Bilateral fractures of the second metatarsals

Answers on p 805.

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Case history

A 45 year old housewife presented with bilateral painful feet. The pain was located over the metatarsals and was increased by walking and decreased on rest. The initial *x* ray was normal and the patient was sent home with advice to rest. A provisional diagnosis of metatarsalgia was made. The patient was reviewed after two months with persistent symptoms and a severe antalgic gait; *x* rays (fig 1) of both

feet revealed callus around both second metatarsals, indicating healing fractures.

Questions

- (1) What type of fractures are these and what is their aetiology?
- (2) Where are the other sites in the foot that stress fractures can occur?
- (3) What would be the best management at this stage?

Figure 1 *x* Ray of feet showing healing metatarsal fractures.

Thyrotoxicosis of a rare aetiology

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Answers on p 806.

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A 28 year old woman was admitted with an eight month history of menstrual irregularities and a four month history of weight loss, haemoptysis, and dyspnoea. She had had menorrhagia for six months, for which a dilatation and curettage was done. Following this, she developed amenorrhoea. Her obstetric history was unremarkable. She was a mother of two (the last childbirth was two years previously). Physical examination revealed bilateral exophthalmos, lid lag, fine tremors in the outstretched fingers, warm and moist skin, and pallor. She had resting tachycardia and tachypnoea. Temperature and blood pressure were normal. There was no goitre. Fine crepitations were heard throughout inspiration and expiration all over the thorax. Haematological studies showed a haemoglobin concentration

of 89 g/l, a leucocyte count of $10.9 \times 10^9/l$, and erythrocyte sedimentation rate of 70 mm/h. Thyroid function tests confirmed the diagnosis of thyrotoxicosis: thyroid stimulating hormone 0.01 mIU/l (reference range 0.35–5.5 mIU/l), T_4 22.4 µg/dl (3.2–12.6), and T_3 2.54 ng/ml (0.6–1.81). The serum biochemical profile was in the normal range. Chest *x* ray showed multiple cannon ball opacities. An ultrasound study of the abdomen was normal. Further studies were performed to establish the aetiology.

Questions

- (1) What is the diagnosis?
- (2) What diagnostic procedure supports it?
- (3) What is the cause of the thyrotoxicosis?

Iatrogenic recurrent severe hypercalcaemia and renal impairment

T Sulkin, A J Krentz

Answers on p 807.

Case 1

A 77 year old woman presented to her general practitioner with a three week history of vague and non-specific symptoms including anorexia, sore throat, and weakness of her legs. Her past medical history included hypertension and osteoarthritis, for which she was receiving bendrofluzide 2.5 mg daily together with ibuprofen (modified release) 1600 mg daily and co-proxamol (dextropropoxyphene and paracetamol) as required. Physical examination revealed an unwell patient with no specific abnormalities. Emergency laboratory investigations showed significant renal impairment with a plasma urea of 24 mmol/l (normal 3.0–6.5) and plasma creatinine concentration of 250 μ mol/l (normal 60–125) together with hyperkalaemia (plasma potassium concentration 5.6 mmol/l (normal 3.5–5.0)). Plasma calcium concentration, corrected for the prevailing plasma albumin concentration,¹ was 3.63 mmol/l (normal 2.15–2.55) with a marginally low plasma phosphate of 0.56 mmol/l (normal 0.7–1.5). Plasma alkaline phosphatase was normal. The patient's erythrocyte sedimentation rate (ESR) was markedly raised at 100 mm/h, and a urinary tract infection was confirmed on microscopy and culture of her urine. Intravenous rehydration, infusion of a single dose of a bisphosphonate (pamidronate), and appropriate antibiotics led to a rapid clinical and biochemical improvement.

Potential causes of hypercalcaemia including multiple myeloma, thyrotoxicosis, and primary hyperparathyroidism were excluded by appropriate laboratory tests; serum parathyroid hormone (PTH) concentration was suppressed at 2.0 pmol/l (normal < 7.3) in the presence of hypercalcaemia. There were no features suggestive of malignant disease. Renal function and plasma calcium had returned to normal and ESR had fallen to 34 mm/h by the time of discharge from hospital. In the absence of a clear cause for the hypercalcaemia and acute renal impairment the patient's thiazide diuretic was discontinued.¹

Later the same year, following the prescription of a loop diuretic (frusemide (furosemide) 40 mg with amiloride 5 mg) for peripheral oedema, she was readmitted with symptoms of acute confusion, anorexia, and dyspepsia. She was clinically dehydrated with a corrected plasma calcium concentration of 3.24 mmol/l and plasma creatinine 166 μ mol/l. Once again, rehydration with intravenous fluids resulted in rapid resolution of the abnormal biochemistry, and a calcium antagonist (amlodipine) was substituted for the diuretic.

The following year she was admitted a third time with similar symptomatology and biochemistry (plasma calcium 3.57 mmol/l). Arterial blood gases confirmed a metabolic alkalosis (pH

7.48 (normal 7.38–7.44), standard bicarbonate 30.3 mmol/l (normal 21–28)). Inorganic phosphate and alkaline phosphatase were normal, as was her ESR; serum PTH was again suppressed at 1.1 pmol/l. On this occasion, direct questioning revealed a long history of symptoms suggestive of reflux oesophagitis for which she had been self medicating with a proprietary medicine. At endoscopy, a large hiatus hernia with oesophagitis was found. The ibuprofen was discontinued and a proton pump inhibitor (omeprazole) begun, with effective symptomatic relief. Following appropriate advice no recurrence of hypercalcaemia has been observed for almost two years.

Case 2

A 64 year old woman was admitted as an emergency with acute confusion. Physical examination revealed bilateral basal crackles. There was a neutrophilia, and a chest infection was diagnosed. Plasma creatinine concentration was 591 μ mol/l with a plasma urea of 28.7 mmol/l. Plasma calcium concentration was 4.29 mmol/l with a low albumin of 26 g/l. The ESR was raised at 74 mm/h. Thyroid function tests were normal. No focus of infection was confirmed. Plasma electrophoresis was normal and urine was negative for Bence-Jones protein. Plasma parathyroid hormone was suppressed at 0.6 pmol/l. Computed tomography of the abdomen (performed because of transiently deranged liver function tests) showed no significant abnormality.

The patient was hydrated with intravenous saline; this led to an improvement in the hypercalcaemia to 3.7 mmol/l within two days. Intravenous pamidronate and continued hydration led to a further fall in plasma calcium concentration during the following days, and by discharge on day 12 renal function was normal. A history of chronic dyspepsia prompted upper gastrointestinal gastroscopy which revealed a large benign ulcer at 32–35 cm. Antral biopsy was positive for *Helicobacter pylori*. A proton pump inhibitor together with *H. pylori* eradication treatment led to resolution of symptoms with no recurrence of hypercalcaemia (plasma calcium 2.48 mmol/l most recently).

¹ Martin TJ, Grill V. Endocrine investigation: hypercalcaemia. *Clin Endocrinol* 1995;42:535–8.

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Questions

- (1) What was the cause of the recurrent hypercalcaemia and renal impairment in these patients?
- (2) What is the appropriate management of this syndrome?
- (3) What are the potential long term sequelae of the untreated syndrome?
- (4) What, if any, was the contribution of the diuretics to the disturbance of calcium metabolism in the first patient?