

Multiple osteolytic bone lesions

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CASE REPORT

A 31-year-old woman of Turkish origin was referred because of hypercalcaemia. She had experienced myalgia and bone pain for years. She further experienced progressive fatigue, poor memory and general muscle weakness. Six months before presentation, she had been diagnosed with vitamin D deficiency, for which supplementation was started. Her symptoms did not improve. Medical consultation during a holiday in Turkey yielded hypercalcaemia.

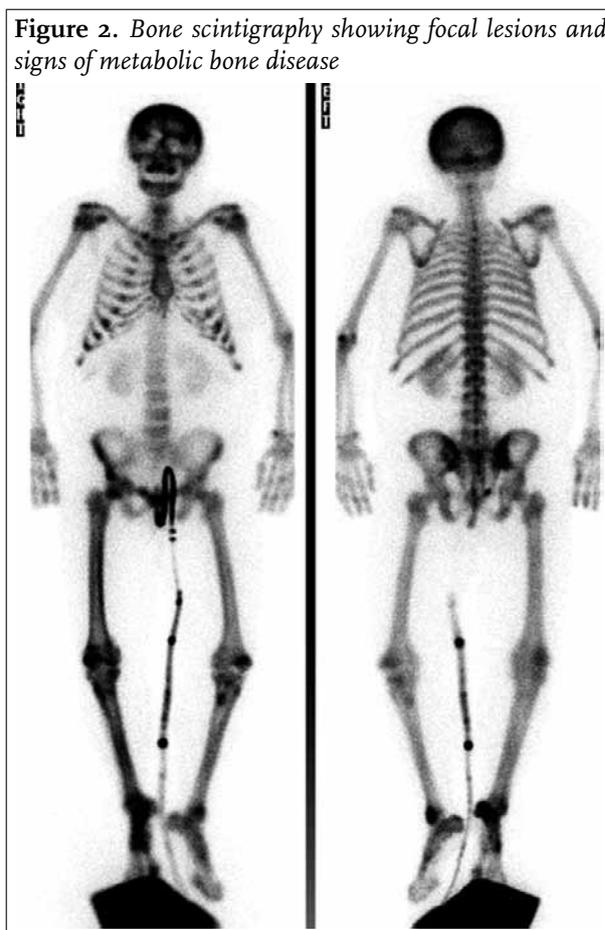
At physical examination she was obese and walked with difficulty. There were no signs of arthritis and motion of the hips was not limited. Laboratory evaluation revealed an albumin-corrected calcium of 3.07 mmol/l (URL 2.65), parathyroid hormone (PTH) 100 pmol/l (URL 6.5), 25 OH vitamin D₃ 18 nmol/l (LRL 35), 1,25 (OH)₂-vitamin D₃ 429 pmol/l (URL 150) and creatinine 50 µmol/l (N 50-90). The alphacalcidol was stopped at presentation because of hypercalcaemia.

The source for PTH hypersecretion was identified as a 3 cm tumour of the left lower parathyroid by tetraphosmin scintigraphy. A pelvic X-ray (*figure 1*) showed a large lytic lesion of the right acetabulum extending into the ilium and

Figure 1. Pelvic X-ray with multiple lytic lesions in the right and left ilium



Figure 2. Bone scintigraphy showing focal lesions and signs of metabolic bone disease



ischium at high risk of fracture; more lytic lesions were present in the right and left ilium. Trabeculae were clearly visible in the neck of both femurs. The bone scintigraphy (*figure 2*) showed multiple focal lesions and accumulation at the skull, costochondral joints and the cortex of the long bones.

WHAT IS YOUR DIAGNOSIS?

See page 403 for the answer to this photo quiz.

ANSWER TO PHOTO QUIZ (PAGE 399)
MULTIPLE OSTEOLYTIC BONE LESIONS

DIAGNOSIS

The combination of hyperparathyroidism and lytic bone lesions raised suspicion of a brown tumour as a consequence of hyperparathyroidism caused by parathyroid adenoma. The initial vitamin D deficiency might have aggravated the hyperparathyroidism. The parathyroid adenoma was surgically removed, resulting in normal PTH levels. A biopsy of the acetabulum lesion confirmed the diagnosis of a brown tumour. In the course of months remineralisation of the bone lesions occurred.

The primary treatment of brown tumours consists of resolving the hyperparathyroidism. Surgical treatment of the bone lesions is only indicated in cases of (high risk for) fracturing.¹

Although clinical features can be highly suggestive for brown tumours, histological evidence is indispensable to rule out malignancy. Histologically, brown tumours are characterised by increased numbers of osteoclasts, cyst formation, signs of increased bone turnover and fibrosis. The chaotic bone matrix does often result in focal necrosis and microfractures. Haemosiderin deposits because of bleeding give these tumours their characteristic brown colour.^{2,3}

Brown tumours are a late feature of osteitis fibrosa cystica. Osteitis fibrosa cystica develops as a consequence

of a disturbed balance between bone formation and degradation due to exposure to high PTH levels. It is radiologically characterised by multiple lytic bone lesions, subperiosteal resorption of the distal phalanges and clavicles, absence of the lamina dura of the teeth, focal demineralisation of the skull and low bone density.³

On bone scintigraphy focal areas of accumulation can be accompanied by features of metabolic bone disease, such as excessive activity of the skull, the costochondral joints (rosary sign) and the cortex of the long bones (tramline phenomenon).⁴

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