BRIEF REPORTS

ACUTE CALCIFIC SUPRASPINATUS TENDINITIS IN A THREE-YEAR-OLD CHILD

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A previously healthy three-year-old boy presented with a 72-hour history of general malaise and refusal to move the right arm. There was no history of injury or of previous symptoms in the limb. On examination, he was unhappy and withdrawn, holding the right arm securely across his stomach with his left hand. He was afebrile when first seen in the accident department but two hours later his temperature was 38.6°C Celsius. There was localised tenderness around the right shoulder and radiographs showed a large, amorphous calcific deposit in the subacromial region with inferior subluxation of the head of the humerus (Fig. 1). His haemoglobin was 11.5 g/dl, the white cell count 12.4 × 10⁹/l and the ESR 43 mm in the first hour. The urea and electrolyte values were normal.

Under general anaesthesia, a wide-bore needle was inserted into the subacromial space and two millilitres of a white toothpaste-like material was aspirated. Because of the size of the calcium deposit, the subacromial space was explored through a short deltoid-splitting incision. An encapsulated white calcareous deposit was dissected from the subacromial space with a small piece of the tendon to which it was attached. There was no sign of infection and inspection of the shoulder joint through a short incision in the superior capsule showed no effusion and a normal appearance despite the subluxation seen on radiographs. Antibiotics were not given and although the arm was rested in a sling, this was rapidly discarded by the child and normal shoulder movements were regained after 14 days.

Immediate Gram stain of the excised material showed no organisms, and cultures (aerobic, anaerobic and for mycobacteria and yeasts) were all negative. The serum calcium, phosphate and alkaline phosphatase and all investigations of renal function including ultrasound scanning were normal. Histology of the tendon and calcium deposit was reported as being consistent with calcific supraspinatus tendinitis.

Discussion. Calcific supraspinatus tendinitis is common in middle-aged patients and is often assumed to be secondary to degenerative change in necrotic tendon. Uhthoff, Sarkar and Maynard (1976) cast doubt on this explanation. They examined 46 excised specimens from patients with calcific supraspinatus tendinitis and observed areas of fibrocartilage and foci of calcification surrounded by phagocytic giant cells and new vessels. They then postulated that fibrocartilage formed in avascular regions of the tendon and that chondrocytes mediated the deposition of calcium. The site of calcium deposits coincided with the region of the tendon shown by cadaveric micro-angiographic studies to be poorly vascularised (Rathbun and Macnab 1970). Our specimen showed similar appearances.

This is the first reported case of acute supraspinatus tendinitis affecting a child of three, an age at which degenerative changes seem unlikely. The child has made a full recovery and radiographs taken one month after operation showed no recurrence of calcification. We postulate the cause to be a localised area of avascularity as described by Uhthoff and his colleagues; there is no theoretical reason why this should not occur in a child.

REFERENCES
