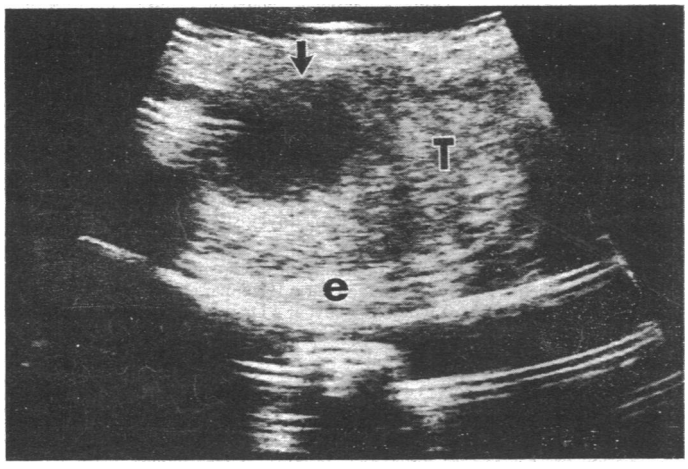
reliable non-invasive diagnostic methods may be excluded with certainty only by undertaking scrotal exploration through an inguinal incision. This approach, though justifiable, does result in a high rate of orchidectomy for benign disease that exceeds 50%, of which epididymal and testicular inflammation account for about 26%.[3] On the other hand, a "wait and see" policy with the administration of more antibiotics often serves only to prolong the patient's misery.

We report on a patient who presented with this dilemma after receiving treatment from his general practitioner, ostensibly for epididymo-orchitis, and in whom the application of scrotal ultrasonography proved to be of value in determining further management that subsequently led to testicular salvage.

Case report

A man aged 35 was referred for urological assessment after complaining of a painful swelling in his scrotum. Two months previously he had been prescribed a week's course of erythromycin by his doctor for suspected epididymo-orchitis. He denied any urinary symptoms. Examination showed a hard mass in the left hemiscrotum from which the testis could not be delineated confidently. A midstream specimen of urine showed sterile pyuria. Serum testicular tumour markers for α fetoprotein and human chorionic gonadotrophin were negative. Scrotal ultrasound imaging using a sector scanner incorporating a 7·5 MHz short focus transducer showed a rounded anechoic area in the upper pole of the testis, which otherwise showed normal echogenicity. The epididymis was thickened (10 mm) and hyperechoic (figure). The features were interpreted as epididymo-orchitis with a localised intratesticular abscess. This diagnosis was confirmed at scrotal exploration by the operative findings of inflammatory adhesions between the tunica vaginalis and testis, a thickened epididymis, and a fluctuant area in the upper part of the testis, the remainder of which felt normal. The abscess was aspirated with a needle, yielding 4 ml of pus from which *Escherichia coli* was later cultured. Cytological examination of the purulent fluid showed no evidence of malignant cells.



Longitudinal ultrasound scan showing thickened hyperechoic epididymis (e), testis (T), and an intratesticular abscess (arrow).

The patient made a rapid recovery and reported no symptoms two months later, at which time the testis was palpably normal, though slightly smaller. A repeat ultrasound examination showed a normal testicular echo pattern.

Comment

Ultrasonography is becoming increasingly popular as a non-invasive technique for the accurate imaging of intrascrotal contents.[4,5] Apart from its obvious ability to differentiate solid from cystic swellings ultrasonography is useful in discriminating between testicular and extratesticular lesions and therefore is of practical importance when clinical examination proves inadequate. In our case ultrasonography was useful not only in elucidating the nature of an indurated scrotal mass but also in detecting the presence of an intratresticular abscess that would otherwise not have been clinically apparent. Treatment based on these findings ensured prompt symptomatic relief and prevented an unnecessary orchidectomy.

An intratesticular abscess should be considered as a possible cause of non-resolving epididymo-orchitis. Its presence can be readily identified by scrotal ultrasonography. The radiological findings, however, must be interpreted in the light of the clinical background. Suspected intratesticular abnormalities should be explored or closely watched by further scanning until resolution occurs. Even though exploration may finally be necessary, the information gained from ultrasonography can guide the surgeon in deciding how he should operate.

We believe that this is the first documented case of testicular salvage after aspiration of an intratesticular abscess diagnosed by ultrasonography in an adult.

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Occupational asthma due to methyl methacrylate in an orthopaedic theatre sister

Acrylates are widely used in the production of polymers such as surface coated resins, plastics, and ion exchange resins. In medicine they are used for dental implants and as bone cement.[1,2] In industry occupational asthma associated with cyanoacrylate based adhesives, including methyl methacrylate, has been described[3]; we describe such a case in a sister who worked in an orthopaedic operating theatre. Two types of bone cement are used in these operating theatres—namely, CMW cement, in which polymethylate methacrylate powder is mixed with monomethyl methacrylate liquid, and Palacos R, in which copolymer methyl acrylate and gentamicin are mixed with methyl methacrylate.

Case report

A 56 year old theatre sister had worked in an orthopaedic operating theatre for 11 years. During this period she had regularly handled CMW bone cement, over the last seven years making about 12 mixes weekly. Before presentation she had handled a new cement, Palacos R with gentamicin. In April 1983 she developed respiratory symptoms characterised by a persistent cough with wheezing and breathlessness. At first there was no clear connection between her asthma and work. She worked on alternate weekends and had two consecutive days off only once every fortnight. In retrospect her asthmatic symptoms were probably better on the evening of the second day away from work. In July she went on holiday, when all her symptoms resolved. When she returned to work they recurred immediately. In December the operating theatre was closed for eight weeks, and again her symptoms resolved. The association between her symptoms and work became apparent to her when her problems again recurred on her return to orthopaedic work. Subsequently she developed wheezing and breathlessness within minutes of mixing bone cement. She was mildly breathless on exertion between these attacks.

She smoked 10-20 cigarettes daily but did not have any history of childhood respiratory illness. Pulmonary function tests when she was not working yielded normal results. Results of skin tests to common environmental allergens were negative. Total serum IgE concentration was 218 U/ml. Bronchial provocation

Environmental methyl methacrylate vapour concentrations (ppm) resulting from mixing of polymeric cement on open trolley and in fume cabinet

Time (s)	Procedure	Cement mixed on open trolley	Cement mixed in fume cabinet
0	Breakage of phial	0	0
15	Addition of liquid cement	126	0
30	Mixing	274	0
45	Mixing	374	0
60	Mixing	164	0
75	Spoonful of cement removed from bowl	126	36*
90	Lid removed from cabinet and bowl extracted		25*
105	Bowl cleaned out	80	76

*Cement removed from fume cabinet.

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.

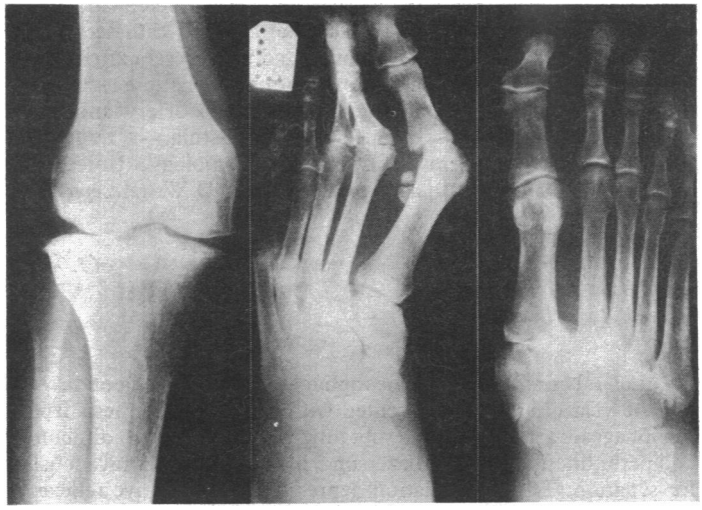
Case report

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.

We thank Drs H M Leather and R Corrall for their help in the management of this patient.

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