International Orthopaedics (SICOT) (2000) 24:88–91

ORIGINAL PAPER

E. Calvo · D. Fernandez-Yruegas · L. Alvarez

Core decompression shortens the duration of pain in bone marrow oedema syndrome

Accepted: 5 January 2000

Abstract We studied nine patients with 12 painful hips without apparent cause but with alteration of signal intensity on magnetic resonance imaging (MRI) consistent with bone-marrow oedema. The patients were randomly assigned to receive conservative or surgical treatment with core decompression. The duration of pain was significantly less in those treated surgically. Histological evaluation of the material obtained from 4 decompressions confirmed bone-marrow oedema without osteoporosis. Bone mineral density studies in 5 patients were normal. Although bone-marrow oedema of the femoral head is usually a self-limiting condition, we suggest that core decompression should be considered, as the symptoms may be prolonged and incapacitating.

Résumé Etude de 12 hanches douloureuses chez 9 patients, sans cause apparente avec une modification du signal RMN compatible avec un oedème de la moelle osseuse. Les patients ont eu, de façon aléatoire, soit un traitement conservateur, soit un forage chirurgical. La durée de la période douloureuse a été significativement plus courte chez les patients opérés. L'étude histologique du matériel obtenu par les 4 forages confirmait la présence d'oedème de la moelle sans signe d'ostéoporose. Le syndrome d'oedème de la moelle osseuse a, habituellement, une durée d'évolution limitée, mais nous proposons néanmoins la pratique du forage puisque, parfois, les symptômes sont prolongés et incapacitants.

Introduction

There are many reports of middle-aged patients with joint symptoms, without a recognisable cause, focal loss of radiodensity on plain X-ray, positive bone scans, and the appearance of bone-marrow oedema on MRI without signs of osteonecrosis. In 1959 Curtis and Kincaid [4] indicated the transient course of the osteopenia and this condition has been named "transient osteoporosis" [11], although it has been given many other names [12]. X-ray signs are variable but the MRI shows an increase in the fluid content of the bone-marrow and the term "bone-marrow oedema syndrome" has been proposed [18]. Most authors report spontaneous regression and various methods of non-operative treatment have been suggested [8, 12]. Some, however, consider it to be an early stage of avascular necrosis, which may progress to collapse of the femoral head [6, 9, 13, 15–17]. As the symptoms may be severe and persistent surgery may be considered and we report the results of core decompression as compared with those of conservative treatment.

E. Calvo (✉) · D. Fernandez-Yruegas · L. Alvarez
Department of Orthopedic Surgery, Fundación Jiménez Díaz,
Universidad Autónoma, Av. Reyes Católicos, 2,
28040 Madrid, Spain
e-mail: ecalvo@fjd.es;
Tel.: +34-91-549 4152, Fax: +34-91-549 4764

Patients and methods

We studied 12 symptomatic hips in 9 male patients with a mean age of 40.3 (36–52) years. No patient suffered from metabolic, neoplastic or inflammatory disorders, and none had a previous history of trauma or steroid therapy.

Plain X-rays and routine full haematological screening were performed in all patients; 5 had bone scans and 5 had bone mineral density studies. The diagnosis was confirmed on MRI scan and the patients were randomly allocated using the statistical package Epi Info v 6.o (CDC Atlanta, Georgia, 1996) to receive either medical or surgical treatment. Core decompression was performed using trephines 8 to 10 mm in 7 hips. In one patient (Case 3), the contralateral hip had been managed non-operatively one year prior to surgical treatment. Bone cylinders suitable for histological examination were obtained in 4 cases. The specimens were fixed in neutral buffered formaldehyde-ethanol solution and embedded in methylmetacrylate without decalcification. Longitudinal sections, 5 µ thick, were stained with Goldner's trichrome and Giemsa stains for microscopic examination. The 5 remaining affected joints were managed conservatively with protected weight bearing and nonsteroideal anti-inflammatory medication.

Symptoms were correlated with MRI changes before and after treatment. Pain was graded as 0 (no pain), 1 (pain on walking), 2 (pain on weight bearing), 3 (pain on hip movement), and 4 (pain at rest). The Mann-Whitney test was used for statistical analysis.

Table 1

Case	Sex Age	Site of lesion	Underlying disorders	X-ray	Bone scan	BMD[a]	Treatment	Severity of pain before treatment	Duration of pain before treatment (months)	Duration of pain after treatment (months)	Follow-up (months)
1	M 52	Hip	–	Normal	+	Normal	Conservative	3	4	8	58
2	M 38	Hip	–	Normal	+	Normal	Core decompression	3	2	0.5	56
3	M 39	Hip bilateral	Obesity	Normal		Normal	Conservative (right hip)	4	1	7	33
							Core decompression (left hip)		1	8	34
4	M 38	Hip	–	Osteopenia		Normal	Core decompression	3	1	1	21
5	M 38	Hip bilateral	–	Normal	+		Conservative	3	0.5 (right hip) 0.5 (left hip)	5 (right hip) 4 (left hip)	25 (right hip) 20 (left hip)
6	M 48	Hip, knee	–	Normal	+		Core decompression	2	1 (hip)	0.5 (hip)	19 (hip)
7	M 36	Hip bilateral	–	Osteopenia (right hip) Normal (left hip)		Normal	Core decompression	3	4 (right hip) 1 (left hip)	0.5 (right hip) 1 (left hip)	15 (right hip) 12 (left hip)
8	M37	Hip	–	Normal			Core decompression	3	4	0.5	7
9	M 37	Hip	–	Osteopenia	+		Conservative	3	2	5	14

[a] Bone mineral density in the femoral neck

Results

Increasing hip pain was the commonest presenting symptom. 6 also had a decreased range of movement. Laboratory tests were always normal. Osteopenia was only seen on X-ray in 3 patients with a long history of pain (Fig. 1). All bone scans showed increased uptake. Bone mineral density studies showed values within normal limits for the Spanish population [5]. The initial MRI study demonstrated changes of bone-marrow oedema in all patients. Spin-echo T1-weighted images showed loss of signal intensity in areas of the head, neck and intertrochanteric region of the femur. On gradient-echo T2 weighted and inversion-recovery images, this abnormal area displayed relatively high signal intensity (Fig. 2). The acetabulum was not involved in any case and none of the hips had the characteristic changes of avascular necrosis. A joint effusion was identified in all cases, but there was no evidence of erosion of bone, cartilaginous defects, or synovial masses.

Nine cases had pain with hip movement. Case 6, which was surgically treated, had pain only when weight-bearing and walking, and case 3, which had one hip managed surgically and the other conservatively, presented with pain at rest. Six cases had complete relief of pain and restoration of a normal range of movement within one month of core decompression, and the remaining patient had only mild thigh pain on walking 8 months after surgery. In those patients treated conservatively the intensity of pain diminished gradually. Symptoms had been present before treatment for an average of 1.7 months (SD 2.8) in the cases undergoing surgery compared with 1.6 months (SD 1.5) for those treated conservatively. This difference is not significant (P=0.43). The mean duration of pain was significantly shorter after decompression than it was after non-operative management: mean 2 months (SD 1.4) compared with 5.8 months (SD 1.6) (P=0.03). The total period of pain, before and after treatment was also significantly shorter (P=0.048) in the patients treated surgically: mean 3.7 months (SD 2.6) when compared with those treated conservatively: mean 7.4 months (SD 2.9). There were no post-operative complications. MRI scans performed three months after surgery showed the core tracks with complete resolution of effusion and marrow changes (Fig. 3). Histological study of the bone cores showed increased fluid with fat necrosis and fragmentation. In some cases there was fibrovascular proliferation. The trabecular bone was viable except in some areas where the osteocyte lacunae were empty. Bone trabeculae showed increased osteoid tissue, mostly covered by active osteoblasts around the trabeculae, indicating increased bone formation. The bone volume intensity was normal with no evidence of osteoporosis.

Discussion

Transient osteoporosis is characterised by joint pain of gradual onset and radiographic osteopenia without asso-

Fig. 1 *Case 8*. Osteopenia and cortical thining of the right hip 4 months following onset of symptoms. The cartilage space is normal

Fig. 2 *Case 7*. Images obtained 6 weeks after core decompression of the right hip and 4 weeks after the onset of pain in left hip. **a** T1-weighted scan shows heterogeneously decreased marrow signal intensity from the head and neck of the left femur. Decompression tracks in the right femur. **b** Increase in the same areas on a gradient-echo T2-weighted image

Fig. 3 *Case 7*: 5 months after decompression of the right hip and 3 months after decompression of the left hip a T1-weighted MRI scan reveals normal signal intensity. The core channels are still visible in the left femur

ciated trauma, immobilisation, sepsis or inflammatory or metabolic disorders.

The hip is the commonest joint affected, but the knee, ankle or foot may be involved [3, 12, 18]. Laboratory values are usually normal, although a slight increase of erythrocyte sedimentation rate has been reported [12]. Transient bone-marrow oedema can occur at any age, but most often occurs in women during the third trimester of pregnancy [4] and middle aged men [2,8]. Osteopenia with preservation of the joint space may be seen on X-ray, and this becomes apparent shortly after the onset of symptoms [2, 7, 8, 12]. Bone scintigraphy demonstrates intense, homogeneous uptake, before X-ray changes are seen [7]. CT scans show loss and thinning of trabeculae [3].

Prior to the availability of MRI, conventional radiography, CT and bone scanning were the only methods available to visualise disorders of bone-marrow. These however, provide limited information. X-rays show mineralised bone, much trabecular bone has to be lost before changes are seen on X-ray or CT scan. Bone scans are more sensitive but lack anatomical detail and the specificity is low. The early diagnosis of this syndrome is thus difficult and must be distinguished from other conditions, specifically avascular necrosis. The delay in diagnosis in the patients in this series illustrates the diagnostic difficulties. The appearance of transient osteoporosis of the hip on MRI was first described by Bloem [1], and was confirmed by others [10, 14]. A few reports have described the MRI characteristics in the knee [7, 18] and foot [3]. The T1-weighted images show loss of signal intensity and the T2-weighted images reveal matching high signal intensity in the marrow. An effusion is commonly present. The abnormal intensity extends from the femoral head and neck to the intertrochanteric region (Fig. 3). Wilson et al. [18] attributed these changes to bone-marrow oedema as the characteristics of the signal intensity are consistent with fluid within the normal fatty marrow. Osteoporosis is rarely seen histologically in these patients [6, 10, 18] and the histological features are also of oedema. We did not find a decrease in bone mineral density in these patients, and for these reasons, we suggest that the term “transient osteoporosis” be replaced by “bone-marrow oedema syndrome”. The etiology, pathophysiology, treatment and outcome in these patients remains controversial. As spontaneous resolution normally occurs, conservative treatment is recommended. Protected weight-bearing and non-steroidal anti-inflammatory medication is the preferred treatment for pain relief [2, 8, 12]. Some authors have suggested that

bone-marrow oedema syndrome may be an early, reversible form of avascular necrosis [6, 9, 10, 13, 15–17]. Based on this concept and because the course of the condition may be as long as 45 months [12], core decompression has been advised [10]. The clinical results of core decompression in our patients have been excellent; it is a simple procedure that can be performed on an outpatient basis, and shortens the duration of symptoms and decreases the possibility of progression to avascular necrosis.

Acknowledgements The authors wish to thank Dr. Félix Manzarbeitia (Department of Histopathology), and Dr. Juan J. Granizo (Department of Statistical Analysis), from the Fundación Jiménez Díaz, for his technical assistance.

References

1. Bloem JL (1988) Transient osteoporosis of the hip: MR imaging. Radiology 167:753–755
2. Bramlett KW, Killian JT, Nasca RJ, Daniel WW (1986) Transient osteoporosis. Clin Orthop 222:197–202
3. Calvo E, Alvarez L, Fernández-Yruegas D, Vallejo C (1997) Transient osteoporosis of the foot. Bone-marrow edema in 4 cases studied with MRI. Acta Orthop Scand 68: 577–580
4. Curtiss PH, Kincaid WE (1959) Transitory demineralization of the hip in pregnancy. J Bone Joint Surg [Am] 41:1327–1332
5. Díaz Curiel M, Carrasco de la Peña JL, Honorato Pérez J, Pérez Cano R, Rapado A, Ruiz Martínez I on behalf of the Multicentre Research Project on Osteoporosis (1997) Study of bone mineral density in lumbar spine and femoral neck in a spanish population. Osteoporosis Int 7: 59–64
6. Dihlmann W, Delling G (1985) Ist die transitorische Hüftosteoporose eine transitorische Osteonekrose? Z Rheumatol 44: 82–86
7. Glockner JF, Sundaram M, Pierron RL (1988) Radiologic case study. Transient migratory osteoporosis of the hip and knee. Orthopedics 21: 594–596
8. Guerra JJ, Steinberg ME (1995) Distinguishing transient osteoporosis from avascular necrosis of the hip. J Bone Joint Surg [Am] 77:616–624
9. Hauzeur JP, Pasteels JL, Schoutens A, Hinsenkamp M, Appelboom T, Chochard I, Perlmutter N (1989) The diagnostic value of magnetic resonance imaging in non-traumatic osteonecrosis of the femoral head. J Bone Joint Surg [Am] 71: 641–649
10. Hofmann S, Engel A, Heuhold A, Leder K, Kramer J, Plenk H (1993) Bone-marrow oedema syndrome and transient osteoporosis of the hip. An MRI controlled study of treatment by core decompression. J Bone Joint Surg [Br] 75:210–216
11. Hunder GG, Kelly PJ (1968) Roentgenologic transient osteoporosis of the hip. A clinical syndrome? Ann Intern Med 68: 539–552
12. Lakhanpal S, Ginsburg WW, Luthra HS, Hunder GG (1987) Transient regional osteoporosis. A study of 56 cases and review of the literature. Ann Intern Med 106:444–450
13. Mitchell DG (1989) Using MR imaging to probe the pathophysiology of osteonecrosis. Radiology 171:25–26
14. Potter H, Moran M, Schneider R, Bansal M, Schreiman J (1992) Magnetic resonance imaging in diagnosis of transient osteoporosis of the hip. Clin Orthop 280: 223–229
15. Robinson H, Hartleben P, Lund G, Schreiman J (1989) Evaluation of magnetic resonance imaging in the diagnosis of osteonecrosis of the femoral head: accuracy compared with radiographs, core biopsy, and intraosseous pressure measurements. J Bone Joint Surg [Am] 71: 650–653
16. Seiler JG, Christie MJ, Homra L (1989) Correlation of the findings of magnetic resonance imaging with those of bone biopsy in patients who have stage-I or II ischemic necrosis of the femoral head. J Bone Joint Surg [Am] 71: 28–32
17. Turner DA, Templeton AC, Selzer PM, Rosemberg AG, Petasnick JP (1989) Femoral capital osteonecrosis: MR finding of diffuse marrow abnormalities without focal lesions. Radiology 171:135–140
18. Wilson AJ, Murphy WA, Hardy DC, Totty WG (1988) Transient osteoporosis: transient bone-marrow edema? Radiology 167:757–760