UNUSUAL AND MEMORABLE.....

doi: 10.1136/ard.2006.053801

Hyperparathyroid bone disease—an unusual and memorable condition

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/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 sestimiibi 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. Superscans 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,¹ commonly described in metastases from carcinoma of the prostate.² 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.³ 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 diaphysial, metaphysial or, rarely, epiphysial.⁴

D J Pradeep

G P R Clunie

R A Watts

R C Nightingale

Rheumatology, Nuclear Medicine

Departments, Ipswich Hospital, Ipswich, UK

Correspondence to: D J Pradeep,
Rheumatology, Nuclear Medicine
Departments, Ipswich Hospital, Ipswich, UK;
drjohnpd@btinternet.com

REFERENCES

1 Hain SF, Fogelman I. Nuclear medicine studies in metabolic bone disease. *Semin Musculoskelet Radiol* 2002;6:323–29.

Figure 1 Scintigraphy in this patient, essentially a superscan (A), reflected the characteristic Lone Ranger sign (B) from excessive cranial uptake and reduced soft tissue background activity.

Figure 2 Brown tumour of hyperparathyroidism.

2 Constable AR, Carnage RW. Recognition of the superscan in prostatic bone scintigraphy. *Br J Radiol* 1981;54:122–5.

3 Silverberg SJ, Bilezikian JP. Clinical presentation of primary hyperparathyroidism in the United States. *The parathyroids*. 2nd edn. New York, NY: Raven Press, 2001;349–60.

4 Brown RF, Murphy SM. Musculoskeletal case 27. Primary hyperparathyroidism induced brown tumour of the third metacarpal. *Can J Surg* 2003;46:122.