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Case report

Humeral greater tuberosity osteolysis as a complication of intraosseous calcification migration: natural history depicted by imaging

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Summary. Migration of calcification within the bone leading to greater tuberosity osteolysis is a peculiar complication of the calcifying tendinitis of the rotator cuff. The case of a 38-year-old woman complaining of right shoulder pain, which had been going on for one year, is hereby described. The evolution of the infraspinatus tendon calcifying tendinitis leading to osteolysis of the greater tuberosity of the humerus is depicted by imaging and, particularly, by the MR and CT features changing over time. In this paper we focus on the importance of both MR and CT exams in the diagnostic process of the different phases of the disease. The correlation between clinical symptoms and imaging features is also helpful for imaging interpretation: the most painful phase corresponds to the migration of the calcification, whereas pain tends to decrease when the osteolysis develops. Awareness of the existence of this condition may prevent unnecessary invasive procedures. (www.actabiomedica.it)

Key words: complication of calcific tendinitis; humeral osteolysis, MRI, CT

Introduction

The migration of calcification within the bone has been described a rather peculiar complication of calcific tendinopathy or, more appropriately, calcium hydroxyapatite crystal deposition disease (1-8). In order of frequency, calcium deposits mainly involve the shoulders, hips, elbows, wrists, and knees, although, the greater tubercle of the humerus is the most common site reported in the literature (2, 4, 5).

Hereby, we are reporting a case in which the effect of the intraosseous calcium penetration leading to osteolysis of the greater tuberosity of the humerus is documented by imaging: particularly, MR signal intensity and CT density changes over a brief period of time are documented.

Knowledge of clinical symptoms and radiological appearance permits to identify this rare condition and, since the imaging findings are quite specific, a confident diagnosis can be reached, thus avoiding biopsy and reducing patients' anxiety by reassuring them about the benignity of this condition.

Case report

A 38-year-old woman complaining of right shoulder pain, mostly affecting her sleep at night, associated with stiffness and weakness of the shoulder during the previous year, was referred to our centre to perform a bone biopsy. She had already undergone CT and MR exams in another diagnostic centre.

She is a lawyer; neither history of trauma nor sport activities were reported.

The physical examination revealed a painful, reduced arc of motion of the right upper limb associ-

ated with mild weakness, resisted external rotation and abduction testing. General physical examination and blood examination values were normal.

On the CT scan, a large semilunar calcification of the infraspinatus tendon adjacent to an increased bone density area of the humeral greater tuberosity was visible; a thin cortical discontinuity was also observed (Fig. 1).

On the MRI, the area of increased bone density displayed a dark signal in all the sequences, and a marked perilesional bone marrow oedema was depicted on STIR sequence (Fig. 2).

A clear diagnosis was not provided and a differential diagnosis, including osteoblastoma, osteoid osteoma, osteochondritis or an infective disease, was proposed.

Based on our retrospective re-evaluation of MR and CT exams, a diagnosis of intraosseous migration of the calcification was suspected; therefore, we decided not to perform a biopsy but to repeat the MR with gadolinium-contrast medium injection in order to more confidently find out the nature of the lesion and, particularly, to rule out an osteoid osteoma.

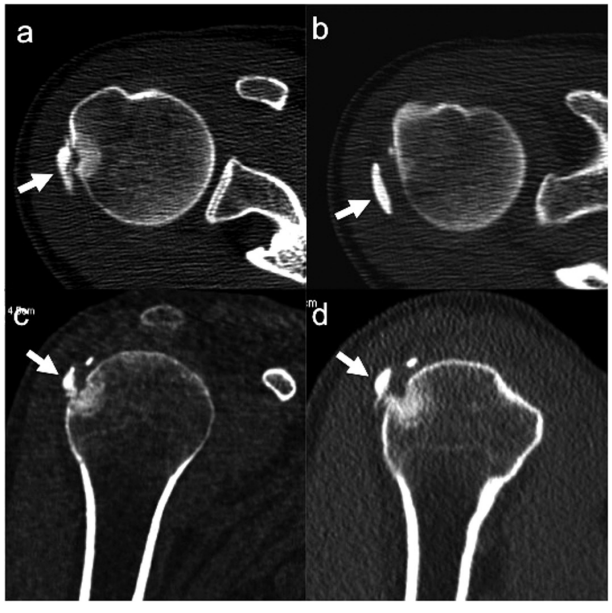

Figure 1. Baseline CT exam, axial (a, b) and oblique reformatted images (c, d). A large, dense, semilunar calcification of the infraspinatus tendon is visible (white arrow). Adjacent to the calcification an increased density area of the humeral greater tuberosity is also depicted as well as a thin cortical discontinuity. Another small rounded calcification is also detectable

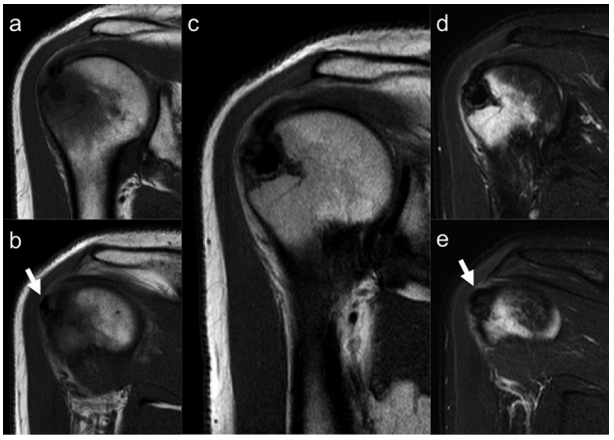

Figure 2. Baseline MR, coronal T1w (a, b), T2w (c) and STIR (d, e). A rounded area of the humeral greater tuberosity with hypointense signal on all the sequences is present; on STIR images a marked perilesional bone marrow oedema is clearly detected. Note the hypointense calcification within the preinsertional portion of the infraspinatus tendon (arrows)

At the follow-up MR performed 3 weeks after the first exams, the imaging findings were completely different. In fact, a pseudocystic lesion with a very bright signal suggestive of a liquid content was depicted at the level of the previously dark area; the bone marrow oedema was reduced and showed diffuse enhancement after contrast injection (Fig. 3). These findings were confirmed on CT images, where the increased bone density area of the humeral head had disappeared and had been replaced by an osteolytic lesion.

Finally, the calcification was markedly reduced and showed a different shape (Fig. 4).

Due to the regression of the oedema, the absence of a mass and the lack of a hypervascular nidus, a bone tumour was confidently ruled out as well as an infective pathology: the final diagnosis was osteolysis of the greater tuberosity as a sequela of intraosseous penetration of the infraspinatus calcification.

At the next follow-up MR exam, after another ten weeks, the findings had slightly changed again: the osteolytic area resulted smaller due to a thin peripheral solid reparative tissue and the surrounding bone marrow oedema had almost disappeared. These findings were confirmed by the CT exam, tailored to the humeral head (Fig. 5).

Accordingly to imaging, clinical symptoms complained by the patient were significantly reduced.

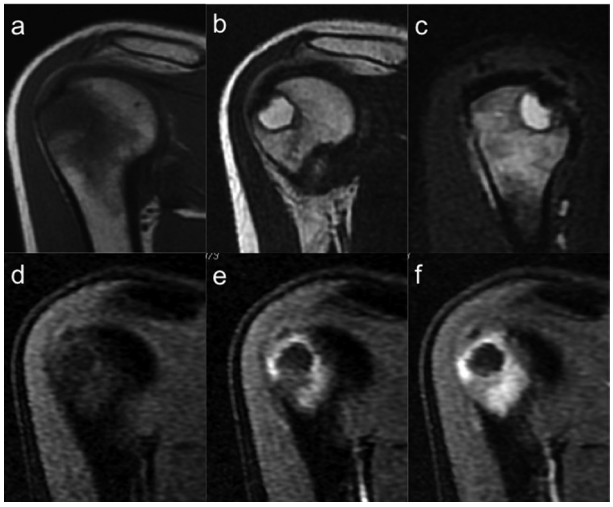

Figure 3. MR follow-up scan after three weeks, coronal T1w (a) and T2w (b), sagittal STIR (c) and 3 different temporal phases of the dynamic contrast study at the same level (d-f). The previously increased bone density area is replaced by a pseudocystic lesion with a liquid content; the perilesional bone marrow oedema is slightly reduced, as shown on the sagittal STIR image (c). The dynamic study shows the contrast enhancement of the bone marrow oedema and no contrast uptake of the cystic area (d-f)

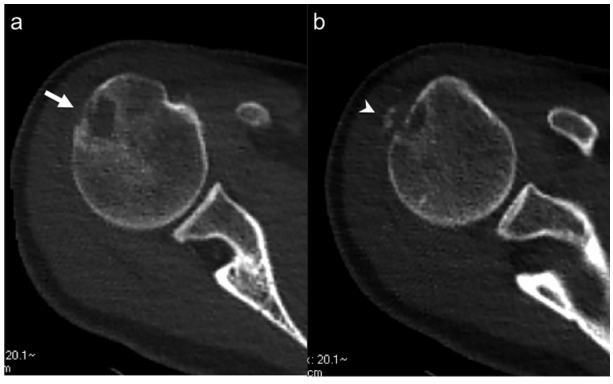

Figure 4. CT follow-up scan after three weeks, axial images (a, b). The increased bone density area of the humeral head had disappeared and replaced by an osteolytic lesion (arrow); the tendon calcification is reduced and shows a different shape (arrowhead)

Discussion

Complications of calcium hydroxyapatite crystal deposition disease of the shoulder include adhesive capsulitis, rotator cuff tears, ossifying tendinitis and greater tuberosity osteolysis (1).

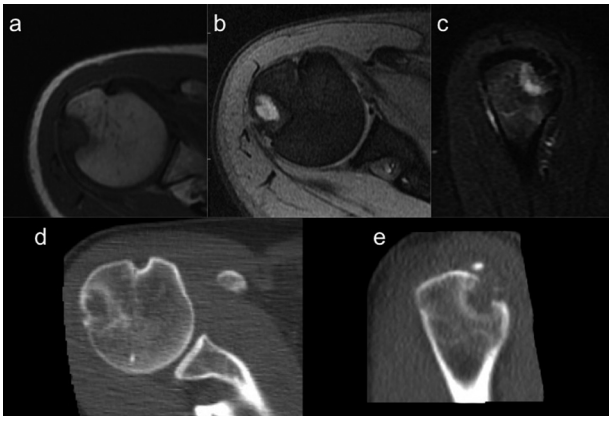

Figure 5. MR follow-up after ten weeks, axial T1w (a) and GRE T2*w (b), sagittal STIR (c) and TC follow-up axial (d) and sagittal (e) reformatted images. Reduction of the pseudocystic lesion, partially replaced by a solid reparative tissue partially calcified (d, e), due to the bone healing process, with further reduction of the perilesional bone marrow oedema, better depicted on STIR (c).

According to the literature, bone involvement in calcific tendinitis of the shoulder is far from uncommon and humeral greater tuberosity is the most affected site reported, although, usually related to the involvement of the supraspinatus tendon (76%), whereas infraspinatus, teres minor and subscapularis tendons are less commonly affected (2).

In our case, imaging documented all the phases before, during and after the development of the humerus greater tuberosity osteolysis due to calcifying tendinitis of the infraspinatus muscle.

First, we wish to focus on the specificity of the pre-osteolysis imaging findings both on CT and MR; a well defined dark MR hypointense area corresponding to an increase in bone CT density area associated with pararticular calcification and bone oedema seem to be very specific of this pathology (3-5). Awareness of the above imaging features significantly restricts the differential diagnosis and makes bone biopsy as well as surgery not needed.

Another important diagnostic key is the demonstration of the continuity between the osseous lesion and the calcific tendinitis. For this reason, we believe that both CT and MR are necessary to diagnose this pathological entity; CT can better demonstrate site and morphology of the calcification as well as cortical

erosion, whereas MR is fundamental to depict bone marrow/soft tissue oedema (3).

We want to stress that also the oedema represents an important diagnostic element and it should not be misinterpreted as a sign of an aggressive lesion and, in fact, Malghem J et al. (6) pointed out that it was visible on MRI in all the cases of cortical erosion in their series.

Marinetti et al. (7) very well describe all these findings in a recent paper and we found their work very useful in order to guide us to make our diagnosis.

The only difference we observed in our case is related to the appearance of the calcification in the reabsorption phase, before the onset of the osteolysis. In fact, no hyperintensity of the calcification was noticeable on MRI. A possible explanation could be that either hyperintense calcifications are not the only ones that may lead to osteolysis or that the hyperintensity of the calcifications represents a brief, temporary appearance, not depicted in our case.

We believe that our case can be useful from a teaching point of view since the natural history of this pathology is here well documented by imaging related to one single subject over a brief period of time. Actually, we found it impressive how fast the development of osteolysis can be; in only 3 weeks imaging findings completely changed.

In fact, on CT, the area first depicted as dense was replaced by a lytic lesion and corresponded to the MR signal intensity modification: the marked hypointense area changed into a strong hyperintense lesion, with fluid-like appearance.

The reduction of the inflammatory reaction expressed by the progressive decrease of the bone marrow oedema well correlated with the clinical symptoms: the pain was strongly reduced.

The possible differential diagnoses for the sclerotic humeral head lesion proposed in the literature include osteoma, osteoblastoma, osteoid osteoma, chondroblastoma, osteonecrosis, chondroma and osteolytic synovial processes (4, 5).

Because of exacerbation of pain during the night, bone marrow oedema and the dark area that could have been a possible nidus, although atypical, the most likely alternative differential diagnosis we needed to rule out was osteoid osteoma; but the lack of an hypervascular nidus in the contrast dynamic study let us to exclude this diagnosis (9, 10).

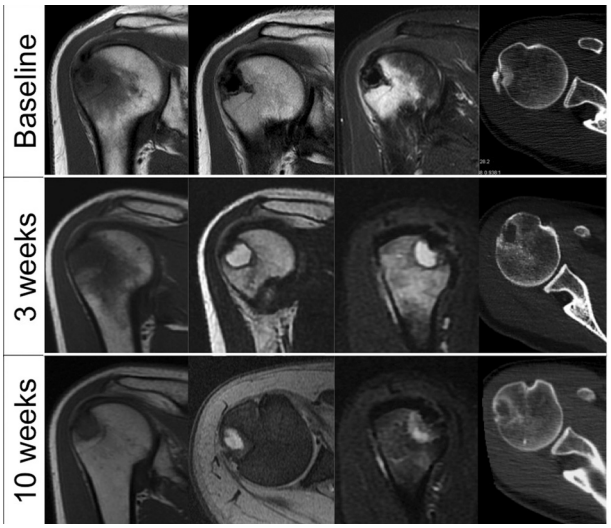

Figure 6. Summary Image. The natural history of the humeral greater tuberosity osteolysis over time; imaging changes depicted by CT and MR

Other differential diagnoses reported are soft tissue sarcoma with bone invasion and metastatic osteolytic lesions (11), as malignant conditions (3, 4).

Anyway, in our case the lack of a soft tissue mass and the overall radiological features, including the absence of significant contrast uptake of the bone lesion were helpful in ruling out an aggressive pathology.

In conclusion, calcifying tendinitis with cortical erosion is far from uncommon and the diagnosis should not be difficult. In the case of erosion and increased density of the greater tuberosity on CT, associated with a low signal intensity lesion on MR with strong perilesional oedema, radiologists should strongly consider the diagnosis of intraosseous migration of calcification as a complication of calcific tendinitis. Eventually, osteolysis is very likely to develop; clinical and imaging follow-up is required and no further invasive diagnostic procedures are needed (3, 12) (Fig. 6).

Conflict of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

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