Clinical picture

Patellar fracture: atypical presentation of parathyroid bone disease

A 21-year-old male was admitted to the orthopaedic and trauma department following a left knee injury. On examination, he appeared dehydrated and had left-sided knee pain. Radiological investigations revealed a left patellar fracture (Figure 1A); in addition, incidental distal femoral and proximal tibial bone cysts were present. Routine serum biochemistry revealed severe hypercalcaemia with a corrected calcium 4.03 mmol/l. Initial management of hypercalcaemia included 4 l/24 hr intravenous

Figure 1. (A) Plain radiograph showing patellar fracture with distal femoral and proximal tibial bone cysts. (B) T99m MIBI nuclear sestimibi scan showing uptake in the region of the right lobe of the thyroid gland (arrow) consistent with a parathyroid adenoma. (C) Resected parathyroid mass confirmed histologically as a benign parathyroid adenoma. (D) Plain radiograph following operative intervention with tension band wiring technique

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(IV) saline (from Day 1), 30 mg IV pamidronate (Day 2) and 40 mg iv furosemide (Day 4), with improvement in serum calcium (3.01 mmol/l on Day 8). Parathyroid hormone (PTH) was significantly elevated consistent with primary hyperparathyroidism, PTH: 1393 pg/ml [Normal range (NR): 15–60 pg/ml]. A T99m sestamibi scan (Figure 1B) revealed uptake in the region of the right lobe of the thyroid gland suggestive of a parathyroid adenoma. Further radiological imaging revealed multiple bone cysts in the appendicular skeleton consistent with classic hyperparathyroid bone disease. Definitive treatment with parathyroid exploration was performed 9 days after admission, revealing a 25 mm inferior parathyroid mass (Figure 1C), which was excised and confirmed histologically as a benign parathyroid adenoma. His post-operative course was complicated by symptomatic hypocalcaemia attributed to hungry bone syndrome, requiring IV calcium gluconate and escalating titration of 1α-Hydroxycholecalciferol with resolution of hypocalcaemia.

Primary hyperparathyroidism is characterized by hypercalcaemia attributable to autonomous overproduction of parathyroid hormone either due to a solitary adenoma, multiple gland hyperplasia or more rarely, neoplasia.¹ High circulating levels of parathyroid hormone result in excessive resorption of calcium and phosphate from bone.² Bone disease is rarely overt, as in this case. Radiographic manifestations are observed in <2% of patients and can include sub-periosteal erosions, diffuse osteoporosis, cystic lesions or ‘brown tumours’.³ However, presentation with a pathological fracture is very uncommon in this age group. A recent cohort study of risk of fracture before and after surgery for primary hyperparathyroidism revealed increased fracture risk up to 10 years before surgery in patients with long standing hyperparathyroidism.⁴ Importantly, fracture risk is attenuated within 1 year after parathyroidectomy, with improved bone mineral density observed with DEXA scanning and in keeping with restoration of bone biomechanical competence.⁴

This case highlights the unusual presentation of patellar fracture arising from classic hyperparathyroid bone disease. When suspecting pathological fractures, in addition to the common causes, such as secondary neoplasm, primary hyperparathyroidism must be considered.

Photographs and text from: A. Walls, Department of Trauma and Orthopaedics, Altnagelvin Area Hospital, Londonderry, UK; P.C. Johnston, Department of Endocrinology and Diabetes, Altnagelvin Area Hospital, Londonderry, UK; K. Kluczewska-Zygan, Department of Radiology, Altnagelvin Area Hospital, Londonderry, UK; M.H. Vazir, Department of Histopathology, Altnagelvin Area Hospital, Londonderry, UK; G.B. McBride, Department of Otologyngology, Altnagelvin Area Hospital, Londonderry, UK; J.R. Lindsay, Department of Endocrinology and Diabetes, Altnagelvin Area Hospital, Londonderry, UK. email: johnrlindsay@hotmail.com

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A.W. and P.C.J. contributed equally to the work.

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References