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Impingement Syndrome: A Review of the Literature

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Abstract

Rotator Cuff tendinopathy (RCT) is a common disorder that poses challenges for effective treatment. Evidence suggests that extrinsic, intrinsic, and combinations of biomechanical mechanisms play a role. There are no significant differences in outcome between conservatively and surgically treated patients with subacromial impingement syndrome (SAIS). For most patients with subacromial impingement syndrome (SAIS), nonsurgical treatment is successful. Surgical intervention is successful in patients who fail nonsurgical treatment. Surgeon experience and intraoperative assessment may guide the method of surgical treatment. Studies have shown that many surgical interventions, including debridement and open and arthroscopic acromioplasty, have been successful. However, there remains a need for high-quality clinical research on the diagnosis and treatment of SAIS.

In this review, there are not figures and outcomes.

Keywords: Subacromial impingement syndrome; Review of Literature

Introduction

Subacromial impingement syndrome (SAIS) is the most common disorder of shoulder, accounting for 44–65% of all complaints of shoulder pain during a physician's office visit [1]. SAIS encompasses a spectrum of subacromial space pathologies including partial thickness rotator cuff tears, rotator cuff tendinosis, calcific tendinitis, and subacromial bursitis. The main consequences of SAIS are functional loss and disability [2]. Subacromial space is defined by the humeral head inferiorly, the anterior edge and under surface of the anterior third of the acromion, coracoacromial ligament and the acromioclavicular joint superiorly. The height of space between acromion and humeral head ranges from 1.0 to 1.5 centimeters as seen on radiographs. Interposed between these two osseous structures are the rotator cuff tendons, the long head of the biceps tendon, the bursa, and the coracoacromial ligament. Any abnormality that disturbs the relationship of these subacromial structures may lead to impingement [3]. Neer described three stages of impingement [4] Stage-I impingement is characterized by edema and hemorrhage of the subacromial bursa and cuff. It is typically found in patients who are less than twenty-five years old. Stage-II impingement represents irreversible changes, such as fibrosis and tendinitis of the rotator cuff, and is typically found in patients who are twenty-five to forty years old. Stage-III impingement is marked by more chronic changes, such as partial or complete tears of the rotator cuff, and usually is seen in patients who are more than forty years old.

Given the high prevalence of this condition, the aim of this review is to evaluate the different etiological theories that may explain SAIS. The different anatomical structures involved in this type of impingement are described; the clinical findings are presented and treatment guidelines are suggested.

Glenohumeral Joint Kinematics

The glenohumeral joint possesses six degrees of freedom, three rotations and three translations.5 With active in vivo glenohumeral abduction in the scapular plane (approximately 30–40° anterior to the frontal plane), the humerus concomitantly externally rotates. External rotation is important for clearance of the greater tuberosity and its associated tissues as it passes under the coracoacromial arch, as well as for relaxation of the capsular ligamentous constraints to allow maximum glenohumeral elevation [6].

Translation of the humeral head in the magnitude of 1–3 mm in the superior direction occurs in the first 30–60° of active glenohumeral scapular plane elevation. After the initial phase of elevation in the scapular plane or frontal plane abduction, the humeral head remains somewhat centered on the glenoid cavity with fluctuations between inferior and superior translations of typically less than 1 mm. The glenohumeral joint demonstrates essentially ball and socket kinematics above approximately 60° of glenohumeral elevation [7].

Superior humeral translation that occurs during the initial phase of elevation appears to be due in part to the cranially directed pull on the head of the humerus by the deltoid muscle [5]. Humeral head translations in the anterior-posterior directions have been less well investigated. Anterior humeral head translations in the magnitude of 2–5 mm have been demonstrated during passive glenohumeral flexion. During active glenohumeral flexion, anterior humeral head translation of less than 1 mm occurs over the course of motion [8].

The height of the subacromial space, from the head of the humerus to the coracoacromial arch, is only 1.0–1.5 cm as seen on radiographs. Changes of this space occur in subjects with healthy shoulders; a decrease in the width of the acromio-humeral interval and an increase in the contact between the inferior acromion and underlying subacromial tissues occur during glenohumeral abduction. Contact pressure and force in the subacromial space has also been demonstrated to increase during glenohumeral abduction. Theoretically, these changes in the subacromial space would be accentuated with an increase in the normal superior and anterior humeral head translation, leading to mechanical compression of the tissues in subacromial space during glenohumeral motion [9].

Scapulothoracic Articulation Kinematics

Scapula and thoracic cage form the scapulothoracic articulation. This articulation is assessed kinematically either two-dimensionally or three-dimensionally. The joint is typically described with five degrees of freedom, three rotations and two translations [5].

The scapula demonstrates a pattern of upward rotation, external rotation, and posterior tilting during glenohumeral elevation. The three-dimensional analysis of scapular motion by van der Helm and Pronk describe scapular upward rotation occurring about an anterior-posterior axis, with the inferior angle of the scapula moving laterally;[10] external rotation occurring about a superior-inferior axis, with the lateral border of the scapula moving posteriorly; posterior tilt occurring about a mediallateral axis, with the inferior angle moving anteriorly.

Less well examined are scapular translations, depicted as scapular positions. Scapular positions can be represented by clavicular rotations about the sternoclavicular joint in two different planes: clavicular elevation/depression for superior/inferior translation and clavicular protraction/retraction for anterior/posterior translation. The assumption is made that motion of the clavicle at the sternoclavicular joint will be in direct relationship to scapular translation, because of the interposed rigid bone (clavicle) between these two joints and the lack of significant motion occurring at the acromioclavicular joint. During glenohumeral elevation the clavicle retracts posteriorly and elevates, putting the scapula in essentially a more superior and posterior position [11].

Subjects with subacromial impingement generally have decreased scapular posterior tilting, decreased upward rotation and increased internal rotation compared to healthy subjects. Weak or dysfunctional scapular musculature, fatigue of the infraspinatus and teres minor and changes in thoracic and cervical spine posture have all demonstrated a change in scapular kinematics [12].

Etiology

SAIS is an encroachment of the subacromial tissues as a result of narrowing of the subacromial space. Mechanisms of rotator cuff (RC) tendinopathy have been classically described as extrinsic, intrinsic or a combination of both. Intrinsic impingement, theorizes that partial or full thickness tendon tears occur as a result of the degenerative process that occurs over time with overuse, tension overload, or trauma of the tendons.5 An alternative theory is that of extrinsic impingement, where inflammation and degeneration of the tendon occur as a result of mechanical compression by structures external to the tendon [4]. A unique subset of extrinsic impingement, internal impingement occurs due to compression of the articular side rather than the bursal side of the RC tendons, between the posterior superior glenoid rim and humerus when the arm is in full external rotation, abduction, and extension. Although internal impingement

can be considered an extrinsic mechanism, narrowing of the subacromial space is not a hallmark finding [12].

Extrinsic Impingement

Extrinsic mechanisms of RC tendinopathy that result in bursal sided RC tendon compression due to narrowing of the subacromial space include anatomical factors, biomechanical factors, or a combination. The acromiohumeral distance (AHD), a linear measure between the acromion and the humeral head used to quantify the subacromial space, has been studied in patients with RC disease using magnetic resonance imaging [13], ultrasonography [14], and radiographs [13], AHD is normally between 7 and 14 mm in healthy shoulders. It is reduced in SAIS patients with the muscles at rest or during muscle activation which functionally narrow the subacromial space. Furthermore, AHD less than 7 mm with the arm at rest is a predictive indicator of less favorable surgical outcome [15].

Anatomical factors

Anatomical factors that may excessively narrow the subacromial space and outlet to the RC tendons include variations in shape of the acromion, orientation of the slope/angle of the acromion or prominent osseous changes to the inferior aspect of the acromio-clavicular (AC) joint or coracoacromial ligament.12 A widely used classification system for acromial shape is flat (type I), curved (type II), or hooked (type III), which was developed from observations of 139 shoulder specimens [3]. Whether acromial shape is congenital or acquired with age remains controversial [12]. Another possible culprit of encroachment into the subacromial space is thickening of the coracoacromial ligament. Significant relationships have been demonstrated between acromion morphology and patient's self-reported shoulder function and the severity of the rotator cuff pathology [5]. However, surgical decompression of coracoacromial arch has only been demonstrated to produce comparable level of successful outcome as non-operative treatment. This suggests that direct encroachment of the subacromial space by the coracoacromial arch soft tissue or bony changes is not the only mechanism of impingement [5,12]. On the other hand, these coraco-acromial arch changes have significant effect on tendon injury when combined with overuse activity. Supporting this theory of a requisite overuse exposure, symptomatic RC disease is more often present in dominant than nondominant shoulders [16].

Biomechanical factors

Biomechanical mechanism of extrinsic SAIS is based on dynamic narrowing of the subacromial space leading to RC tendon compression secondary to superior translation of the humeral head or aberrant scapular motion that causes the acromion to move inferiorly. These include shortening of the posterior-inferior glenohumeral joint capsule and decreased RC muscle performance [5,12].

Posterior capsule

Posterior capsular tightness may cause changes in glenohumeral kinematics leading to SAIS. When posterior capsular tightness was surgically induced in cadavers, there was an in increase in superior and anterior humeral head translations during passive glenohumeral flexion. Excessive superior and anterior humeral head translations can decrease the size of the subacromial space, leading to increased mechanical compression of the subacromial structures.9 Glenohumeral internal rotation range of motion and horizontal adduction at 90° of elevation are reliable clinical measures that potentially assess posterior capsule length. Furthermore, stretching to address impairments of posterior shoulder tightness has been identified as an important component to rehabilitation for patients with RC tendinopathy [17].

Scapular musculature

Aberrant scapular muscle activity has been identified in patients with SAIS and been directly linked to abnormal scapular kinematics. Of particular interest are the relative contributions of the upper and lower serratus anterior muscles and trapezius muscles, found to stabilize the scapula and induce scapular upward rotation, external rotation, and/or posterior tilt to potentially allow the humeral head to clear the acromion with elevation [18]. These individuals have decreased muscle performance of the trapezius and serratus anterior in terms of force output [19], muscle balance/ratios,19 electromyographical activity [18], and latencies in activation [20].

Relatively small changes in the muscle performance of the scapulothoracic muscles can alter the position of the scapula at a fixed angle of humeral elevation and, in theory, affect the length-tension relationship (point on the length-tension curve) of the RC muscles and the subacromial space [12].

Spine

A relatively small increase in thoracic spine flexion has resulted in a more elevated and anteriorly tilted scapula at rest, and less upward rotation and posterior tilt during glenohumeral elevation. An increase in thoracic spine flexion has also resulted in a decrease in the amount of elevation of the glenohumeral joint and a decrease in the amount of force generated at 90° of glenohumeral scapular plane abduction [21].

Rotator Cuff Musculature

The supraspinatus along with the other rotator cuff muscles of teres minor, infraspinatus, and subscapularis serve to maintain the congruent contact between the humeral head and the glenoid fossa by producing a compressive force during glenohumeral movements.5 Weakness or dysfunctional rotator cuff musculature can lead to changes in glenohumeral and scapulothoracic kinematics. Excessive superior translation of the humeral head resulting from rotator cuff weakness can lead to a decrease in the subacromial space during elevation, and thus increased mechanical compression of the subacromial contents [22].

Clinical Evaluation

History

Although impingement symptoms may arise following trauma, the pain more typically develops insidiously over a period of weeks to months. The pain is typically localized to the anterolateral acromion and frequently radiates to the lateral mid-humerus. Patients usually complain of pain at night, exacerbated by lying on the involved shoulder, or sleeping with the arm overhead. Normal daily activities such as combing one's hair or reaching up into a cupboard become painful. Weakness and stiffness may also be encountered, but they are usually secondary to pain [2].

Physical examination

In their systematic analysis, Papadonikolakis, et al. [23] concluded that the physical findings used to diagnose the impingement syndrome, i.e., the Neer sign (pain on forced flexion), the Hawkins sign (pain on internal rotation with the arm elevated to 90_), and the Neer injection test (relief of pain on the Neer sign after subacromial injection of local anesthetic) may be sensitive, but are not specific. The average sensitivity (and standard deviation) of the Neer sign was 76±11%, while the average specificity was 36±22%. The respective values for the Hawkins sign were 80±11% and 41±19%. In their meta-analysis, Hegedus, et al. [24] concluded that neither the Neer nor the Hawkins sign had diagnostic utility for impingement.

Imaging

Standard radiographs including internal and external rotation anteroposterior, scapular Y, axillary, and Supraspinatus outlet views are important for the thorough evaluation of shoulder pain. These plain radiographs may show characteristic changes of rotator cuff disease, including subacromial osteophytes, subacromial sclerosis, cystic changes of the greater tuberosity, and narrowing of the acromiohumeral distance, they are not definitive [15,25].

Magnetic resonance imaging (MRI) provides detail of potential sites of subacromial impingement through the supraspinatus outlet. Ossification of the coracoacromial ligament (CAL) or presence of a subacromial spur can be best identified in the sagittal oblique plane; however, differentiation of a pathologic spur and the normal CAL can be difficult. MRI also may demonstrate findings of subacromial/subdeltoid bursitis. Findings that indicate this condition include bursal thickness >3 mm, the presence of fluid medial to the acromioclavicular joint, and the presence of fluid in the anterior aspect of the bursa. Typically, MRI is performed with the arm adducted; however, this position does not recreate the position of impingement [26].

Treatment and Conclusion

Many treatments are available for impingement syndrome such as physical therapy, shock-wave therapy, medication, and surgery. In the last decade, several (systematic) reviews on treatment for impingement syndrome were published [27–31]. These reviews compared the effectiveness of treatments on a variety of outcome measures, including pain, range of movement, functional limitations, and return to work. Hence, the conclusion on effectiveness of various treatments was primarily based on the combination of these outcome measures.

There is strong evidence that extracorporeal shock-wave therapy is no more effective than placebo,32,33 moderate evidence that ultrasound therapy is no more effective than placebo [34], and limited evidence that laser is no more effective than placebo with regard to functional limitations.35With regard to the improvement in functional limitations there is limited evidence that exercise is more effective than no intervention [36], and moderate evidence that exercise combined with manual therapy is more effective than exercise alone [37].

There is limited evidence for the effectiveness of the following interventions: exercise is more effective than no intervention on functional limitations, oral diclofenac is more effective than analgesic injections, both on functional limitations and on ability to work after 1 year. On the short term, arthroscopic acromioplasty is more effective than open acromioplasty with regard to functional limitations and return to work. However, moderate evidence exists that on the long term open and arthroscopic acromioplasty are equally effective with regard to functional limitations [38].

A systematic review by Dorrestijn, et al. led the authors to conclude that according to the best-evidence synthesis, however, there is no evidence from the available randomized controlled trials for differences in outcome in pain and shoulder function between conservatively and surgically

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treated patients with subacromial impingement syndrome [39]. However, several observational studies report a significantly better outcome in operated-on patients who had not responded to non-operative measures and who had a short symptom duration compared with those who had prolonged symptoms before surgery [38].

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