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Review article

Rotator cuff tear: A detailed update

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Abstract

Rotator cuff tear has been a known entity for orthopaedic surgeons for more than two hundred years. Although the exact pathogenesis is controversial, a combination of intrinsic factors proposed by Codman and extrinsic factors theorized by Neer is likely responsible for most rotator cuff tears. Magnetic resonance imaging remains the gold standard for the diagnosis of rotator cuff tears, but the emergence of ultrasound has revolutionized the diagnostic capability. Even though mini-open rotator cuff repair is still commonly performed, and results are comparable to arthroscopic repair, all-arthroscopic repair of rotator cuff tear is now fast becoming a standard care for rotator cuff repair. Appropriate knowledge of pathology and healing pattern of cuff, strong and biological repair techniques, better suture anchors, and gradual rehabilitation of postcuff repair have led to good to excellent outcome after repair. As the healing of degenerative cuff tear remains unpredictable, the role of biological agents such as platelet-rich plasma and stem cells for postcuff repair augmentation is still under evaluation. The role of scaffolds in massive cuff tear is also being probed.

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Relevant anatomic details

Rotator cuff tendons form confluence with the joint capsule and coracohumeral ligament, and insert on the humeral tuberosity. The subscapularis (SC) is the largest and strongest cuff muscle with the upper 60% of the insertion being tendinous, and the lower 40% being muscular with footprint attachment varying from 24 to 40 mm in the superoinferior and 16–20 mm in the mediolateral directions. The supraspinatus (SS), infraspinatus (IS), and teres minor (TM) are inserted over the superior, middle, and inferior facets on to the greater tuberosity with a mean anteroposterior length of 37.8 mm, mean medial–lateral distance of 14.7 mm, and mean area of 6.2 cm².Dugas et al¹ suggested that the restoration of this footprint might increase the likelihood of normal

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healing and subsequent normal function. The SS muscle has two bellies, namely, anterior and posterior. The anterior belly has a larger physiologic cross-sectional area $(140 \pm 43 \text{ mm}^2)$ and a smaller tendon cross-sectional area $(26.4 \pm 11.3 \text{ mm}^2)$ as compared with the posterior belly, which has a smaller cross-sectional area $(62 \pm 25 \text{ mm}^2)$ but a larger tendon crosssectional area $(31.2 \pm 10.1 \text{ mm}^2)$. The anterior belly has an intramuscular fusiform core tendon, whereas the posterior belly is *strap like* with no intramuscular tendon.² Therefore, the peculiar anatomy of the anterior muscle with a larger belly, a fusiform cross section, and an intramuscular fusiform core tendon is responsible for the major contractile force generation by the SS and is primarily responsible for arm abduction and humeral head depression.² It may also help explain why many patients who have SS tear with intact anterior tendon on the footprint retain their ability to flex forward and abduct their arm. Therefore, incorporation of the anterior tendon into rotator cuff tendon repair is an important aspect to enable transmission of the major portion of contractile loads, which subsequently improves functional outcome.² On the downside,

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stronger leading edge of the SS forms the *stress riser zone* between the anterior and remaining mid-posterior fibres. This may explain why more than 90% of degenerative tears start and propagate from this junction. The IS tendon bends in its insertion anteriorly and covers the largest part of the greater tuberosity compared with the SS insertion.³ Histologically, there are five interlocked distinct layers of the SS and IS tendons.^{4,5} Differential anatomy between various layers initiates stress risers and can cause intrasubstance tears that are two times as common as bursal- or articular-side tears. Robust collagen on the bursal side makes it more resistant to tensile stress. Therefore, the stress-failure point of the articular side is half of bursal side.^{6–8}

The primary function of the rotator cuff is to keep the head of the humerus depressed and centred into the glenoid fossa permitting a single centre of rotation, while allowing efficient abduction or forward elevation of the arm.^{9,10} This centring in the glenoid by the cuff is achieved by balancing the force couples around the glenohumeral joint. A force couple is a pair of forces that act on an object and cause it to rotate. The shoulder joint has two force couples, namely, coronal and transverse (Fig. 1). The coronal force couple, which was first described by Inman et al,¹¹ is a result of the balance of moments created by the deltoid versus inferior rotator cuff (IS, SC, and TM). The coronal force couple is said to be balanced only if the line of action of the rotator cuff force is below the centre of rotation of the humeral head so that it can oppose the moment created by the deltoid muscle. A balanced coronal force couple maintains adequate fulcrum for the glenohumeral joint motion. The transverse force couple, which was described by Burkhart,^{12,13} is a balanced moment between the anterior SC and posterior IS-TM muscles. Burkhart emphasized the role of transverse force couple, which is imbalanced in massive cuff tear wherein the large tears of the SS gradually involve the IS too, in weakening the posterior cuff. This leads to imbalanced transverse as well as coronal force couples, leading to posterosuperior migration of the head and inability to maintain stable fulcrum of motion. Parsons et al¹⁴ in their study on nine cadaveric human shoulders emphasized that peak joint reaction forces (JRFs) drop by 10-11% in case of incomplete or complete tear of the SS. However, the peak

abduction angle attained was almost similar to intact shoulder as concavity compression through the action of remaining cuff was sufficient to provide a fixed fulcrum for concentric rotation of the glenohumeral joint. However, the peak JRF further dropped by another 50% when tear propagated into the posteroinferior cuff. This disrupts the transverse force couple, and the deltoid is unable to achieve maximum abduction. Burkhart^{12,13} established that restoration of transverse and coronal force couples by partial repair of tendons (IS and SC) in case of massive cuff tear can give surprisingly good results.

Aetiology of cuff tear

Cuff tears are either traumatic or degenerative. Traumatic tears are due to significant trauma, whereas degenerative tears are far more frequent and multifactorial in aetiology. The rotator cuff is weakened by both extrinsic and intrinsic factors, leading to gradual failure of tendon with or without superimposed acute injury, which finally results in full-thickness tear (Fig. 2).

Extrinsic theory

Neer's classic work advocated extrinsic factors for rotator cuff tendon failure in which during forward elevation of the shoulder, the anterior part of the cuff abuts against the coracoacromial (CA) arch and leads to impingement, tendonitis, and tear.¹⁵ Neer's theory got a boost when Bigliani et al¹⁶ proposed that downsloping acromion in the sagittal plane can impinge upon the anterior cuff, and could cause cuff tear. Bigliani et al classified the acromial morphology into three types, namely, type I (flat undersurface), type II (curved), and type III (hooked); (Fig. 3). Several authors confirmed close relationship between hooked acromion and cuff tear.¹⁷⁻²⁰ Wang et al²¹ concluded that patients with Bigliani type II and type III acromion are poor responders to conservative treatment for impingement syndrome. Various other factors such as the presence of acromial spur,^{22,23} acromion slope,^{24,25} CA ligament,^{26–28} os acromiale,^{29–31} and acromioclavicular joint spur^{32,33} also contribute towards extrinsic compression. Recently, Nyffeler et al introduced the acromion index as a measurement of lateral extension of the acromion, which is



Fig. 1. (A) Coronal force couple. (B) Transverse force couple. D = deltoid muscle force; I/Tm = infraspinatus/teres minor muscle force; SSc = subscapularis muscle force.

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