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 using a FITC-Scan flow meter (Becton Dickinson, Mountain View, California, USA). The following antibodies were analysed: T4, T8, T4, B5, B4, B1 (Coulter Clone); LeuM5, Leu15, Leu4, IL-2, Leu17 and Leu8 (Becton Dickinson); anti-LFA 1 and anti-β2LFA 1B (Janssen); Cris 1 (Dr R Viliea, Hospital Clinic Provincial, Barcelona); FMC7 (Sera-Lab); 10B8 (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.1 Our case was a mantle zone lymphoma in leukaemic phase, which is a rare form of B cell chronic lymphoid leukaemia (B-CLL). Cytoplasmic immunoglobulins, usually κ light chain, were found in 40% of the cases. The lymphoma cells characteristically 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 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 should be 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.

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 1.14 × 10^9/l (normal range 4×10^9–10×10^9/l) and 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 3×10^9/l) and the platelet count 142 × 10^9/l. Serum calcium was 3.66 mmol/l (normal range 2.50–2.80 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 0.5–3 μg/l) and vitamin D concentration was 10 μmol/l (normal range 15–100 μmol/l). A chest x-ray was of normal size and density. Bone scan showed no evidence of bone lesions, and all of the bone biopsies were normal. A diagnosis of CLE was made.

Treatment with chlorambucil 6 mg/day, prednisolone 40 mg/day and intravenous fluids was begun, and after three days the calcium had fallen to 3.0 mmol/l. Intravenous mithramycin (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 a year after the final 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 secondary malignancy. During the next six months further lytic lesions developed in association with severe generalised osteoporosis. Crush fractures of several long bones developed. 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 protein was present.

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

The prognosis for patients with advanced disease and normal or low serum parathyroid hormone activity was generally measured in weeks since initial diagnosis at both the CLE and the hypercalcaemia. In contrast, hypercalcaemia complicating early stage disease or secondary to hyperparathyroidism may be associated with survival for several years.44

Hypercalcaemia and osteolytic bone lesions may complicate CLE. 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 salmonellosis
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 hae-moptysis, hoarseness, and continuous left shoulder pain. There was no history of recent foreign travel, nor diarrhoeal illness in the patient or his 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 border. The chest x-ray was not notable (figure). 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 in the first hour. A fall in the white cell count of 109/l, a urine sample for mycobacteria was processed, and 250 ml of 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

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Computed tomogram of the thorax showing aneurysmal dilatation of the thoracic aorta containing thrombus.

As aortography is a false aneurysm of the aortic arch which had originated at the left subclavian artery.

At thoracotomy an apparent hole in the aorta was oversewn with removal of slough from a false aneurysmal cavity adjacent to the upper lobe of the left lung. Cultures from pericardial tissue, the wound drain, and stools grew Salmonella enteritidis. He was given intravenous ampicillin 2 g four times a day. While apparently recovering, he suddenly collapsed and died 18 days after surgery from a massive haemoptysis. At necropsy it was impossible to identify the source of blood loss. The left lung contained a ragged cavity partly lined with thrombus in its upper lobe, with no evidence of vascular patency into this area. There was a small true aneurysm between the left carotid and subclavian ostia where a suture line was intact. The aorta showed moderate to severe atherosclerosis. Although Salmonella thoracic aortitis is rare, salmonellas have been incriminated in 18-35% of infected aeurysms.1 The elderly are particularly liable to the complications of salmonella bacteremia, with 25-30 developing an endocardial infection.2 Clinical presentation may be with chronic sepsis unresponsive to antibiotics,3 or with features of a primary focus, such as osteomyelitis. Alternatively, the symptoms may relate to the presence of an aneurysm or its rupture. Frequently there may be no such signs, even after rupture.4 A review of 34 cases of infected aortic aneurysms reported fever in all cases, positive blood cultures in 53%, and preoperative rupture in 79%. In 73%, of cases the patient was over 60 years old.5 The tendency for Gram negative infections to progress rapidly and rupture6 makes urgent surgery imperative following diagnosis. If not treated surgically, myotic aneurysms are invariably fatal.7 Successful management requires prompt diagnosis, appropriate antibiotics based on culture sensitivities, and intraoperative Gram stain with culture of the aorta and contents. Wide resection of infected tissue is necessary, with extra-anatomic grafting through clean tissue planes.8 Cholecystectomy has been advocated if Salmonella is identified preoperatively, as the biliary tree is often a sanctuary for organisms and for continued sepsis.9

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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 even 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,4 who showed that the actual risks of metastasis after needle 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.5 According to us, the evolution of a golden rule for needle biopsy: one pass with a fine needle (22 gauge or larger) through normal parenchyma.6 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 two increased the seeding by a factor of 60 (without improving diagnostic efficiency).7,8

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 case1 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 emphasize the risk factors and how they can be avoided. Large cutting needles, in particular, should not be used when cancer is suspected.

References


Drs Denton and Cotton comment: We are grateful to Dr Roussel for his comments, and we agree with several points, including his assessment of risk factors. While it is true that most of the measures given should reduce the risk of needle track metastasis, we would dispute his suggestion of maintaining suction while withdrawing the