tration was 87 g/l, platelet count was 123 × 10^9/l and lactic dehydrogenase was 939 U/l.

Surface membrane markers were identified by indirect immunofluorescence and examined with a FACScan flow cytometer (Becton Dickinson, Mountain View, California, USA). The following antibodies were analysed: T3, T4, T8, J5, B4, B1 (Coulter Clone); LeuM5, Leu15, Leu14, IL-2, Leu17 and Leu8 (Becton Dickinson); anti-LFA 1 and anti-βLFA 1B (Janssen); Cris 1 (Dr R Vilella, Hospital Clinic Provincial, Barcelona); FMC7 (Sera-Lab); I0B8 (Immunotech); and surface immunoglobulins (Kallestad). Mouse rosettes were also sought. Detailed results are shown in the table.

B cell chronic lymphoid leukaemias comprise a broad spectrum of lymphoid proliferations classified according to the cytological and phenotypic features of the leukaemic cells.[3] Our case was a mantle zone lymphoma in leukaemic phase, which is a rare form of B cell chronic lymphoid leukaemia (B-CLL). Cytologically, the leukaemic cells had pronounced heterogeneity of size and a fairly pleomorphic appearance. The surface marker analysis of the leukaemic cells (table) showed a monoclonal B cell proliferation that was not characteristic of classic B-CLL. Surface immunoglobulin was strong, FMC7 was positive, and there was no formation of mouse rosettes. All these features differ from typical B-CLL leukaemia but resemble the surface phenotype of prolymphocytic leukaemia and that of follicular lymphoma in leukaemic phase. Overall, it seems that the characteristic phenotypic profile of mantle zone lymphoma in the leukaemic phase includes strong surface immunoglobulin and positivity for FMC7 and CD5. Reactivity with CD10 and mouse rosette formation is variable. Data on the antibodies Leu8, CD11, CD22, CD23, CD25 and CD38 are scarce. Further studies are needed to clarify precisely the phenotype of this particular lymphoid leukaemia.

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Hypercalcaemia and osteolytic lesions associated with chronic lymphatic leukaemia (CLL)

Case 1

A 72 year old man had cervical and axillary lymphadenopathy and an enlarged spleen palpable 1 cm below the left costal margin. A blood count showed that his haemoglobin concentration was 11·5 g/dl (normal range 12·5–16 g/dl), his white cell count was 114·0 × 10^9/l (normal range 4·0–10·0 × 10^9/l), his lymphocytes were 105 × 10^9/l and his platelet count 250 × 10^9/l (normal range 150–400 × 10^9/l). A biochemical screen, including that for serum calcium concentration, was normal. A bone marrow aspirate and trephine biopsy specimen showed diffuse infiltration with small mature lymphocytes, and chronic lymphatic leukaemia (CLL) was diagnosed. The disease was easily controlled by short, intermittent courses of chlorambucil.

Three years from diagnosis and while not receiving treatment, the patient was admitted with a two week history of thirst, malaise, and vomiting. Examination showed that he was dehydrated, had enlarged cervical lymph nodes, an enlarged liver palpable 3 cm below the right costal margin and an enlarged spleen palpable 4 cm below the left costal margin. The haemoglobin concentration was 9·1 g/dl, the white cell count 14·8 (small mature lymphocytes 9·1 × 10^9/l, pro-lymphocytes 3·9 × 10^9/l), and the platelet count 142 × 10^9/l. Serum calcium was 3·66 mmol/l (normal range 2·20–2·65 mmol/l), phosphate 0·9 mmol/l (normal range 0·70–1·30 mmol/l), and alkaline phosphatase activity 101 IU/l (normal range 28–142 IU/l). Serum albumin was 34 g/l (normal range 35–45 g/l). The urea, creatinine, and electrolyte concentrations were normal. The serum parathormone concentration was < 0·1 μg/l (normal range < 0·5 μg/l) and vitamin D concentration was 10 μmol/l (normal range 15–100 μmol/l). A skeletal survey showed generalised osteoporosis and multiple lytic lesions throughout the skull. No serum or urinary paraprotein was detected.

Treatment with chlorambucil 6 mg/day, prednisolone 40 mg/day, frusemide 40 mg/day and intravenous fluids was begun, and after three days the calcium had fallen to 3·0 mmol/l. Intravenous mithramyacin (25 μg/kg/day) for three days was given, after which the calcium concentration was 2·05 mmol/l. Two weeks later a further course of mithramycin was necessary as the calcium concentration had risen to 3·7 mmol/l. A further short-lived response was achieved but three weeks later the patient fell, fractured his femur and pelvis, and died shortly afterwards from bronchopneumonia.

Case 2

A 70 year old woman had Binet stage A CLL. No treatment was needed for four years after which short intermittent courses of chlorambucil controlled a rising lymphocyte count and lymphadenopathy.

About six years after diagnosis she fell and fractured the left humerus. Radiographs showed lytic lesions at the site of fracture and also throughout the skeleton. She had progressed to stage C CLL at this time. There was no evidence of a second primary malignancy. During the next six months further lytic lesions developed in association with severe generalised osteopenia. Crush fractures of several vertebrae occurred. Death from bronchopneumonia ensued 10 months after she fractured her humerus.

Biochemistry screens (including serum calcium, phosphate, and alkaline phosphatase) were normal throughout the last year of life and no serum or urinary paraprotein was present.

Hypercalcaemia is a rare complication of CLL which occurs most frequently in the setting of advanced disease.[1] Hypercalcaemia, however, has also been reported in patients with early stage disease but in many of these patients coincidental primary hyperparathyroidism has been found.[2] Where hyperparathyroidism is not detected the cause of the hypercalcaemia has been attributed to increased osteoclastic activity secondary to secretion of osteoclast activating factor by malignant lymphocytes.[3]

The prognosis for patients with advanced disease and normal or low serum parathyroid hormone activity was generally measured in weeks despite treatment directed at both the CLL and the hypercalcaemia.[1] In contrast, hypercalcaemia complicating early stage disease or secondary to hyperparathyroidism may be associated with survival for several years.[2 4 5]

Hypercalcaemia and osteolytic bone lesions may complicate CLL. The prognosis is generally poor but primary hyperparathyroidism should be excluded as this group of patients, if correctly treated, fare much better.

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Thoracic aortitis due to salmonella

Case report

A 62 year old college lecturer was admitted with a six month history of night sweats, arthralgia, and lethargy. Two weeks before admission he developed haemoptysis, hoarseness, and continuous left shoulder pain. There was no history of recent foreign travel, nor diarrhoeal illness in the patient or family, nor a notable medical history. On examination he had fluctuating fever up to 38·5°C. His blood pressure was 110/80 mm Hg in both arms with a systolic murmur at the left sternal edge and a pericardial rub. A chest *x* ray picture, which had been normal four months earlier, showed a left hilar mass. His white cell count was raised at 18·6 × 10^9/l, with an erythrocyte sedimentation rate of 116 mm/hr. C3d was moderately increased at 17 units/ml and an IgG cryoprotein was detected. Six blood cultures and culture of urine were negative. At bronchoscopy the left vocal cord was seen to be paralysed, with extrinsic compression of the trachea and left main bronchus. Culture of bronchial washings was negative. A computed tomogram of the thorax (figure) showed aneurysmal dilatation of the thoracic aorta; this was confirmed at