AVASCULAR NECROSIS OF THE FEMORAL HEAD IN HIV-INFECTED PATIENTS

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Osteonecrosis of the femoral head is uncommon and may be associated with other conditions, including hypertriglyceridaemia (Lee, Corcoran and Parson 1980) and, less often, the presence of antiphospholipid antibodies (Asherson et al 1989). We report two cases in HIV-infected patients, in whom hypertriglyceridaemia and anticardiolipin antibodies were found respectively.

Case 1. A 26-year-old man, an HIV-seropositive drug addict, gave a 20-month history of groin pain and recent difficulties in walking. He was at stage III of the disease (Center for Disease Control 1986). He had no history of trauma, alcohol abuse, steroid intake, or other chronic infection. Movements of his left hip were limited and radiography showed a typical osteonecrosis of the left femoral head. MRI showed a normal right femoral head. His total lymphocyte count was $1600/\text{mm}^3$ with 250 T4 cells/ mm^3 . Hypertriglyceridaemia ranged from 2.27 to 2.30 mmol/l (normal, 0.70 to 1.70). Levels of serum cholesterol, fasting glucose, and uric acid were normal. Electrophoresis of haemoglobin showed no abnormality. The patient was treated by left total hip replacement, which was still satisfactory after a two-year follow-up.

Case 2. A 27-year-old man, also an HIV-seropositive drug addict, gave a two-year history of right groin pain, and increasing difficulty in walking. There was no history of trauma, alcohol abuse, or corticosteroid therapy; he also was at stage III of the disease.

Hip movement was restricted, and osteonecrosis of the right femoral head was evident on radiographs. Early osteonecrosis of the left femoral head was also detected both by $^{99\text{m}}\text{Tc}$ bone scan and CT scans. His total lymphocyte count was $3200/\text{mm}^3$ with 960 T4 cells/ mm^3 . Anticardiolipin antibodies (IgG) were present at 126 IU/l (normal, < 20 IU/l). Haemoglobin electrophoresis was normal. The patient was successfully treated by right

total hip arthroplasty with no further complications at one-year follow-up.

Discussion. Aseptic osteonecrosis is probably an ischaemic disease (Lee et al 1980). Although no specific aetiological factor can be identified in most patients, many precipitating factors or conditions have been identified. These include local trauma, barotrauma, chronic alcoholism, sickle-cell anaemia, systemic lupus erythematosus, steroid therapy, and haemodialysis (Ficat 1985). Known metabolic risk factors include hypertriglyceridaemia (Lee et al 1980), present in case 1, and anticardiolipin antibodies (Canoso, Zon and Groopman 1987), found in case 2.

Hypertriglyceridaemia is more common in HIV-infected patients than in the seronegative population and is found in asymptomatic subjects, which suggests that it is due to HIV itself (Grunfeld et al 1989). Low levels of anticardiolipin antibodies are frequently detected in HIV-infected patients (Canoso et al 1987). Antiphospholipid antibodies are a well-recognised risk factor for venous and arterial thrombosis, and an association between osteonecrosis and the so-called 'primary' antiphospholipid antibody syndrome has been reported (Asherson et al 1989).

We conclude that osteonecrosis of the femoral head may be linked to HIV-associated metabolic and immunological disturbances. HIV infection must therefore be added to the list of risk factors for avascular bone necrosis.

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