CASE OF THE MONTH

An unusual cause of bone marrow oedema in the acetabulum

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Case presentation

A 37-year-old male with no significant past medical history presented at the orthopaedic surgery department with gradually increasing pain in the left hip for 3 months. 3 days prior to presentation the pain exacerbated acutely. There was absence of any previous history of trauma.

Clinical examination revealed an antalgic gait and reduced range of motion of the left hip but no signs of inflammation. The patient's laboratory results were all within normal limits. Bone scintigraphy using ⁹⁹Tc^m-methylene diphosphonate (MDP) was performed which revealed increased homogeneous tracer uptake of the proximal part of the left acetabulum and less intense but more diffuse uptake in the other parts of the left hip joint (Figure 1).

Subsequent MRI was performed and demonstrated bone marrow oedema (BMO) in the acetabulum and the femoral head and neck (Figure 2). The *T*₁ weighted images (*T*₁WI) (Figure 2a) showed marked hypointensity with subtle subchondral sparing of the normal bone marrow intensity of the acetabulum. Predominant hyperintensity of the acetabular marrow (Figure 2b) extending to the iliopubic region and sacroiliac joint was seen on fat-suppressed *T*₂ weighted images (*T*₂WI). There was also diffuse but less intense signal increase in the intertrochanteric area extending to the femoral head and neck (Figure 2b). Subtle sparing of normal fatty bone marrow was present in the subchondral area of the acetabulum (Figure 3). After intravenous contrast administration (Figure 2c) diffuse enhancement of the bone marrow in the acetabulum and proximal femur was seen.

In order to exclude a stress fracture or malignancy additional CT was performed (Figure 4). There was no evidence of fracture or frank osteolysis but hazy

delineation of the bone trabeculae was seen in the acetabulum, femoral head and neck on the left side compared with the right side. In addition a thin but preserved subchondral bone plate of the femoral head was demonstrated.

What is the most likely diagnosis at this stage of the disease? Do you recommend further imaging or biopsy?

Figure 1. Bone scintigraphy (anterior view) showed intense uptake in the left acetabular region and less avid uptake in the femoral head and neck compared with the right side.

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Figure 2. (a–c) Coronal MRI of the left hip at first presentation (T_1 weighted imaging (T_1 WI), fat-suppressed T_2 weighted imaging (T_2 WI) and T_1 WI after intravenous injection of gadolinium chelate, respectively). (a) The T_1 WI showed hypointensity (asterisk) with subtle subchondral sparing (arrow) of the normal bone marrow intensity of the acetabulum. (b) Fat-suppressed T_2 WI showed predominant hyperintensity of the acetabular marrow (asterisk) and less intense signal increase in the intertrochanteric area extending to the femoral head and neck was seen (arrow). (c) Contrast-enhanced T_1 WI of acetabular marrow (asterisk) and proximal femur (arrow) was seen. (d–f) Coronal MRI 2 months later (T_1 WI, fat-suppressed T_2 WI and T_1 WI after intravenous injection of gadolinium chelate, respectively) showed regression of the signal intensity changes in the acetabulum (asterisk) and femoral head and neck (arrow).

Diagnosis

A definitive diagnosis at this stage was not forthcoming. MRI showed a pattern of BMO, consisting of low-signal intensity on T_1 WI and high signal intensity on STIR or fat-suppressed T_2 WI corresponding with increased interstitial water content. The differential diagnosis for BMO is extensive [1]. BMO is a non-specific imaging finding with variable aetiology. Three major pathogenic groups to consider are ischaemic, mechanical and reactive aetiologies. Careful analysis of the following imaging signs may allow a more specific diagnosis of BMO syndrome (BMOS): (1) the absence of a tumoural lesion causing focal osteolysis on CT; (2) simultaneous involvement of the acetabulum and the femoral head-neck region; (3) the intact aspect although hazy delineation of bone trabeculae of the acetabulum, femoral head and neck with a thin but preserved subchondral bone plate on CT; (4) the relative preservation of the subchondral bone marrow on T_1 WI; and (5) the resolution of oedema on follow up MRI [2, 3].

Because BMOS is usually a self-limiting condition in which the natural course of the bone marrow oedema is transient, conservative management was employed and follow-up MRI was recommended. MRI 2 months later

Figure 3. Sagittal T_1 weighted imaging at initial presentation. Subtle sparing of normal fatty bone marrow was present in the subchondral area of the acetabulum (arrowheads).

Figure 4. Coronal CT image of pelvis in bone window setting at initial presentation. Hazy delineation of the bone trabeculae was seen in the acetabulum (arrow), femoral head and neck (arrowhead) on the left side compared with the right side. A thin but preserved subchondral bone plate of the femoral head was demonstrated (double arrowhead).

(Figure 2) showed marked regression of the signal intensity changes in the acetabulum (Figure 2d–f), femoral head and neck (Figure 2d–f), on T_1 WI and T_2 WI (Figure 2d,e) and after contrast administration (Figure 2f).

Discussion

BMOS is a self-limiting clinical syndrome also referred to as “transient osteoporosis”, “localised transient osteoporosis”, “regional migratory osteoporosis”, “algodystrophy”, “transient marrow oedema” and “transient bone marrow oedema”. The aetiology of BMOS remains controversial and the pathophysiology of the condition is poorly understood [4]. Terms such as transient osteoporosis, localised transient osteoporosis and regional migratory osteoporosis were historically used to describe osteopenia which is an important imaging feature of the syndrome. More recently it was recognised that the presence of diffuse BMO on MRI was a more constant feature even in the absence of radiographically detectable osteopenia.

BMOS is usually seen in middle-aged men presenting with acute hip pain and functional disability without history of trauma. Women are mostly affected during the third trimester of pregnancy or the immediate postpartum period [4]. The hip is most frequently involved with a pattern of diffuse BMO in the femoral head and intertrochanteric region. Our case, however, demonstrates a less typical location of BMOS with primarily acetabular and only minor femoral involvement. Acetabular involvement is reported in approximately 17% of patients, as reported by Malizos et al [5]. To our knowledge only one case report has been published with isolated acetabular involvement [6].

Different imaging modalities such as bone scintigraphy, conventional radiographs, CT and MRI can help the radiologist to make the correct diagnosis and avoid invasive biopsy procedures. $^{99}\text{Tc}^{\text{m}}$ -MDP scintigraphy shows non-specific but homogeneous tracer uptake representing the increased blood flow in combination with an increased osteoblastic activity [4]. Osteopenia can be detected on conventional radiographs but this feature is time

dependent as a considerable amount of trabecular bone is needed in order to become apparent on imaging. CT sometimes demonstrates early diffuse demineralisation whereas initial radiographs may be normal. A thin but preserved subchondral bone plate can be documented if the femoral head is affected [2]. MRI is the imaging modality of choice for evaluation of BMOS. For early diagnosis of this syndrome the presence of BMO is often diagnostic. Moreover, meticulous analysis of the subchondral area reveals prognostic information. Subchondral preservation of the fatty bone marrow is described in approximately 23% of the patients and may predict a good outcome with reversibility of bone marrow changes in 100% of cases [3, 5]. In our case, this phenomenon was best seen on sagittal T_1 WI, whereas other authors preferred the use of T_2 WI or contrast-enhanced T_1 WI to exclude subchondral insufficiency fractures or contour deformity of the epiphysis [3].

BMOS is usually self-limiting and may be treated conservatively. Some authors have advocated protected weight-bearing as there is a risk of pathological fracture secondary to the osteoporosis, although this therapy has not yet been proven [7]. The differential diagnosis of BMOS may be a radiological challenge. If the aetiology is not clear on initial imaging, time is an important factor to distinguish transient bone marrow changes, early avascular necrosis or other causes of BMO. Although not routinely required for the diagnosis of BMOS, CT may be helpful in specific scenarios, such as the differential diagnosis of reactive BMO owing to a benign bone tumour (osteoid osteoma) or chronic recurrent multifocal osteomyelitis (CRMO). In osteoid osteoma, CT will better demonstrate the nidus than MRI and in cases of CRMO, a combination of lytic areas and sclerosis will be seen [8].

Further discussion of the differential diagnosis is beyond the scope of this brief report. In summary, the imaging clues to diagnosis in our case are the following: (1) the absence of a tumoural lesion causing focal osteolysis; (2) simultaneous involvement of the acetabulum and the femoral head-neck region of which the latter is a typical location; (3) the intact aspect although hazy delineation of

bone trabeculae of the acetabulum, femoral head and neck with a thin but preserved subchondral bone plate on CT; (4) relative preservation of the subchondral bone marrow on T_1 WI; and (5) the resolution of oedema on follow up MRI.

Conclusion

BMOS is a distinct clinicoradiological entity which should be recognised by the radiologist in order to avoid unnecessary invasive biopsy procedures.

References

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