Final diagnosis

Peripheral neuropathy secondary to pyogenic liver abscess.

Keywords: peripheral neuropathy; hepatic abscess

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Hypercalcaemia with radiographic abnormalities in chronic myeloid leukaemia

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A 56-year-old woman with untreated chronic myeloid leukaemia presented with a history of weakness, easy fatigability and fever of 6 weeks duration. She was emaciated, pale, had sternal tenderness and a few palpable lymph nodes in the cervical, axillary and the inguinal areas. General physical examination showed splenomegaly (2 cm below the left costal margin). Laboratory investigations revealed a haemoglobin level of 8.5 g/dl; a total leucocyte count of $125 \times 10^9/l$ with 35% neutrophils, 4% lymphocytes, 1% monocytes, 37% eosinophils, 2% basophils, 1% promyelocytes, 10% myelocytes, 6% metamyelocytes, and 4% blasts, and there were 3–4 nucleated red blood cells per 100 white blood cells. The peripheral blood smear showed microcytic and elliptocytic red blood cells. The platelet count was $200 \times 10^9/l$. The serum biochemistry showed a serum calcium of 3.6 mmol/l, serum albumin 26 g/l, serum phosphorous 1.3 mmol/l and serum alkaline phosphatase 241.4 IU/l (normal 21.3–92.3 IU/l). Estimation of the 24-hour urine sample showed a calcium excretion of 660 mg (normal 100–300 mg) and a phosphorous excretion of 1580 mg (normal 600–1200 mg). The bone marrow of this patient was hypercellular with depressed erythropoiesis and a differential count of 12% blasts, 2% promyelocytes, 2% myelocytes, 3% metamyelocytes, 44% neutrophils, 3% lymphocytes, 2% monocytes and 32% eosinophils. The leucocyte alkaline phosphatase score was 42 (normal 35–140) and the trephine biopsy disclosed coarse deposition of reticulin. Photographs of the X-rays and a Technetium^{99m} bone scan images are displayed in figures 1 and 2, respectively.

Figure 1 X-ray of the skull, lateral view

Figure 2 Technetium^{99m} bone scan of the pelvis

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Questions

- 1 What do the haematological parameters and the bone marrow findings of this patient indicate?
- 2 What does the serum biochemistry and analysis of the 24-hour urinary sample disclose? List the different conditions that can cause this state.
- 3 What do the X-ray and the radionuclide scan depict? List the conditions that can manifest with such a picture and give their appropriate treatments.

Answers

QUESTION 1

The haematological profile of this patient with chronic myeloid leukaemia (CML) shows anaemia, leucocytosis and a differential leucocytic count compatible with the basic diagnosis. In addition, eosinophilia and the presence of some immature red cells in the peripheral blood smear may indicate either conversion to the accelerated phase, blast crisis or the presence of myelofibrosis. The diagnosis of CML in the accelerated phase with myelofibrosis is confirmed by the presence of a normal leucocytic alkaline phosphatase score, eosinophilia, anaemia, myelofibrosis and an increased number of blast cells in the marrow (not a blast crisis, in which the blast plus promyelocyte cell count should be more than 30%).^{1,2}

QUESTION 2

The serum biochemistry shows hypercalcaemia with a normal serum phosphorous and raised serum alkaline phosphatase. This profile can occur only in three states:

- excessive parathyroid activity (primary or due to ectopic production)
 - humoral hypercalcaemia of malignancy due to the production of a parathyroid hormone related peptide
 - direct osteolytic activity by malignant cells.
- The state of excessive parathyroid activity and that of its related peptide are accompanied by a low urinary excretion of calcium and hyperphosphaturia. Thus, the high serum calcium in this patient is attributed to direct osteolytic tumour invasion by leukaemia cells.

QUESTION 3

The X-ray of this patient shows generalised osteopenia and numerous lytic lesions in the skull while the radionuclide scan shows many spots of increased radiotracer uptake in the pelvis (multiple foci) indicating the existence of generalised osteoclastic bone resorption. Such a picture can occur with multiple myeloma, hyperparathyroidism and osteolytic secondaries. Myeloma can be discounted on the basis of a normal serum electrophoresis and bone marrow showing only 4% plasma cells, while hyperparathyroidism is ruled out for the reasons mentioned above. Thus, this picture has been caused by direct osteolytic activity of leukaemia cells.

Malignancy-related hypercalcaemia can be adequately treated by hydration to restore the intravascular volume, loop diuretics to enhance calcium excretion in the urine and glucocorticoids for their antitumour effects, until specific treatment for the underlying tumour can be administered. In this case the ideal treatment would be a bone marrow transplantation, pro-

Summary points

- hypercalcaemia as a metabolic emergency occurs in 0.5% of hospitalised patients
- malignancy-related hypercalcaemia usually occurs as a complication of specific tumours (lung, head, neck, breast and oesophagus)
- haematological malignancies account for 10% of all tumour-associated hypercalcaemia
- hypercalcaemia can occur in the accelerated or the blastic phase of chronic myeloid leukaemia and can be adequately treated by hydration, loop diuretics and glucocorticoids

vided that an HLA-matched donor is available. Anticancer drugs like hydroxyurea or cytarabine can be used to control the raised cell count and interferon-alpha in high doses for its antiproliferative effects.

Discussion

Hypercalcaemia is one of the common metabolic emergencies occurring in 0.5% of hospitalised patients.³ Malignancy-related hypercalcaemia is usually seen complicating specific types of tumours, ie, carcinomas of lung, head, neck, breast and oesophagus.³ Carcinoma of the breast accounts for nearly 20–25% of cases, and together with lung carcinoma, the figure approaches 60%.^{3,4}

Haematological malignancies account for 10% of cases of tumour-associated hypercalcaemia, the majority of which are leukaemias, myelomas, lymphomas and HTLV-I associated leukaemias/lymphomas.³

Hypercalcaemia associated with CML, with or without destructive bone lesions, is uncommon.^{3,5} This hypercalcaemia is usually attributed to bone destruction by the leukaemic cells⁶ but ectopic production of parathyroid hormone has been seen in one case, while another case report has documented increased levels of urinary cyclic adenosine monophosphate.⁷ In contrast, most of the other reported cases of CML presenting with hypercalcaemia were conspicuous by normal levels of parathyroid hormone and cyclic AMP.^{5,8,9} The survival time of CML patients after the recognition of hypercalcaemia is reported to be from a few days to 7 months.^{5–10}

Final diagnosis

Malignancy-related hypercalcaemia in a patient with chronic myeloid leukaemia.

Keywords: malignancy-related hypercalcaemia; chronic myeloid leukaemia

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Renal calculi following superior mesenteric artery occlusion

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A 48-year-old woman developed an occlusion of the superior mesenteric artery resulting in infarction of the jejunum, ileum and proximal colon up to the splenic flexure. She underwent extensive small intestinal resection followed by a jejuno-colic anastomosis; the residual jejunal length was 35 cm. She was morbidly obese prior to intestinal resection (weight 102 kg; BMI 45.7) and steadily lost weight following the surgery. Initially, she experienced marked diarrhoea post-resection; this improved until she was experiencing four to eight bowel movements daily.

She re-presented two years later with painless haematuria when she was found to have multiple calculi in the left kidney. She underwent lithotripsy following which a temporary renal stent was inserted for six months. Baseline serum electrolytes, calcium and liver profiles as well as 24-hour urine calcium and urate excretion at this time were normal. She had significant renal impairment with subnormal creatinine clearance as well as raised serum creatinine and urea following this episode, which resolved partially. She was advised to go on a low-calcium diet.

She was re-evaluated three years following intestinal resection when her body weight was 53.5 kg (BMI 23.6). She continued to have frequent bowel movements and was almost certainly mal-absorbing. She had noticed foul smelling and pale faeces which were difficult to flush. Cholestyramine therapy had no effect on the diarrhoea although some relief was obtained with codeine phosphate.

Questions

- 1 What is the cause of the renal calculi in this woman?
- 2 Comment on the dietary advice she was following at the time of presentation.
- 3 What further investigations would you perform?
- 4 What further therapy would you consider?

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