromioclavicular and glenohumeral joint. The glenohumeral joint is the most mobile in the body and relies on the static and dynamic stabilisers to remain centred during its large range of motion. Dynamic stabilisers relate to the muscles around the shoulder including the rotator cuff.
The pathology of subacromial impingement generally relates to a chronic repetitive mechanical process in which the conjoint tendon of the rotator cuff undergoes repetitive compression and micro trauma as it passes under the coracoacromial arch [3]. As the arm is abducted or rotated the subacromial space width changes and the cuff becomes increasingly compressed. The supraspinatus is in closest contact to the anterior inferior border of the acromion in 90 degrees of abduction with 45 degrees internal rotation [4]. Patients with impingement tend to externally rotate the arm in order to allow the cuff to occupy the widest part of the subacromial space thereby relieving the symptoms.

The supraspinatus tendon is the most commonly implicated rotator cuff muscle in shoulder impingement. It mainly derives its blood supply from the anterior circumflex humeral and suprascapular arteries. Within the supraspinatus tendon, near its insertion at the greater tuberosity, there is an avascular or ‘Critical’ zone. It is here ‘impingement’ usually occurs and this zone has been found to increase in area with advancing age [5-7].

Neer outlined 3 stages of shoulder impingement:

Stage 1 described a process of acute inflammation, oedema, and hemorrhage of the rotator cuff conjoint tendon. This stage affects younger patients normally aged below 25 years of age and is usually reversible with conservative treatment alone.

Stage 2 affects patients between 25-40 years of age and represents a continuation of the process, outlined in stage 1, to a more irreversible form. As the tendon becomes swollen there is increased friction further perpetuating the problem. In this stage, the rotator cuff tendon undergoes fibrosis and tendinitis.

Stage 3 affects older patients usually over the age of 40 years. The key factor in stage is that there is an actual mechanical disruption of the rotator cuff tendon in the form of either partial or complete cuff tears. In this stage, changes also occur in the coracoacromial arch such as osteophyte formation, which may also reduce the subacromial space [8].

Aside from subacromial impingement, other less common forms of shoulder impingement include subcoracoid impingement and Internal Impingement.

Subcoracoid impingement is a relatively rare cause of anterior shoulder pain. Symptoms occur when there is impingement of the subscapularis on the coracoid process and lesser tuberosity of the humerus. It typically presents with anterior shoulder joint pain in activities involving forward flexion, adduction and internal rotation. It tends to occur in patients with a prominent coracoid in forward flexion and internal rotation of the shoulder [9, 10].

Internal or posterosuperior impingement syndrome has an unclear aetiology with a number of hypotheses being suggested. Examples of these include initial anterior shoulder instability or micro-instability, contracture of the posterior capsule or scapular dyskinesis. This particular form of impingement however seems to occur when the arm is abducted extended and externally rotated causing internal contact of the posterior rotator cuff with the posterosuperior aspect of the glenoid [11].

CLASSIFICATION

The mechanisms involved in leading to impingement syndrome are likely to be a multifactorial in origin. Shoulder impingement itself has been classified however into primary and secondary types, with primary shoulder impingement relating to a mechanical compromise of the rotator cuff as a result of either intrinsic factors, extrinsic factors or a combination of both with the precise interplay between the two not always being entirely clear.

Intrinsic causes relate to a problem with the tendon itself which occur secondary to either an acute or chronic processes. The degenerative cuff constitutes the commonest cause of cuff failure and usually occurs in the older individual following a chronic repetitive process which has been previously outlined.

Acute causes relate to either a traumatic event or secondary to calcific tendonitis both of which may damage to the rotator cuff and lead to partial or complete tears. Traumatic cuff failure occurs when the upper limb is subject to a violent force and the rotator cuff subsequently sustains a traumatic tear. In some instances the tendons may avulse their bony attachment. Calcific tendinitis of the shoulder is a process involving calcium deposition commonly in the rotator cuff tendon and can present as acute cause of shoulder pain. The pathogenesis is not completely clear but the calcified mass that occurs within the tendon can lead to impingement under the subacromial arch [12-14] and subsequent problems for the rotator cuff.

The shape of the acromion, the attachment of the coracoacromial ligament and changes in the acromioclavicular joint have all been implicated as causes for extrinsic causes of primary shoulder impingement. With regards to the acromion in particular, Bigliani classified the acromion into three categories with a progressive increase in the incidence of impingement from a type 1 to type 3 acromion. Type 1 relates to the acromion being flat in shape, Type 2 reflects a more curved acromion which lies parallel to the humeral head and a Type III acromion is where the edge of the acromion is hooked and therefore may impinge on the rotator cuff on elevation of the arm [3]. It has been demonstrated that patients with a type 3 acromion have increased risk of impingent. Other extrinsic causes include osteophytes under the acromioclavicular joint secondary to osteoarthritis, subacromial bursitis and a thickened coracoacromial ligament, among others. These all reduce the subacromial space and can lead to cuff impingement and failure.

Any cause which leads to a dysfunction of either glenohumeral and/or scapulothoracic movement may lead to subacromial impingement also. In athletes where repetitive overhead activity is required, the act of throwing may subsequently lead to the pathological process outlined by Neer. Secondary impingement is usually associated with repetitive overhead activity resulting in glenohumeral instability [15, 16]. Ludewig et al. performed a study looking at translation of the humerus in patients with shoulder impingement. They concluded that the identified kinematic deviations in their study were consistent with possible reductions of the subacromial space and therefore may contribute to the development or progression of shoulder instability.
impingement symptoms [17]. Struyf et al. reviewed the literature in scapular positioning between unimpaired shoulders and in shoulder impingement. They found differences between these groups and although the literature was inconsistent regarding scapular resting position. During shoulder elevation, they found most researchers agreed that the scapula tilts posteriorly and rotates both upward and externally. In patients with shoulder impingement however they found that there was a decreased upward scapular rotation, a decreased posterior tilt, and a decrease in external rotation [18-20].

**ASSESSMENT AND INVESTIGATIONS FOR SHOULDER IMPINGEMENT**

Diagnosing shoulder impingement syndrome involves performing a structured clinical assessment followed by various imaging modalities to assess the pathology or stage of the condition.

There are various ‘special’ tests for eliciting shoulder impingement and rotator cuff tears. Tests include Hawkins test, Neers test and Jobes test among many others. In identifying shoulder impingement Hawkins test appears to be more sensitive whereas Neers test has been found to be more specific [21, 22].

**IMAGING MODALITIES**

Clinical assessment is important but where impingement or rotator cuff pathology is identified or suggested, imaging is justified. This should however be tailored to the individual patient with specific reference to expected patient outcome.

Plain x-rays are usually normal in an acute rotator cuff injury [23]. Xrays however should form the basis for investigating the painful shoulder and shoulder impingement syndrome. They may demonstrate, subacromial spurs or anomalies of the acromion. They are also important in the differential diagnosis of shoulder impingement syndrome and demonstrate calcifying tendinitis, fractures and neoplasm.

Ultrasound (U/S) has been widely used for the evaluation of the shoulder, mainly for rotator cuff pathology. Ultrasonographic evaluation for rotator cuff tears were first described by Crass and Middleton in 1984 [23, 24]. It has been shown to be a sensitive and accurate method of identifying patients with full-thickness tears of the rotator cuff and Dynamic ultrasound can help confirm, but not exclude, a clinical diagnosis of impingement [23-25]. Although relatively inexpensive and non-invasive evaluation tool, the main issues regarding ultrasound relate to the inter-observer variability in the demonstration of rotator cuff tears.

Although not universally accepted, MR arthrography is the most sensitive and specific technique for diagnosing both full- and partial-thickness rotator cuff tears [26]. U/S and MRI are comparable to each other in both sensitivity and specificity in the diagnosis of full-thickness rotator cuff tears. MR arthrography and Ultrasound appear to be more accurate for the detection of partial-thickness tears than MRI. Given the large difference in cost of MRI and U/S scanning, U/S may be the most cost-effective diagnostic method for identification of full-thickness tears in a specialist hospital setting. To add further weight to this argument a study by Rutten et al. found that MRI scanning following routine shoulder US was requested in only 5.2% of the patients in their study. MRI therefore justifiably may be reserved for doubtful or complex cases, in which delineation of adjacent structures is mandatory prior to surgical intervention [25-27].

**TREATMENT**

Once clinical assessment and the appropriate investigations have been reviewed, management of the condition must be tailored to each individual case. The natural course of shoulder impingement is poorly described and there are a variety of treatment options available. These can be categorised into non operative treatment modalities which includes; analgesia, steroid injection, shock-wave therapy and physical therapy, and operative treatment which includes both arthroscopic and open procedures.

A review on the treatment of impingement syndrome by Faber et al. found that for functional limitations, there is strong evidence that extracorporeal shock-wave therapy is not effective, moderate evidence exists that exercise combined with manual therapy is more effective than exercise alone and that ultrasound is not effective. They also found that open and arthroscopic acromioplasty are equally effective on the long term [28].

**CONSERVATIVE MANAGEMENT**

Studies show that conservative management of shoulder impingement syndrome resolves the problem in 70-90% of patients [9, 11]. In symptomatic patients a course of conservative management is widely accepted as first line management but the time frame for this is variable and a point of controversy. Most surgeons generally tend to observe patients for a 6 month period however based on individual patient factors this can vary. For patients older than 50, a longer period of conservative treatment may be warranted but when managing these cases in the athletic or young individuals (<40 years) with acute trauma and injury to the rotator cuff, surgical intervention should be considered at an earlier time as successful repair allows these particular patients to return to pre-injury level of function [28-31].

There is a general consensus also that a comprehensive and supervised rehabilitation programme is the first line of treatment of the shoulder impingement syndrome [28, 32, 33]. However with regards to physical therapy, Desmeules et al. reported a lack of uniformity in defining, evaluating, and treating shoulder impingement. They found that the few trials that were moderately well designed offered limited evidence to support the efficacy of physical therapy in shoulder impingement syndrome [34].

Oral NSAIDS have been used extensively to treat pain and more recently, the use of topical NSAIDS in form gels or patches have also gained in popularity. A review by Brett et al. looking at the effectiveness of NSAIDs in the treatment of tendinopathy found the current evidence demonstrated a role for them in reducing tendinopathy related pain in the short term of between 7–14 days [35]. In particular relation to acute shoulder bursitis/tendinosis they also suggested that oral and local NSAIDs both appeared to be an effective treatment. There was little evidence however to either support or refute the use of NSAIDS in the longer term treatment. However the complications associated with these
particular drugs warrants caution in their longer term use [35].

Previous reviews of the use of corticosteroid injections in shoulders have found conflicting results and there is an underlying concern regarding potential damage to the rotator cuff following repeated injections into the subacromial space. Most surgeons appear to agree that limiting these steroid subacromial injections to three within a period of 12 months. A study by Bhatia et al. found that subacromial impingement should not be considered a causative factor in rotator cuff tears. Subacromial injections of corticosteroids were shown to be effective however in the improvement of rotator cuff tendonitis for up to a 9-month period post injection which probably makes it more effective than NSAID medication [35-37].

**OPERATIVE TREATMENT**

When conservative management fails to relieve the symptoms or a complete cuff rupture is identified, operative intervention may be warranted, particularly in the younger patient group. Where there has been a traumatic injury to the rotator cuff early surgery has been found to be associated with better results with regard to shoulder function [38].

With regards to surgery in shoulder impingement the two structures that need to be addressed are the acromion and the rotator cuff itself. Neer was the first to popularise acromioplasty for the treatment of shoulder impingement. He emphasised that resecting the antero-inferior portion of the acromion would increase the volume of the subacromial space and therefore decrease the degree of impingement of the supraspinatus tendon under the acromion. Neer also described the indications for acromioplasty as being long-term disability from chronic bursitis and either partial tears or complete tears of the supraspinatus [2, 8].

The main issues surrounding operative treatment relate to whether these procedures should be done open or arthroscopically and in the presence of tears, should the rotator cuff be debrided or repaired.

Outcomes following arthroscopic subacromial decompression surgery (ASAD) have been found to be similar to that of open surgery. For persistent stage II primary impingement, arthroscopic subacromial decompression allows earlier rehabilitation, less scarring and less deltoid morbidity than open decompression because complete detachment of the deltoid is not performed. ASAD procedures are however more technically demanding and have a longer learning curve [39, 40]. Arthroscopic surgery appears to be more favourable due to its advantages over open surgery which are particularly important in the cases of younger and/or athletic patients in allowing them to return to pre-injury/competitive activity levels [29, 31, 40].

Debridement of partial tears is thought to promote healing of the tendon. In the case of elite athletes, Strauss et al. performed a systematic review of the arthroscopic management of partial thickness rotator cuff tears. They reviewed 16 clinical studies and found significant variation in the results obtained after the arthroscopic management of partial-thickness rotator cuff tears. The available data however revealed that tears involving <50% of the tendon could be treated, with good result, by debridement of the tendon with or without a formal acromioplasty, although subsequent tear progression may occur. When the tear was greater than 50%, surgical intervention focusing on repair was successful [41].

When considering complete tears, particularly in the older patient group, massive complete tears are technically difficult to repair because of the sizeable deficit and generally poorer bone quality in this particular patient group. Melillo et al. found that although several studies demonstrated satisfactory results in the short to mid-term following debridement, the three long-term studies currently available all reported that these results deteriorate significantly with time and are not acceptable. It is therefore felt, that where possible in the case of complete rotator cuff rupture, repair of the rotator cuff remains the treatment option of choice [42].

**SUMMARY**

Following a detailed clinical examination, further investigation and management must be tailored to the individual patient. Ultrasound scan appears to be a justifiable and cost effective assessment tool following plain radiographs when assessing the rotator cuff, with MRI scans being reserved for more complex cases. A period of observed conservative management including the use of NSAIDs, physiotherapy with or without the use of subacromial steroid injections is a well-established and accepted practice. However in young patients or following any traumatic injury to the rotator cuff surgery should be considered early. If surgery is to be performed this should be done arthroscopically and in the case of complete rotator cuff rupture the tendon should be repaired where possible.

**ABBREVIATIONS**

ASAD = Arthroscopic Subacromial Decompression
U/S = Ultrasound
MRI = Magnetic resonance imaging
NSAIDs = Non-steroidal anti-inflammatories

**CONFLICT OF INTEREST**

The authors confirm that this article content has no conflict of interest.

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