histological changes of sarcoidosis. She was followed for a further four years without further incident. At no time during the 11 years that she was under supervision was evidence found to suggest a sarcoid lesion anywhere else than in her right lung and right breast.

Comment

Goldman has recently drawn attention to the rare occurrence of tuberculosis in breast tissue.² This case may help to remind clinicians of the need to consider granulomas in the differential diagnosis of mammary disorders.

I thank Dr R I K Elliott for providing the photomicrograph.

¹ Scadding, J G, *Sarcoidosis*, p 385. London, Eyre and Spottiswoode, 1967.

² Goldman, K P, *Tubercle*, 1978, **59**, 41.

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Transient myeloid and erythroid aplasia associated with infectious hepatitis

Bone marrow aplasia is an uncommon but serious sequel to acute infectious hepatitis. This form of aplasia occurs predominantly in teenagers and young adults, typically running a protracted course with a mortality of 85% during the first 16 months in 174 cases reported up to 1975.¹ Early bone marrow transplantation has been advocated because of this ominous prognosis.² We have, however, observed a case of marrow depression associated with acute infectious hepatitis which displayed features departing considerably from the reported pattern: the aplasia did not affect megakaryocytes, and complete haematological recovery occurred after four weeks of myeloid and erythroid aplasia.

Case report

A 26-year-old woman in the third trimester of her second pregnancy developed polyarthralgia, malaise, and maculopapular rash, followed after five weeks by features of acute hepatitis. The liver was moderately enlarged and there was no splenomegaly or lymphadenopathy. Sole exposure to drugs had been penicillin administered during the preceding week. Plasma concentration of bilirubin was 20 $\mu\text{mol/l}$, alkaline phosphatase 370 IU/l, and

aspartate transaminase 220 IU/l. Radioimmunoassay showed no hepatitis B surface antigen or antibody. IgA antibody to hepatitis A was detected, but there was no IgM antibody, indicating past but not recent infection with hepatitis A.³ No rise in titre occurred in antibodies to toxoplasma or cytomegalovirus, and no antinuclear factor or heterophile antibody was detected. Antibody to Epstein-Barr virus was not determined. Liver function deteriorated further, and three weeks later a healthy premature infant was delivered. At this time the patient developed fever, and severe neutropenia was detected. Marrow aspiration and trephine showed considerable erythroid and complete myeloid depression but normal numbers of megakaryocytes. Severe neutropenia persisted for 27 days and was associated with high fever. Treatment consisted of intravenous antibiotics, bowel sterilisation, and 17 leucocyte transfusions. Extensive postpartum haemorrhage secondary to clotting factor deficiency was treated successfully with fresh frozen plasma. The platelet count remained normal throughout the illness except for a transient fall during the haemorrhage, and was attributed to effects of transfusion with 26 units of blood in four days. Weekly studies showed no change in the bone marrow until neutrophils reappeared in peripheral blood on day 27. The aspirate then showed 33% erythroblasts and 62% granulocyte precursors. The fever resolved, and normal blood counts were permanently restored after two days. Resolution of abnormal liver function required three months before serum alkaline phosphatase became normal. Needle biopsy of the liver on day 28 showed a picture consistent with resolving acute viral hepatitis.

Comment

Marrow depression is a well-recognised sequel to acute infectious hepatitis, but this case indicates that there may be greater variability in the marrow injury and its course than had been appreciated.^{1,2} No explanation for the deviation from the usual pattern of protracted aplasia affecting all cell series was evident from the features of the preceding hepatic illness, which satisfied accepted criteria.^{1,2} Serological evidence indicated that the hepatitis was not due to hepatitis B, which was consistent with previous experience,¹ but due to non-A, non-B viral hepatitis.³ The spontaneous recovery of haemopoiesis clearly indicates that progression to protracted aplasia is not inevitable, and that in our patient early marrow transplantation would have been inappropriate. In one other case aplasia affected only myeloid series, but death from sepsis supervened,⁴ so that it remains to be established whether persistence of one or two cell series predisposes to earlier haemopoietic recovery.

One possible pathogenic mechanism is an autoimmune process initiated by viral infection and directed against haemopoietic tissue,⁵ although this case provided no supportive evidence as recovery occurred in the absence of glucocorticoid or cytotoxic immunosuppressive treatment.

We thank Dr B Dwyer for referring this patient.

¹ Hagler, L, *et al*, *Medicine*, 1975, **54**, 139.

² Camitta, B M, *et al*, *Blood*, 1974, **43**, 473.

³ Lehman, N I, and Gust, I D, *Medical Journal of Australia*, 1977, **2**, 731.

⁴ Dische, F E, and Golding, J R, *British Medical Journal*, 1957, **2**, 738.

⁵ Royal Marsden Hospital Bone-Marrow Transplantation Team, *Lancet*, 1977, **2**, 742.

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Course of peripheral blood indices relative to time of delivery.

Intermittent ophthalmoplegia

Intermittent severe ophthalmoplegia with complete normality between attacks occurred over several years in a woman. We present evidence of an unrecognised part of the range of dysthyroid eye disease, and the case shows the initial diagnostic confusion that may occur.

Case report

A 35-year-old housewife was referred to us in 1975 with a two-year history of recurrent episodes of ophthalmoplegia with painful, red, oedematous eyes;