LETTERS TO THE EDITOR

Idiopathic osteonecrosis of the hip in twins

The pathogenesis of osteonecrosis is not completely understood.¹⁻³ Circulatory impairment leading to bone necrosis is one of the principal features. Many clinical disorders have been associated with osteonecrosis but in most cases a specific pathogenic mechanism remains unclear. Several mechanisms may explain the interruption of the blood supply such as, embolism and thrombosis, mechanical vascular interruption, injury to or pressure on vessel walls and venous occlusion.⁴ A minority of patients exposed to the same risk factor, such as high dose steroid therapy, will develop an osteonecrosis of the hip.⁹ Familial cases of idiopathic necrosis may give new insight into the pathogenesis of osteonecrosis. We report idiopathic osteonecrosis of the hip in homozygous twin brothers. This condition in twins has not been reported previously.

Patient 1, a 25 year old white electrician, was admitted to hospital in June 1988 because of persistent right hip pain over a period of six months. Radiographs of the right hip (fig 1) showed stage III osteonecrosis; the left hip was normal. MRI of the left hip showed a dark band of hypointensity surrounding a zone of high signal intensity (fig 2). Core biopsy revealed typical features of advanced osteonecrosis. Investigations failed to find any risk factors for the disease. HLA typing was A1, A2, B27, B40. An increase in right hip pain required total hip arthroplasty at the end of 1988.

Figure 1 Radiograph of the pelvis of patient 1 showing stage III osteonecrosis of the right hip, with normal aspect of left hip.

Figure 2 MRI of patients 1. Frontal view. T1 sequence TR 44, TE 15. Confirmation of osteonecrosis of right hip. Band of hypointensity of left hip.

Figure 3 Radiographs of right and left hip of patient 2 showing osteonecrosis stage II left hip and stage III right hip.

Patient 2, twin brother of patient 1, was a railway employee. He had bilateral hip osteonecrosis which was diagnosed in March 1989. Radiographs of the pelvis showed stage 2 for the left hip and stage 3 for the right hip according to the classification by Ficat and Arlet⁵ (fig 3). He experienced pain from both hips six months and one year later. His past medical and social history was uneventful. No risk factor was found. HLA typing was HLA A1, A2, B27, B40. A core decompression of the left hip was performed which revealed histological lesions typical of aseptic osteonecrosis. Because of persistent pain and functional impairment, the patient had bilateral hip arthroplasty in October 1990 and February 1991. Both patients had normal serum lipids, serum uric acid, liver tests, renal function, normal haemoglobin electrophoresis. Both patients denied alcohol consumption. Repeat gamma glutamyl transferase test were normal.

There have been no previous reports of familial idiopathic osteonecrosis. Itoman⁶ described homozygous twin sisters with systemic lupus erythematosus who developed bilateral hip osteonecrosis. Both sisters had received high doses corticosteroid therapy. In our patients, no risk factors were identified that could have triggered osteonecrosis. Neither patient had an abnormal stature, joint laxity or hip dysplasia and they were not involved in heavy work, or trauma before osteonecrosis was diagnosed.

In population studies eight to 20% of patients with osteonecrosis do not have identifiable risk factors.⁷ In patients exposed to classic risk factor such as high dose steroid therapy only a small percentage develop symptomatic osteonecrosis.^{1,10,11}

In our patients the radiographs and MRI demonstrated classic osteonecrosis. Histological examination showed typical signs of trabecular necrosis with new bone formation on dead trabeculae, suggesting a normal repair process in these patients. As no examination of the arterial network of the femoral head was performed, the possibility of an abnormal vascular distribution to the femoral head remains undefined.

In conclusion, our observation of spontaneous osteonecrosis in identical twin

brothers suggest a genetic predisposition in the pathogenesis of idiopathic osteonecrosis. Further familial survey of such patients is needed to confirm a genetic predisposition in cases of idiopathic aseptic osteonecrosis of bone.

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