Bone lesions in chronic granulocytic leukaemia

Sir,

Bone involvement is seen commonly in acute leukaemia but occurs rarely in chronic granulocytic leukaemia (CGL). Among 370 patients with CGL seen between July 1984 to June 1988, 14 (3.7%) developed bone disease. Bone involvement occurred within 1–120 months (median 24 months) of diagnosis and presented with severe localized bone pains (13/14), bony swelling (6/14), difficulty in walking (4/14) and paraplegia (1/14). The various sites were: spine-4, pelvis-3, skull-3, tibia-4, and femur, mandible, sternum, talus in one patient each. The lesions were single in 10 and multiple in 4 patients. Radiologically the lesions were osteolytic permeative in 10, multiple punched out in 2 and due to partial collapse of the spine in 4 patients. Peripheral blood and bone marrow examination revealed: chronic phase in 8 patients and accelerated phase and lymphoid blast crisis in 4 and 2 patients respectively. Serum calcium was normal in all patients on at least two repeated occasions. Local radiotherapy in 11 patients resulted in significant relief of symptoms. Three patients were lost to follow-up after the diagnosis of bone lesions.

Three to ten per cent of CGL patients may develop bone involvement during the course of the disease. The most common presenting symptom is localized bone pain followed by bony swelling. Rarely, patients may present with neurological deficits, pathological fracture, or hypercalcaemia. The ends of long bones, the spine, skull and pelvis are commonly involved. Sternum, mandible and metacarpal may, as seen here, be rarely involved. The lesions may be single, multiple, discrete, punched out or ill-defined. Osteolytic lesions are commonest. Rarely, there may be osteosclerotic or mixed lytic-sclerotic lesions due to myelofibrosis which may complicate the course of CGL. Bone disease due to infection, an unrelated second neoplasm and infarction should also be kept in mind.

Bone lesions are most commonly seen in the late stage of the disease during accelerated phase/blast crisis. Their association with the chronic phase is rare. Interestingly, 8 out of our 14 patients were in the chronic phase with bone involvement. It is speculated that presence of a bone lesion due to leukaemic infiltrates in the chronic phase of CGL may indicate the initial site of clonal proliferation extending later into the remainder of haemopoietic tissue—a situation similar to extramedullary blast crisis described for infiltrates into skin, lymph nodes, breast, meninges etc. This is further supported by the development of a blast crisis in 6 of these patients within 2–12 months. We used hydroxyurea in the chronic phase patients. Whether the use of aggressive chemotherapy in these chronic phase patients could have delayed the onset of blast crisis remains speculative. Here, localized radiotherapy resulted in marked symptomatic relief in all patients, suggesting that this should be used in addition to systemic chemotherapy.

Our experience and other publications suggest that once the bone lesions develop the course is rapidly progressive. The development of blast transformation results in a rapid clinical deterioration and short survival.

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References

Salmonella enteritidis empyema complicating lupus pleuritis

Sir,

In the absence of concurrent pulmonary infection pleural empyema caused by non-typhoid salmonella is a rare event. In two recent reviews only 16 cases were recorded.

Systemic lupus erythematosus (SLE) predisposes to severe salmonella infections, which frequently localize at a site of clinical SLE involvement. Colonization of sterile serous effusions by salmonella has been reported in other diseases, but not in SLE. We report a patient with long standing lupus pleural effusion who developed bilateral pleural empyema caused by Salmonella enteritidis.

A 26 year old man had SLE diagnosed in 1983. Lupus manifestations had been intestinal vasculitis, arthritis, polyserositis, cutaneous lesions, proteinuria, and interstitial cystitis. Treatment had included prednisone and azathioprine for long periods.

In March 1989 he complained of exertional dyspnoea and a chest X-ray showed bilateral pleural effusion. The pleural fluid was not analysed. The corticosteroid dose was increased, but the effusions persisted. Nine months later, the patient presented with a 5-day history of fever, cough, right pleuritic pain and dyspnoea at rest. A chest
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