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Case Report

THE PROGRESSIVE COMPLEX NATURE OF ROTATOR CUFF TEAR ARTHROPATHY

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ABSTRACT

A small number of patients with a chronic rotator cuff tear appear to progress to the clinical entity known as rotator cuff tear arthropathy (RCTA). There are various theories concerning the etiology and progression to account for the findings associated with this clinical condition. A broad spectrum of pathology may be present. Progressive destruction of soft tissues, cartilage, and bone leading to increasing shoulder disability has been documented. Significant complications such as instability or bleeding are rarely associated with RCTA. We present an extreme case of RCTA with extensive destruction and unusual shoulder pathology that has not been previously reported. Of even more concern was the finding of acute and subacute hemorrhage from major vascular erosion requiring emergency treatment. There appears to be little or no limits to the extent of pathology in some individuals with this condition.

Keywords: Rotator cuff tear arthropathy, Ecchymoses, Instability, Hemarthrosis, Vascular erosion, Acromial artery.

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INTRODUCTION

Rotator cuff tear arthropathy (RCTA) is a distinct clinical entity defined by the presence of three critical features. Rotator cuff insufficiency, degenerative glenohumeral joint arthritis, and superior migration of the humeral head are all present to various degrees [1,2]. There is a growing spectrum of additional shoulder pathology associated with RCTA. Progressive destruction of soft tissues, cartilage, and bone leading to increasing shoulder disability has been documented. We present an unusual case of RCTA with unique shoulder pathology that has not been previously reported.

CASE REPORT

A 63-year-old right-handed dominant Caucasian male presented with a 9-year history of progressive stiffness of both shoulders requiring no medical treatment. Four months before our evaluation, the patient was treated in a local emergency room for a spontaneous dislocation of his right shoulder in an anterior subcoracoid direction. He subsequently experienced multiple recurrent dislocations of his right shoulder that he was able to reduce on his own. He had increasing left shoulder pain for several weeks, but no dislocations. Visual analog scale pain score was 8/10 in the right shoulder and 10/10 in the left.

Clinical examination revealed a thin statured male with prominent swelling and a positive "fluid sign" [3] in both the right and left shoulders. The skin over the left shoulder showed erythema and pigmentation changes of resolving ecchymoses (Fig. 1). Severe supraspinatus and infraspinatus muscle atrophy and "Popeye" signs demonstrating biceps deformity were present in both shoulders. The right humeral head by palpation was dislocated anteriorly, but was reducible by inferior traction with internal rotation. The patient demonstrated active redislocation by deltoid contraction. The left humeral head was fixed in an anterior position and could not be reduced. Pseudoparalysis with profound weakness of external rotation and internal rotation of both shoulders was noted as well. Deltoid function was normal in both shoulders. Active and passive glenohumeral range of motion of both shoulders was severely limited and was accompanied by audible and palpable painful crepitus. Cervical spine examination found no abnormalities.

Plain radiographs (Fig. 2) showed femoralization and anterosuperior escape of both humeral heads. Prominent glenoid and coracoid

erosion and significant erosion of the distal clavicle and acromion (acetabularization) with discontinuity along with multiple amorphous soft-tissue calcifications and ossifications were also present in both shoulders.

The left shoulder magnetic resonance imaging (MRI) (Fig. 3) showed very large joint effusion with marked distention of the glenohumeral capsule. Large synovial masses were present in the axillary, anterior capsular, and subcoracoid recesses with extensive synovial osteochondromatosis also noted. Disruption of the long head of the biceps tendon and diffuse severe muscle atrophy along with chronic full-thickness disruption of the supraspinatus, infraspinatus, and the majority of the subscapularis tendons were notably present. Glenoid and coracoid erosion, erosion and remodeling of the undersurface of the acromion, and partial erosion of the distal clavicle appeared quite prominent. The right shoulder MRI showed similar findings, but less complex signal and less glenoid erosion.

Computerized tomography (CT) scans of both the left and right shoulders showed marked erosion of the glenoid, humeral head, acromion, and clavicle. The left shoulder scan (Fig. 4) showed soft-tissue density consistent with hemorrhage and anterior humeral head positioning.

A treatment plan was developed for the more symptomatic left shoulder. The patient was admitted through the emergency room for control of a sudden increase in pain and swelling of his left shoulder the day before scheduled surgery. His hemoglobin dropped to 7.2 g/dl with hematocrit of 21.3%. Coagulation studies were in the normal range. There were no signs of external blood loss.

The patient was taken to the operating room. Through an anterior deltopectoral approach, a bulging, capsule-like structure with deep purple coloration was incised. A large acute and subacute hematoma measuring approximately 750 cc including abundant rice-like bodies was evacuated under pressure. Bursa-like synovial masses spotted with synovial based bodies were noted. Pulsatile bleeding was encountered from the acromial branch of the thoracoacromial artery near a remnant of eroded coracoid tip. This was ligated and controlled.

The long head of the biceps, conjoined tendon, and all rotator cuff tendons were absent. The distal clavicle as well as the anterior acromion



Fig. 1: Physical appearance left shoulder

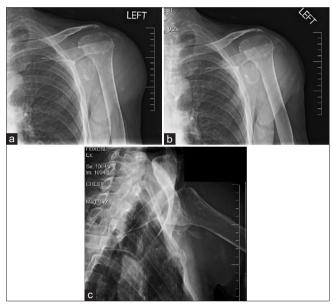


Fig. 2: Plain radiographs left shoulder: (a) AP projection. (b) Rotation projection. (c) Transthoracic lateral

were absent from erosion. The glenoid showed a large anterior erosive defect with an absent coracoid process.

Bone of the humeral head was fixed as autograft to fill the glenoid defect. A reverse total shoulder arthroplasty was performed with a large 40 mm glenosphere. The patient received 5 units of packed red blood cells pre and intraoperatively and 5 additional units postoperatively.

Postoperatively, the patient was maintained in a sling and swathe shoulder immobilizer. Within 3 days of surgery, the patient's pain score improved to a level 3/10. A CT scan done at 3 months postoperatively showed secure glenoid fixation and incorporation of the bone graft. The patient unexpectedly passed away from unrelated causes following this examination.

DISCUSSION

It has been estimated that only 4% of patients with full-thickness rotator cuff tears develop true cuff tear arthropathy. The arthropathy most likely progresses on the basis of mechanical and nutritional factors with a progressive loss of both dynamic and static stabilizers, leading to increasing shoulder instability and destruction [1]. The role

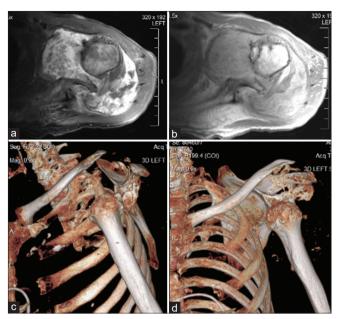


Fig. 3: MRI scan left shoulder: (a) Axial T2. (b) Axial T1. (c) Transthoracic reconstruction. (d) AP reconstruction

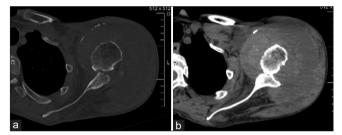


Fig. 4: CT scan left shoulder: (a) Soft-tissue window. (b) Bone window

of crystal deposition and inflammatory factors is unclear and may also play a role in some cases [4,5].

There are a number of complications that have been associated with RCTA. Shoulder swelling is the most common and often of chronic nature. A large collection of synovial fluid may be present and is often streaked with blood. A number of patients will demonstrate different stages of subcutaneous ecchymoses about the shoulder as a clinical finding.

More significant bleeding as in spontaneous hemarthrosis of the shoulder is a much less frequently reported complication of RCTA. Patients usually present with a sudden onset of severe shoulder pain and swelling with no history of trauma. Most often non-operative treatment has been recommended including both diagnostic and therapeutic aspirations [6-9].

Several sources of bleeding have been suggested. Bleeding from various boney erosions appears to be the likely source in most cases [2]. Erosive boney bleeding has been documented in two reports [10,11]. In another unrelated case, angiography revealed numerous small vessel pooling of contrast, but no bleeding points were found at the time of arthroscopic surgery [7].

We found two cases of hemarthrosis requiring transfusion [9,12]. Kang *et al.* [12] found bleeding from a pseudoaneurysm resulting from humeral head erosion at the tip of the coracoid. Collectively our findings would suggest that more significant medial erosion in an unstable

shoulder with RCTA resulted in coracoid destruction and the acromial artery erosion in our case.

We found Seebauer functional and biomechanical classification of cuff tear arthropathy [13] to be helpful in understanding our findings and making recommendations. Most investigators would agree that the extensive pathology exhibited in our case is most unusual. More recently, Seebauer, himself, has suggested the term "geriatric shoulder" [14] when encountering this degree of pathology and has proposed a modification of his original classification to include Type III A and B pathology. These Type III cases would include those cases considered very progressed and difficult to treat. Type III B being the most difficult case with chronic "fixed" anterior dislocation as appeared to be the finding in our patient's left shoulder.

CONCLUSION

RCTA may on rare occasion progress to extensive shoulder destruction as demonstrated in this case. There appears to be little or no limits to the extent of pathology in some individuals. Reverse shoulder arthroplasty has provided some treatment solutions, but challenges continue when confronted with extensive disease.

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CONFLICTS OF INTEREST AND FINANCIAL DISCLOSURE

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ETHICAL APPROVAL

The author has obtained patient consent for the publication of this article and any photographs used.

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