Idiopathic chondrolysis of the hip

A case report

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Summary

A report of a case of idiopathic chondrolysis of the right hip in a 13-year-old Black girl is presented. Radiographs and microbiological and biochemical investigations excluded all other diseases. Cartilage and synovium were removed at biopsy of the right hip. Macroscopic and histopathological studies showed the typical features of idiopathic chondrolysis. Transmission electron microscopy confirmed the histological findings. Scanning electron microscopy highlighted the irregular pitted and fibrillated surface of the cartilage. Immunofluorescent studies on cartilage and synovium were negative. The possible pathogenesis of the disease is discussed, but the cause still remains unknown.

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Idiopathic chondrolysis was first reported by Jones[1] in 1971, when 9 cases of chondrolysis of the hip joint, not associated with slipped capital femoral epiphysis or trauma, were described. However, a possible aetiological factor — prolonged immobilization — was present in 2 cases. Since then Golding,[2] Heppenstall *et al.*[3] Moule and Golding,[4] Wenger *et al.*,[5] Duncan *et al.*[6,7] and Sivanantham and Kutty[8] have all reported cases of idiopathic chondrolysis of the hip.

Case report

A 13-year-old pubertal Black girl from Natal was referred from an outlying hospital 8 months after the insidious onset of a limp and pain in the right hip. There was no history of trauma and no signs of systemic illness, and none of the other joints were affected. Examination of the hip revealed a fixed flexion deformity of 40°, a flexion range of 40 - 80°, no adduction, an abduction deformity of 40°, 10° internal rotation and 10° external rotation.

Radiographs of the pelvis showed a decreased joint space of the right hip, peri-articular osteoporosis, blurring of the femoral head and acetabular margin and an increase in the diameter of the femoral head and neck, as well as a mild degree of protrusio. The lateral radiograph of the hip showed no evidence of a slipped capital femoral epiphysis (Figs 1 and 2). Radiographs of the chest and all other joints were normal.

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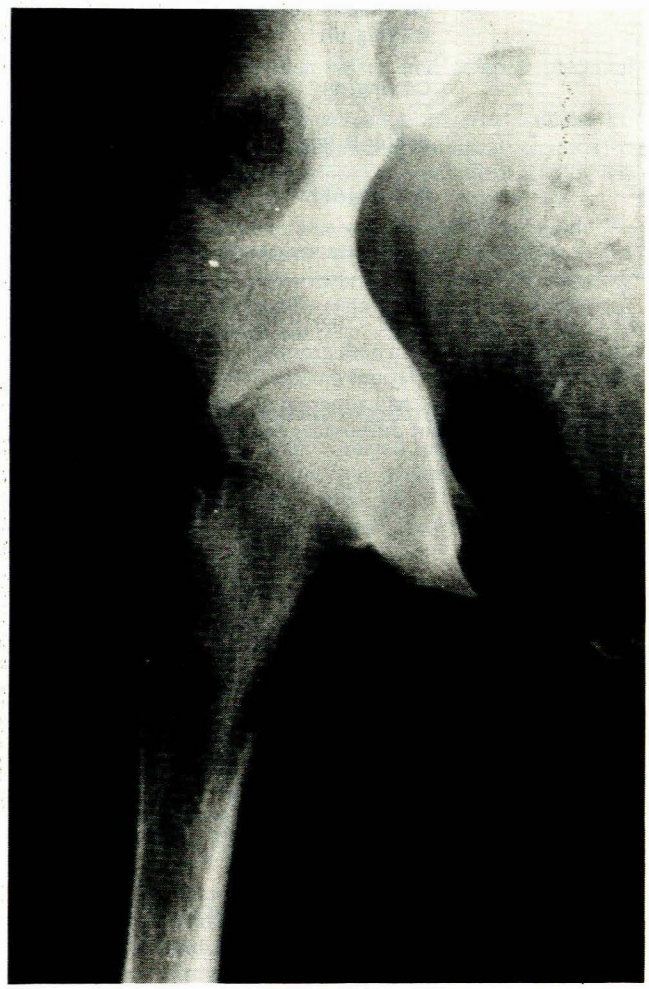

Fig. 1. The initial radiograph of the right hip showing the features of idiopathic chondrolysis.

The following laboratory tests were performed: haemoglobin estimation, white cell and platelet counts and determination of the ESR and C-reactive protein, rheumatoid factor, antinuclear factor and anti-DNA levels; reactive protein reagin, fluorescent treponemal antibody and gonococcal complement fixation tests; typhoid, *Brucella*, *Rickettsia* and *Yersinia* antibody tests; examination of the sputum for acid-fast bacilli; determination of the urea, electrolyte, glucose, uric acid, calcium, phosphate, cholesterol, bilirubin (total and direct), alkaline phosphatase, aspartate transaminase, alanine transaminase, γ-glutamyl-transferase, total protein, albumin, globulin, IgG, IgA, IgM and C3 levels; protein electrophoresis; analysis of urinary protein and 17-ketosteroids, estimation of 17ß-oestradiol, luteinizing hormone, follicle-stimulating hormone, cortisol and salicylate values; and tests for suspected haemoglobinopathies. All results were uniformly normal or negative. The patient remained apyrexial throughout her stay in hospital.

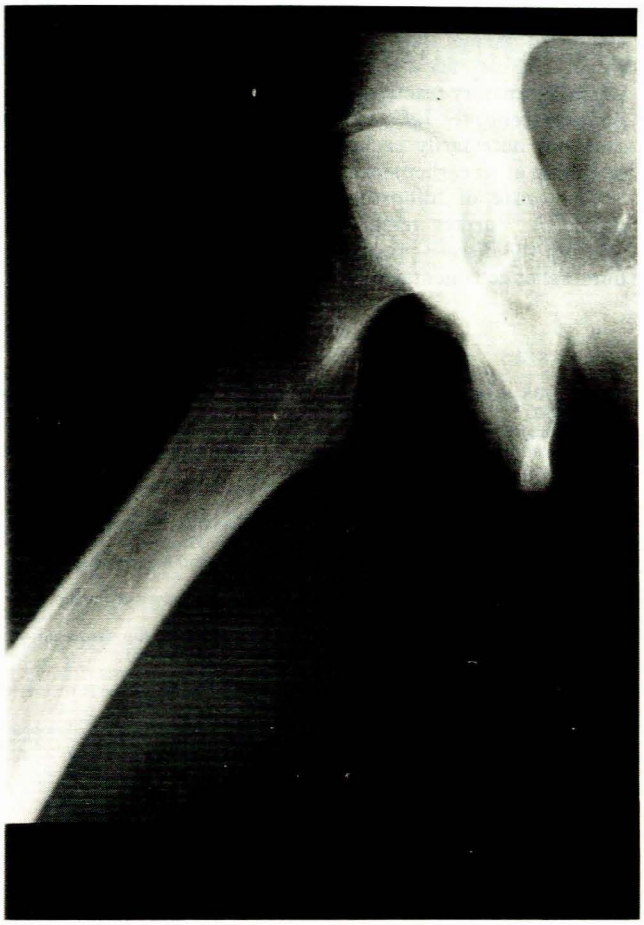

Fig. 2. A lateral radiograph of the right hip.

Arthrotomy of the right hip revealed a dry joint; no synovial fluid was obtainable, but sterile saline was injected into the joint and aspirated for bacteriological, viral and tuberculosis cultures. All were negative. The synovium was thick and oedematous. It was grey and lustreless with a pitted and fibrillated surface.

Histopathological studies of the synovium revealed evidence of a chronic nonspecific synovitis (Fig. 3). The articular cartilage was vacuolated and the surface was irregular; in some areas chondrocytes showed degenerative changes with nuclear pyknosis (Figs 4 and 5). There were no signs of tuberculosis or fungal lesions.

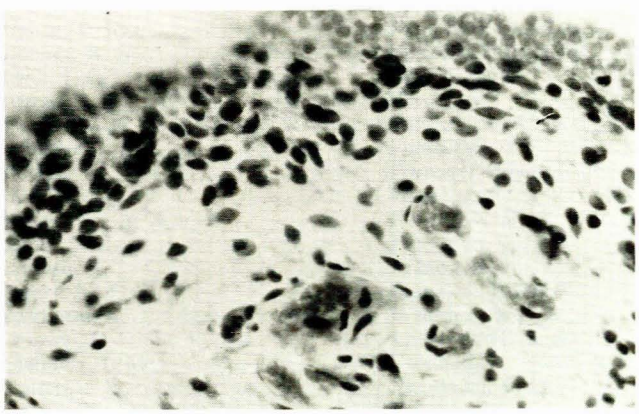

Fig. 3. The synovial membrane of the right hip, showing oedema, hyperaemia and a scanty infiltrate of lymphocytes and plasma cells (H and E x 800).

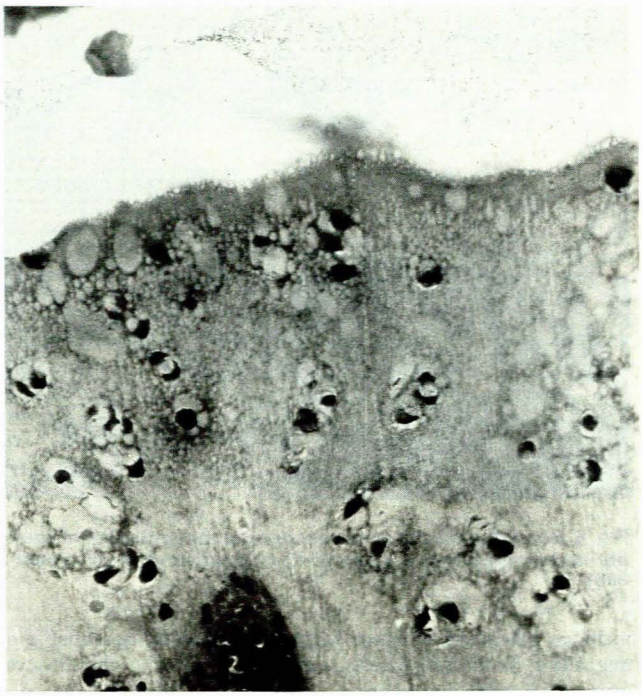

Fig. 4. The articular cartilage, showing vacuolar changes in the matrix towards the surface and chondrocytes undergoing degenerative changes with nuclear pyknosis (H and E x 600).

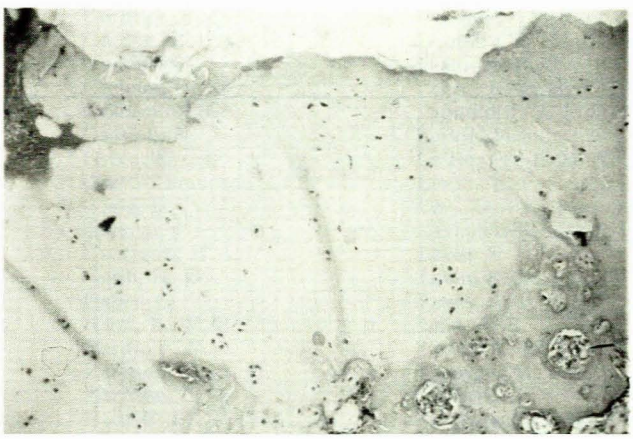

Fig. 5. The articular cartilage, showing an irregular surface (H and E x 200).

Transmission electron microscopy demonstrated necrotic chondrocytes and a frayed articular surface. The scanning electron microscope highlighted the features of an irregular, fibrillated and pitted surface (Fig. 6).

Immunofluorescent studies of the synovium and cartilage for IgG, IgM, IgA, C3 and fibrinogen were negative.

The patient was treated with skin traction of 2 kg, salicylates and bed rest for 2 weeks. She was then mobilized in bed with a range of movement exercises. Her affected hip became free of pain, but mobility did not improve significantly.

Discussion

Golding[2] and Mankin *et al.*[9] suggested that idiopathic chondrolysis is basically an auto-immune phenomenon. In a study of idiopathic chondrolysis of the knee Herman *et al.*[10] demonstrated spontaneous T-cell mitogens which augmented cartilage-

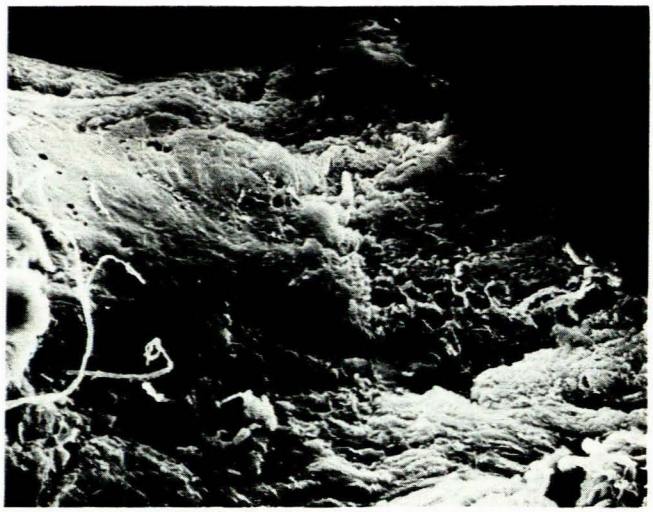

Fig. 6. Scanning electron microscope demonstrates the irregular fibrillated and pitted surface (critical point-dried gold-plated x 300).

induced release of certain factors believed to be capable of degrading articular proteoglycans and inhibiting chondrocyte glycosaminoglycan synthesis.

The matrix of collagen is antigenic, but this is normally masked by the avascular nature and conformation of the cartilage. In idiopathic chondrolysis the cartilage is disrupted and there is often an associated pannus. It is difficult to explain why such a rapidly progressive cartilage disease develops with such a mild surrounding inflammatory reaction. Our studies showed negative immunofluorescence for IgG, IgM, IgA, C3 and fibrinogen, but this does not necessarily exclude an immunopathological process operating at an earlier stage of the disease.

The cause of idiopathic chondrolysis of the hip is still unknown. Further research should be aimed at elucidating whether the disease process is non-immune or whether in fact humoral or cell-mediated factors play a role in its development.

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