Idiopathic osteonecrosis of the hip in twins

The pathogenesis of osteonecrosis is not completely understood. 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. A minority of patients exposed to the same risk factor, such as high dose steroid therapy, will develop an osteonecrosis of the hip. Familial cases of idiopathic necrosis may give new insight into the pathogenesis of osteonecrosis. We report idiopathic osteonecrosis of the hip in homozous 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 hyposignal 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.

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

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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