ABSTRACT

A 25-year-old woman with end-stage renal failure presented with subcutaneous calcinosis cutis that grew rapidly in both hands. Radiographs showed subcutaneous lobulated calcific deposits. Magnetic resonance imaging revealed a heterogeneous mass encasing segments of the abductor pollicis longus and extensor pollicis brevis tendons. Excision of the masses was performed. Histopathology revealed amorphous calcified deposits in fibrous tissue and a foreign body reaction. There was no evidence of a tubercular lesion. Further investigation revealed the presence of hyperphosphataemia and secondary hyperparathyroidism, despite a normal serum calcium level. Oral phosphate-lowering agents failed to control the condition, and recurrence was noted 6 months later. The patient finally underwent parathyroidectomy and has had no further recurrence.

Key words: calcinosis; kidney failure, chronic

INTRODUCTION

Calcinosis cutis (also known as calciphylaxis) is a rare manifestation of systemic calcinosis characterised by precipitation and deposition of calcium and phosphate salts in the dermis and subcutaneous tissues. We report a rare case of subcutaneous calcinosis cutis that grew rapidly in both hands of a 25-year-old woman with end-stage renal failure.

CASE REPORT

In April 2007, a 25-year-old woman presented with a 3-month history of swelling over the left dorsoradial wrist. She had end-stage renal failure of unknown cause and had been having peritoneal dialysis for 2 years. The swelling was soft and mobile, and grew rapidly from 2 cm to 4 cm in 2 months, but she had neither pain, skin changes, nor regional lymphadenopathy. Radiographs revealed subcutaneous lobulated calcific deposits (Fig. 1). At that time, the patient refused surgical excision. Over the next 6 months, more swellings developed over
the left little finger and right thumb (Fig. 1). Magnetic resonance imaging revealed a heterogeneous mass encasing segments of the abductor pollicis longus and extensor pollicis brevis tendons (Fig. 2). In view of the rapidly growing nature of the masses in an immune-compromised patient, the differential diagnoses included soft-tissue tuberculosis, atypical mycobacterial infection with calcification, giant cell tumour of tendon sheath, fibrous histiocytoma, and epithelioid sarcoma.

Intra-operatively, the left wrist mass ruptured and leaked caseating fluid (Fig. 3). Streptomycin sulphate (2 g) with water was used for irrigation. No mycobacterial species was cultured. One branch of the superficial radial nerve was embedded within the mass and thus sacrificed. The extensor pollicis longus tendon, radial artery and vein, and dermis were not involved. The left little finger mass was tightly adherent to the extensor sagittal band, but the digital neurovascular bundle was spared. The right thumb mass was over the palmar interphalangeal joint. Complete excision of all masses was achieved.

Histopathology revealed amorphous calcified deposits in fibrous tissue and a foreign body reaction. There was no evidence of a tubercular lesion. A diagnosis of calcinosis cutis was made.

The patient had a normal serum calcium level (2.5

Figure 1  Radiographs showing (a) a 3-cm calcific mass over the left radial wrist (b) the mass increases to 5 cm and becomes more radiodense 6 months later. Another 3-cm mass is noted over the left little finger, and a right thumb mass is forming.

Figure 2  T2-weighted magnetic resonance image of the left wrist showing a heterogenous mass encasing a segment of abductor pollicis longus and extensor pollicis brevis tendons.

Figure 3  Intra-operative photographs showing leakage of caseating fluid after puncture and the mass adhering to the extensor pollicis brevis tendon.

Figure 4  Calcification and foreign body reaction in the surrounding soft tissue (H&E, x125).
mmol/l), but she had endured a hyperphosphataemia of 4.0 mmol/l (normal range, 0.8–1.5 mmol/l) for 2 years. The calcium phosphate product was markedly elevated at 10 mmol²/l² (normal range, 1.7–4.0 mmol²/l²). She was diagnosed to have secondary hyperparathyroidism, and her serum parathyroid hormone level was 27 µmol/l (normal range, 1.6–6.9 µmol/l). The patient was treated with an oral phosphate-lowering agent (lanthanum carbonate hydrate). Recurrence of calcinosis cutis was noted 6 months later. The patient finally underwent parathyroidectomy, and has had no further recurrence.

**DISCUSSION**

Cutaneous lesions are typically found on the abdomen, buttocks, or thighs, and present as firm, tender papules, nodules, or plaques that may periodically discharge chalky material. In severe cases, progressive deposition of calcium in the skin and mural calcification of small blood vessels with associated thrombosis and necrosis can be a life-threatening illness. Depending on its aetiology and pathogenic mechanism, extraosseous calcification is classified into 3 main groups: dystrophic, metastatic, and idiopathic. Some cases may be iatrogenic, with extravasation of calcium gluconate. The more common dystrophic form results from the deposition of calcium salts in tissue previously damaged by inflammatory, degenerative, neoplastic, or hereditary mechanisms and occurs in patients with normal serum calcium and phosphate levels. In contrast, metastatic calcification is associated with abnormal systemic metabolism and serum levels of calcium and/or phosphate. It occurs primarily in patients with renal failure with primary or secondary hyperparathyroidism, vitamin D intoxication, and milk-alkali syndrome.

Calcinosis cutis occurs in approximately 1% of patients with end-stage renal disease undergoing chronic dialysis, usually after a short period of dialysis (median, 4 years). It is associated with elevated levels of plasma calcium-phosphate product (>5.50 mmol²/l²) and increased serum phosphate concentrations. Skin and subcutaneous mineralisation may decrease if the serum phosphate level decreases. Dietary control of phosphate intake and oral phosphate-binding agents can be used as treatment. Parathyroidectomy may be needed in cases of secondary hyperparathyroidism. In our patient, secondary hyperparathyroidism was not noted before excision of the masses. In cases of a calcified mass in the subcutaneous tissue, investigation of serum parathyroid hormone, calcium, and phosphate levels should be included, with a view to revealing the presence of hyperparathyroidism or any other underlying disease process. Parathyroidectomy should have been performed earlier to better control the hyperparathyroidism, in which case the calcinosis lesions may have regressed, especially when phosphate-lowering treatment failed to control the condition.

**REFERENCES**