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CASE REPORT

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Calcifying tendinitis of the rotator cuff with cortical bone erosion

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Abstract Calcifying tendinitis occurs most commonly in the rotator cuff tendons, particularly involving the supraspinatus tendon insertion, and is often asymptomatic. Cortical erosion secondary to calcifying tendinitis has been reported in multiple locations, including in the rotator cuff tendons. We present a pathologically proven case of symptomatic calcifying tendinitis involving the infraspinatus tendon with cortical erosion with correlative radiographic, CT, and MR findings. The importance of considering this diagnosis when evaluating lytic lesions of the humerus and the imaging differential diagnosis of calcifying tendinitis and cortical erosion are discussed.

Keywords Infraspinatus · Calcification · Bone erosion · MRI · CT

Introduction

Calcifying tendinitis, or calcific tendinopathy, is a self-limited disorder of unclear etiology, which is characterized by the formation of calcium hydroxyapatite in the tendon, potentially followed by its spontaneous resorption, and then subsequent tendon healing [1, 2]. This condition has been reported to occur most often in the rotator cuff tendons but has also been described to involve tendons at other sites of the body, such as the pectoralis major, gluteus maximus, gluteus medius, adductor magnus, and deltoid insertions [3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14]. At these other sites, cortical erosion of nearby bone has been noted, potentially mimicking a neoplastic process. This phenomenon of cortical erosion due to concretions has previously been less often described to occur in both the femur and humerus. We present another case of calcifying tendinitis, but involving the infraspinatus tendon with cortical erosion of the greater tuberosity, which we believe is new to the current literature.

Case report

An otherwise healthy thirty-year-old left-hand dominant avid volleyball player presented with a 3-month history of right shoulder pain of insidious onset, exacerbated by overhead activities and running. There was no specific injury. He also complained of frequent night pain that awoke him from his sleep. Although his pain improved with rest and application of ice, he was unable to play volleyball or engage in any strenuous activity. Review of systems was negative including no weight loss, fever, sweats, or chills.

Physical examination revealed a mildly painful but normal arc of motion, with a positive Neer impingement sign and a markedly positive O'Brien's sign. He had mild weakness with resisted external rotation and abduction testing. His belly press and liftoff tests were normal, and he demonstrated no signs of instability.

Initial radiographs of the right shoulder revealed ill-defined calcifications in the soft tissues adjacent to the greater tuberosity of the humerus, consistent with calcifying tendinitis with a thin rim of lucency surrounding a separate intraosseous lytic area with a focus of faint calcification (Fig. 1). Subsequent magnetic resonance imaging showed a low signal intensity rounded structure in the superolateral humeral head with a surrounding thin rim of fluid and adjacent bone marrow edema (Fig. 2). These findings correlated with the faint calcification and lucency in the humerus on radiographs and were separate from areas of calcification that were

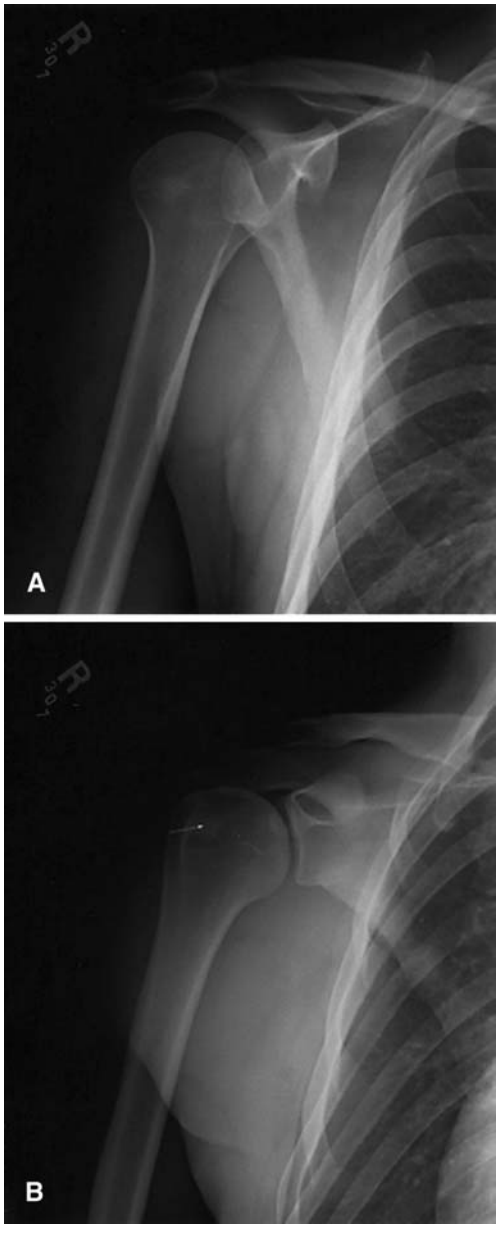

Fig. 1A, B Ill-defined calcifications in the soft tissues adjacent to the greater tuberosity of the humerus are consistent with calcific tendinitis (*arrow*). A thin rim of lucency surrounds a separate intraosseous focus of faint calcification (*arrowhead*)

external to the bone. Differential diagnosis at this point included calcific tendinitis with cortical erosion, osteochondroma, osteoid osteoma or osteoblastoma, osteonecrosis, or chondroblastoma. Computed tomography examination was performed to help differentiate an extraosseous from an intraosseous etiology. CT appearance demonstrated calcification in the humeral head that appeared to be eroding into the humeral head cortex (Fig. 3). The extraosseous calcification had a distribution of a "comet tail," suggesting a tendinous distribution.

Because of his continued symptoms of pain and the suspicious radiographic appearance of this lesion on both radiographic and advanced imaging (CT/MRI) in the proximal humerus, the patient underwent an open biopsy of the right proximal humeral lesion. Operative findings demonstrated cortical erosion of the posterior aspect of the greater tuberosity. The rotator cuff overlying this area was noted to be deficient and associated with a small defect in the infraspinatus tendon with longitudinal fraying. The lesion itself was whitish, red, and friable with a hard paste-like consistency.

Frozen sections of the tissue were nondiagnostic but demonstrated fibrous tissue with typical calcific deposits of calcific tendinitis, both with stromal tissue containing calcifications. Final pathology revealed thin soft tissue and invading into bone. The bone was reactive and nonneoplastic (Fig. 4).

At 6-month follow-up, the patient was pain-free with full shoulder range of motion and normal rotator cuff strength. Repeat radiographs showed no evidence of recurrent calcifying tendinitis.

Discussion

Calcifying tendinitis is a common disorder, which can occur in tendons and at the insertion of muscles. It is most common in the shoulder, especially in female patients in their 4th to 6th decades of life and is characterized by deposits of hydroxyapatite that affect the rotator cuff tendon [2, 14]. These calcifications may be incidental radiographic findings in asymptomatic patients and are usually located 1 to 2 cm from the insertion of the supraspinatus tendon on the greater tuberosity. Theories regarding the cause of calcifying tendinitis include soft tissue degeneration, soft tissue necrosis, trauma, and hypoxia-induced metaplasia of the less perfused tendon insertion to fibrocartilage with resultant calcifications [2]. Uhthoff and Loehr recognize three stages of calcifying tendinitis: precalcific, calcific, and postcalcific [1]. Approximately 50% of patients with calcifying tendinitis have shoulder pain and limited mobility [15]. However, calcifying tendinitis does not commonly cause cortical bone erosion.

Hayes et al. first described calcifying tendinitis with cortical erosion in the pectoralis major, gluteus maximus, and adductor magnus tendons [7]. Calcifying tendinitis with cortical erosion has since been described in other reports and at other insertions—the pectoralis major, gluteus maximus, gluteus medius, adductor magnus, deltoid, linea aspera, and bicipital groove [3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14]. There have been nine cases reported in the literature which involve the shoulder [3, 5, 6, 7, 9, 14]. A retrospective review of 50 cases of osseous involvement in calcific tendinitis was recently published where humeral erosions at the tuberosities were encountered in 11 patients [14].

Hypotheses of bone erosion include active inflammation and local vascularization at the tendon insertion or mechanical effects of muscle traction [5]. It is unknown why some areas appear more predisposed to erosion.

Calcifying tendinitis with cortical erosion could be a difficult diagnosis to make and is an important entity to recognize. One needs to be aware that the cortical erosion and periosteal reaction in this entity can appear somewhat

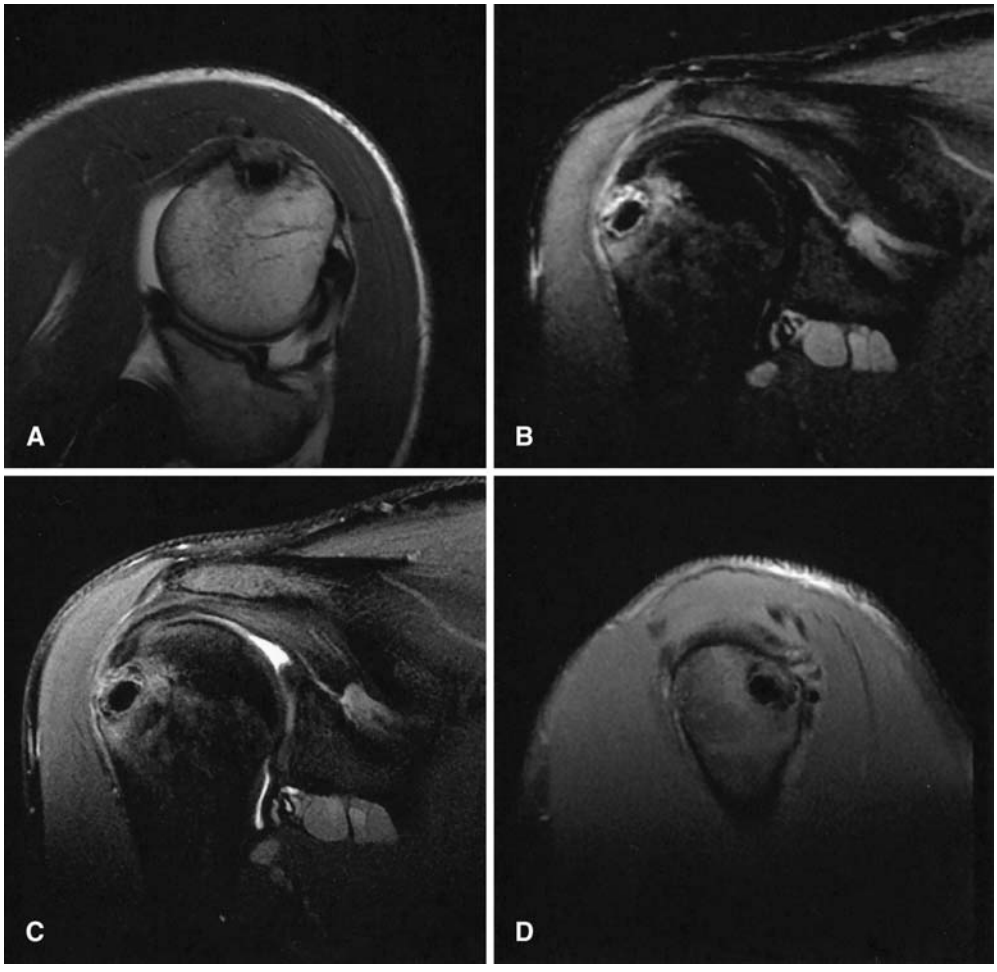

Fig. 2 **A** Axial PD FSE (TR 2033, TE 18.38), **B** coronal STIR (TR 3267, TE 9.288), and **C** coronal PD FSE with fat suppression (TR 2000, TE 29.88) **D** sagittal PD with fat suppression (TR 800, TE 14) direct MR arthrographic images show calcific tendinitis at the insertion of the infraspinatus tendon insertion. There is a low signal intensity rounded structure in the superolateral humeral head with a surrounding thin rim of fluid and adjacent bone marrow edema. Findings correlate with the faint calcification and lucency in the humerus on radiographs and are separate from areas of calcification that are external to the bone consistent with calcific tendinitis. Differential diagnosis included calcific tendinitis with cortical erosion, osteoid osteoma or osteoblastoma, osteonecrosis, or chondroblastoma (epiphyseal lesions)

aggressive on both radiographs and advanced imaging. In the presented case, the differential diagnosis included primarily benign conditions, particularly osteoma, osteoblastoma/osteoid osteoma, and chondroblastoma. Erosion secondary to calcific tendinitis is also important to consider in the differential diagnosis of epiphyseal lesions, which also include osteonecrosis, chondroma, erosion from synovial processes, soft tissue sarcoma with secondary bone invasion, and metastatic lesions. Associated periosteal reaction may cause confusion with extracortical lesions but the location at a tendon insertion and the "comet tail" appearance of the calcification may be helpful methods of distinction. In a large case series, the comet tail appearance was only noted in 51% [6, 14]. The sclerotic margins of the lesion may indicate a slow process. Also, the lack of soft tissue mass and the small size of the lesion may help make the diagnosis. CT appears best suited for confirmation of the intratendinous location of the calcification. Awareness of the possibility of cortical erosion due to calcifying tendinitis may avert unnecessary surgery.

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