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Subacromial impingement syndrome

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Abstract

Subacromial impingement syndrome (SAIS) represents a spectrum of pathology ranging from subacromial bursitis to rotator cuff tendinopathy and full-thickness rotator cuff tears. The relationship between subacromial impingement and rotator cuff disease in the etiology of rotator cuff injury is a matter of debate. However, the etiology is multi-factorial, and it has been attributed to both extrinsic and intrinsic mechanisms. Management includes physical therapy, injections, and, for some patients, surgery. No high-quality randomized controlled trials are available so far to provide possible evidence for differences in outcome of different treatment strategies. There remains a need for high-quality clinical research on the diagnosis and treatment of SAIS.

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. 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.

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.

Neer described three stages of impingement. 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. With active in vivo glenohumeral abduction in the scapular plane (approximately 30-40º 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.

Translation of the humeral head in the magnitude of 1-3 mm in the superior direction occurs in the first 30-60º 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 60º of glenohumeral elevation.

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. 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.

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 occurs 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.

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.

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; external rotation occurring about a superior-inferior axis, with the lateral border of the scapula moving posteriorly; posterior tilt occurring about a medial-lateral 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/depres-
son for superior/inferior translation and clav-
icular protraction/retraction for anterior/poste-
rior 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 scapu-
lar 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 subacromi-
al tissues as a result of narrowing of the sub-
acromial space. Mechanisms of rotator cuff
(RC) tendinopathy have been classically
described as extrinsic, intrinsic or a combina-
tion 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 over-
load, 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 exten-
sion. Although internal impingement can be
considered an extrinsic mechanism, narrow-
ning of the subacromial space is not a hallmark
finding.12

### Extrinsic Impingement

Extrinsic mechanisms of RC tendinopathy
that result in bursal sided RC tendon compres-
sion due to narrowing of the subacromial
space include anatomical factors, biomechni-
cal factors, or a combination. The acromio-
humeral 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 ultrasonogra-
phy,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 func-
tionally narrow the subacromial space.
Furthermore, AHD less than 7 mm with the arm
at rest is a predictive indicator of less
favorable surgical outcome.13

### Anatomical factors

Anatomical factors that may excessively nar-
row 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 coracohumeral 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.2 Whether acromial
shape is congenital or acquired with age
remains controversial.12 Another possible cul-
prit of encroachment into the subacromial
space is thickening of the coracoacromial liga-
ment. 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 coraco-
arcomial arch has only been demonstrated to
produce comparable level of successful out-
come as non-operative treatment. This sug-
gests that direct encroachment of the subacro-
mial space by the coracoacromial arch soft tis-
ue or bony changes is not the only mecha-

### Biomechanical factors

Biomechanical mechanism of extrinsic SAIS
is based on dynamic narrowing of the subacro-
mial 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
crease 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 sub-
acromial structures.7 Glenohumeral internal
rotation range of motion and horizontal adduc-
tion at 90° of elevation are reliable clinical
measures that potentially assess posterior cap-
sule length. Furthermore, stretching to
address impairments of posterior shoulder
tightness has been identified as an important
component to rehabilitation for patients with
RC tendinopathy.12

### Scapular musculature

Aberrant scapular muscle activity has been
identified in patients with SAIS and been
directly linked to abnormal scapular kinemat-
ics. Of particular interest are the relative con-
tributions 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.14 These
individuals have decreased muscle perfor-
ance of the trapezius and serratus anterior in
terms of force output,12 muscle balance/ratios,13
electromyographical activity,13 and

### 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 tho-
racic 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 gleno-
humeral scapular plane abduction.21

### Rotator cuff musculature

The supraspinatus along with the other
rotator cuff muscles of teres minor, infraspin-
atus, and subscapularis serve to maintain the
congruent contact between the humeral head
and the glenoid fossa by producing a compres-
sive force during glenohumeral movements.5
Weakness or dysfunctional rotator cuff muscu-

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[page 80] [Orthopedic Reviews 2012; 4:e18]
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, Hegeduš 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 liga ment (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

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,22,23 moderate evidence that ultrasound therapy is no more effective than placebo,24 and limited evidence that laser is no more effective than placebo with regard to functional limitations.23 With regard to the improvement in functional limitations there is limited evidence that exercise is more effective than no intervention,34 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 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.34

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 treated patients with subacromial impingement syndrome.27 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.18

Summary

RC tendinopathy 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. For most patients with 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.

References