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Leukaemic tumefaction in soft tissues

All types of leukaemia may give rise to tumours in sites not normally associated with haemopoiesis or lymphopoiesis. Chloroma (granulocytic sarcoma) results from the localised proliferation of myelogenous leukaemia cells; lymphocytic sarcoma derives from the cells of acute or chronic lymphocytic leukaemia. Extramedullary acute lymphocytic leukaemia occurs most frequently in the central nervous system and gonads,¹ and is found in a high proportion of patients. In contrast, pigmented (chloromas) and non-pigmented granulocytic sarcomas may occur anywhere in the body, and their incidence is low: one study found granulocytic sarcomas in only 3.1% of 478 patients with acute or chronic myelogenous leukaemia.² Granulocytic sarcomas are found most often in bones, particularly those of the face and cranium. The ribs, vertebrae, and pelvis may also be affected, but granulocytic sarcomas of the long bones are seen less often. The brain and spinal cord are never directly invaded, though extradural tumours may cause neurological symptoms. Tumour formation is rare in organs such as the liver, pancreas, thyroid, and breasts.³⁻⁶

Why there should be both generalised and localised phases of leukaemic cell growth remains unknown. Growth of a single tumour might be determined by local factors, but the presence of multiple deposits in different sites makes this explanation unlikely. Factors related to the leukaemic cells themselves seem more probable determinants. Differentiation of normal haemopoietic cells is associated with the development and loss of cell-surface antigens.⁷⁻⁸ Normal haemopoietic stem cells in the peripheral blood of the mouse, for example, are antigenically different from those in the bone marrow,⁹ which suggests that cell-surface properties may be one factor determining the release of stem cells from the marrow cavity. Furthermore, studies of Moloneyvirus leukaemia in mice have shown that interactions with viruses may result in the appearance of virus-specific surface antigens, which may vary in density in individual leukaemias.^{10,11} Indeed, some forms of virus-induced murine leukaemia may start as a solid tumour.¹² Thus one possible explanation for the different distributions of leukaemic cells might be the acquisition of inappropriate differentiation-associated antigens or virus-associated antigens.

The occurrence of extramedullary acute lymphoblastic leukaemia in pharmacological "sanctuaries" such as the central nervous system and testis,¹ and, less frequently, the anterior chamber of the eye,¹³ remains an important clinical complication. Vigorous measures are taken routinely nowadays to eradicate these cells. In contrast, though tumours have occasionally been reported to antedate the appearance of leukaemia,^{14,15} most granulocytic sarcomas are found at necropsy. There is no firm evidence for the preferential occurrence of granulocytic sarcomas in sanctuary sites, though the tendency for acute myelogenous leukaemia to recur at an original extramedullary site¹⁶ suggests that the tumour cells may be protected or resistant to chemotherapy. Since the unpredictable nature of granulocytic sarcomas does not permit prophylactic treatment they have to be treated locally as and when they are discovered. On the rare occasions that a granulocytic sarcoma presents without evidence of leukaemic change in the bone marrow, the leukaemia-free marrow might in theory be harvested in advance of medullary disease and stored in liquid nitrogen. More aggressive chemotherapy could then be given, with later infusion of the patient's own haemopoietic stem cells.

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Correction

Interferon: therapeutic fact or fiction for the '80s?

We much regret that an error occurred in the Regular Review on "Interferon: therapeutic fact or fiction for the '80s?" by G M Scott and D A J Tyrrell (28 June, p 1558). In fact, in the study of renal transplant recipients given interferon as a prophylaxis against reactivation of viral infections,²⁸ interferon was given twice weekly, not daily as stated in the text and table.