ACTA
ORTHOPAEDICA
et
TRAUMATOLOGICA
TURCICA

Acta Orthop Traumatol Turc 2010;44(4):340-343
doi:10.3944/AOTT.2010.2364

# Migrating bone marrow edema syndrome: a cause of recurring knee pain

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Bone marrow edema syndrome is a condition of unknown etiology, presenting with painful limping. It is characterized by normal radiographs, but magnetic resonance imaging findings change with bone marrow edema. When there is osteopenia in the radiographs, the condition is called transient osteoporosis. The term migratory bone marrow edema syndrome is used when there is involvement of another joint, or another compartment in the same joint, which typically occurs within 6 months of onset of primary symptoms. Here, a case of migratory bone marrow edema syndrome in a 47-year-old male patient, which was conservatively managed, is reported.

**Key words:** Alendronate; bone marrow edema syndrome; knee; magnetic resonance imaging; regional migratory osteoporosis.

Transient osteoporosis or bone marrow edema syndrome (BMES) is a condition of unknown etiology, which affects middle-aged men and women in the third trimester of pregnancy. Although the condition is of unknown etiology, many unproven theories have been proposed.

The term regional migratory osteoporosis (RMO) is used when there is transient osteoporosis with migratory features with involvement of another joint, which occurs usually within 6 months of the onset of primary symptoms.[1,2] RMO typically migrates to adjacent joints and most commonly affects the knee and ankle. Conservatism is the mainstay of treatment, but the duration of symptoms can be reduced by oral or intravenous bisphosphonates as per various reports.[1,3-5]

## Case report

A 47-year-old male general medical practitioner presented in the outpatient clinic with pain in the left knee of one month duration. He did not give any history of trauma. He was non-smoker, and there was no history of alcohol or drug abuse. He had tenderness over the femoral attachment of the medial collateral ligament. There was no laxity of the cruciates or collateral ligaments of the knee. He had full range of movements on presentation. The pain was progressive, and he developed antalgic gait.

His blood investigations including erythrocyte sedimentation, bone profile, liver function tests were normal. He was negative for rheumatoid factor and HLA B27. His initial radiographs were normal.

The patient presented back after few days with severe pain and restriction of flexion of the knee (0-100°). Computed tomography (CT) showed features of osteopenia (Fig. 1), with minimal increase in soft tissue shadow over the medial femoral condyle. The bone density was measured with Hounsfield units (HU units), which showed +39.7 values in the left

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**Submitted:** October 10, 2009 **Accepted:** April 6, 2010

medial femoral condyle as compared to +138.3 units in the right knee. Magnetic resonance imaging (MRI) of the left knee showed reduced signal intensity in T1W images and increased signal intensity on T2W images (Fig. 2a and 2b).

The patient was given alendronate (Fosamax 70 mg; Merck Serona, Middle East, Amman, Jordan) once weekly and diclofenac sodium (Olfen 100 mg capsules; Diclogesic Retard, Dar Al Dawa, Amman, Jordan) once daily. He stayed non-weight bearing on the left knee with a pair of axillary crutches. Patient improved over a period of eight weeks and started full weight bearing by three months. Since the symptoms resolved in 3 months, oral alendronate was stopped.

Pain started in the left knee after an asymptomatic period of 3 months. His radiographs of the knee were normal again. A repeat MRI showed almost normal left medial femoral condyle, but there were changes typical of BMES in the lateral femoral condyle (Fig. 3a and 3b) A diagnosis of migratory BMES syndrome was made, and the patient was managed conservatively as before, with oral alendronate continued for 6 months.

By 6 months patient returned back to normal with no pain on weight bearing. For being a physician, he refused further radiological studies. There was no recurrence of symptoms at one-year follow-up.

The patient was informed that data regarding his case would be submitted for publication.

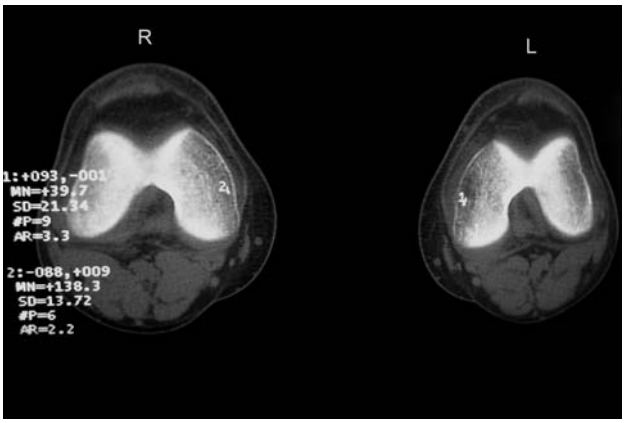


**Fig. 1.** Transverse CT images showing relative osteopenia of the medial femoral condyle in the left knee.

## Discussion

First described by Curtiss and Kincaid in 1959 as transitory demineralization of the hip in pregnancy, transient osteoporosis is a self limiting condition which resolves without leaving any residual effects.[5,6] Duncan et al.[7] in 1969 coined the term regional migratory osteoporosis for polyarticular variant of the disease. The term BMES was first used by Wilson et al.[8] in 1988. If there is no osteopenia, the diagnosis becomes transient BMES.[9,10] Both regional osteoporosis and RMO are part of the same spectrum. The term transient BMES is used when there is no radiological osteopenia. The condition, if there is radiological osteopenia, is called transient osteoporosis.

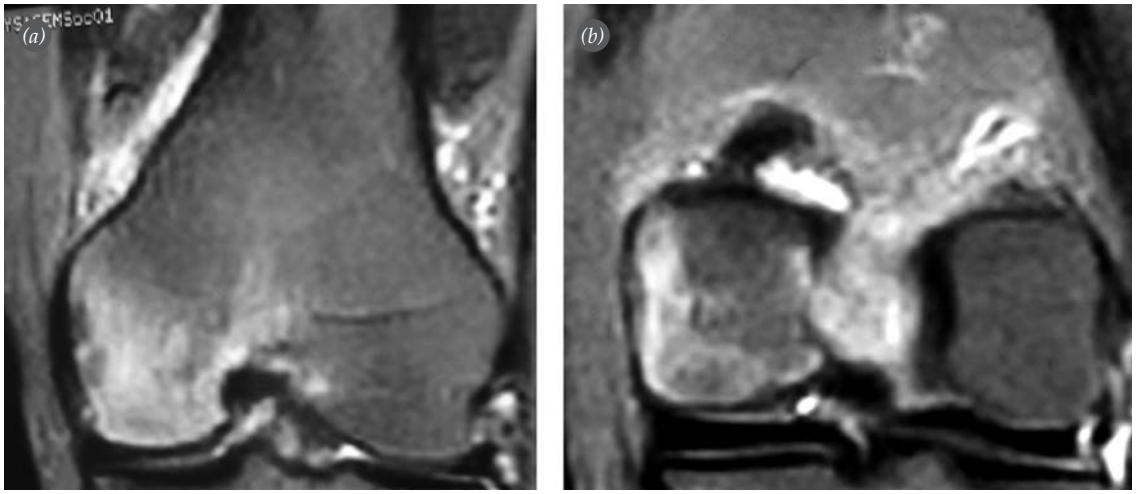


**Fig. 2.** **(a)** Coronal short-tau inversion recovery (STIR) and **(b)** transverse MR image showing bone marrow edema in the medial femoral condyle.

The disease is self-limiting in nature.[11] Symptoms are known to subside by 3-9 months.[11] Pain typically occurs on loading of the joint. Disease typically affects middle aged men (2/3 of the cases) and women in the third trimester of pregnancy.[7] It is characterized by spontaneous or gradual onset of pain, which is progressive over a period of several weeks. The condition improves even without treatment over a period of weeks to few months.

RMO predominantly involve the ankle, foot, and knee; and less commonly the hip.[2,8] It can migrate from one compartment in the joint to another.[8] Shifting of the lesion from one condyle to another could be due to pain on loading and spontaneous off loading of the compartment by the patient or off loading with help of bracing.[8]

BMES occurs with spontaneous onset of pain without prior history of trauma,[5,12] though there are reports of BMES occurring after a traumatic insult to the knee.[9,11] There may be joint line tenderness, soft tissue swelling, and effusion.[1] Recurrence of symptoms in an adjacent joint or intra-articular migration in the same joint with similar clinical and radiologic features suggests the diagnosis of RMO.[1] In most patients secondary joint involvement occurs soon after the primary joint is affected, usually within one year.[1]

Trauma has been documented as a causative factor in transient osteoporosis.[9,11,13] Microvascular injury causing tissue ischemia, bone marrow edema and cell death has been proposed as another cause.[6,11,14] BMES due to mechanical derangement resulting in abnormal loading of a compartment of the knee is limited to the subchondral bone, but doesn't explain the horizontal shifting of the lesion.[5] The theory of regional, diffuse and reversible ischemia of unknown etiology proposed by Aigner et al.[5] is reasonable. Neurogenic compression theory, obstruction to venous return resulting in local hyperemia, ischemic injury to the bone marrow are some of the etiological causes reported.[7] Ischemia is considered the most probable cause of transient BMES.[15] Ischemic phase is followed by reactive hyperemia and vasodilatation, and thereby increased intraosseous marrow pressure. This intraosseous elevated pressure produce the symptoms of the patients.[15]

The pathology could be vasomotor response similar to reflex sympathetic dystrophy (RSD),[9,11] but sensory symptoms are typically lacking and history of trauma may not present. Unlike in RMO, patients with RSD leave behind some permanent disability if not treated properly.[11]

Since the trabecular and cortical bone is spared, plain radiographs and CT scan are typically negative.[11] Radiological signs of osteopenia develop within few weeks, usually 4-8 weeks.[13] In the case report by El Masry et al.[9] CT scan showed marked cortical resorption of the distal femur. Focal demineralization is better appreciated as multiple focal lucencies in the CT than in the radiographs.[1]

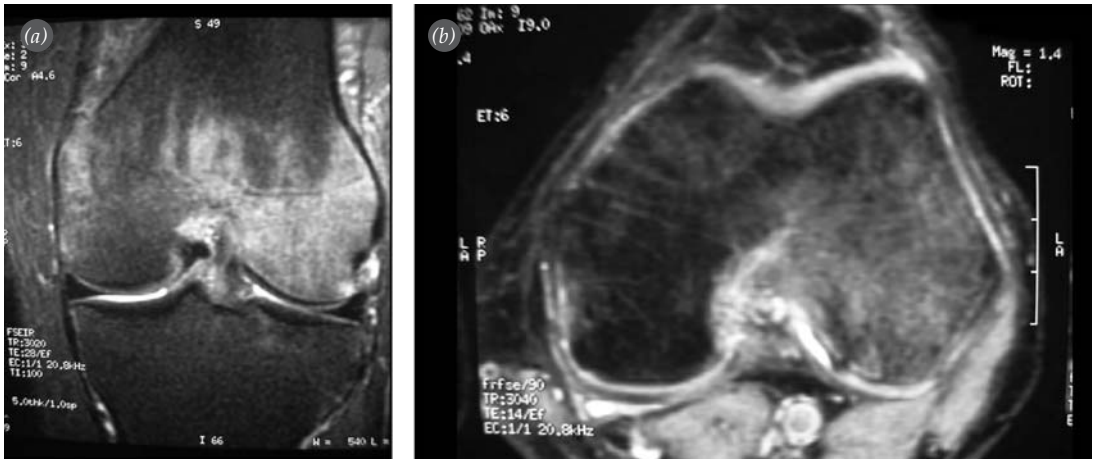


**Fig. 3.** **(a)** Coronal short-tau inversion recovery (STIR) MR image showing resolution of edema in the medial femoral condyle and development of edema in the lateral femoral condyle. **(b)** Transverse MR image showing bone marrow edema in the lateral femoral condyle.

BMES shows reduced signal intensity in T1W images and increased signal intensity on T2W images in MRI, with ill defined margins.[1,3,6] Term of transient BMES is used to describe any patient in whom a reversible bone marrow edema pattern is seen in the MRI.[13]

Well-defined demarcation zone typical of avascular necrosis is not found in transient osteoporosis.[14] The knee changes reported were mostly in the lateral condyle.[16]

Oral or intravenous bisphosphonates and calcitonin, and iloprost (vasoactive prostacyclin analogue) have been used by many authors with beneficial effects.[1,3-5] Core decompression that was reported to produce immediate pain relief by decompressing the raised intramedullary pressure, over treats a seemingly benign condition with natural regression in few weeks to months.[1,5-7,13,16]

One differential diagnosis thought of was spontaneous osteonecrosis of the medial femoral condyle.[17] Subchondral area of low signal intensity on T2W images are longer (>14 mm) and thicker (>4 mm) in early irreversible osteonecrosis.[13] The quantitative assessment helps in differentiation of the two lesions.[13] Focal underlying subchondral defects are seen in osteonecrosis, differentiating it from transient BMES, though diffuse changes in the signal intensity are more apparent in the subchondral location.[13] Signal intensity changes in BMES extent from the metaphysis to the epiphysis, whereas in neoplasm and osteomyelitis it is usually located in the metaphysis.[14] Moreover, soft tissue enhancement is more marked in neoplasm and infection.

As a conclusion, transient osteoporosis in the knee is a disease of exclusion. BMES is considered as the MRI equivalent of transient osteoporosis. Follow-up MRI scans are not required as the symptoms resolve in 3-6 months, but MRI may be performed to reassure the patient and surgeon.[6]

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