

Migratory symptomatic bone marrow oedema of the knee : A case report and literature review

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Bone marrow oedema is a common finding on MRI. Differential diagnosis includes many different clinical entities. We hereby present a case of migrating symptomatic bone marrow oedema from the medial to the lateral femoral condyle after which spontaneous resolution of both clinical and radiographic findings occurred. The role of altered weight-bearing due to therapeutic use of an unloading brace as a potential causative factor in the pathogenesis of migrating bone marrow oedema remains unclear. The literature on the topic is reviewed.

Keywords: bone marrow oedema; MR Imaging; knee; transient osteoporosis; regional migratory osteoporosis.

INTRODUCTION

Bone marrow oedema on MRI may be associated with a range of distinct pathologies. It is however a nonspecific sign and therefore it remains a challenge for both the orthopaedic surgeon and the radiologist to interpret the radiological findings in a broader clinical and pathological context. Bone marrow oedema syndrome is a self limiting condition of symptomatic bone marrow oedema without any clear causative factor such as a trauma, infection or infarction. Clinical signs include pain aggravated by weight bearing, joint effusion and usually no restriction in range of motion of the joint. Osteopenia may be present on plain radiographs.

We present a case of bone marrow oedema shifting from one femoral condyle to the other within the ipsilateral knee joint.

CASE REPORT

A 43-year-old male presented at our outpatient clinic with a sudden onset medial right knee pain. He did not recall any trauma. There was no pain at rest or at night. The use of non steroidal anti-inflammatory drugs did not give any pain relief. The complaints worsened on weight bearing. His medical history was unremarkable and he did not take any medication. Clinical examination showed normal limb alignment. There was a slight effusion in the right knee, a full range of motion and normal stability. McMurray's test was painful over the medial joint line although no snap was felt. Palpation was painful over the medial joint line and the medial femoral condyle.

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Fig. 1. — MRI of the right knee

a. Sagittal T1-weighted MR image showing a faint subchondral hypointense bandlike structure at the medial femur condyle, representing a subchondral insufficiency fracture (white arrow). The lesion is surrounded by an ill-defined area of bone marrow oedema (asterisks).

b. This area of BMO is better appreciated on the coronal fat suppressed T2-weighted MR image (asterisks).

Standard radiographs of the knee were normal. Further diagnostic imaging was performed by MRI. This showed an ill-defined area of bone marrow oedema at the medial femoral condyle and a subchondral insufficiency fracture (fig 1). Since no cartilage defects were seen, the patient was treated with non-steroidal anti-inflammatory drugs and a valgus unloader brace in order to minimize the forces acting on the medial knee compartment.

Three months later both the subjective symptoms and clinical examination had normalised. A further MRI however revealed bone marrow oedema at the lateral femoral condyle, whereas the lesions at the medial femoral condyle were no longer visible (fig 2). We therefore changed the valgus unloader brace to a neutral brace.

Follow-up examination after another three months showed complete resolution of clinical symptoms as well as absence of signal changes on MRI.

DISCUSSION

Bone marrow oedema (BMO) is a nonspecific anomaly frequently encountered on MRI (3,9). It may be related to several different pathologies.

BMO is defined by increased signal intensity on T2-weighted images, especially when using fat-suppression techniques. After intravenous administration of contrast agents, enhancement of the lesion is seen, indicating hypervascularity.

Histological examination of an area of BMO shows an increased presence of fluid, both intraand extracellular within the bone marrow (15). This increase in intra- and extracellular fluid causes an increased pressure in the bone marrow (2,7,11), which may be responsible for the pain experienced by the patient. The pain typically increases during mechanical loading and when tapping the affected area.



Fig. 2. — MR examination of the right knee three months after the onset of the treatment.

Coronal fat suppressed T2-weighted MR image showed disappearance of BMO at the medial femoral condyle. However,

pearance of BMO at the medial femoral condyle. However, there was an extensive area of BMO at the lateral femoral condyle (asterisks).

Areas of BMO usually are not visible on plain radiographs and CT scan since the process is limited to the bone marrow and the trabecular and cortical bone is typically spared. Bone scintigraphy is a sensitive technique but suffers from a poor spatial resolution.

Hofmann et al (10) published a clear and helpful review on the differential diagnosis of BMO. They classified it into three distinct categories, based on aetiology: ischaemic, mechanical and reactive BMO. Each category is then subdivided into different clinical entities (table I). Mechanical BMO implicates that either a trauma or a chronic overloading has occurred. Neither trauma nor malalignment in any plane was present in our case. Nevertheless, stress related BMO, though most often occurring in combination with cartilage loss, can be observed without any arthritic changes of the joint. Therefore, it may be a possible cause of BMO in our case. The presence of a subtle subchondral insufficiency fracture (SIF) is an argument in favour of a mechanical aetiology in our case.

Table I. — Differential diagnosis in BMO. Modified from Hofmann *et al.* (10)

1. Ischaemic BMO

- a. Osteonecrosis
- b. Bone Marrow Oedema Syndrome
- c. Osteochondrosis dissecans
- d. Complex Regional Pain Syndrome

2. Mechanical BMO

- a. Bone Contusion
- b. Microfracture
- c. Stress related BMO
- d. Stress fracture

3. Reactive BMO

- a. Arthritis
- b. Osteoarthrosis (Cartilage loss)
- c. Postoperative BMO
- d. Tumour-related BMO

In reactive BMO, the BMO is a symptom of an underlying disease or prior surgical procedure, and the cause of the BMO is clear in most cases. There were no arguments for any arthritic, postoperative or tumour-related changes in the case presented.

In ischaemic BMO, the differential diagnosis between osteonecrosis and osteochondrosis dissecans on one side and bone marrow oedema syndrome (BMOS) on the other is based on MRI findings. Whereas in the former, there is a clear demarcation of the zone of oedema, such a demarcation zone is absent in the latter. A subchondral fracture may occur in osteonecrosis and osteochondrosis dissecans as well as in BMOS.

We believe that the findings in our case are consistent with a BMOS, migrating from the medial to the lateral condyle.

There still is ongoing discussion about BMOS. The term 'transient bone marrow oedema syndrome' was first used in 1988 by Wilson *et al* (18) to describe patients with BMO without any signs of osteopenia on plain radiographs. When however there are signs of osteopenia visible on plain radiographs the pathology is described as 'localized transient osteoporosis' (LTO) which was first described by Curtis and Kinciad in 1959 (6). It is well known that transient osteoporosis may have a migrating presentation, migrating from one joint to another or shifting within a single joint, similar

to the migration pattern in our case. In recent literature, 63 reports of migratory transient osteo-porosis have been described, which have been reviewed by Cahir and Toms (4). Whether these entities are to be considered as different presentations of the same pathology, or as distinct pathologic entities, is still a matter of debate. We believe that the distinction between regional osteoporosis and transient BMOS is rather artificial and that they are both part of the spectrum of the same pathology.

Aigner *et al* (1) recently reported 8 cases of patients with intra-articular shifting BMOS of the knee; 25% showed signs of mild osteopenia, supporting the former statement that both BMOS and transient osteoporosis are features of the same pathology. The same goes for the series of Moosikasuwan *et al* (14) in which two out of five patients with shifting BMO of the knee showed signs of osteopenia, and one patient's plain radiograph could not be evaluated.

Another argument to support our opinion is that a significant amount of trabecular mineral loss must occur before it can be detected on plain radiographs (5). Therefore one can assume that there is an underestimation of the occurrence of osteopenia in the reported cases of BMOS, including ours (4,12).

The aetiology of the bone marrow oedema and especially the shifting from one condyle to another within the knee is still under discussion. It has been stated that the knee is confined to develop BMO in each condyle subsequently, because of weight shifting from one condyle to another as BMO sets in (12). This weight shifting can occur spontaneously because of the pain or due to bracing as in the case discussed here.

We treated our patient with peroral NSAID and partial weightbearing by means of an unloading brace. No reports where found in the literature on the use of unloading braces in the treatment of TBMO. The role of the unloading of the medial compartment, and thus the loading of the lateral compartment, in the shifting of the BME from one compartment to the other remains unclear. On one hand, Schweitzer *et al* (17) demonstrated that altered weightbearing may be a cause of BMO in the loaded compartment. On the other hand many cases of spontaneous shifting of BMO from one condyle

to the other over a period of 2-4 months have been reported in literature (1,4,12,14,19).

Most authors prefer a conservative treatment, consisting of partial weight bearing and oral administration of NSAID as was performed in the case here presented. After all it is a self-limiting disease. Corticosteroids have not been proven to alter the course of the disease (13). Intravenous administration of ibandronate, a nitrogen-containing bisphosphonate, was proven to be effective in the treatment of LTO in three patients (16). Both an increasing bone mineral density and a substantial pain relief were reported.

Surgical intervention by means of core decompression has been proposed and performed with good results (8). However, care must be taken to avoid fracture since osteopenia may already be present.

CONCLUSION

Bone marrow oedema remains a clinical and radiological enigma. Before the diagnosis of BMOS or LTO is established, other entities such as AVN and osteochondrosis dissecans must have been excluded. Shifting of BMO on follow-up MR examinations, with or without radiographic osteopenia, may suggest a migratory BMOS or migratory osteoporosis. We believe that both transient migratory BMOS and transient osteoporosis are part of the spectrum of the same pathology. However, further studies are needed in order to confirm this theory.

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