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Rotator Cuff Tendonitis

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Introduction

Injuries to the rotator cuff (RC) range from simple contusions and tendonitis to chronic tendinopathy, partial tears, and full-thickness tears (PTTs versus FTTs). RC pathology can affect any subset of patient populations, from the casual "weekend warrior" to elite-level professional athletes. Similarly, RC pathology is seen across all age populations.[1][2]

Etiology

While subacromial impingement syndrome (SIS) is the most common cause of shoulder pain, rotator cuff (RC) tendonitis is often seen in association with shoulder impingement. RC tendonitis can present in the acute setting following injury or, chronically, as a result of repetitive overuse activities or sport-related demands [1][2][3].

Acute Rotator Cuff Tendonitis

Acutely, RC tendonitis afflicts athletes at all levels of competition. Acute injuries often occur secondary to direct trauma to the shoulder in contact sports, poor throwing mechanics in overhead sports (i.e., baseball, javelin throwers), or from falls on an outstretched arm.[1]

Chronic Rotator Cuff Tendinopathy

Chronically, RC tendinopathy can occur secondary to a variety of proposed mechanisms:

Extrinsic compression

The extrinsic theory of mechanical impingement and pathologic contact between the undersurface of the acromion and the RC results in repetitive injury to the cuff. RC tendinopathy results in weakened areas of the cuff, eventually resulting in PTTs and/or FTTs. The mechanical compression can occur secondary to a degenerative bursa, acromial spurring, and predisposing acromial morphologies (i.e., the hooked-type acromion). Theories were popularized and modified by Watson-Jones, Neer, and Bigliani.[2][4][5]

Intrinsic mechanisms

Several theories exist to support intrinsic degeneration of the cuff as the primary source of shoulder impingement. In general, the intrinsic degenerative theories cite that cuff degeneration eventually compromises the overall stability of the glenohumeral joint. Once compromised, the humeral head migrates superiorly, and the subacromial space decreases in size. Thus, the cuff becomes susceptible to *secondary* extrinsic compressive forces, ultimately leading to cuff degeneration, tendinopathy, and tearing.[2][6]

- *Vascular changes* - Advocates for intrinsic degenerative theories cite focal vascular adaptations that occur secondary to age-related changes and intrinsic cuff failure from repetitive eccentric forces directly

articular side that lacks blood vessel supply 1 cm proximal to its insertional footprint.[7] Rudzki et al. demonstrated an age-dependent manner of increasing hypovascular regions in these distal cuff regions as well.[8] Controversy proposed by other studies, however, supports that the attritional areas develop secondary to the preceding impingement mechanisms. Subsequently, external impingement (EI) leads to blood vessel damage, ensuing ischemia, tenocyte apoptosis, gross tendinopathy, and attritional cuff damage.[2] Furthermore, many studies cite increased vascularity in focal areas of the cuff, and the hypervascularity has been associated with age-related changes, tendinopathy, and PTTs and/or FTTs.[9][10]

- *Age, sex, and genetics* - Histologically, age-related RC changes include collagen fiber disorientation and myxoid degeneration.[11] The literature favors increasing frequencies of RC abnormalities with increasing age. The frequency increases from 5% to 10% in patients younger than 20 years of age, to 30% to 35% in those in their sixth and seventh decades of life, topping out at 60% to 65% in patients over 80 years of age.[6][12][13]
- *Tensile forces* - A study by Budoff et al. proposed that the primary mode of failure of the cuff occurs intrinsically within the cuff itself as it repeatedly withstands significant eccentric tensile forces during physical activity.[14]

Epidemiology

Shoulder pain comprises the third-leading musculoskeletal complaint for physician office visits. Following spine and knee complaints, shoulder complaints account for 4.5 million visits and $3 billion in associated healthcare costs in the United States alone.[15] With a lifetime prevalence rate of 67%, shoulder pain is most commonly caused by SIS and/or RC pathologies.[2][6][12][13] Rotator cuff (RC) injuries afflict patient populations in an age-dependent fashion. Prevalence rates increase from 5% to 10% in patients younger than 20 years of age to over 60% in patients over age 80 years old.[6]

Pathophysiology

Acute rotator cuff (RC) tendonitis can occur secondary to direct blows to the shoulder, poor throwing mechanics in overhead sports, or from falls on an outstretched arm.[1]

Tendinopathy ensues after repetitive RC injury triggers a recurrent pathological cycle that results in acute on chronic tendonitis, increasing levels of tendinopathy and tendinosis, and ultimately, PTTs and/or FTTs to varying degrees of tear sizes and retraction. The exact pathogenesis of RC tears still remains controversial, but most clinicians agree the underlying mechanism is comprised of a combination of extrinsic impingement from structures surrounding the cuff and intrinsic degeneration from changes within the tendon itself.[16]

Histopathology

Rotator cuff (RC) tendinopathic histologic changes include the following [9][10][11][13][17][9]:

- Rounded tenocytes (apoptosis)
- Extracellular matrix disorganization and myxoid degeneration
- Vascular changes (focal hypervascularity; focal hypovascular regions as well)
- Reduced total cellularity
- Calcified depositions
- Collagen fiber thinning
- Degenerative acromion, coracoacromial ligament (CAL)

History and Physical

A comprehensive history should be obtained by clinicians evaluating patients with acute or chronic shoulder pain. Characteristics of a history of potential rotator cuff (RC) injury include:

- Acute RC tendonitis: history of trauma and/or acute on chronic exacerbation

presentation

- symptom exacerbation with overhead activity
- pain at night

A thorough history includes current or history of sports participation (as well as specific positions played in each sport), occupational history and current status of employment, hand dominance, any history of injury/trauma to the shoulder(s) and/or neck, and any relevant surgical history.

Physical Examination Pearls

C-spine/neck exam**[18]**:

Co-existing cervical radiculopathy should be ruled out in any situation where neck and/or shoulder pathology is in consideration. Observation of neck posturing, muscular symmetry, palpable tenderness, and active/passive ROM should be evaluated. Special tests that are helpful in this regard include the Spurling maneuver, myelopathic testing, reflex testing, and a comprehensive neurovascular exam.

Shoulder exam[2][6][19][20][21]**:**

Clinicians must observe the overall shoulder girdle for assessment of symmetry, shoulder posturing, and overall muscle bulk and symmetry. Scapular winging should also be ruled out. The skin should be observed for the presence of any previous surgical incisions, lacerations, scars, erythema or induration.

After the observational component of the physical examination, the shoulder should be palpated for any areas of tenderness. Classically, RC tendonitis correlates with anterolateral tenderness to palpation. Next, the active and passive ROM of both shoulders is documented. In cases of RC tendonitis, patients may demonstrate compromised active ROM but should exhibit full passive ROM. In the absence of advanced degenerative changes affecting the glenohumeral joint, limited passive ROM is considered diagnostic for adhesive capsulitis and involves a separate treatment algorithm from RC tendinopathy/impingement.

The clinician can assess motor strength grading for C5 to T1 nerve roots in addition to specific RC muscle strength testing. Specifically, RC strength and/or pathology can be assessed via the following examinations:

1. Supraspinatus (SS)

- **Jobe's test**: a positive test is pain/weakness with resisted downward pressure while the patient's shoulder is at 90 degrees of forward flexion and abduction in the scapular plane with the thumb pointing toward the floor.
- **Drop arm test**: the patient's shoulder is brought into a position of 90 degrees of shoulder abduction in the scapular plane. The examiner initially supports the limb and then instructs the patient to slowly adduct the arm to the side of the body. A positive test includes the patient's inability to maintain the abducted position of the shoulder and/or an inability to adduct the arm to the side of the trunk in a controlled manner.

2. Infraspinatus (IS)

- **Strength testing** is performed while the shoulder is positioned against the side of the trunk, the elbow is flexed to 90 degrees, and the patient is asked to externally rotate (ER) the arm while the examiner resists this movement.
- **External rotation lag sign**: the examiner positions the patient's shoulder in the same position, and while holding the wrist, the arm is brought into maximum ER. The test is positive if the patient's shoulder drifts into internal rotation (IR) once the examiner removes the supportive ER force at the wrist.

3. Teres Minor (TM)

- **Strength testing** is performed while the shoulder positioned at 90 degrees of abduction and the elbow is also flexed to 90 degrees. Teres minor (TM) is best isolated for strength testing in this position while ER is resisted by the examiner.

shoulder under support. A positive test occurs when the patient is unable to hold this position, and the arm drifts into IR once the examiner removes the supportive ER force.

4. Subscapularis (SubSc)

- **IR lag sign**: the examiner passively brings the patient's shoulder behind the trunk (about 20 degrees of extension) with the elbow flexed to 90 degrees. The examiner passively IRs the shoulder by lifting the dorsum of the handoff of the patient's back while supporting the elbow and wrist. A positive test occurs when the patient is unable to maintain this position once the examiner releases support at the wrist (i.e., the arm is not maintained in IR, and the dorsum of the hand drifts toward the back).
- **Passive ER ROM**: a partial or complete tear of the subscapularis (SubSc) can manifest as an increase in passive ER compared to the contralateral shoulder.
- **Lift-off test**: more sensitive/specific for lower SubSc pathology. In the same position as the IR lag sign position, the examiner places the patient's dorsum of the hand against the lower back and then resists the patient's ability to lift the dorsum of the hand away from the lower back.
- **Belly press**: more sensitive/specific for upper subscapularis pathology. The examiner has the patient's arm at 90 degrees of elbow flexion, and IR testing is performed by the patient pressing the palm of his/her hand against the belly, bringing the elbow in front of the plane of the trunk. The elbow is initially supported by the examiner, and a positive test occurs if the elbow is not maintained in this position upon the examiner removing the supportive force.

5. External impingement/SIS

- **Neer impingement sign**: positive if the patient reports pain with passive shoulder forward flexion beyond 90 degrees.
- **Neer impingement test**: positive test occurs after a subacromial injection is given by the examiner and the patient reports improved symptoms upon repeating the forced passive forward flexion beyond 90 degrees.
- **Hawkins test**: positive test occurs with the examiner passively positioning the shoulder and elbow at 90 degrees of flexion in front of the body; the patient will report pain when the examiner passively IR's the shoulder.

6. Internal impingement

- **Internal impingement test**: the patient is placed in a supine position and the shoulder is brought into terminal abduction and external rotation; a positive test consists of the reproduction of the patient's pain.

Evaluation

Radiographic imaging should be obtained in all patients with acute or chronic shoulder pain.

Radiographs[2]

Recommended imaging includes a true anteroposterior (AP) image of the glenohumeral joint (i.e., the "Grashey" view). The true AP image is taken with the patient rotated between 30 and 45 degrees offset the cassette in the coronal plane. Alternatively, the beam can be rotated while the patient remains neutral in the coronal plane. The distance between the acromion and the humeral head (i.e., the acromiohumeral interval) can be calculated. A normal interval is between 7 and 14 mm, and this interval is decreased in cases of advanced degenerative arthritis and RCA.

Other radiographic imaging includes a 30-degree caudal tilt view that can be performed to visualize the presence of acromial spurring. In addition, the "scapular Y" or "supraspinatus outlet" view is used to determine acromial morphology.

Pertinent findings

The following are the most common radiographic changes associated with rotator cuff (RC) pathology:

- ***RCA:*** Proximal humeral migration and decreases in the acromiohumeral interval to <7mm

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    - osteophytes on the acromion, proximal humerus and/or glenoid are often seen in cases of advanced disease
    - calcification of the CAL and/or coracohumeral ligament (CHL)
    - greater tuberosity cystic degeneration
    - AC joint arthritis
- ***"Hooked" acromion:*** best appreciated on the supraspinatus outlet view
- ***Os acromiale:*** best seen on an axillary lateral radiograph

Ultrasound[6]

Ultrasound (US) is an often-underutilized imaging modality to detect RC tendon and muscle belly integrity. In 2011, a meta-analysis of over 6,000 shoulders revealed a sensitivity of 0.96 and specificity of 0.93 in assessing shoulders for PTTs or FTTs.

Magnetic Resonance Imaging[2]

Magnetic resonance imaging (MRI) is useful in evaluating the overall degree of RC pathology. MRI can be helpful in providing more accurate cuff tear details, including partial- versus full-thickness tears, the extent and size of the tear(s), location, and degree of retraction. In cases of chronic RC pathology, the cuff can be assessed for fatty degenerative changes on the T1-weighted sagittal sequence series.

Subdeltoid and/or subacromial bursitis can also be evident and are important considerations as potential sources of pain. In addition, the acromioclavicular (AC) joint, acromial morphology, and long head of the biceps (LHB) tendon integrity are better appreciated. A systematic approach to reviewing shoulder MRIs is important, especially when correlating the MRI findings with the patient reported symptoms and clinical examination.

Treatment / Management

Given the complex nature of rotator cuff (RC) tendonitis, we recommend the treatment and management be broken down into the following categories:

Group 1: Partial-Thickness (PTT) or Full-Thickness RC Tears (FTTs), Asymptomatic Patient *[22]*

Patients presenting with MRI-evidence of PTTs or FTTs often present without any symptoms. The most recent American Academy of Orthopaedic Surgeons (AAOS) clinical practice guideline (CPG) summary reported the growing awareness of incidental RC pathology revealed via shoulder MRIs in asymptomatic patient populations. Although there is evidence of increasing prevalence of RC disease in the aging population, there is no reliable evidence that surgical intervention prevents tear propagation or the development of clinical symptoms. Thus, the committee recommended symptomatic management via nonoperative modalities alone.

Group 2: Partial-Thickness (PTT), Symptomatic Patients*[22][23][22]*

Patients presenting with symptoms of EI/SIS in the absence of FTTs are first managed with nonoperative treatment modalities. There is no agreed upon time interval of when is most appropriate to proceed with surgical intervention in this particular group of patients. The literature ranges from 3 months to 18 months. Surgical intervention should be individually tailored based on the patient's symptoms, improvement with nonoperative modalities, and overall goals of the patient.

Group 3: Chronic RC Tears, Symptomatic Patients*[22]*

The AAOS CPG reported a "weak" recommendation grade secondary to limited available evidence in the literature comparing rotator cuff repair (RCR) to continued nonoperative treatment modalities in this subset of patients. Certainly, the overall clinical picture must be considered, and the treatment tailored to the individual patient in each scenario.

Nonoperative RCS Treatment Modalities*[22][23]*

Physical therapy (PT)

of PTTs, patients can still be managed with PT alone.

- PT modalities include aggressive RC and periscapular stabilizer strengthening programs, as well as ROM exercises.

Anti-inflammatory medications

- First-line nonoperative management also includes the use of non-steroidal anti-inflammatory (NSAIDs) medications in conjunction with PT modalities.
- Although the AAOS CPG reported inconclusive evidence for the use of NSAIDs, iontophoresis, transcutaneous electrical nerve stimulations (TENS), and other similar therapy modalities in the presence of FTTs, the committee reported a "moderate" recommendation grade for exercises and/or NSAIDs in the presence of RCS symptoms in the absence of FTTs.

Cortisone injections

- For RC tendonitis, the most utilized injection includes one into the subacromial space. Patients can experience symptomatic relief almost instantaneously after the injection is given, allowing them to participate in subsequent PT therapy sessions ideally.

Rest/Activity modifications

- Patients benefit from an initial period of rest from the exacerbating activity (occupation or sport), especially repetitive overhead activity and heavy lifting.

Surgical Management

RCS surgical techniques range from debridement, subacromial decompression (SAD), and/or acromioplasty to RC debridement and, when indicated, RC bursal- or articular-sided tear completion with RCR. The latter will not be discussed in this review. Assuming no RC FTTs are present, the extent of surgical management for external impingement/SIS alone includes[22][23][24]:

Subacromial decompression

- Extensive debridement of the subacromial space is beneficial in patients with persistent symptoms of EI/SIS after at least 4 to 6 months of failed nonoperative modalities.
- Comprehensive bursectomy allows for thorough and more accurate evaluation of the bursal side of the cuff itself.
- CAL debridement is recommended in the setting of substantial CAL fraying and/or calcification as this is considered an additional source of impingement.
- A meta-analysis of 9 studies comparing open versus arthroscopic procedures yielded equivalent surgical times, outcomes, and complication rates at 1-year followup; the arthroscopic cohort returned to work quicker compared to the open cohort.

Acromioplasty

- Shaving the undersurface of the acromion, especially in the setting of significant spurring, improves the environment surrounding the cuff and allows additional clearance distance between the acromion and cuff itself throughout mid-arc and terminal range of motion (ROM) and impingement positions.
- In the case of hooked acromion morphologies, care is taken to debride this area with a shaver, burr, or rasp to flatten the undersurface.
- The anterior extent of the acromioplasty is demarcated by the anterior deltoid origin. This area should be respected in the debridement process. The anteroinferior region of the acromion is a common site of spurring and causes impingement symptoms in these patients.

Os Acromiale

- In the case of persistent symptoms, a two-stage procedure is often utilized. First, the os acromiale is fused using bone grafting techniques, followed by a formal acromioplasty after healing is achieved.

Differential Diagnosis

The differential diagnosis for chronic shoulder pain includes several etiologies:

Impingement

- External/SIS
- Subcoracoid
- Calcific tendonitis
- Internal (including SLAP lesions, glenohumeral internal rotation deficit (GIRD), Little league shoulder, posterior labral tears)

Rotator Cuff (RC) Pathology

- Tendonitis (acute), tendinopathy (chronic or acute on chronic)
- Partial- versus full-thickness tears (PTTs versus FTTs)
- RCA

Degenerative

- Advanced DJD, often associated with RCA
- Glenohumeral arthritis
- Adhesive capsulitis
- Avascular necrosis (AVN)
- Scapulothoracic crepitus

Proximal Biceps

- Subluxation–often seen in association with SubSc injuries
- Tendonitis and tendinopathy

AC Joint Conditions

- AC separation
- Distal clavicle osteolysis
- AC arthritis

Instability

- Unidirectional instability–seen in association with an inciting event/dislocation (anterior, posterior, inferior)
- Multidirectional instability (MDI)
- Associated labral injuries/pathology

Neurovascular Conditions

- Suprascapular neuropathy – can be associated with paralabral cyst at the spinoglenoid notch
- Scapular winging–medial or lateral
- Brachial neuritis
- Thoracic outlet syndrome (TOS)
- Quadrilateral space syndrome

Other Conditions

- Os acromiale
- Muscle ruptures (pectoralis major, deltoid, latissimus dorsi)
- Fracture (acute injury or pain resulting from long-standing deformity, malunion, or nonunion)

Prognosis

The majority of patients with rotator cuff (RC) tendinopathy in the absence of FTTs improve with nonoperative management. The most recent AAOS CPGs touted a "moderate" recommendation grade for initial treatment of NSAIDs and/or exercises programs based on multiple level II studies in the literature.

Complications

Complications associated with rotator cuff syndrome (RCS) are best broken down into nonoperative- versus operative-related complications:

Nonoperative Management

- Persistent pain/recurrent symptoms
- In the setting of PTTs, there is at least a theoretical risk of tear propagation, lack of healing, fatty infiltration, atrophy, and retraction.
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    - Overall a controversial topic, a 2017 study analyzed independent risk factors for symptomatic RC tear progression over a 19-month period of nonoperatively managed shoulders; risk factors for tear progression included:
        -
            - The initial presence of an FTT
            - Medium-sized cuff tears (1 to 3 cm)
            - Smoking
            - While PTTs were included in the study, the presence of a PTT was not a risk factor for cuff tear progression
- In the setting of chronic/atrophic tears, especially with RC tear propagation, degenerative joint disease and RCA ensue

Surgical Management

- Surgical treatment tends to be most effective in patients that have failed or reported persistent or worsening symptoms despite at least 4-6 months of exhaustive nonoperative treatment modalities
- The standard risks of surgery, including recurrent pain/symptoms, infection, stiffness, neurovascular injury, and risks associated with anesthetic use
- SAD/acromioplasty
    -
    - Deltoid dysfunction: can occur secondary to failed deltoid repair following an open acromioplasty or excessive debridement during arthroscopy
    - Anterosuperior escape: occurs secondary to aggressive CAL release–the coracoacromial arch and suspensory system becomes compromised, and with CAL release in the setting of massive, retracted, and irreparable RC tears, the humeral head migrates superiorly and anteriorly to compromise patient functional outcomes

Postoperative and Rehabilitation Care

ROM rehabilitation parameters. Clinicians should recognize the risk of overutilization of sling immobilization, especially when soft tissue repair protocols do not have to be respected.

Initially, patients are instructed to avoid heavy lifting and exercises to facilitate soft tissue healing. Cryotherapy devices are often applied for the first 10 to 14 days postoperatively. Physical therapy is often started postoperatively once the sling is discontinued.

Full active ROM should be achieved by 3 to 6 weeks maximum. If applicable, return to sport-specific skills at 6 to 8 weeks as tolerated.

Consultations

Primary care medical doctors can manage the majority of rotator cuff syndrome (RCS) cases, especially along the nonoperative management spectrum. After these modalities are exhausted, an appropriate referral can be made to an orthopedic surgeon. While the nonoperative management is not conclusively managed with a supervised physical therapy protocol, a supervised protocol is recommended in the postoperative setting to regain maximum ROM and full strength.

Deterrence and Patient Education

Patients should be educated about the condition as well as the possibility of continued chronic pain following surgical management for persistent impingement/rotator cuff (RC) tendonitis symptoms.

Enhancing Healthcare Team Outcomes

Rotator cuff (RC) tendonitis is a clinical entity consisting of a wide range of clinical symptoms ranging in severity from mild shoulder impingement and can advance in the long-term setting to progressive partial thickness cuff tears (PTTs) and/or full-thickness cuff tears (FTTs). When clinicians are working up acute or chronic shoulder pain, it is imperative to correlate the clinical examination with radiographic imaging, MRI, and response to nonoperative treatment modalities. The latter consists of physical therapy, NSAIDs, rest/activity modification, and injections. Surgical management is considered after impingement symptoms fail to resolve or worsen after all other management modalities are exhausted. Referral to an orthopedic surgeon should be considered, especially with long-standing persistent symptoms.

Questions

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References

1. Weiss LJ, Wang D, Hendel M, Buzzerio P, Rodeo SA. Management of Rotator Cuff Injuries in the Elite Athlete. Curr Rev Musculoskelet Med. 2018 Mar;11(1):102-112. [PMC free article: PMC5825345] [PubMed: 29332181]

2. Harrison AK, Flatow EL. Subacromial impingement syndrome. J Am Acad Orthop Surg. 2011 Nov;19(11):701-8. [PubMed: 22052646]

3. Greenberg DL. Evaluation and treatment of shoulder pain. Med. Clin. North Am. 2014 May;98(3):487-504. [PubMed: 24758957]

4. Bigliani LU, Levine WN. Subacromial impingement syndrome. J Bone Joint Surg Am. 1997 Dec;79(12):1854-68. [PubMed: 9409800]

5. Neer CS. Impingement lesions. Clin. Orthop. Relat. Res. 1983 Mar;(173):70-7. [PubMed: 6825348]

6. Sambandam SN, Khanna V, Gul A, Mounasamy V. Rotator cuff tears: An evidence based approach. World J Orthop. 2015 Dec 18;6(11):902-18. [PMC free article: PMC4686437] [PubMed: 26716086]

7. Lohr JF, Uhthoff HK. The microvascular pattern of the supraspinatus tendon. Clin. Orthop. Relat. Res. 1990 May;(254):35-8. [PubMed: 2323147]

8. Rudzki JR, Adler RS, Warren RF, Kadrmas WR, Verma N, Pearle AD, Lyman S, Fealy S. Contrast-enhanced ultrasound characterization of the vascularity of the rotator cuff tendon: age- and activity-related changes in the intact asymptomatic rotator cuff. J Shoulder Elbow Surg. 2008 Jan-Feb;17(1 Suppl):96S-100S. [PubMed: 18069013]

9. Buck FM, Grehn H, Hilbe M, Pfirrmann CW, Manzanell S, Hodler J. Magnetic resonance histologic correlation in rotator cuff tendons. J Magn Reson Imaging. 2010 Jul;32(1):165-72. [PubMed: 20578021]

10. Feeney MS, O'dowd J, Kay EW, Colville J. Glenohumeral articular cartilage changes in rotator cuff disease. J Shoulder Elbow Surg. 2003 Jan-Feb;12(1):20-3. [PubMed: 12610481]

11. Longo UG, Franceschi F, Ruzzini L, Rabitti C, Morini S, Maffulli N, Forriol F, Denaro V. Light microscopic histology of supraspinatus tendon ruptures. Knee Surg Sports Traumatol Arthrosc. 2007 Nov;15(11):1390-4. [PubMed: 17721701]

12. Gumina S, Candela V, Passaretti D, Latino G, Venditto T, Mariani L, Santilli V. The association between body fat and rotator cuff tear: the influence on rotator cuff tear sizes. J Shoulder Elbow Surg. 2014 Nov;23(11):1669-74. [PubMed: 24906904]

13. Dean BJ, Franklin SL, Carr AJ. A systematic review of the histological and molecular changes in rotator cuff disease. Bone Joint Res. 2012 Jul;1(7):158-66. [PMC free article: PMC3626275] [PubMed: 23610686]

14. Budoff JE, Nirschl RP, Guidi EJ. Débridement of partial-thickness tears of the rotator cuff without acromioplasty. Long-term follow-up and review of the literature. J Bone Joint Surg Am. 1998 May;80(5):733-48. [PubMed: 9611036]

15. Oh LS, Wolf BR, Hall MP, Levy BA, Marx RG. Indications for rotator cuff repair: a systematic review. Clin. Orthop. Relat. Res. 2007 Feb;455:52-63. [PubMed: 17179786]

16. Yang H, Lee HJ, Lee YG, Kim JW, Kim YS. Integrity of the Untorn Articular-Sided Tendon in Bursal-Sided Partial-Thickness Rotator Cuff Tear: A Comparative Study of Apoptotic Activity in Torn and Untorn Layers. Am J Sports Med. 2018 Aug;46(10):2478-2485. [PubMed: 29975554]

17. Pozzi F, Seitz AL, Plummer HA, Chow K, Bashford GR, Michener LA. Supraspinatus tendon micromorphology in individuals with subacromial pain syndrome. J Hand Ther. 2017 Apr - Jun;30(2):214-220. [PubMed: 28502699]

18. Linaker CH, Walker-Bone K. Shoulder disorders and occupation. Best Pract Res Clin Rheumatol. 2015 Jun;29(3):405-23. [PMC free article: PMC4836557] [PubMed: 26612238]

19. Doxey R, Thiese MS, Hegmann KT. Reliability of Common Provocative Tests for Shoulder Tendinitis. J. Occup. Environ. Med. 2018 Dec;60(12):1063-1066. [PubMed: 30096066]

20. Bakhsh W, Nicandri G. Anatomy and Physical Examination of the Shoulder. Sports Med Arthrosc Rev. 2018 Sep;26(3):e10-e22. [PubMed: 30059442]

21. Holmes RE, Barfield WR, Woolf SK. Clinical evaluation of nonarthritic shoulder pain: Diagnosis and treatment. Phys Sportsmed. 2015 Jul;43(3):262-8. [PubMed: 25622930]

22. Tashjian RZ. AAOS clinical practice guideline: optimizing the management of rotator cuff problems. J Am Acad Orthop Surg. 2011 Jun;19(6):380-3. [PubMed: 21628649]

23. Wolff AB, Sethi P, Sutton KM, Covey AS, Magit DP, Medvecky M. Partial-thickness rotator cuff tears. J Am Acad Orthop Surg. 2006 Dec;14(13):715-25. [PubMed: 17148619]

24. Paavola M, Malmivaara A, Taimela S, Kanto K, Inkinen J, Kalske J, Sinisaari I, Savolainen V, Ranstam J, Järvinen TLN., Finnish Subacromial Impingement Arthroscopy Controlled Trial (FIMPACT) Investigators. Subacromial decompression versus diagnostic arthroscopy for shoulder impingement: randomised, placebo

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