Acute Calcific Tendonitis of the Shoulder Mimicking Septic Arthritis

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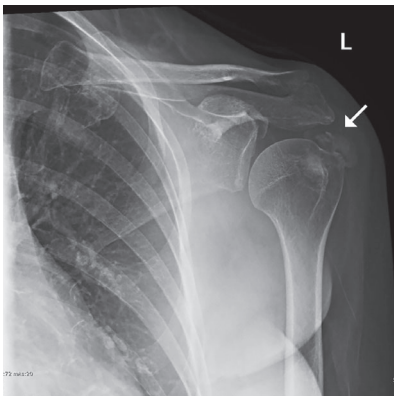
Calcific tendonitis is a relatively common condition that is found in 2–10% of the general population [1,2]. Approximately half of the affected patients become symptomatic, typically with chronic shoulder pain aggravated by arm motion. In some patients, acute, severe pain is the first symptom of calcific shoulder tendonitis. In these acute cases, patients often seek urgent medical care due to the intense pain and severe limitation of shoulder motion. Traditionally, this acute episode is considered to represent the resorptive phase of the disease, which results in most patients disappearance of the calcium deposits and remodeling of the tendon structure [3]. We describe three patients in which the clinical picture was confounded by additional findings indicating severe systemic inflammatory response simulating a septic shoulder.

PATIENT DESCRIPTION

PATIENT 1

A 60-year-old female with no significant past medical history presented in the orthopedic outpatient clinic complaining of increasing left shoulder pain for the past week as well as fever up to 38.0°C. The physical examination revealed severely reduced and painful range of motion (ROM) as well as severe tenderness over the anterior and lateral aspects of the shoulder. Laboratory tests revealed erythrocyte sedimentation rate (ESR) of 20 mm in the first hour, C-reactive protein (CRP) of 3.6 mg% (normal < 1.0 mg %), and a normal white blood cell count. Plain radiographs of the affected shoulder showed calcifications in the subacromial area, with fluffy appearance [Figure 1]. The patient was treated with non-steroidal anti-inflammatory medication (NSAID). In a follow-up examination 2 days later, the pain had mildly subsided, ROM was still severely limited, ESR increased to 40 mm, CRP rose to 8.3 mg%, while the white blood cell count (WBC) remained normal. In order to exclude septic arthritis as a possible cause for the patient's symptoms, A computed tomography (CT) scan with intravenous contrast was performed. The CT scan demonstrated calcified deposits within the supraspinatus tendon, but no articular effusion. The patient continued taking NSAIDS and analgesics and was discharged from the hospital. One and a half months later, the patient complained of only minimal pain, shoulder ROM returned to 85% of normal and shoulder X-ray showed nearly complete resorption of the calcification. At 2 years follow up, the patient had no shoulder pain, a full range of shoulder motion had returned, and no calcific deposits were found in the shoulder X-ray.

Figure 1. Calcific tendonitis, the resorptive phase. An anteroposterior radiograph of the left shoulder of a 60-year-old female with an opaque calcium deposit superimposed on the upper part of the lesser tuberosity representing a resting calcification in the infraspinatus tendon and a large cloud of radio-opaque substance between the acromion and the greater tuberosity (arrow) representing calcium depositions spreading from the supraspinatus tendon into the subacromial bursa



PATIENT 2

A 48-year-old male with a past medical history of diabetes mellitus and ischemic heart disease was seen in the outpatient shoulder clinic due to severe left shoulder pain of 4 days duration and mild fever. The patient recalled having chronic shoulder pain a year earlier that was diagnosed as calcific tendonitis. He received subacromial injection 10 months prior to the current episode, which partially improved his symptoms. On examination the patient had severe painful limitation in shoulder ROM, marked tenderness over the shoulder, and mildly elevated temperature of 37.8°C. Shoulder X-ray showed calcium deposits within the subacromial area. Due to the elevated temperature, blood tests were taken, which showed elevated ESR of 56 mm in the first hour, CRP of 4.0 mg% (normal < 1 mg%) and an elevated WBC

of 11,100. In order to rule-out septic shoulder, an intravenous (IV) contrast enhanced shoulder CT scan was performed. Calcific deposits within the supraspinatus tendon and surrounding hyperemia were seen, but no evidence of increased joint fluid or joint inflammation was found. Calcifications in the rotator cuff were noted to be fluffy, with ill-defined margins. The patient was treated with NSAID and discharged within a day. At one week follow up he regained about 80% of his baseline range of motion and pain dramatically subsided.

PATIENT 3

A 57-year-old female was seen in the emergency department for severe acute right shoulder pain beginning 2 days prior to admission. On examination the patient had a temperature of 38.3°C, with severe painful restriction of shoulder ROM and diffuse tenderness around the shoulder. Blood tests showed WBC count of 14,000, ESR of 42 and CRP of 6.1. An ultrasound evaluation of the shoulder revealed calcifications of the rotator cuff tendons but no signs of joint effusion. The patient was treated with NSAID and rest. In a follow-up examination 4 days later, pain was still severe, mild improvement in ROM was noticed while WBC count decreased to 11,000 and CRP to 5 while ESR increased to 68. Patient continued with NSAID and rest with significant improvement within the next week. At 6 months follow up the patient had no shoulder pain and no calcific deposits on shoulder X-ray.

COMMENT

We describe three patients in whom the clinical picture of acute calcified tendonitis had been confounded by additional findings indicating severe systemic inflammatory response simulating septic shoulder. We have found only one report of a similar case in the literature [4]. Knowledge of this condition is important for the treating physician to arrive at the correct diagnosis.

Calcified tendonitis may be clinically silent or create chronic impingement like symptoms in the remainder [1]. The resorptive phase of calcific tendonitis, which represents the end of the calcific stage, is marked by a local inflammatory process involving neo-angiogenesis around the calcium deposits, recruitment and activation of monocytes and macrophages, secretion of pro-inflammatory cytokines, and further recruitment of reactive cells [3]. The resultant tissue edema with increased tendon volume may cause acute symptoms. Clinically, patients may present at this stage with poorly localized, severe pain around the shoulder and significant limitation in range of motion, both passive and active. Patients often seek urgent medical care due to the abrupt and intolerable nature of the symptoms. Following the symptomatic period, which may range from a few days to a few weeks, most patients experience clinical recovery, with remodeling of tendon tissue, as evident in biopsies from the site of calcium deposits. Radiologically, the leakage of tensioned calcium deposits from the tendon is evident on the plain radiographs as a fuzzy or cloudy radio-opacity in the area of the rotator cuff tendons. This finding is different from the well-bordered deposits in the chronic state of calcific stage [5]. Radiographs of the shoulder, if taken few months later, would show complete or partial disappearance of the calcium deposits.

The intense acute and severe nature of symptoms may resemble the clinical appearance of septic arthritis. The lack of systemic signs, such as fever, leukocytosis, and elevated ESR and CRP, as well as the radiologic findings of calcified material in the subacromial region help to differentiate between the two. However, there is a subgroup of patients, described in this study, in whom the resorption of the calcific deposits produces systemic involvement (fever, elevated ESR, CRP, and leukocytosis), which may confound the diagnostic process, and mislead the treating physician.

In these cases, the diagnosis of calcifying tendonitis of the rotator cuff was established early in the patient's evaluation, using plain radiographs. The absence of current history of local trauma, local injection into the shoulder area or another concurrent infection reduced significantly the possibility of septic shoulder. To evaluate the shoulder for an effusion, which is common sign in septic arthritis or bursitis, an IV contrasted CT scan was conducted in two patients and a sonographic evaluation was performed on the third patient. Neither of the radiographs demonstrated any obvious fluid collection. The combination of the current medical history and the lack of shoulder effusion in the context of sizable calcified tendonitis allowed us to conclude that it was a systemic involvement of the calcific tendonitis and not septic arthritis, thus treating the patients with NSAID and observation.

Ross et al. [4] reported a case of acute calcifying tendinitis of the shoulder mimicking infection. In that case report the 28-year-old female had, in addition to severe shoulder pain, borderline ESR and mildly elevated CRP. No significant event preceded the symptoms. Due to the high index of suspicion, and despite the established diagnosis of calcifying tendinitis, the patient underwent urgent an magnetic resonance imaging scan, which demonstrated glenohumeral effusion that was thereafter aspirated and yielded a thick and cloudy fluid with 50,000 WBC per μL. Urgent arthroscopy was performed, and antibiotic treatment was initiated for 3 days, while cultures remained sterile. In this case, the pressurized calcium material penetrated the joint capsule into the glenohumeral joint creating synovitis in the glenohumeral joint and significant effusion.

Ultrasound examination is an accepted and commonly used modality to evaluate shoulder pathology. As such, it can accurately and easily locate the calcific deposits within the shoulder rotator cuff and can demonstrate any fluid collection in the subacromial bursae or glenohumeral joint. It can also allow for immediate aspiration of any fluid collection if found. Therefore, we prefer to use the ultrasound as our imaging modality in such cases.

CONCLUSIONS

Calcified tendonitis, in its final resorptive stage, may present itself as acute shoulder pain with signs of systemic inflammato-

ry reaction, which may mimic septic arthritis. Knowledge of this possibility may help clinicians to correctly diagnose the patient and avoid unnecessary treatment and procedures.

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References

1. Sansone V, Maiorano E, Galluzzo A, Pascale V. Calcific tendinopathy of the shoulder: clinical perspectives into the mechanisms, pathogenesis, and treatment. *Orthop Res Rev* 2018; 10: 63-72.
2. Louwerens JK, Sierevelt IN, van Hove RP, van den Bekerom MP, van Noort A. Prevalence of calcific deposits within the rotator cuff tendons in adults with and without subacromial pain syndrome: clinical and radiologic analysis of 1219 patients. *J Shoulder Elbow Surg* 2015; 24 (10): 1588-93.
3. Darrieutort-Laffite C, Blanchard F, Le Goff B. Calcific tendonitis of the rotator cuff: From formation to resorption. *Joint Bone Spine* 2018; 85 (6): 687-92.
4. Ross G, Cooper J, Minos L, Steinmann SP. Acute calcific tendinitis of the shoulder mimicking infection: arthroscopic evaluation and treatment--a case report. *Am J Orthop (Belle Mead NJ)* 2006; 35 (12): 572-4.
5. Gosens T, Hofstee DJ. Calcifying tendinitis of the shoulder: advances in imaging and management. *Curr Rheumatol Rep* 2009; 11 (2): 129-34.

Capsule

CD8+ T cells contribute to survival in patients with COVID-19 and hematologic cancer

Patients with cancer have high mortality from coronavirus disease-2019 (COVID-19), and the immune parameters that dictate clinical outcomes remain unknown. In a cohort of 100 patients with cancer who were hospitalized for COVID-19, **Bange** et al. noted that patients with hematologic cancer had higher mortality relative to patients with solid cancer. In two additional cohorts, flow cytometric and serologic analyses demonstrated that patients with solid cancer and patients without cancer had a similar immune phenotype during acute COVID-19, whereas patients with hematologic cancer had impairment of B cells and severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2)-specific antibody responses. Despite the impaired humoral immunity and high mortality in patients with hematologic cancer who also have COVID-19, those with a greater number of CD8 T cells had improved survival, including those treated with anti-CD20 therapy. Furthermore, 77% of patients with hematologic cancer had detectable SARS-CoV-2-specific T cell responses. Thus, CD8 T cells might influence recovery from COVID-19 when humoral immunity is deficient. These observations suggest that CD8 T cell responses to vaccination might provide protection in patients with hematologic cancer even in the setting of limited humoral responses.

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Capsule

Neutralizing antibody levels are highly predictive of immune protection from obesity accelerates hair thinning by stem cell-centric converging mechanisms

Obesity is a worldwide epidemic that predisposes individuals to many age-associated diseases, but its exact effects on organ dysfunction are largely unknown. Hair follicles, which are mini-epithelial organs that grow hair, are miniaturized by ageing to cause hair loss through the depletion of hair follicle stem cells (HFSCs). **Morinaga** and co-authors reported that obesity-induced stress, such as that induced by a high-fat diet (HFD), targets HFSCs to accelerate hair thinning. Chronologic gene expression analysis revealed that HFD feeding for 4 consecutive days in young mice directed activated HFSCs toward epidermal keratinization by generating excess reactive oxygen species but did not reduce the pool of HFSCs. Integrative analysis using stem cell fate tracing, epigenetics, and reverse genetics showed that further feeding with an HFD subsequently induced lipid droplets and NF-κB activation within HFSCs via autocrine and/or paracrine IL-1R signalling. These integrated factors converge on the marked inhibition of sonic hedgehog (SHH) signal transduction in HFSCs, thereby further depleting lipid-laden HFSCs through their aberrant differentiation and inducing hair follicle miniaturization and eventual hair loss. Conversely, transgenic or pharmacological activation of SHH rescued HFD-induced hair loss. These data collectively demonstrate that stem cell inflammatory signals induced by obesity robustly represses organ regeneration signals to accelerate the miniaturization of mini-organs, and suggests the importance of daily prevention of organ dysfunction.

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