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Can imaging determine if a rotator cuff tear is traumatic?

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The purpose of this review paper is to clarify if imaging studies can determine whether a rotator cuff tear is traumatic or atraumatic in aetiology. The impetus for this review is the important issue of entitlement for Accident Compensation Corporation (ACC)-funded treatment in New Zealand.

In this paper we review specific information that can be reported on plain X-rays, ultrasound scans, CT and MRI scans. Literature searches have been undertaken to investigate radiological features that have been speculated to be associated with atraumatic rotator cuff disease. The historical and recent relevant research has been reviewed. Conclusions are based on literature evidence. The review includes some definitions and grading systems so the reader may better understand the topic.

Acromial morphology

Historical—In 1934 Codman, considered acromial morphology was a potential cause of rotator cuff tendon pathology.[1] Codman's conclusions were based on observation of cadaver shoulders.

In 1972 Neer published his results of anterior acromioplasty in 50 shoulders. He postulated that impingement on the tendinous portion of the rotator cuff by the coracoacromial ligament and anterior acromion was responsible for the syndrome of chronic impingement. It is important to note however that this study excluded acute traumatic tears, there was no radiographic assessment of the acromion and the study did not prove causation.[2]

Bigliani and Morrison presented a cadaveric study on the morphology of the acromion in 1986. They examined 140 cadaveric shoulders with a mean age of 74 years.[3] They identified three types of acromion with scapular lateral X-rays. 17.1% were type 1 (flat), 42.9 % type 2 (curved) and 39.3% type 3 (hooked). 24 % of the cadaver shoulders had a full thickness rotator cuff tear. 3 % of the rotator cuff tears were in type 1 acromions, 24 % in type 2 and 70% in type 3 respectively. The type 2 (curved) acromion was the most common. Only 8 of the 60 specimens with type 2 acromions had a full thickness rotator cuff tear (13%). There was no information regarding pre-morbid history of shoulder symptoms or trauma.

Another acromial morphological variation that has been considered to have an association with rotator cuff pathology is the os acromiale. This is an anatomical variation in which the ossification centres of the acromion do not fuse with bone. In 1984 Mudge et al published a review of 8 patients with rotator cuff tears who also had os acromiale.[4]

Recent research—A prerequisite for a radiological classification to be useful is the classification being reliable and reproducible. Stehle et al's cadaveric radiological study demonstrated significant variation in the appearance of the acromion with projectional changes.[5] Bright et al examined reliability of assessment of acromial morphology concluding that there was '*unacceptable variability of interpretation and grading of radiographs*'.[6]

Ogawa et al examined the relationship of subacromial spurs to rotator cuff tear and tear morphology in cadavers, patients with cuff tears and a control group. Subacromial spurs were classified as small (<5 mm), medium (5 mm–9 mm) and large (≥10 mm).[7] A large amount of information is presented in the results and many aspects are relevant to this paper. Spur size increased with increasing age. The authors concluded '*The presence of a small spur has no diagnostic value for rotator cuff tears. Spurs measuring 5 mm or more, however, are of diagnostic value because of the high rate of association with bursal-side tear, complete tears limited to the supraspinatus tendon, or massive tears.*'[7]

Pearsall et al, however, found no association between acromial morphology and rotator cuff tears. They concluded that acromial shape and AC joint changes are not predictive of a full thickness rotator cuff tear. Standard shoulder radiographs in these patients however, did demonstrate greater tuberosity sclerosis, osteophytes, subchondral cysts, and osteolysis that are not noted in patients without shoulder symptoms.

Gill et al[9] identified a strong association between rotator cuff pathology and acromial morphology when no adjustments for patients age were made. However stratified univariate analysis did not demonstrate an independent association between full thickness rotator cuff tears and type 3 acromions in patients over 50 years of age.[9] The authors concluded that '*Previous reports linking full-thickness cuff tears to acromial morphology may simply have been describing the association of type III acromions and cuff tears with aging, rather than a true causal relationship.*'[7]

The association between os acromiale and cuff pathology has been re-examined by Ouellette et al in 2007; 42 patients with os acromiale who were compared with age and gender matched controls with MRI.[10] They were unable to demonstrate an association between os acromiale and tears of the rotator cuff.

Conclusions—The historical concept that acromial morphology is a significant contributor is based upon clinical and cadaver observations, not studies designed to assess causation. More recent studies have demonstrated that acromial morphology is unreliably and inconsistently assessed by radiographs. There is an increasing incidence of subacromial spurs and rotator cuff pathology with normal aging. Although there are conflicting conclusions in different studies, some studies report that patients with a large subacromial spur are more likely to have rotator cuff pathology. Evidence of a causal relationship is lacking. An os acromiale cannot be considered an aetiological factor in rotator cuff tears.

Acromioclavicular joint arthrosis

Historical—Neer, in his landmark paper of 1972, mentioned that excrescences on the under surface of the AC joint may potentially impinge upon the cuff.

Recent research—Shubin Stein et al examined the MRI appearances of symptomatic and asymptomatic AC joints.[11] Reactive bone oedema was a more reliable predictor of symptoms from the AC joint than was the severity of AC arthritis on MRI.

Relevant to our review is the extremely high prevalence of AC arthritis on imaging studies. The prevalence of AC joint arthritis using MRI was 82% in asymptomatic shoulders. In those less than 30 years, 68% had MRI features of arthritis, while the prevalence rose to 93% in those over 30 years. The authors concluded that radiological AC joint arthritis may not be clinically relevant.[11]

Needell et al[12] demonstrated AC joint arthritis correlated more closely with age than MRI evident tendon abnormalities. In their study, AC joint arthritis was seen in 89% of shoulders in those over 40 years and 76% of shoulders overall.[12] Approximately two-thirds of those with normal tendons had AC arthritis.

Conclusions—Contemporary studies show that radiographic appearances of AC joint arthrosis are common and increase in prevalence with age. Imaging findings of AC arthrosis can be considered normal for age in patients over 40 years of age and may be found as an asymptomatic finding in younger age groups as well. There is no evidence that AC arthritis causes rotator cuff tears or indicates the presence of a rotator cuff tear.

Greater tuberosity changes

Historical—In 1934 Codman, in a cadaver study, described an association between partial thickness rotator cuff tears and eburnation at the sulcus, or interval between the articular cartilage and greater tuberosity.[1,13] No radiographic correlation was made. In 1964 Cotton et al performed a cadaver study with radiology and dissection.[14]

The authors concluded that cysts were indicative of tears. Sclerosis and cortical thickening in the absence of other changes were not found to be a significant predictor of cuff tearing. Unfortunately only 6/136 of the radio graphically normal shoulders were dissected for comparison. Other groups have reported similar findings associating tuberosity sclerosis, cortical thickening and cyst change with rotator cuff tears. Unfortunately, the majority of these are either anecdotal or lack controls.[15]

Recent research—In 1999, Huang et al published an MRI and radiological study titled 'Greater tuberosity changes as revealed by radiography: Lack of clinical usefulness in patients with rotator cuff disease.'[15] The authors concluded '*Cortical thickening of the greater tuberosity and subcortical sclerosis are not associated with rotator cuff disease. For some observers, identifying cyst-like lesions is associated with rotator cuff disease, but the clinical usefulness of the observation is limited by high interobserver variability and poor positive predictive value.*'[15]

McCualey et al examined bone marrow oedema in the greater tuberosity on MRI scans. Bone marrow oedema was a rare finding. The authors concluded that traumatic rotator cuff tear with oedema may result from avulsion forces on the supraspinatus insertion.[16]

Williams et al examined the relationship greater tuberosity cysts and both age and rotator cuff tears. Cysts were present in 70% of patients and were 7 times more frequent in the posterior aspect of the tuberosity than the anterior aspect of the tuberosity. Tuberosity cysts and rotator cuff tears did not appear to be related.[17]

In 2007, Fritz et al published on the relationship of greater tuberosity cysts and rotator cuff disorders. MRI better demonstrates the position of the cysts than plain X-rays.

Posterior cysts were more common, occurring in 56.7% of shoulders and showed no correlation with age or rotator cuff disorders.

Anterior cysts occurred in 22.7% of shoulders and were strongly associated with rotator cuff disorders with 94% of patients with anterior cysts having a rotator cuff disorder (p<0.001). They defined rotator cuff disorders as including tendinopathy (a surgical diagnosis of the appearance of the tendon), partial thickness tears and full thickness tears. With regard to full thickness tears of the rotator cuff, 48% of those with anterior cysts had a rotator cuff tear.[13]

Conclusions—Greater tuberosity cysts are common. The location of cysts is more accurately determined on MRI or CT scans than plain X-rays. Posterior cysts are more common and show no correlation with rotator cuff disorders. Anterior cysts are less common and are probably associated with the presence of rotator cuff disorders. Full thickness rotator cuff tears were found in 48% of patients with anterior greater tuberosity cysts in one study of symptomatic shoulders evaluated by MRI and surgery. Cortical thickening and subcortical sclerosis are not seen more frequently in shoulders with rotator cuff disease than those without rotator cuff disease.

Reduced acromiohumeral interval

Historical—The acromiohumeral interval (AHI) has been used in the assessment of potential rotator cuff disease since Golding recommended its routine measurement on AP shoulder radiographs in 1962 . [18] Cotton used it as one of his radiological diagnostic criteria in his cadaver study. [14]

Recent research—Gruber et al published level 1 evidence in 2009, demonstrating that the assessment of AHI on standardized anteroposterior X-rays is reliable and reproducible.[18] This study did not assess the effect of radiographic projectional errors. The patient was standing with their arm beside them in external rotation and the palm of the hand facing forwards.

Recently Saupe et al examined the relationship between the AHI and abnormalities of the rotator cuff tendons as assessed by MR arthrography.[19] Of those with an AHI less than or equal to 7 mm, 90% had a full thickness supraspinatus tear, 67% a full thickness infraspinatus tear and 43% a full thickness subscapularis tear. Additionally 71% showed MR evidence of fatty atrophy.[19]

Nove-Josserand et al examined the factors affecting the AHI and coracohumeral interval (CHI) in patients with rotator cuff tears having surgery. The AHI was assessed on 20° caudal tilt AP X-rays, with the arm in neutral rotation with the patient relaxed. A distance of less than 7 mm was considered abnormal. The CHI was assessed on preoperative CT scans, measuring the narrowest distance between the tip of the coracoid process and the humeral head. The position of the arm was not found to significantly alter this distance. A distance of less than 6 mm was considered abnormal.[20]

Multiple tendon tears were more likely to have a reduced AHI with 45% of combined supraspinatus, infraspinatus and subscapularis tears having a reduced AHI. No shoulder with an isolated rupture of supraspinatus or subscapularis had a reduced AHI. A reduced CHI was present in 24% of patients with a combined tear of supraspinatus and subscapularis, but was only present in 5% of shoulders with no subscapularis tears and 7% of patients with an isolated subscapularis tear. Fatty degeneration Grade 3 or worse was associated with reduced AHI and CHI. Shoulders with symptoms for more than 5 years were more likely to have a reduced AHI (p=0.002). More than one-third of the patients with more than 5 years symptoms had a reduced AHI. [20]

Conclusions—Recent studies support the long held assumption that reduced AHI is correlated with rotator cuff tears. Measurement is reliably reproducible on standardized X-rays. Reduced AHI is associated with both fatty atrophy and symptoms that may be of more than 5 years duration.

Cuff tear arthropathy

Historical—The first documented description of the typical changes of the entity now known as rotator cuff arthropathy was by Adams and Smith in the 1850s.[21, 22]

In 1983 Neer et al introduced and defined the term 'cuff tear arthropathy', the essential features being the presence of a rotator cuff tear and glenohumeral osteoarthritis, characterized by the presence of a massive rotator cuff tear, superior humeral head migration, acetabulisation of the acromion, collapse of the humeral head articular surface and erosion of the superior glenoid.[21,23]

Neer hypothesised that both mechanical and nutritional factors were responsible for the development of cuff tear arthropathy and estimated the prevalence of cuff tear arthropathy to be approximately 4% of patients with rotator cuff tendon tears. The duration of symptoms in his series prior to ranged between 2 and 20 years, with a mean of 9.8 years.

In 1981 an association between a cuff tear arthropathy-like condition and microscopic basic calcium phosphate crystals deposited in the synovium and adjacent structures was described and is known as Milwaukee shoulder.[21,22]

Hamada et al, in 1990[24] , proposed a grading system for RCA based primarily on reduced acromiohumeral interval (AHI) as follows:

Grade 1—AHI >6 mm

Grade 2—AHI < or equal to 5 mm

Grade 3—Acetabulisation of the acromion

Grade 4—Narrowing of the glenohumeral joint

Grade 5—Humeral head collapse

Recent research—Rockwood et al followed shoulders with massive irreparable tears of supraspinatus and infraspinatus post surgery for an average of 6.5 years.[25] None progressed to cuff tear arthropathy. Apoil and Augereau, however, reported that more than 25% of 56 shoulders that had undergone debridement of a degenerative lesion of the rotator cuff had developed cuff tear arthropathy 10 years after surgery.[21]

In 2007 Zingg et al reported the outcomes of patients with massive rotator cuff tears treated non operatively, followed for a mean of 4 years. Tears were classified as reparable if the fatty infiltration was stage 2 or lower and the AHI was 7 mm or more and irreparable if the fatty infiltration was stage 3 or greater and the AHI was less than 7 mm. As in other publications the classification of a traumatic tear was based on the patient history of a traumatic event. 84% reported a traumatic event.

The time between acute injuries and diagnosis averaged 23 months. Although most patients in this group maintained satisfactory shoulder function, the radiological appearances deteriorated significantly. Over the follow-up period (mean 4 years), glenohumeral osteoarthritis progressed (p=0.014), the AHI decreased by 2.6 mm (p=0.01), fatty infiltration increased by one grade and the size of the tear increased (p=0.003). Half of the rotator cuff tears that were graded as reparable initially were graded irreparable at final follow up.[26]

Rotator cuff tears in primary osteoarthritis are uncommon. Edwards et al reported an incidence of 7% partial thickness tears and 7% full thickness tears in patients having shoulder replacements for primary osteoarthritis.[27]

Conclusions—Cuff tear arthropathy appears to be an uncommon sequelae of massive rotator cuff tears that develops over many years. The exact incidence of cuff tear arthropathy following massive rotator cuff tears and the duration from tear to onset of significant arthropathy is long but unknown.

Fatty muscle degeneration

Early research—CT and MRI were initially used to assess muscles in neuromuscular disease and spinal disorders. In 1989, Goutallier et al presented on fatty muscle degeneration.[28] The authors proposed the following 5 grade staging system, using CT scans to assess the rotator cuff muscles

Stage 0—Normal muscle, no fatty streak

Stage 1—The muscle contains some fatty streaks

Stage 2—The fatty infiltration is important, but there is muscle than fat

Stage 3—There is as much fat as muscle

Stage 4—More fat than muscle is present

In 1994, Goutallier et al[29] reported that significant fatty degeneration of infraspinatus was associated with severe functional impairment and that fatty degeneration of infraspinatus did not improve after repair. The authors concluded that '*it is probably better to operate on wide tears before irreversible muscle damage*'. Their data suggests that significant (worse than stage 2) degeneration of infraspinatus was uncommon in tears that has been symptomatic for less than 6 months although they commented '*successive preoperative CT scans performed in more recent patients have shown that the infraspinatus can degenerate in several months or even weeks.*'[29]

Nakagaki et al published a histological cadaveric study in 1996[30] concluding that '*the fatty degeneration in the supraspinatus muscle after cuff tear was found to have a strong association with the degree of retraction of the tendon fibres*'.

Recent research—Oh et al evaluated the reliability of MRI arthrography and CT arthrography. MRI arthrography had more interobserver reliability than CT arthrography. Interobserver and intraobserver reliability was poor.[31] The investigators chose to assess fatty degeneration on oblique sagittal CT and T1 weighted MRI images where the scapular body, spine and base of the coracoid process form a 'Y'.

Williams et al investigated the most reliable plane of imaging to identify fatty infiltration of supraspinatus.[32] They recommended the axial plane, which had good intraobserver agreement and moderate interobserver agreement. The authors also evaluated the 'tangent sign' described by Zanetti et al.[33] The tangent sign is evaluated on the sagittal plane at the most lateral image where the scapular spine is in contact with the scapular body. A line is drawn tangential to the scapular spine and the coracoid. The tangent sign is positive if the supraspinatus does not reach above this line.

Williams et al found a positive tangent sign is an indicator of muscle atrophy and advanced fatty infiltration. The authors described a new sign called the 'fish backbone sign'. In this sign the supraspinatus looks like a fish backbone in the axial view and this indicates Goutallier grade 3 fatty infiltration.[32]

Berthouet et al reviewed massive rotator cuff tears in patients younger than 65 years.[34] They did not find a correlation between AHI and duration of symptoms. There was a significantly higher rate of fatty infiltration of the infraspinatus muscle (> stage 2) in patients with a long duration of symptoms ($p<0.05$). There was a significantly higher rate of infraspinatus fatty degeneration in patients with no history of trauma ($p<0.05$). However, 25% of patients with short duration symptoms and a history of trauma had greater than Goutallier stage 2 fatty infiltration in infraspinatus. The authors placed importance on the patient's history of trauma and duration of symptoms to define the study groups.

Conclusions—The Goutallier classification system is used to stage fatty degeneration of the rotator cuff muscles. CT and MRI can be used to assess fatty degeneration. The axial image is best for evaluating supraspinatus. However, it should be noted that the potential for interobserver and intraobserver variation is high, especially with a variety of imaging sequences and observer experience. Significant infraspinatus fatty degeneration, greater than Goutallier stage 2, is more common if symptoms have been present for more than 6 months, although it may occur with a duration of symptoms of less than 6 months.

Bursal changes

Historical—We were unable to find literature indicating that bursal thickening was a sign of non traumatic rotator cuff disease.

Recent research—Teefey et al investigated the sonographic differences in acute and chronic full thickness rotator cuff tears.[35] Once again the diagnosis of an acute rotator cuff tear (RCT) was based on the patient's history.

'*An acute RCT was considered to be present when (1) the clinical history revealed a distinct injury within 6 months from the time of operation in a previously asymptomatic shoulder and (2) the operative findings showed blunt, frayed cuff edges, tendon quality and thickness comparable to those of an intact cuff, and a freely mobile cuff.*'

Seventy-five percent of patients with a midsubstance tear had an acute tear. Sixty-four percent of patients with joint or bursal fluid had an acute tear; 80% of patients with a non visualised rotator cuff due to a massive tear had a chronic tear; and 73% of patients with no sonographic evidence of bursal or joint fluid had a chronic tear.[35]

Li et al studied ultrasound appearances of bursal thickening in Stage I and Stage II subacromial impingement.[36] They defined Neer's classification of impingement as follows:

Stage I—Reversible oedema and haemorrhage in the bursa

Stage II—Fibrosis and thickening of subacromial soft tissue and sometimes a partial rupture of the rotator cuff

Stage III—Complete rupture of the rotator cuff

The Neer classification of impingement classifies the pathology causing symptoms defined as impingement symptoms and not the aetiology (traumatic v non traumatic). Li's et al reported that the normal subacromial bursal thickness is considered to be less than 2 mm.

Conclusions—A mid substance rotator cuff tear, or the presence of bursal fluid in a rotator cuff tear are more commonly present in an acute tear. Absence of bursal fluid and visible cuff tissue are more likely signs of a chronic tear. There is no information regarding bursal thickening in patients with symptoms of Stage I or Stage II impingement that indicates whether the impingement is traumatic or non traumatic in aetiology.

Tendon retraction

Gerber et al examined the effect of tendon release and delayed repair on the structure of the rotator cuff muscle in sheep.[37] The authors concluded

"Rotator cuff tendon tears lead to substantial and progressive muscular changes with a severity that is proportional to the amount of musculotendinous retraction. If muscular function is to be preserved, a repair may need to be performed before marked retraction has occurred or new or different techniques for repair need to be developed."

Gerber's group later used the same model to examine how some of the retraction occurs. They found the tendon end retracts into the muscle the musculotendinous junction shifting more distal relative to the tendon.[38]

Braune reported the intraoperative appearances of acute traumatic, chronic traumatic and atraumatic rotator cuff tears in the German literature. One of the factors he examined was tendon retraction. Once again the criteria used to define a tear as traumatic was an acute traumatic incident, pain free and healthy shoulder before accident, spontaneous constantly painful shoulder after accident. An acute traumatic tear was defined as less than 12 weeks from trauma and a chronic traumatic tear more than 12 weeks.

Patients with no known trauma were considered to have degenerative tears. They noted the location of the tear, tear size and retraction. The quality of the tendon end and changes in the long head of biceps, were also noted. Retraction was graded on as follows:

Grade I—Retraction to half the distance between the greater tuberosity and the level of the neck of the humerus

Grade II—Retraction to the centre of the neck of humerus

Grade III—Retraction to between the centre of neck of humerus and the glenoid

Grade IV—retraction beyond the glenoid

Although numbers in each study group were small, Braune did not find retraction beyond the glenoid in any patients in the acute traumatic group. Isolated subscapularis tears were only seen in the traumatic groups. Haematoma may be seen in the acute traumatic group. Subluxation of the long head of biceps was more common in the traumatic group (p=0.007). Partial and complete tears of the biceps were more common in the degenerative group. Synovitis of the biceps was seen in all 3 groups.[39]

Conclusions—Little has been written about tendon retraction. Animal studies demonstrate significant tendon retraction occurring within one hour of rotator cuff tendon release, whereas a human study reports that retraction beyond the glenoid rim was not seen within 12 weeks of injury. Anatomical differences in the animal models may contribute to these different observations.

Partial thickness tears

Historical—Codman described an articular surface partial thickness tear, which he labelled a 'rim rent' tear. His hypothesis was that intrinsic tendon degeneration was the primary lesion.[1,40,41] Neer instead favoured a process extrinsic to the tendon itself as primarily responsible for tears and proposed that subacromial impingement was the primary cause of tears.[2]

Rothman and Parke in 1965 described a 'critical zone' of hypovascularity of the supraspinatus tendon near its humeral attachment.[40] They confirmed a relatively avascular zone distally and found that the earliest changes of 'degeneration' occurred in this zone. They emphasised that their study did not produce evidence of causation of degeneration.[42]

In 1990 Lohr and Uhtoff reported zone of relative hypovascularity to be more pronounced on the articular surface of the tendon, extending from the myotendinous junction to within a few millimetres of the humerus. Their cadaveric study published in 1990, demonstrated a hypovascular zone extending from the myotendinous junction to within 5mm of the humeral head insertion on the articular surface of the tendon. The bursal surface in comparison has a rich vascular supply.[43]

Current research—The relative incidence of partial thickness tears in clinical studies however differs, with numerous reports of articular surface tears being 2 to 3 times more common than bursal surface tears.[40, 41]

Nakajima et al in 1994 compared the histological and *ex vivo* biomechanical properties of the articular and bursal surface fibres of suprapinatus, demonstrating more organised tendon bundles on the bursal surface.[44] Biomechanical analysis showed that the bursal surface fibres had greater deformity and tensile strength. This may explain the increased incidence of partial thickness articular surface tears following a traumatic event.[40]

Ko et al,[45] in a prospective histological study, concluded that articular surface tears are likely related to intrinsic factors while bursal surface tears are associated with subacromial impingement.

Payne et al performed a retrospective analysis of athletes under the age of 40 who had undergone arthroscopic treatment for partial thickness cuff tears.[46] Two main groups were identified; Group A patients had a history of an acute traumatic event and Group B consisted of overhead throwing athletes with an insidious onset of non traumatic shoulder pain. Seventy-nine percent of the partial tears in Group A were of the articular surface and 97% of the tears in Group B were of the articular surface.

A recent meta- analysis by de Jesus et al compared MR arthrography (MRA), conventional MR (MRI) and ultrasound (US) for diagnosis of full and partial thickness cuff tears.[47] MRA performed significantly best and MRI and US performed similarly. The sensitivity for full thickness and partial thickness tears respectively were 95% and 86% for MRA, 92% and 64% for MRI and 92% and 66% for US. The specificity for full and partial tears was 99% and 95% for MRA, 93% and 92% for MRI and 94% and 86% for US. The authors stress that the analysis encompasses studies performed over a wide range of time and that this may influence findings as technical improvements and expertise improve in each modality.[47] The accuracy of US in particular is highly operator dependent, especially for small partial tears.

Conclusions—The aetiology of partial thickness rotator cuff tears is complex, not fully understood and almost certainly multifactorial. It appears likely that intrinsic tendon factors including differences in tendon layer mechanical characteristics and alterations in tendon structure with age, external impingement and trauma all have a part to play in the aetiology of partial thickness tears. It is clear that trauma can cause both articular surface and bursal surface tears and this has been reported in normal tendons of young athletes. It is also probable that intrinsic tendon changes and extrinsic impingement have a part to play in articular surface and bursal surface tears respectively.

MR Arthrography remains the gold standard for the assessment of rotator cuff tears, but standard MRI and US in appropriate hands have good accuracy.

Calcific tendinitis

Historical—Calcific tendinitis of the rotator cuff refers to a syndrome of shoulder pain associated with calcific deposits within the rotator cuff tendons. Codman proposed tissue hypoxia as the primary event leading to the development of tendon calcification in 1934.[1] This remains a popular theory.[49] The reported rates of coexistence of cuff tears and calcification in the literature have varied greatly, with some authors reporting that the two entities are virtually mutually exclusive while others report an association.[50]

In 1993, Jim et al in 1993 examined the incidence of cuff tears in 81 symptomatic patients with calcification and reported that a small (not defined) rather than a large amount of calcium was more likely to be associated with a cuff tear.[50] A deficiency of these and other studies is that they fail to distinguish between calcific tendinitis and degenerative calcification.

Recent research—Hamada et al demonstrated that crystals were composed of carbonate apatite.[51] Recent extensive reviews on the subject by Uhthoff and Hurt emphasise that the actual aetiology of calcific tendinitis is unknown.[48,49] Uhthoff and Loehr in 1976 proposed a three stage process. The initial *precalcific* stage, the site of future calcification is proposed to undergo cartilaginous metaplasia. The second *calcific stage* is proposed to consist of three distinct phases itself. During the formative phase, calcium crystals are deposited. During the resting phase, no new calcium deposits are laid down. Finally during the resorptive phase calcium deposits are reabsorbed. This is accompanied by an inflammatory infiltrate. During the *post calcific stage,* the tendon is repaired. The calcific and post calcific stages are often associated with pain, which is most severe during the resorptive phase.[49]

Most authors have found that rotator cuff tears and calcification rarely coexist. Loew et al studied 75 patients with calcific tendonitis with MRI scans and found only 1 patient had a partial thickness cuff tear. [52] Rotator cuff tears in calcific tendinosis are sufficiently rare that Gotoh et al published a case report in Skeletal Radiology in 2003 of a patient with calcific tendinosis that progressed to rotator cuff tear.[53]

Dystrophic calcification in comparison consists of linear calcific deposits at the insertions of tendons.[48,49] These are thought to be secondary to degenerative changes in tendons.

Conclusions—Calcific tendinosis of the rotator cuff is a condition of unknown aetiology. Tissue hypoxia near the rotator cuff insertion appears to be involved in the pathogenesis. Most studies report that cuff tears are uncommon in association with calcific tendinosis. Studies frequently fail to distinguish between different types of cuff calcification. This likely contributes to the large reported differences.

Tendinopathy

Tendinopathy is a complex subject beyond the scope of this imaging review. Lewis, in a review of rotator cuff tendinopathy in the British Journal of Sports Medicine 2009 noted that '*tendinopathy is a generic term without aetiological, biochemical or histological implications and is used to describe pathology in, and pain arising from, a tendon.'* Lewis's extensive literature review concludes that the pathogenesis of rotator cuff tendinopathy is multifactorial and results form a combination of intrinsic, extrinsic and environmental factors. Intrinsic degenerative changes in a tendon may occur due to overuse or overload of a tendon. Extrinsic compression from subacromial impingement may sometimes occur as a secondary phenomenon when there is tendon dysfunction.[54]

Although the aetiology of tendinosis is likely multifactorial, age does appear to have a role in some cases. The peak age group for rotator cuff tendinopathy is the 5th to 7th decades.[55]

Is there an accepted definition of tendinopathy on imaging studies? Is there interobserver and intraobserver reliability in reporting tendinopathy on imaging? Does tendinopathy on imaging correlate to tendinopathy histologically or biochemically? Can some tendinopathy changes be considered normal for age in older subjects? Does the presence of tendinopathy mean that a subsequent rotator cuff tear is wholly or substantially due to degeneration? We have not been able to satisfactorily answer these questions from our literature review

Conclusions—Studies reporting imaging findings in traumatic rotator cuff tears use a history of a traumatic event with onset of symptoms as the criteria for diagnosing a traumatic rotator cuff tear. The clinical history is important.

Age related changes occur in the shoulder and rotator cuff. Some changes can be considered normal for age. These include;

- Subacromial spur formation[7,9]
- AC joint arthrosis[11,12]
- Possibly tendinopathy but definitions and data lacking.[55]

There are some signs on imaging that indicate chronicity. The definition of chronicity varies from months to years in different conditions and studies. When these are present, in a patient with a recent history of a traumatic event, the symptoms may be substantially due to a pre-existing condition. This may be more likely if the traumatic event is a low energy event, more in keeping with an activity of daily living. It may also be more relevant if the onset of symptoms was a significant time after the perceived traumatic event. Signs that commonly, but not always, indicate chronicity include;

- Cuff tear arthropathy[21,22,24,26]
- Decreased Acromiohumeral interval of 7 mm or less[19,20]
- Fatty muscle degeneration[28,29,34]
- Tendon retraction beyond glenoid rim[39] Animal studies report immediate significant retraction however[37]
- Anterior greater tuberosity cysts[13]

There are some imaging features that cannot be considered significantly associated with rotator cuff tears or are unreliable in their appearance or reporting. These include;

- Acromial morphology[5,6,8,10]
- Os Acromiale[10]
- AC joint changes[11]
- Greater tuberosity sclerosis[15]
- Greater tuberosity posterior cysts1[3,15,17]
- Calcific tendinosis[52,53]

There are some features that, when present, do indicate the tear is likely to be traumatic. These include:

- Bone oedema in the greater tuberosity with supraspinatus tear[16]
- Midsubstance tear[35]
- Bursal fluid, haematoma or debris present[35,39]
- Isolated subscapularis tears[39]

None of the listed features are 'absolute'. Imaging features should be interpreted in the context of the patient's age, history of injury and history of symptoms when considering the aetiology of a rotator cuff tear.

Competing interests: None.

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