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: T4, T8, T1, B4, B1 (Coulter Clone); LeuM5, Leu15, Leu4, IL-2, Leu17 and Leu8 (Becton Dickinson); anti-LFA1 and anti-β3-LFA 1B (Janssen); Cris 1 (Dr R Villela, Hospital Clinic Provincial, Barcelona); FMC7 (Sera-Lab); 1088 (Immunotech); and surface immunoglobulins (Kallestad). Mouse rosettes were also sought. Detailed results are shown in the table.

B cell chronic lymphoid leukemias comprise a broad spectrum of lymphoid proliferations classified according to the cytological and phenotypic features of the leukemic cells. 1 Our case was a mantle zone lymphoma in leukaemic phase, which is a rare form of B cell chronic lymphoid leukemia (B-CLL). Cytologically, leukemic cells had pronounced heterogeneity of size and a fairly pleomorphic appearance. The surface marker analysis of the leukemic cells (table) showed a monoclonal B cell proliferation that was strongly positive for the surface membrane IgM 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 leukemia but resemble the surface phenotype of prolymphocytic leukemia and that of follicular lymphoma in leukaemic phase. Overall, it seems that the characteristic phenotypic profile of mantle zone lymphoma in the leukaemic phase is a strong surface membrane IgM 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 leukemia.

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: 12.5–16.5 g/dl), his white cell count was 1.14 × 10^9/l (normal range 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 weight loss. Examination showed 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 2.15–2.65 mmol/l), serum phosphate 0.9 mmol/l (normal range 0.70–1.30 mmol/ml), 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 mmol/l (normal range 15–100 μmol/l). A bone scintigraphy showed focal bone lesions of porosis and multiple lytic lesions throughout the skull. No serum or urinary paraprotein was detected.

Treatment with chlorambucil 6 mg/day, prednisolone 40 mg/day, frusemid 40 mg/day and intravenous fluids was begun, and after three days the calcium had fallen to 3.0 mmol/l. Intravenous thiamylamin (25 μg/kg/day) for three days was given, after which the calcium concentration was 2.95 mmol/l. Two weeks later a further course of thiamylamin 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.

Thoracic aorticosis 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 his contacts, nor a notable medical history. On examination he had fluctuating fever up to 38°C. His blood pressure was 110/80 mm Hg in both arms with a systolic murmur at the left sternal edge and a pectoral rub, a crepitant picture, which had been normal for 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/h. Urinalysis showed 200,000 white cells/l and 150 red cells/l, with a proteinuria of 0.8 g/day. A chest radiograph showed a left hilar mass, large para-aortic lymph nodes, and pleural effusion on the left. A chest and abdominal CT scan showed a left hilar mass, enlarged lymph nodes, and a pleural effusion on the left. A transbronchial biopsy of the left hilum revealed a catarrhal bronchitis with an acute inflammatory cell infiltrate. A diagnosis of salmonella aortitis was made.

The patient was treated with levofloxacin 500 mg/day for seven days, followed by azithromycin 500 mg/day and rifampicin 600 mg/day for 12 weeks. He made a full recovery and was discharged home after 4 months. Follow up three months later showed no recurrence of his symptoms.
MATTERS ARISING

Risk of metastasis during fine needle aspiration

Denton et al. expressed the opinion that there is a systematic underestimation of the risk of metastasis during needle biopsy.¹ This topic is indeed worthy of discussion.² The true incidence of these accidents, however, is and probably always will be impossible to assess. Not all cases are diagnosed, nor are they reported: it seems remarkable that not one case of peritoneal metastasis after needle biopsy has ever been reported. The variability of survival is also of great importance: 20%, of the reported subcutaneous metastases are detected after four years or more.³

Good indications of the true incidence were given by Smith,¹ who showed that the actual risks of metastasis after needleling were very low (of the order of 0.5/10 000). Bleeding and sepsis after needle biopsy are at least 10 times more common than metastasis.

Puzzled by the question of the number of metastases and being unable to obtain a satisfactory scientific answer, we thought it more relevant to examine the circumstances in which they occurred and found that the occurrence of metastasis seemed to be associated with large needles, core biopsy devices, high numbers of passes, and absence of normal parenchyma covering the tumour.²

Accordingly, we evolved a golden rule for needle biopsy: one pass with a fine needle (22 gauge or larger) through normal parenchyma.² This seems to be well advised because we were unable to find any report of metastasis in such circumstances.

When it can be calculated, the risk of metastasis seems to grow exponentially—for instance, increasing the needle diameter by a factor of 2 increased the seeding by a factor of 60² (without improving diagnostic efficiency).²,³

In our opinion good practice is that needle biopsies of solid masses should be performed by (i) trained teams, (ii) only when taking decisions about the patient's management, (iii) through normal parenchyma, whenever possible, respecting anatomical boundaries, (iv) always with a fine non-cutting needle, (v) the sampling has to be done under suction, which must be maintained when withdrawing the needle, (vi) the sample quality has to be checked later to keep the number of passes to the very minimum.

In our opinion the case² referred to accumulated risk factors, and should, in no way, be used to affirm that the rate of metastasis after needle biopsy, and especially fine needle aspiration, is higher than is usually thought. It could serve, instead, to restate the risk factors and how they can be avoided. Large cutting needles, in particular, should not be used when cancer is suspected.

**References**
