I thank Professor R W Stout for permission to report this case and for his helpful advice.

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Hypercalcaemia during resolution of calcinosis in juvenile dermatomyositis

Calcinosis is common in juvenile dermatomyositis and may regress spontaneously. We report a case of juvenile dermatomyositis in which regression of pelvic calcinosis was associated with life threatening hypercalcaemia.

Case report

An 11 year old white boy was diagnosed as having dermatomyositis when he presented with proximal muscle weakness, a heliotrope rash, and a scaly dermatitis on the extensor aspect of his knuckles. Muscle enzyme activities were raised (serum creatine phosphokinase activity 331 U/l (normal 5-115 U/l)), and electromyographic findings were consistent with myopathy. He was treated with prednisone with good effect but required increasing doses after several relapses over the subsequent seven years.

In November 1980, at the age of 17, he sustained a minor blow to his left thigh and shortly after noted a painful lump in his left groin. Radiography showed calcinosis in the soft tissues around the left hip and thigh. This became more extensive over the next three months with resulting restriction in the movement of his hip. He developed anaemia and thrombocytosis; the erythrocyte sedimentation rate was 30 mm in the first hour and serum creatine phosphokinase activity 215 U/l. Serum calcium and parathyroid hormone concentrations were not raised. In February 1981 the lump in his groin became fluctuant, and about 100 ml of turbid material flecked with calcium was drained surgically, leaving a chronic sinus and severe residual calcinosis.

In January 1982 he developed a fever, vomiting, and malaise together with a tender swelling over his left buttock. Serum calcium concentration was 3.1 mmol/l (12 mg/100 ml) (normal 2.07-2.52 mmol/l (8.3-10 mg/100 ml)), serum phosphate concentration 1.59 mmol/l (4.9 mg/100 ml) (normal 0.9-1.6 mmol/l (2.8-5 mg/100 ml)), and serum albumin concentration 31 g/l. The urine calcium concentration was 6.6 mmol (265 mg)/24 h (normal 1.2-6.2 mmol (48-248 mg)/24 h), serum parathyroid hormone concentration less than 0.05 µg/l (normal 0.1-0.35 µg/l), and serum 1,25-dihydroxycholecalciferol concentration 24 pmol/l (10 ng/l) (normal 70-132 pmol/l (29-55 ng/l)). He was treated with saline diuresis and increased doses of prednisone, but his serum calcium concentration remained raised.

Three weeks later, after complaining of weakness, nausea, constipation, and aching in the jaw, he had several grand mal convulsions and then remained unconscious. Serum calcium concentration was 3.53 mmol/l (14 mg/100 ml) and serum magnesium concentration 0.6 mmol/l (1.5 mg/100 ml) (normal 0.7-1.4 mmol/l (1.7-3.4 mg/100 ml)). A computed tomogram of the brain was normal, but electroencephalography showed a severe abnormality over both cerebral hemispheres. He was treated with saline diuresis, phenytoin, and prednisone 100 mg daily. After 36 hours he awoke suddenly, and over the next few days he recovered fully, although his serum calcium concentration remained raised at 2.6-3.2 mmol/l (10.4-12.8 mg/100 ml). Acute hypercalciuria of up to 30 mmol (1.2 g)/24 h was noted together with phosphaturia (index of phosphate excretion +0.45 (normal -0.3 to +0.3)), but urine hydroxyproline excretion was low at 0.08 mmol (10.5 mg)/24 h (normal 0.0-0.38 mmol (50 mg)/24 h), indicating that calcium was not being mobilised from bone.

Treatment was started with aminohydroxypropylidenediphosphonate 10 mg/kg/day. After one month, during which he remained well, the area of inflammation over his left buttock ulcerated and discharged large amounts of calcific material. The serum calcium concentration rose to 4.1 mmol/l

Top: Computed tomogram obtained in February 1981, showing extensive deposits of calcium density in extensor and adductor muscles of left thigh, extending to affect subcutaneous fat (adjacent unaffected thigh muscles are hypertrophied). Bottom: Computed tomogram obtained in March 1983, showing complete reabsorption of calcification; muscles in thigh are normal size.

(16.4 mg/100 ml) but fell to 2.9 mmol/l (11.6 mg/100 ml) with saline diuresis. The ulcer continued to drain for two days, discharging roughly 400 ml, and then gradually healed with appreciable reduction in the swelling around the hip together with radiological evidence of regression of the calcinosis (figure). Subsequently he remained well with complete clinical and radiological regression of the calcinosis and normal serum calcium and parathyroid hormone concentrations. Steroids were gradually withdrawn. He showed no further evidence of dermatomyositis and achieved full puberty.

Comment

Calcinosis has an estimated incidence of up to 74% in patients who survive the active phase of juvenile dermatomyositis.¹ Episodes of skin ulceration with extrusion of calcific debris are common and may be associated with cellulitis and systemic reaction, as in our patient. Spontaneous regression of calcinosis has been well documented,² but associated hypercalcaemia has not previously been described. In our patient the suppressed serum parathyroid hormone concentration and low vitamin D and urine hydroxyproline concentrations together with extreme hypercalciuria and phosphaturia suggest that the hypercalcaemia developed as a result of spontaneous mobilisation of calcium from the calcific mass in the pelvis. Treatment with aminohydroxypropylidenediphosphonate did not appear to affect the serum calcium concentration and probably did not influence the outcome.

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