tests were carried out on consecutive days using concentrations found during occupational exposure. On the control day water was added to the cement and mixed; on the active challenge day methyl methacrylate was mixed with cement. The technique used was identical with the normal procedure in the operating theatre, and the period of exposure each day was two minutes. Because of the colour and odour of methyl methacrylate it was not possible to "blind" the patient on the active challenge day. A late asthmatic reaction occurred after challenge with methyl methacrylate, starting six hours after with a maximal fall in forced expiratory volume in one second of 25% 13 hours after the challenge. Environmental concentrations of methyl methacrylate were monitored with an infrared gas analyser (Miran model 104) when the cement was mixed on an open trolley and inside a cabinet connected to an extraction system (table).

Comment

The mixing of monomethyl methacrylate liquid with polymethylate methacrylate powder leads to a brief period of a high concentration of methyl methacrylate in the atmosphere. This cement is widely used in orthopaedic work, and the same people usually handle it repetitively. The peak concentration of methyl methacrylate occurs during the first 90 seconds of mixing. Although the atmospheric concentrations do not exceed the threshold limit value for this chemical (100 ppm), brief but repeated exposure to high peak concentrations of a known pulmonary sensitiser is undesirable. Our study shows that with a small cheap fume cabinet, connected either to the theatre extractor system or to an acrylabsorber cartridge, the atmospheric concentration of methyl methacrylate can be reduced to an acceptable level; use of a fume cupboard for the initial mixing process is strongly recommended.

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Polyarthropathy associated with Cushing's disease

Arthropathy associated with Cushing's disease is rare. To our knowledge only five cases have been described,[1-4] all with aseptic necrosis of the femoral head but not a polyarthropathy. We report a patient with longstanding Cushing's disease who developed multiple joint lesions secondary to her disease and who, despite successful treatment of her Cushing's disease, remained severely handicapped by her arthropathy.

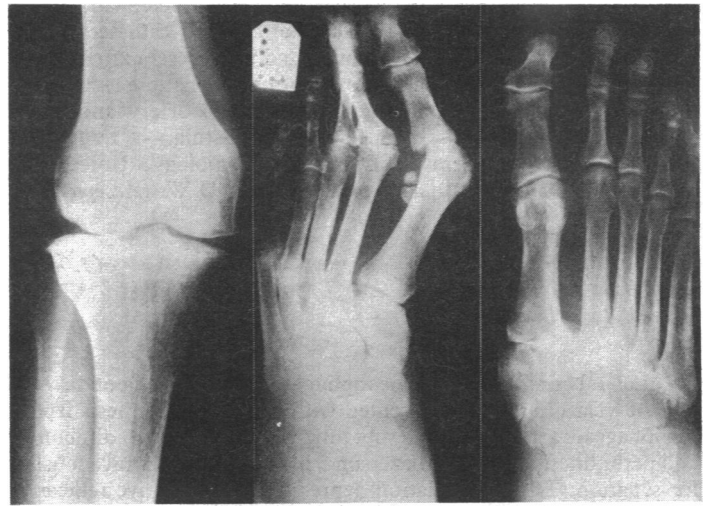
Case report

In 1965, at the age of 32, this patient underwent a routine examination and was noted to be hypertensive, plethoric, hirsute, and suffering from amenorrhoea. Cushing's disease was diagnosed on the basis of raised urinary oxygenic and oxosteroid values and plasma cortisol concentration, which was suppressed by high but not by low dose dexamethasone.

In 1967 she suffered two miscarriages but a year later had a successful pregnancy. A further miscarriage followed in 1969 and a second successful pregnancy in 1970. In 1975 she complained of pain in the right ankle, right knee, and low back. She began to limp and the longitudinal arch of the right foot collapsed. Both wrists became deformed with bony swelling and pain. In 1978 investigations showed a raised plasma cortisol value with loss of diurnal variation (am 635 nmol/l (22·86 μg/100 ml); pm 718 nmol/l (25·85 μg/100 ml); normal: am 193-690 (7-25); pm 83-153 (3-5·5)), which did not fall 12 hours after administration of 1 mg of dexamethasone. Plasma adrenocorticotrophic hormone concentration was 63 ng/l (normal 10-80), suggesting pituitary dependent Cushing's disease. In 1980 a scintiscan of her adrenals showed bilateral hyperplasia.

In 1982 she was referred for a rheumatological opinion. There were asymmetrical bony deformites of her fingers and wrists, but they showed a good range of movement and no synovitis. A large right olecranon bursa was present and her elbows and knees were hyperextensible. The hip joints were normal. The right longitudinal arch had collapsed. There was pronounced thoracolumbar scoliosis but she could flex forward and place her palms on the floor. Radiography of the affected joints, including the spine, confirmed the destructive changes and osteoporosis but no bony erosions were seen. Serum urate concentrations, Wassermann reaction, liver function test values, antinuclear factor, and latex fixation were all either normal or negative. In 1983 she developed acute pain and an effusion of the right knee; aspiration yielded 10 ml of viscous clear fluid with a low cell count and no crystals; radiography confirmed aseptic necrosis of the lateral femoral condyle (see figure).

Because of her deteriorating joint function she underwent bilateral adrenalectomy and radiotherapeutic pituitary ablation. This controlled her Cushing's disease, her hypertension settled, and she suffered no further episodes of bony aseptic necrosis.



Left: Anteroposterior radiograph of right knee showing aseptic necrosis of the lateral condyle and "tramline" calcification in the posterior tibial artery. Right: Radiograph of feet showing destruction of right tarsometatarsal joints, deformity of tarsal bones, and secondary osteoarthritic change. Note march fractures in left fourth and fifth metatarsals and aseptic necrosis of left second metatarsal head.

Comment

This is the first report of Cushing's disease (including iatrogenic cases) resulting in polyarthropathy. The aetiology was probably multifactorial but included bony aseptic necrosis at several sites. Hypercorticism leads to depression of osteoblastic activity and osteoporosis.[5] In animals with iatrogenic Cushing's syndrome it has been suggested that aseptic necrosis of bone is the result of fat emboli from fatty liver change. Patients with iatrogenic Cushing's syndrome have been shown to be hyperlipidaemic, but as yet there is no evidence that they are predisposed to fat emboli. Mechanical changes occurring as a result of ligamentous laxity in our patient may have been a factor in determining the extent of her arthropathy. She was hypermobile but whether this was a result of her Cushing's disease is uncertain. There was no family history of hypermobility and no other cause for her polyarthropathy was found.

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