Lymphadenopathy
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Pombo
Proposals for the classification of chronic (mature) monoclonal B cell leukaemias.
The chronic lymphoid lymphoma in mantle had arisen 10 months from
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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: 12.5-16.0 g/dl), his white cell count was 13.4 x 10^9/l (normal range 4.0-10.0 x 10^9/l), his lymphocytes were 105 x 10^9/l and his platelet count 250 x 10^9/l (normal range: 150-400 x 10^9/l). A biochemical screen, including that for serum calcium concentra-
tion, 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 x 10^9/l, prolymphocytes 3.9 x 10^9/l), and the platelet count 142 x 10^9/l. Serum calcium was 3.66 mmol/l (normal range 2.50-2.65 mmol/l), serum 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 serum, creatinine and electrolyte concentra-
tions were normal. The serum parathormone concentration was 0.01 mg/l (normal range 0.5-5 mg/l) and vitamin D concentration was 10.0 mmol/l (normal range 15-100 mmol/l). A skeletal survey and bone biopsy showed osteoporosis and multiple 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 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 after 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 secondary malignancy.

During the next six months further lytic lesions developed in association with severe generalised osteopenia. Crush fractures of several vertebrae 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 paraprotein was present.

Hypercalcaemia is a rare complication of CLL which occurs most frequently in the setting of advanced disease. Hypercalcaemia, however, has also been reported in patients with early stage disease but in many of these patients coincidental primary hyperpara-
thyroidism has been found. Where hyper-
parathyroidism 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.

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. In contrast, hypercalcaemia complicating early stage dis-
tease or secondary to hyperparathyroidism may be associated with survival for several years. Hypercalcaemia and osteolytic bone lesions may complicate CLL. The prognosis is generally poor but primary hyperpara-
thyroidism 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 his family, nor a notable medical history. On examina-
tion 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 on chest x-ray picture, which had been normal four months earlier, showed a left hilar mass. His white cell count was raised at 18 x 10^9/l, with an erythrocyte sedimentation rate of 116 mm/hr and a serum CRP of 150 mg/l. Special study for the presence of rheumatoid factor (RF) and positive antinuclear antibody (ANA) was negative. Urinalysis showed 95% of the urinary output was in the form of red cells and casts, and no microhaematuria was detected. Blood cultures were negative. On echocardiography the left ventricular wall was normal, and no evidence of pericardial effusion was noted.

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


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 will always 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 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.5

Accordingly, we evolved 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 100 (without improving diagnostic efficiency).6,7

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

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3 Smith EH. The hazards of fine needle aspiration biopsy. Ultrason Imaging 1984;10:529-34.
Thoracic aortitis due to Salmonella.

M J Steiger and I D Johnston

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