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Case Report

Atypical Presentation of Intra-Articular Osteoid Osteoma in the Proximal Femur: A Difficult Diagnosis

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ABSTRACT

Osteoid osteoma is among the commonest bone tumors, primarily affecting young subjects. Often localized in the diaphysis cortex of long bones, the disease has a well-described symptomatology and imagery of choice for diagnosis. When in a different location, the diagnosis is less evident. We describe a case herein of an intra-articular osteoid osteoma of the hip misdiagnosed as a femoro-acetabular impingement and treated by means of hip arthroscopy.

INTRODUCTION

Osteoid Osteomas (OOs) are benign tumors consisting of a hypervascular inflammatory lytic nidus in the cortex of long bones. They are defined as benign Stage 2 lesions according to Musculoskeletal Society (MSTS) Staging. They often respond to aspirin, tending to disappear following several years of conservative management. The imagery of choice is Computed Tomography (CT), able to reveal the hypolucent nidus surrounded by an area of reactive sclerosis and periosteal thickening. Positron-Emission Tomography (PET) can reveal a double density sign, with the nidus the most intense surrounded by the less-intense bony sclerosis, giving the image of a halo. Magnetic Resonance Imaging (MRI) demonstrates the nidus with the surrounding bone edema. These findings may differ in cases involving atypical osteoma location.

CASE REPORT

A 19-year-old soccer player presented at consultation because of residual pain in the left hip 1 year after undergoing arthroscopy for femoroacetabular impingement. The patient had left inguinal nonirradiating pain, exacerbated by walking to the point of limping. Past pre-operative imagery revealed superior acetabular and posterior femoral-head osteophytes, suggesting pincer impingement and cam deformity. Post-operative imagery revealed excision of the superior osteophyte (Figure 1A, Figure 1B). In spite of this, the patient suffered from persistent pain, and a pelvic MRI was prescribed (Figure 2), revealing joint edema and synovial hypertrophy and an anterior cortical femoral neck lesion on STIR sequences. The lesion was hypolucent, surrounded by a hyperlucent rim. CT was performed for further evaluation, revealing a millimetric lesion that was slightly hyper intense and surrounded by a narrow osteolytic rim with no perinidal sclerotic bone reaction. Nevertheless, radiology was performed to confirm the diagnosis of an OO, with subsequent radiofrequency ablation. The patient was examined 5 months later and reported complete alleviation of pain and improvement in functional status. Physical examination showed a significantly improved range of motion for all movements except in internal rotation, where the hip remained stiff. Retrospectively, the lesion was not apparent on MRI prior to arthroscopy. On the other hand, the scan (Figure 3A) did reveal a subtle osteolytic lesion without the evident osteoid calcification that was present on the post-arthroscopy scan (Figure 3B). This could have been mistaken for an artefact.

DISCUSSION

Anatomically, OOs are typically located in the diaphysis cortex of long bones. In these locations, they present typical symptomatology and require normal imagery. In atypical locations, the diagnosis may become less evident. For example, patients with intraarticular hip lesions have less night pain [1], and imaging-based diagnosis proves more challenging. CT is not able to detect the sclerotic rim around the nidus, which has been attributed to absence of the bone-forming layer of the periosteum called the cambium layer [2]. The reactive sclerosis, however, may be a little distant [3]. X-ray can reveal distant reactive bony hypertrophy of the femoral neck, suggesting cam deformity. This can contribute to a patient's symptomatology, leading physicians away from the true cause. MRI can reveal labral tears, which are associated with cam deformity [4,5]. Bone edema can conceal the nidus, giving the appearance of regional osteoporosis or

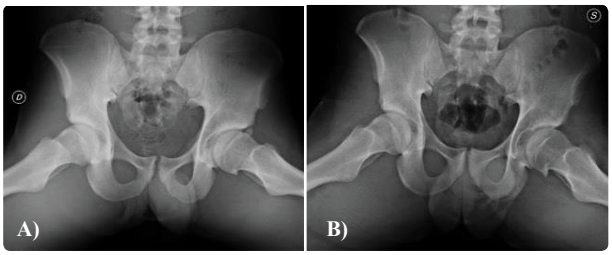

Figure 1: (A) Anteroposterior view of the pelvis with the hips in extension, revealing superior acetabular osteophytes and posterior femoral cam deformity on the left
(B) Excision of the acetabular osteophyte.

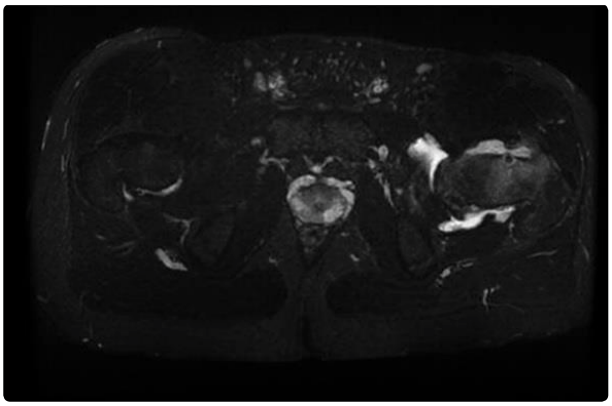

Figure 2: Axial cut of a T2-weighted image of the pelvis, revealing anterior cortical hypolucent lesion with concomitant bone and joint edema.

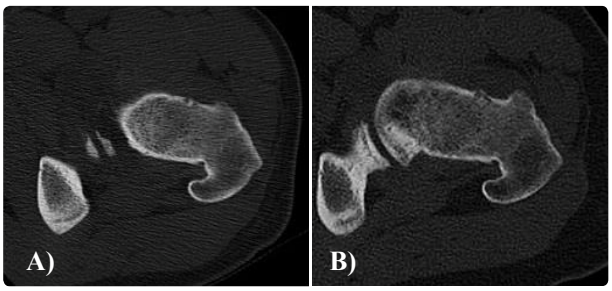

Figure 3: (A) Small intracortical lesion of the anterior border of the femoral neck shortly following manifestation of the patient's symptoms
(B) Increased size of the lesion with more osteoid calcification, yet no perinidal bone sclerosis.

an aggressive process [6,7]. The edema may extend to the synovium and soft tissues, mimicking inflammatory monarthritis or synovitis [8-11]. A small nidus can be masked by the intense signal exhibited by a bone edema on MRI [12], and a smaller MRI field is sometimes necessary, including half-moon sign assessment, as suggested by Klontas *et al.* [12,13]. Moreover, bone scintigraphy may not have diagnostic value in intra-articular lesions [14], and when lesions affect the feet or hands, they may be misdiagnosed as complex regional reflex dystrophy [15]. Symptoms can be acutely aggravated when OO contents perforate into the joint following cartilage erosion [16,17]. Briefly, OOs can occur anywhere in the bone, typically mimicking the usual pathologies of their surrounding area.

Children can also be affected, and can exhibit such an intense reactive bony sclerosis that this may obscure the site of the hypolucent nidus. In these cases, the CT scan slices need to be thinner than 2-3mm or the lesion may be missed [2,18]. With lesions located near the epiphysis, patients may present with limb-length discrepancy and axial deformation [14,19].

Sometimes, OOs may be mistaken for Brodie's abscesses. Osteoid nidus calcification, if present, looks the same as a sequestrum on imaging. On gadolinium-enhanced MRI, however, the nidus can be revealed as hypervascular and hyperintense, and the abscess may become a fistula, showing a break in the rim [20].

OOs may also be present in cancellous bone, the lesion potentially not associated with any reactive bone sclerosis process. CT scanning in these cases do not show the perinidal sclerotic contour. In fact, histologically, OOs can be intracortical (mainly), subperiosteal (e.g. talus) or cancellous. When cancellous, CT only reveals a small area of decreased bone density, while scintigraphy may not be able to detect the perinidal halo of a reactive bony sclerosis [14]. In contrast, dynamic contrast-enhanced MRI can reveal the early peak and washout flow of a hypervascular nidus, proving different from that of the surrounding bone edema, and may thus offer an alternative option in detecting atypical OOs [21,22].

Nevertheless, OOs remain benign tumors known to naturally regress over several years, with the use of medical management reported in several studies [23,24]. The regression may be caused by clotting of the vascularity inside the nidus [25]. In healed OOs, the sclerosis and cortical thickening may not disappear for many years, thus creating the potential for error [26,27]. Furthermore, the morphological appearance of femoroacetabular impingements does not necessarily mean they are the cause of hip pain, and careful assessment is thus essential [28].

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