BONE-MARROW OEDEMA SYNDROME AND TRANSIENT OSTEOPOROSIS OF THE HIP

AN MRI-CONTROLLED STUDY OF TREATMENT BY CORE DECOMPRESSION

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Bone-marrow oedema syndrome (BMOS) of the hip gives a characteristic MRI pattern, in association with severe pain, non-specific focal loss of radiological density and a positive bone scan. In our MRI-controlled study, nine patients with non-traumatic BMOS in ten hips all had core decompression. Bone-marrow pressure measurements and intraosseous venography in five cases showed pathological values.

All patients had immediate relief of pain, with return of MRI signals to normal after three months. Regular review was continued for at least 24 months with serial clinical radiological and MRI assessment. At a mean follow-up of 33 months all patients remained free of pain with normal radiographs and MR scans. The histological evaluation of undecalcified sections obtained from eight core decompressions confirmed the presence of bone-marrow oedema, with necrotic and reparative processes involving bone and marrow similar to those of early avascular necrosis but with no evidence of 'osteoporosis'.

These findings support the assumption that BMOS may be the initial phase of non-traumatic avascular necrosis.

In most patients BMOS will have a self-limiting course, but the duration of symptoms may be reduced by core decompression treatment.

There have been several recent reports of patients with hip pain, focal loss of radiodensity, positive bone scans and the appearance of bone-marrow oedema on MRI, without the specific MRI signs of avascular necrosis (AVN). This syndrome has been described as 'transient marrow oedema syndrome' (Wilson et al 1988) and as 'transient osteoporosis' (Bloem 1988; Takatori et al 1991; Grimm et al 1991; Urbanski, De Lange and Eschenroeder 1991). All the authors regard it as a condition which regresses spontaneously after 6 to 12 months and needs no surgical intervention. Another group of authors, however, consider it to be an early phase of AVN which, without surgical treatment, may progress to full AVN with collapse of the femoral head (Mitchell 1989; Turner et al 1989; Hofmann et al 1991).

It is generally accepted that the change in MRI is caused by an increase in fluid in the marrow cavities of the femoral head. We therefore propose that the term 'bone-marrow oedema syndrome' (BMOS) be used for this condition. Its aetiology and possible role in the pathogenesis of AVN are poorly understood, but we are now able to report for the first time histomorphological verification of BMOS and the accompanying changes in bone and marrow.

PATIENTS AND METHODS

From 1986 to 1989, we diagnosed BMOS with no other specific signs of AVN in MRI scans of ten hips in nine patients. All had pain and a reduced range of movement at the hip, with plain radiographs showing a normal joint space. Some of the radiographs showed non-specific radiolucency. Bone scans with $^{99m}$Tc-MDP were carried...
out on six patients; all were positive, with the region of increased uptake corresponding to the extent of bone-marrow oedema shown by MRI (Table I). The MRI appearance of BMOS is non-specific, and it was necessary to exclude stress fracture, bone bruising, osteomyelitis, acute sickle-cell crisis and neoplasm (Vogler and Murphy 1988).

All nine patients were male, with an average age of 44 years (40 to 55). None had any disease definitely associated with AVN (Hungerford 1979), and none had received cortisone therapy or had a history of injury. Eight patients, however, had one or more risk factors for AVN, such as alcohol abuse, nicotine consumption, or hyperuricaemia (Hungerford 1979; Table I).

At the initial diagnosis and at the latest review, the hips of all patients were graded using the Harris hip score (Harris 1969). At these times and at each follow-up at 6- to 12-month intervals, standard anteroposterior and frog- legateral radiographs were made and staged according to Steinberg et al (1984). MRI was performed at all examinations, using a 0.5 Tesla imaging system (Gyroscan, Philips, and in some cases a 1.5 Tesla system (Magneton, Siemens, Germany) with a body coil in the coronal plane in spin echo technique. In six cases a surface coil with sagittal plane was also used. The T1-weighted images were obtained with four acquisitions (TR 550 to 700 ms and TE 15 to 30 ms) and the T2-weighted images with two acquisitions (TR 2000 to 2500 ms and TE 50 to 100 ms). Section thickness was 3 to 5 mm with a 0.5 mm interval and an imaging matrix of 256 × 256 mm. The MRI results were classified according to Mitchell et al (1989).

In all ten hips the pain was resistant to conservative therapy, and core decompression treatment was undertaken. During this we performed the functional exploration of bone as described by Ficat (1985), including measurements of bone-marrow pressure, intraosseous venography and bone biopsy using trephines ranging from 3 to 10 mm in diameter (Table I). We obtained bone cylinders suitable for histological evaluation in eight cases. The specimens were fixed in neutral buffered formaldehyde-ethanol solution and embedded without decalcification in methylmethacrylate. Longitudinal, 5 μm-thick sections were stained with Goldner's trichrome and Giemsa stains for microscopic examination. Microradiographs of consecutive 100 μm-thick ground sections were used for microdensitometry (Plenk 1986). The eight cases were categorised according to the four types of AVN proposed by Ficat (1985).

**RESULTS**

In all ten cases the typical appearance of BMOS was visible by MRI at the first examination (Figs 1 to 6). The T1-weighted images showed loss of signal intensity in areas in the head, neck and intertrochanteric region of the femur, but in no case was the acetabulum involved. The areas of low signal intensity were hypointense relative to the subcutaneous fatty tissue and to the bone marrow of the same contralateral area, and were well demarcated from the normal bone marrow in the distal part of the affected femur (Figs 1b, 1d and 5a). In the T2-weighted images, these areas showed a significant increase in signal intensity and were then isointense or hyperintense as compared with normal bone marrow. Six hips showed a second-degree joint effusion (Mitchell et al 1989) and the other four a third-degree effusion (Figs 1c, 5b and Table II). None of the hips had the specific MRI patterns of AVN (subchondral focal signal alterations surrounded by a ring of low signal intensity on all sequences) either in the coronal body-coil image or in the sagittal surface-coil image used in some cases.

In one patient (case 6) the contralateral hip had been treated by core decompression for radiological and

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**Table I. Details of nine patients with bone marrow oedema syndrome of the hip**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Side affected</th>
<th>Contralateral side</th>
<th>Risk factors</th>
<th>Duration of pain* (mth)</th>
<th>Follow-up (mth)</th>
<th>Trephine diameter (mm)</th>
<th>Number of MR scans</th>
<th>Bone biop.†</th>
<th>Functional exploration of bone†</th>
<th>Histology (Ficat type 1 to 4)</th>
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* before treatment † bone marrow pressure in mmHg in trochanter/neck/head of femur and venography result (+ or -) ‡ affected contralateral side of patient 9
histological AVN two years before the second hip was included in our study. In another patient with BMOS in one hip (case 9), the contralateral hip became involved 28 months later (case 10).

Table II. Comparison of clinical, radiological and MR findings before core decompression (Pre) and at latest follow-up (Post)

<table>
<thead>
<tr>
<th>Case</th>
<th>Harris score</th>
<th>Radiological stage</th>
<th>MRI classification*</th>
<th>Joint fluid (Grade 0 to 3)</th>
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<td>10</td>
<td>63</td>
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* E = bone marrow oedema, N = normal signal
† affected contralateral side of patient

Clinical symptoms had been present for an average of four months before the diagnosis was made (Table I). The Harris hip score before treatment was moderate in two hips and poor in eight. Radiologically, four hips were in stage I, and six in stage II, with a more or less pronounced loss of radiodensity of the femoral heads but a normal joint space (Fig. 1a). We could not distinguish between stages 0 and 1 because a bone scan had not been performed in all cases (Table II).

The bone cores from trephining, which had usually been broken into two or three portions during collection and preparation (Fig. 3), showed that the marrow was filled with a pale- to dark-staining homogeneous fluid, consistent with BMOS. There were fragmented necrotic fat cells and the remnants of haemopoietic marrow showed necrosis. In some areas haemopoietic or fatty marrow had been replaced by fibroblastic proliferation, a fibrous matrix, and new dilated vessels, suggesting an active repair process (Fig. 4).

The bone of the trabeculae appeared to be viable except in some areas in the central portions of enlarged trabeculae in which there were empty osteocyte lacunae. In all cases there were extended osteoid seams, mostly covered by active osteoblasts around the trabeculae (Fig. 4) though osteoclastic resorption was rarely found. By

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![Fig. 1a](image1.png)

Fig. 1a - Radiograph showing stage II changes in the right hip with loss of radiodensity of the femoral head but a normal joint space. Figure 1b - MRI with 0.5 Tesla T1-weighted image showing irregular areas of decreased signal intensity in the head, neck and intertrochanteric region of the right femur. There is no focal lesion specific for avascular necrosis and there is a low signal intensity at the fovea centralis (arrow). Figure 1c - T2-weighted image in the same plane showing irregular high signal intensity in the area corresponding to the regions of low signal intensity of Figure 1b. A grade 3 joint effusion is indicated by the high signal intensity surrounding the femoral head and neck (arrows). Figure 1d - T1-weighted image in the axial plane shows decreased signal intensity of the entire right femoral head. The left femoral head shows normal signal intensity and there is low signal intensity in both foveae centrales (arrows).
the Arlet and Durroux classification (Ficat 1985), one of the eight hips showed type 1 changes, three type 2 and four type 3 (Table I). The bone trabeculae had normal volume density and no signs of 'osteoporosis'. Quantitative microradiography, however, showed a distinct loss of hydroxyapatite content in all cases, as compared with healthy, age-matched control femoral heads.

In five cases, measurement of bone-marrow pressure showed pathological values and intrasosseous venography was also positive (Table I). Since the basic bone-marrow pressure was already elevated, no stress tests were performed.

All patients had complete relief of pain and restoration of a nearly normal range of movement within one week of core decompression. There were no post-operative complications. After six weeks of partial weight-bearing all the patients were able to return to work.

In eight hips, the first follow-up MRI was performed three months after surgery; all eight showed normal signals. The grade 3 joint effusions had diminished in all cases (Figs 2a, b, c and 6a,b). Further follow-up examination of all ten cases at 6- to 12-month intervals showed normal MRI signal intensity in all and the patients had remained pain-free and able to work.

The latest review of all the patients was made after an average of 33 months (Table I). All the hips showed a

Case 4. MRI with 0.5 Tesla after core decompression treatment. Figure 2a – T1-weighted image three months after core decompression showing a significant increase in the signal intensity of the right femoral head and neck compared with Figure 1b. No focal lesions are visible. The core track is clearly seen as an uneven area of low signal intensity. Figure 2b – T2-weighted image confirms the normal signal intensity of the entire right femoral head and neck. The core track shows high signal intensity, and there is a residual grade 2 joint effusion (arrows). Figure 2c – Three months after core decompression a T1-weighted image demonstrates the increase in signal intensity of the right femoral head in comparison with Figure 1d. The central core track shows a low signal intensity (arrow). Figure 2d – Twenty-six months after core decompression a coronal T1-weighted MRI of the right hip shows completely normal signal intensity in comparison with the left side. The core track is still visible, but shows signs of repair.

Case 4. Contact microradiographs from corresponding 100 μm-thick ground sections of the peripheral (a) and central (b) portions of the core biopsy. Structural damage can be seen in both portions. The aluminium-step wedge at the bottom is used for quantitative microdensitometry.
marked improvement in Harris hip score; all were excellent and seven scored 100 points. The average score had risen from 48 to 98 (Table II). Radiologically, the four hips originally in stage I remained unchanged, and all six stage II hips had improved to stage I (Table II). On MRI, all cases showed completely normal signals (Figs 2d, 6c).

DISCUSSION

The more or less evident focal loss of radiodensity in the affected femoral head and neck and its later spontaneous recovery in this syndrome prompted Hunder and Kelly (1968) to use the term 'transient osteoporosis'. Since MRI has been increasingly used to elucidate obscure skeletal disorders, a distinct type of localised marrow oedema has been repeatedly demonstrated by MRI in patients suffering from this syndrome (Bloem 1988; Grimm et al 1991; Hofmann 1991; Takatori et al 1991; Urbanski et al 1991). Because of this, Wilson et al proposed in 1988 that the term 'transient osteoporosis' be replaced by 'transient marrow oedema syndrome'. One year later, however, Turner et al (1989) showed that six patients with the MRI changes of BMOS were actually suffering from AVN. Other authors also described the typical BMOS pattern in cases of AVN (Hauzeur et al 1989; Robinson et al 1989; Seiler, Christie and Homra 1989). Despite this, most authors still believe that BMOS is a distinct self-limiting disease which is associated with the reflex sympathetic dystrophy syndrome (Lequesne 1968) and is often seen in the last trimester of pregnancy (Shifrin et al 1987). There is still controversy about the aetiology, pathophysiology and outcome of the disease.

Some studies of fully developed AVN of the femoral head have shown that the characteristic MR scans correlate well with the histological findings (Mitchell et al 1989; Seiler et al 1989), but we could find no previous histological correlation for BMOS. We were able to demonstrate increased interstitial fluid in the bone-marrow spaces, and also found bone and marrow changes

![Fig. 4a](image1.png)  
**Fig. 4a**  
Case 4. Undecalciﬁed microtome sections from the peripheral portion of the core biopsy. Figure 4a – Dark grey osteoid seams with the trabecular surfaces partly covered by active osteoblasts (OBL). Homogeneous material is seen in the marrow cavities, resembling marrow oedema (BMO), fat cell fragmentation (FF), or ﬁbrous marrow regeneration (FM) (trichrome Goldner stain, × 60). Figure 4b – Vital bone trabeculae are partly covered by active osteoblasts (OBL), leading to new bone formation (NB). The homogeneous extracellular material is now dark staining (basophilic), with marrow oedema (BMO) between fat cells and remnants of haemopoietic marrow (HM) (Giemsa stain, × 60).

![Fig. 5a](image2.png)  
**Fig. 5a**  
Case 2, preoperative findings. Figure 5a – MRI with 0.5 Tesla T1-weighted image, showing irregular loss of signal intensity in the right femur. Figure 5b – T2-weighted image showing irregular increase in signal intensity, with patchy hyperintensity as compared with the contralateral side. There is a grade 2 joint effusion (arrows).
which were similar to those of early AVN, as described by Ficat (1985) and others (Hungerford 1979; Hauzer et al 1989) in femoral heads with the typical focal signs of AVN. Other authors have presented histological evidence of early AVN in cases of radiologically proven 'transient osteoporosis' (Hunder and Kelly 1968; Dihlmann and Delling 1985) and we were able to prove AVN histologically in the seven cases with type 2 or type 3 changes.

Since there was no evidence of osteoporosis in our biopsies, the term 'transient osteoporosis' no longer seems appropriate. The abundant osteoid seams, the mineral
developing pathophysiology seems to be the repair
capacity of the femoral head; on this depends whether
restoration will take place or full necrosis develop
(Glimcher and Kenzora 1979).

The concept that BMOS can be transient does not
contradict our view that it may be an early phase of
AVN. The spontaneous healing after 6 to 12 months in
most cases indicates only that there has been limited
necrotic damage to the bone, and that the full develop-
ment of AVN was prevented by an adequate repair
mechanism (Rutishauser et al 1960; Glimcher and
Kenzora 1979). Spontaneous repair cannot always pre-

![Fig. 6a](image1)

![Fig. 6b](image2)

![Fig. 6c](image3)

Case 2. MRI with 0.5 Tesla, three months after core decompression. Figure 6a – T1-weighted image showing nearly normal signal intensity in the right femur. The core channel has different signal intensities in its peripheral and central parts. Figure 6b – T2-weighted image showing isointense signal intensity compared with the contralateral side. There are small residual signal changes in the subchondral area (arrow). Figure 6c – At 35 months, the T1-weighted image shows completely normal signal intensity in comparison with the left side. The core channel is no longer visible.

loss detected by quantitative microradiography, and the
marrow changes in our cases may be responsible for the
focal loss of radiodensity (Hofmann 1991).

The development of oedema of the bone marrow,
and the increased intramedullary pressure, both suggest
that the cause may be disturbed venous outflow, rather
than an interrupted arterial supply (Rutishauser, Rhoner
and Held 1960). The recently published canine model for
early AVN (Brody et al 1991) seems inappropriate for
idiopathic AVN, because acute ischaemia was used
rather than venous stasis. Furthermore, only decalcified
sections were investigated and it is not surprising
therefore that no BMO was observed. BMO appears to
be a feature of the initial phase of idiopathic AVN,
causong tissue necrosis on the one hand and increased
bone formation on the other. The crucial feature of the
vent this development and Turner et al (1989) confirmed
this when they reported three cases with BMOS which
developed AVN during follow-up.

The patients that we have reported did not differ in
symptoms, radiographs, bone scans or MRI from those
with 'transient osteoporosis' presented by other authors.
The unusual feature was that continued symptoms led us
to perform core decompression which we and others
routinely use to treat early cases of AVN. The clinical
results of core decompression in our patients were
excellent; they all had immediate relief of pain. Sudden
relief of pain after core decompression in early AVN has
been well described (Hungerford 1979; Ficat 1985; Seiler
et al 1989), and the relationship between elevated bone-
marrow pressure and pain has been established by
Lemperg and Arnoldi (1978). Our finding that the MRI
signals returned to normal at the first follow-up examination confirms that BMO has completely disappeared after three months, and we therefore assume that the bone-marrow pressures have also become normal. The disappearance of the joint effusion without puncture of the capsule suggests that this too was secondary to the elevated bone-marrow pressure.

At latest follow-up only three patients complained of slight pain after unusual physical activity (Table II), but none had any other hip symptoms. Both plain radiographs and MRI continued to confirm the lack of progression of the disease and the adequate repair capacity of the femoral head. The 'residual stage' MRI appearance, with subchondral fibrotic or necrotic areas, which has been described after conservative treatment of 'transient osteoporosis' (Grimm et al 1991), was not found in our cases. This provides a further indication of the therapeutic effect of core decompression.

The use of different sizes of trephine for core decompression did not influence our results. The larger 8 mm trephines provided us with excellent histological material, but 3 mm trephines seem large enough for decompression in BMOS. Their use would largely eliminate the risk of fracture or fragmentation of the femoral head which is described as a rare, but possible, complication of this operation (Learmonth, Maloon and Dall 1990).

We have to admit that we operated on cases which would probably have improved under conservative treatment, but usually only after such long periods as 6 to 12 months (Grimm et al 1991). Since the average duration of clinical symptoms was dramatically reduced by this surgery, we advocate the use of core decompression for non-traumatic BMOS of the femoral head, after the exclusion of the other differential diagnoses. We consider that the risk of progression to full AVN with collapse of the femoral head after conservative treatment is considerably greater than the minimal surgical risk of core decompression.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

REFERENCES


