An 81-year-old man presented with reduced mobility and severe proximal muscle pain and weakness. He reported having frequent falls. His medical history included osteoarthritis of the knees, hypertension and L2 vertebral wedge collapse in 1987. A clinical examination showed swollen ankles and synovitis in his left knee and bilateral quadriceps muscle wasting. Muscle power was grade 4 or 5, and tone, reflexes and sensation were normal.

The results of investigations were as follows: corrected calcium, 4.02 mmol/l (2.10–2.60); phosphate, 0.71 mmol/l (0.80–1.40); alkaline phosphatase, 782 u/l; parathyroid hormone, 190.1 pmol/l (0.95–5.7); and urinary calcium, 12.85 mmol/l/day (1.6–8.8).

A diagnosis of longstanding parathyroid bone disease complicating primary hyperparathyroidism was made on the basis of plain radiographs and bone scintigraphy. A neck ultrasound and TC-99m sestimibi scanning located a swelling around the left upper pole of the thyroid. A mass was removed by minimally invasive parathyroidectomy, and histology showed a benign adenoma.

Scintigraphy in this patient, essentially a superscan (fig 1A), reflected the characteristic Lone Ranger sign (fig 1B) from excessive cranial uptake and reduced soft tissue background activity. Supercans are caused by diffuse increased skeletal uptake secondary to increased bone turnover, giving an apparently normal or negative scan but suggesting reduced or absent soft tissue or renal uptake, commonly described in metastases from carcinoma of the prostate. They are also seen in metabolic bone disease and in haematological disorders.

Hyperparathyroid bone disease is also becoming increasingly rare, occurring in <5% of patients with primary hyperparathyroidism in the USA. Brown tumours (fig 2) are more common in primary (3%) than in secondary (2%) hyperparathyroidism. They are well-defined, solitary, or multifocal lytic lesions provoking little bone formation. They may be diaphyseal, metaphyseal or, rarely, epiphyseal.

REFERENCES