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MUSCULOSKELETAL DISORDER OF THE SHOULDER: ANATOMY, ETIOLOGIC AND PATHOLOGICAL FACTORS INVOLVED IN ROTATOR CUFF TENDINOPATHY

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Abstract:
A review was conducted to synthesize the available research literature on the pathogenesis of rotator cuff tendinopathy. Musculoskeletal disorders of the shoulder are extremely common. An understanding of the anatomy and biomechanics of the rotator cuff may provide some insight into the pathologic process involved in this tendinopathy. The patho etiology of rotator cuff failure is multifactorial and results from a combination of intrinsic, extrinsic and environmental factors. Profound changes within the subacromial bursa are strongly related to the pathology and resulting symptoms. A considerable body of research is necessary to more fully understand the etiology and pathogenic factors of rotator cuff tendinopathy Profound changes within the subacromial bursa are strongly related to the pathology and resulting symptoms. A considerable body of research is necessary to more fully understand the etiology and pathohistology of rotator cuff tendinopathy and its relationship with bursal pathology.

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INTRODUCTION:

Musculoskeletal disorders of the shoulder are extremely common, with reports of prevalence ranging from one in three people experiencing shoulder pain at some stage of their lives[1] to approximately half the population experiencing at least one episode of shoulder pain annually[2].

Anatomy The shoulder joint allows the greatest range of motion of any joint in the body[3].This large amount of motion primarily consists of movement of the scapulothoracic articulation and the glenohumeral joint. To allow such large motions, the glenohumeral joint must be relatively unconstrained. The rotator cuff is composed of four muscle/tendon units: the subscapularis anteriorly, the supraspinatus superiorly, and the infraspinatus and teres minor posteroinferiorly. These four muscle/tendon units are the primary dynamic stabilizers of the joint. The supraspinatus tendon is most frequently involved[4].The supraspinatus glides beneath this arch through all planes of humeral elevation and has been shown to come into contact with this arch, depending on the elevation and rotation of the humerus.

Subacromial Bursa

Bursae are comprised of fibrous, areolar and adipose tissue as well as synovial cells. They function primarily to reduce friction during movement. Approximately seven to eight bursae have been identified around the shoulder[5].The subacromial bursa (SAB), the largest bursa in the body, separates the coracoacromial arch and deltoid above and the rotator cuff tendons below, and acts to reduce friction during shoulder movement. The SAB is innervated by the suprascapular nerve posteriorly and by the lateral pectoral nerve anteriorly (C5,6).The presence of pressure detectors under the coracoacromial ligament suggests that a reflex system involving the rotator cuff may control displacement of the humeral head. Although conventional radiographic films assist in the evaluation of the painful shoulder, there are no specific plain-radiographic diagnostic features of rotator cuff tendinopathy or tears.

Plain radiographic examination should include an anteroposterior view in the plane of the scapula and an axillary view of the shoulder. Specialized views, such as the supraspinatus outlet view, can help delineate acromioclavicular arthritis or supraspinatus outlet narrowing. When properly obtained, these views can define the anterior extension of the acromion beyond the clavicle, the presence of a spur, or cystic changes in the acromioclavicular (AC) joint of the shoulder[6].

Ultrasound evaluation of the shoulder has been reported to be an accurate and cost-effective noninvasive screening tool for the diagnosis of full-thickness rotator cuff tears, but its use in partial tears and tendinopathy is limited[7]. Furthermore, accuracy of ultrasonography is highly dependent on the experience of the ultrasonographer and the type of equipment used. Magnetic resonance imaging (MRI) has become an established technique for diagnoses of shoulder pathology.

Etiology

Although the precise pathogenesis of rotator cuff disease is unknown, it is generally accepted that the etiology of rotator cuff tendinopathy is multifactorial—
a combination of intrinsic and extrinsic factors. Which of these mechanisms are the primary and secondary factors in a particular patient, however, is a controversial topic. The intrinsic mechanism is defined as a tendon injury that originates within the tendon from intrinsic inferior tissue mechanical properties, direct tendon overload, intrinsic degeneration, or other insult. On the other hand, the extrinsic mechanism is associated with damage to the tendon through compression against surrounding structures[8].

Intrinsic conditions include traumatic, reactive, or degenerative changes originating in the substance of the supraspinatus. Specifically, the proposed causes of intrinsic degeneration are primarily the microvascular supply, aging, and tensile overload.There is evidence to both support and refute this suggestion, however. The major vascular supply to the rotator cuff is derived from the ascending branch of the anterior circumflex humeral artery, the acromial branch of thoracoacromial artery, as well as the suprascapular and posterior humeral circumflex arteries[9]. The negative effect that age has on tendon mechanical properties is less controversial. Histologic studies have shown degenerative changes characterized by calcification, fibrovascular proliferation, and microtears in elderly individuals but not in younger subjects. Furthermore, the healing potential of elderly individuals is thought to be decreased in tendons and would therefore make elderly individuals more susceptible to a tensile overload mechanism of injury. Although it is generally accepted that younger tissues heal better and faster, there appear to be no studies that specifically correlate age with a reduced healing potential for tendons.

Tension overload is another intrinsic injury mechanism that can result in damage to the supraspinatus tendon. Several studies have been conducted to delineate the mechanisms that may be responsible for the different types of tension

overload tears. It was found that the modulus of elasticity is lower and the ultimate strain and stress is greater on the bursal side rather than the articular side[9]. This suggests that if the joint and the bursal sides of the supraspinatus tendon are subjected to the same loads, the joint-side will be more susceptible to failure. These studies are limited, however, in that the supraspinatus tendon must be divided to obtain the properties, which does not take into account the interactions between the two sides. To address this limitation, a novel, nondestructive method to measure intra tendinous strain using MRI has been developed. Using this method, it was found that intratendinous strain fields increased with increasing glenohumeral joint angle, but that the strain did not vary between the articular and bursal sides[10]. Taken together, these findings may indicate that individuals who are involved in overhead activities may be susceptible to articular side tears due to tendon overload Results of this preliminary study revealed high stress concentrations about the critical zone, with tears potentiating on the bursal side, articular side, or within the tendon. More complicated models will undoubtedly be developed for further analysis.
Extrinsic causes of rotator cuff tendinopathy can best be summarized as subacromial impingement lesions. Compression of the tendons can occur through bony impingement or direct pressure through surrounding soft tissues Neer's studies revealed that the anterior third of the acromion was responsible for impingement on the structures of the subacromial space. The overall incidence of full-thickness tears in the elderly was 34%. Three acromial types were defined in their cadaveric study: Type I, which were flat, had an incidence of 17%; Type II, which were curved, had an incidence of 43%; and Type III which were hooked, had an incidence of 39%. The Type III acromion was present in the majority of rotator cuff tears. Subsequent clinical studies revealed a correlation between Type III acromions seen on radiographs and rotator cuff tears, with treatment being directed at increasing the space between the coracoacromial arch[11].

Rotator Cuff Tendinopathy

Tendinopathy is a generic term without etiological, biochemical and histological implications and is used to describe pathology in, and pain arising from, a tendon. The theories of the pathogenesis of rotator cuff tendinopathy may be divided into extrinsic and intrinsic causes and combinations of both.

Intrinsic theories

Intrinsic tendinopathy is defined as tendon pathology that originates within the tendon, usually as a consequence ofoveruse or overload (including compression). Increases and changes in collagen, proteoglycans, vascularity and cells have been described in tendon pathology.In painful and degenerate rotator cuff, increases in type III collagen were found[12] which could reduce the strength of the tendon tissue. Amyloid deposits within the rotator cuff have been observed, suggesting irreversible structural change.

Evidence suggests that intrinsic degeneration within the rotator cuff is the principal factor in the pathogenesis of rotator cuff tears. Perry et al[13] reported high concentrations of vascular endothelial growth factor, which is involved in the formation and growth of new blood vessels. Bursal specimens taken from patients with constant shoulder pain were reported as having inflammatory cell infiltration whereas those with pain only on movement did not. The movement of the subunits of the rotator cuff tendons will subject the tendon to internal compressive forces and fibrocartilage develops in these regions. In the supraspinatus less relative strain occurs on the articular side compared with the superior aspect tendon during abduction. Fibrocartilage is less capable than tendon of withstanding tensional load. In the supraspinatus, Nakajima et al[14] identified tendon fibres of a smaller cross-sectional area on the articular side compared with the superior bursal-side fibres, which had a reduced ability to withstand strain.

The two different sections of the tendon were stretched to the point of rupture and the joint-side fibres ruptured with half the force required for the bursal-side fibres. The supraspinatus and infraspinatus tendons insert into the cable, which is located approximately 1.5 cm from the insertion of the fibres into the humerus. The cable lies at 90u to the long axis of the tendon fibres. Between the cable and the humerus are the thinner and structurally weaker insertional fibres of the tendons, known as the rotator crescent. This cable is much thicker (4.7 mm) than the crescent (1.8 mm). The thinner crescent is located within the hypovascular region (critical zone) described by Codman[15].The muscle and tendon fibres medial to the cable may act through the cable to produce humeral movement, and by doing so the cable may stress-shield the crescent tissues. Due to the function of the cable, the rotator crescent tissues are relatively underloaded and potentially more prone to degeneration and tearing.Overuse, where the physiological limit of the tendon unit is surpassed, results in pain, weakness and structural failure. In addition, the patho-aetiology conceivably occurs primarily as a result of intrinsic failure in the articular-side fibres of the tendon, possibly due to a combination of stress shielding and internal compression in this region. The decreased function in the rotator cuff may lead to

superior translation of the humeral head and irritation of bursal tissue, increasing the strain on the coracoacromial ligament and the acromion.

Extrinsic theories

Neer[16] argued that 95% of all rotator cuff pathology was caused by irritation from the anteroinferior aspect of the acromion onto the superior aspect of the rotator cuff. Neer argued that if conservative treatments, such as anti-inflammatory medicationsand injections and physiotherapy, did not alleviate the symptoms then removal of the anteroinferior aspect of the acromion (acromioplasty) was necessary. Although acromioplasty has become one of the most common surgical procedures performed on the shoulder,recent evidence questions the involvement of the acromion in the development of rotator cuff tendinopathy[17]. Success rates of 80–90% following subacromial decompression have been recorded.

When acromioplasty was compared with conservative care (physiotherapy exercises and pain relief), surgery appeared to be no more beneficial clinically at 6, 12 or 48 months. Improvement reported in the surgical studies may be due either to the procedure or to the extensive period of relative rest and reduction in activity following the procedure. There is also the distinct possibility that the operation itself is a placebo or that the benefit is related more to the removal of the bursa than the acromion[18]. The questionable benefit of performing an acromioplasty as part of arthroscopic repair of the rotator cuff also exists. Hyvonen et al[19] reported that acromioplasty does not prevent the progression of impingement syndrome to a rotator cuff tear.After open acromioplasty, shoulders that were symptom-free following the surgical procedure had all become painful and demonstrated signs of rotator cuff degeneration after 5 years. Neer argued that, in addition to the acromion, the coracoacromial ligament may contribute to external impingement. Edelson and Taitz reported that traction spurs were seen at the insertion of the coracoacromial ligament in 18% of 200 acromion specimens with no changes on the corresponding coracoid process. The spurs may have been isolated to the acromion because the coracoid process has a greater area of insertion to distribute the tensile forces. Free nerve endings and neovascularity have been identified in the coracoacromial ligament of subjects with clinically diagnosed subacromial impingement syndrome when compared with subjects undergoing anterior shoulder stabilisation procedures.These changes may have occurred as a result of chronic strain and suggest that the ligament itself may be a source of symptoms..Mechanical abrasion from the under surface of the acromion or coracoacromial ligament should result in abrasion to the superior (bursal-side) surface of the rotator cuff; however, this is not found during surgery. Two studies have shown that the majority of partial-thickness tears were intrasubstance or jointside tendon tears and not on the upper bursal side of the tendon adjacent to the acromion.The bias toward joint-side rotator cuff pathology has led a number of investigators to conclude that acromially initiated rotator cuff pathology does not occur arguing that extrinsic impingement (and not intrinsic failure) causes pathology on the articular side of the tendon. This model of impingement is known as internal impingementor superior and posterosuperior impingement, reflecting the belief that the rotator cuff is mechanically damaged by compression between different parts of the superior aspect of the glenoid fossa and the greater tuberosity of the humerus.Although the concept of internal impingement has gained clinical acceptance, substantial ongoing biomechanical, laboratory, imaging and clinical research is required. Wuelker et al concluded that the inferior components of the rotator cuff counteract the tendency of the deltoid and the supraspinatus to superiorly translate the humerus and thus reduce subacromial pressure. Subacromial pressure varies with arm position, and maximal subacromial pressure (66.9 mmHg) was reported at 90u abduction, with slightly less pressure recorded at 90u flexion (61.1 mmHg). Subacromial pressure was more than three times greater in shoulder internal rotation (35.6 mmHg) than in external rotation (13.7 mmHg), These findings question the appropriateness of the simultaneous prescription of both internal and external rotation exercise for patients with a diagnosis of rotator cuff tendinopathy or impingement.

CONCLUSION:

The etiology and pathological factors of rotator cuff failure is multifactorial and results from a combination of intrinsic, extrinsic and environmental factors. The specialised morphology of the rotator cuff, together with the effects of stress shielding, may contribute to the development of rotator cuff tendinopathy. Acute and chronic tendon overload may also result in increased volume in the confined subacromial space, which may have a potentially catabolic effect on intratendinous and bursal homeostasis and may trigger the cascade of cytokines, neuropeptides and other chemicals that have been identified within the tendon and bursal tissue. Both tendon overloading and underloading will influence the balance of MMPs and TIMPs and have a detrimental effect on normal tendon remodelling. The tissues medial to the rotator cable may act through the cable to produce movement and the more lateral tissues of

the rotator crescent may be stress-shielded, and this may be more pronounced in the articular-side fibres. A considerable body of research is necessary to more fully understand the rotator cuff tendinopathy and its relationship with bursal pathology. Once this knowledge exists, more effective management will become available.

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