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CASE REPORT

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Vanishing osteosclerotic lesion of the humeral head

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Abstract We report on the spontaneous disappearance of a dense round lesion from within the greater tuberosity of the humerus. The patient was treated with oral non-steroidal anti-inflammatory agents for symptoms of subacromial bursitis. Symptoms resolved in 10 days and the lesion had vanished when radiographed 3 months later. We surmise the lesion to represent intraosseous crystal deposition.

Key words Osteosclerotic lesion · Calcification · Hydroxyapatite · Humerus · Disappearance

Introduction

An isolated round dense sclerotic lesion of bone generates a differential diagnosis and its management may range from doing nothing more, as in a bone island, or investigating, if thought to represent a symptomatic or clinically significant bone lesion. We report on the spontaneous disappearance of such a lesion and discuss its possible cause and the mechanism of disappearance.

Case report

A 58-year-old woman presented with pain and tenderness of the right shoulder of 3 weeks' duration. Physical examination revealed painful active and passive abduction-internal rotation without any decrease of muscular strength. The cervical spine was normal on examination.

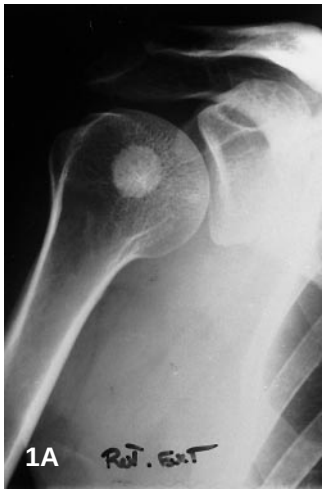
Anteroposterior plain radiographs of the shoulder demonstrated a homogeneous, high-density, 15-mm round lesion with regular and well-defined outlines in the greater tuberosity and a thin curvilinear, ill-defined, low-density calcification in the expected position of the infraspinatus tendon (Fig. 1). A bone scan performed 4 h after injection of HMDP-^{99m}Tc showed an intense focus of radiotracer accumulation in the greater tuberosity (Fig. 2). MRI confirmed the intraosseous location of the lesion, which appeared as a round focus of signal void on T1- and T2-weighted images before and after intravenous contrast injection. These latter scans showed some thickening of the subacromial bursa, which contained fluid (Fig. 3).

Physical examination and laboratory studies revealed no abnormalities. The pain improved after 10 days of oral non-steroidal anti-inflammatory medication.

A plain radiograph taken 3 months later showed complete disappearance of the intraosseous calcified lesion, which was replaced by a radiolucency in the greater tuberosity (Fig. 4). On MR images the low T1 and low T2 were replaced by low T1 and high T2 signals (Fig. 5). CT showed a round focus of cancellous bone resorption filled by a density that measured 8 HU, containing minimal dotted residual calcification. On reformatted coronal CT scan, a thin transcortical channel was perceptible from the former site of calcification to the posterosuperior aspect of the greater tuberosity, near the insertion of the infraspinatus tendon (Fig. 6).

Discussion

The swift and spontaneous disappearance of the mineralized lesion of the greater tuberosity rules out a diagnosis of true osteosclerotic lesion



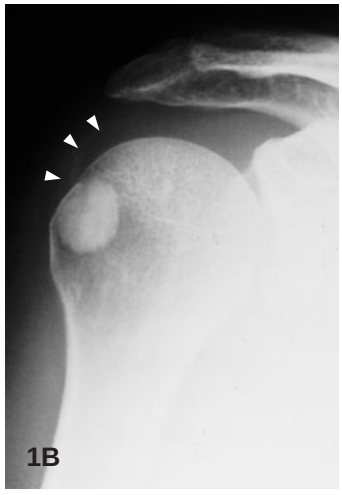


Fig. 1A, B Anteroposterior plain film radiographs of the right shoulder. **A** External rotation shows sclerotic lesion in humerus. **B** Internal rotation shows curvilinear calcifications near the infraspinatus tendon (*arrows*)

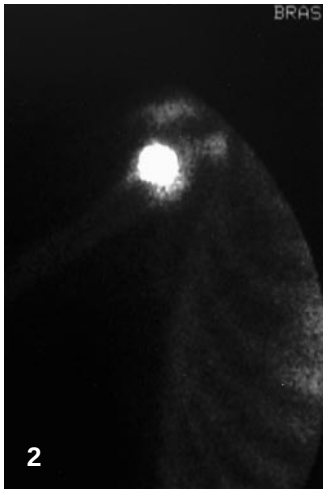


Fig. 2 HMDP-^{99m}Tc bone scan. Intense intraosseous radiotracer accumulation is evident at the site of radiographic abnormality

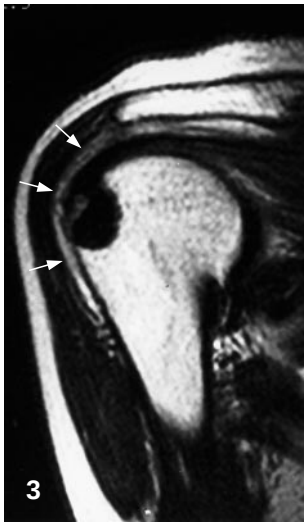


Fig. 3 Coronal MR image of the right shoulder. T2-weighted fast spin echo image (3700/100) shows low-signal intraosseous lesion with thickening of the subacromial bursa (*arrows*)

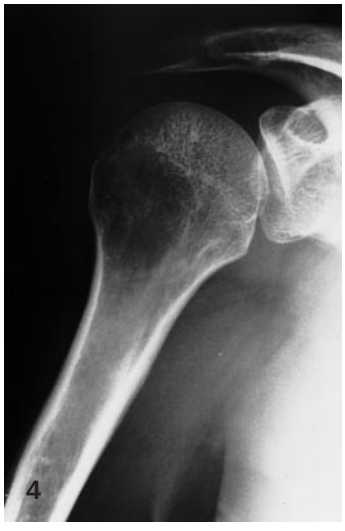
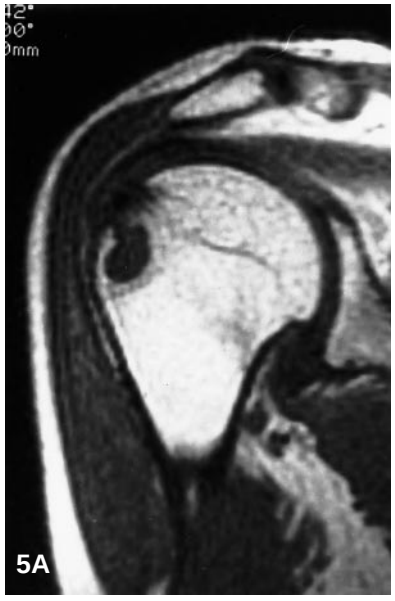
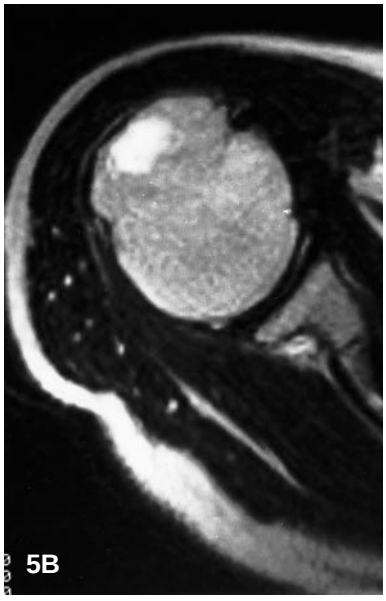


Fig. 4 Internal rotation plain film radiograph 3 months later. The dense lesion of the greater tuberosity has disappeared and is replaced by an osteolytic focus of abnormality

such as bone island, osteoid osteoma, or osteoblastic metastasis [1]. The presence on the first set of radiographs of a low-attenuation calcification along the course of the infraspinatus tendon (Fig. 1B), associated with subacromial bursitis by MR imging, suggests hydroxyapatite crystal deposition. The lack of biopsy or proof from crystal aspiration leaves open to speculation the type of crystal deposition. Nevertheless, the only reasonable hypothesis for the bone lesion seems to be an intraosseous amorphous crystal deposition, probably hydroxyapatite, near the insertion of the infraspinatus tendon. There are two possible explanations for the disappearance of the intraosseous calcification: either resorption into the bone itself, or an evacuation into the subacromial bursa or the articular cavity through the thin cortical channel. The latter is probably more plausible in view of the transcortical channel and initial pain.

Intratendinous hydroxyapatite crystal deposition is a very common condition, especially in the rotator cuff tendons [2]. Even if the pathophysiology is unclear, the natural history of such lesions is well known, with sub-bursal or intrabursal rupture and resorption, generally associated with clinical events like severe pain and tenderness. Cortical erosions have been described with such calcifications in locations such as the femoral insertion of gluteus maximus and the humeral insertion of the pectoralis major [3]. Moseley describes occasional operative discovery of intraosseous calcification with extension from the supraspinatus tendon to the greater tuberosity, but without radiological evidence [4]. A conspicuous, spontaneously disappearing intraosseous calcified lesion without continuity with intratendinous calcification has not yet been reported to our knowledge. This finding has to be added to the spectrum of findings associated with crystal deposition diseases. Treatment of symptoms with oral nonsteroidal anti-inflammatory agents and time would appear to result in disappearance of such intraosseous lesions, just as with their more familiar tendinous counterparts.





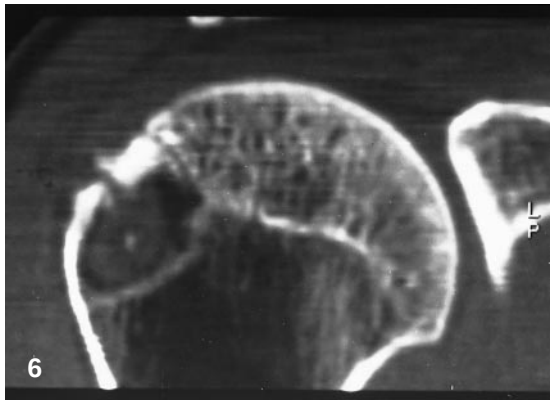


Fig. 5 MR examination 3 months later. **A** Coronal T1-weighted spin echo image (450/20) shows low-signal lesion. **B** Axial T2-weighted fast spin echo image (3700/100) now shows an intraosseous lesion with high signal

Fig. 6 Reformatted coronal unenhanced CT scan, 3 months later. There is a narrow channel of cortical disruption with slightly increased density in the cancellous bone and a single tiny focus of calcification

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ANNOUNCEMENTS

15th Annual Masters Diagnostic Radiology Conference
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