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Case Report

Case report: Treatment of bone marrow edema of femoral head with hyperbaric oxygen therapy

Tarun Sahni [a,*], *Shweta Gupta* [b]

[a] *Senior Consultant, Internal & Hyperbaric Medicine, Indraprastha Apollo Hospital, New Delhi 110076, India*
[b] *Clinical Research Manager, Hyperbaric Oxygen Unit, Indraprastha Apollo Hospital, New Delhi 110076, India*

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ABSTRACT

Bone marrow edema syndrome is a rare condition of unclear etiology that is characterized by hip pain, limited osteopenia on plain radiography, and characteristic MRI findings. Although the etiology and pathogenesis of bone marrow edema syndrome are not currently known, different mechanisms have been proposed, including microvascular injury, venous obstruction, secondary localized hyperemia, abnormal mechanical stress, metabolic causes, neurogenic compression, etc.

Hyperbaric oxygen therapy (HBO) appears to be effective in treating bone marrow edema syndrome, resulting in an accelerated recovery of hip function compared to pharmacological therapy alone.

The primary effects of HBO in bone marrow edema are threefold. Firstly, by improving oxygen tension in tissue fluids at the site of bone necrosis, which prevents further loss of ischemic bone. Secondly, HBO-induced vasoconstriction decreases edema allowing better perfusion to the injury site, and thirdly, by enhancing reparative process by providing an improved oxygen environment for osteoclastic function, neovascularization and osteogenesis.

Here, we report a case of bone marrow edema syndrome of hip in a 32-year-old male patient, which was conservatively managed.

1. Introduction

The term 'bone marrow edema' was first introduced in 1988 characterized by ill-defined areas of decreased signal intensity on T1-weighted MRI and increased signal intensity on T2-weighted images (Table 1).[1–3] Bone marrow edema syndrome (BMES) of the hip is discussed as a possible early reversible stage of avascular necrosis, but the incidence of progression from BMES to avascular necrosis is still unclear.[4]

BMES of the hip is a recently identified clinical entity that has been described as transient osteoporosis or algodystrophy hip affecting middle-aged men.[5] The most common site affected is the hip joint, with the left hip more frequently involved than the right. Bilateral involvement occurs in only 25–30% of cases. Up to 40% of patients have involvement of

* *Corresponding author*. Tel.: +91 9810038010; fax: +91 011 26823629.
E-mail address: aimhu@live.in (T. Sahni).

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Table 1 – MR features of BMES of the femoral head on SE T1-weighted images.

Location	At least one portion of femoral head
Outline	Blurred contour
Homogeneity	Homogeneous, no low/high signal intensity within the lesion on T1 WI
Signal intensity	Moderate

other joints, such as lower-extremity joints, upper-extremity joints, and the spine, with migration to multiple sites or recurrence seen in 25–50% of this population.[4–6]

Diagnosis is made by excluding other possible causes of hip pain. The clinical course is benign, and spontaneous recovery usually occurs over 2–12 months. All current treatment options are considered as symptomatic therapy since a causative influence on the pathogenetic vascular disturbance has not yet been reported.[6,7]

Because of the reversibility of the disease, treatment usually consists of avoiding load on the hips as well as the use of nonsteroidal anti-inflammatory drugs (NSAIDs), bisphosphonates, and prostacyclin and hyperbaric oxygen therapy (HBO), which can improve local hemodynamic characteristics.[8,9]

2. HBO helps in management of bone marrow edema by

1. Improving oxygen tension in tissue fluids at site of bone necrosis. This prevents further loss of ischemic bone cells.
2. Enhancing reparative process by providing improved oxygen environment for osteoclastic function, neovascularization, and osteogenesis (improving microcirculation).
3. Decreasing tissue edema.
4. Stimulating angiogenesis.
5. Decreasing intraosseous pressure within femoral head.

3. Case report

An Arabic national is a 32-year-old male, nonsmoking doctor, who presented to our hyperbaric medicine unit with primary complaints of pain around the left hip joint and difficulty in walking.

He had experienced a sudden onset of diffuse pain around the left hip joint for the past 4 months with no history of trauma or any other systemic illness. Pain was not accompanied by fever, skin rash, urethritis, etc. Pain was aggravated by walking and prolonged standing.

The pain caused the patient to alter his gait, but was able to walk with assistance. The visual analog scale (VAS) pain score at that time was 7–8 on a 10-point scale with 1 indicating the least pain and 10 indicating the worst pain. On physical examination, range of motion of the left hip was limited and painful in all ranges. Laboratory data and NCV/EMG findings were normal.

Blood work ruled out gout and X-ray hip revealed sclerosis with osteophytes seen in the right hip joint suggestive of degenerative changes but left hip appeared normal (Fig. 1). MRI

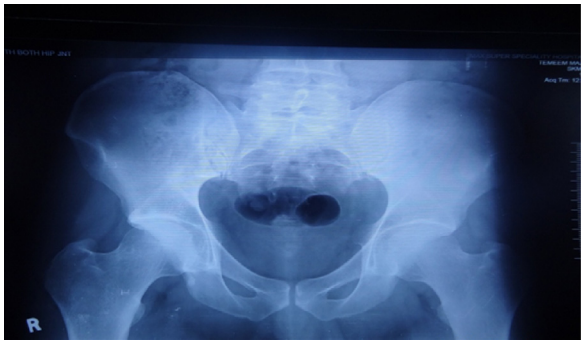

Fig. 1 – X-ray of pelvis.

reported underlying bone disorder 'early osteomyelitis with inflammatory changes' in left hip (Fig. 2).

He was advised to take analgesics, NSAIDs, and supplements to alleviate pain and reduce inflammation.

He was asked to repeat MRI after 8 weeks of this pharmacological regime. Following 4 weeks of therapy, his condition improved with mild to moderate pain on prolonged walking with a stick reducing to 5–6 on VAS. Orthopedic advised to continue the treatment and to repeat MRI after 4 weeks. MRI showed extension of inflammatory changes to involve >50% of head and >50% of neck of hip joint. MRI pelvis revealed marked 'bone marrow edema' at the femoral head.

He was advised for further investigations including bone and DEXA scan, which revealed avascular necrosis of femur head (Fig. 3) and mild osteopenia with slightly increased fracture risk, respectively.

He was then advised for HBO along with other conservative treatment. 50 sessions of HBO were given at 2.4 ATA for 90 min with 60 min of oxygen breathing. He showed good improvement with no pain on rest (VAS = 0); on walking, pain reduced to 2 on VAS and mild pain with VAS of 4–5 on heavy exertion. He walked without support and had improved general well-being.

After 2 months of treatment, MRI reported reduction in intensity of osteoblastic activity in head, neck, and trochanteric region of left femur suggesting good response to treatment (Fig. 4).

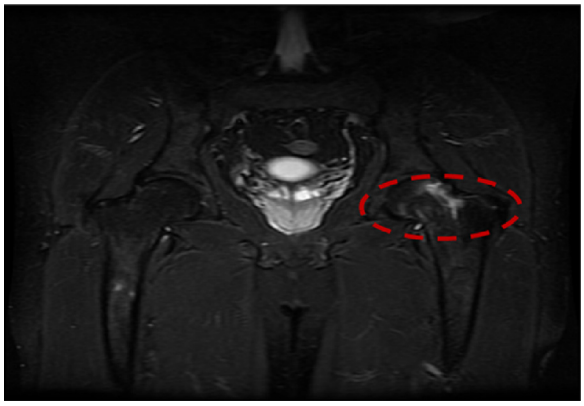

Fig. 2 – MRI of pelvis.