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TEST YOURSELF: ANSWER

Shoulder pain

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Answer: Subscapularis calcific tendinopathy with cortical erosion.

MRI shows a very prominent area of lobular signal void consistent with calcification in the distal aspect of the subscapularis tendon (Figure A). It is extending over the bicipital groove and has eroded into the lesser tuberosity (Figure B) where there is a poorly defined area of low signal associated with extensive edema-like marrow change (Figure C and D). The features are consistent with calcific subscapularis tendinopathy with secondary bone erosion, which was confirmed by CT. The remainder of the cuff tendons are intact and muscle bulk is well preserved. No other abnormality was seen.

Calcific tendinopathy is a relatively common condition characterized by abnormal deposition of calcium hydroxyapatite crystals within a tendon. It usually involves the supraspinatus tendon, accounting for approximately 7% of patients who present with shoulder pain. It is commonly seen in patients in the fourth and fifth decades of life, and there is a slight predilection towards females. Risk factors include thyroid and estrogen metabolism disorders, and life-style factors such as alcohol consumption and obesity [1]. The exact etiology is unclear, but it has been hypothesized that hypoxia-induced fibrocartilaginous metaplasia of the critical zone, tissue necrosis, and trauma can all contribute to the formation of intra-tendinous calcification [2, 3].

Moseley has described the natural history of calcific tendinopathy based on surgically treated rotator cuff tendinopathies [4]. The initial stage is an asymptomatic 'silent' phase where the calcium deposition is powder-like. This leads to the mechanical phase, characterized by an increase in size of the calcium deposition. During the hyperemic dissolution phase, sub-bursal rupture can occur where the calcium dissolves between the tendon and bursa interface. Intrabursal rupture can follow when the calcium disseminates through the bursal lining, causing a marked inflammatory response. Cortical erosion and intraosseous migration of calcium have previously been described at various anatomical sites including the supraspinatus, infraspinatus, pectoralis major, and gluteus maximus tendons [5–8]. Erosion is seen on MRI as low signal intensity on T1-weighted imaging and heterogeneously high signal intensity on T2-weighted imaging. It is associated with adjacent bone marrow and soft tissue edema that can mimic aggressive osteitis, especially since calcium is poorly visualized on MRI studies [6, 9]. It is hypothesized that inflammation and hypervascularity lead to bone resorption and mechanical pull at the inflamed tendon insertion can exacerbate the cortical erosion [10]. Although laboratory tests are usually normal, an acute phase can be associated with raised leukocyte, erythrocyte sedimentation rate (ESR), and C-reactive proteins (CRP) with associated clinical symptoms of fever, swelling, and erythema, which in conjunction with abnormal image features can lead to an erroneous diagnosis of infection or aggressive neoplasm [11]. Fleming et al. have demonstrated that histology can show chondroid metaplasia, which can be confused for chondrosarcoma [12]. It is therefore imperative to review previous radiographs and CT with multi-planar reconstructions to visualize the calcium along the tendon and a close working relationship with the pathologist is crucial.

Calcific tendinopathy with cortical erosion and intraosseous migration has been described in the literature, but involvement of the subscapularis tendon is rare. MRI features can mimic infection and aggressive neoplasm. It is therefore important to be aware of the MRI appearances and correlate with radiographs and CT to assess for tendon calcification to arrive at a correct diagnosis and avoid unnecessary biopsy.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

The case presentation can be found at https://doi.org/10.1007/s00256-018-2967-8

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