series cells. These latter markers have recently been reviewed by Crocker and Burnett.2 The preparations usually bind to "X-hapten"—that is, 3-fucosyl-N-areetyl-lactosamine (3FNa) and include Leu M1, 3C4, VEP8 and 9, and AGF 4-48.2 The epitopes labelled by these antibodies, however, are widespread in many tissues, including those of epithelial type.2 It is interesting to presume that shared epitopes imply a common ontogeny, but, it would be interesting to speculate on a common ancestry between granulocytes and Reed-Sternberg and Hodgkin's cells.

Accordingly, we applied two antisera to cathepsin G3 and leucocyte elastase4 to a series of 35 cases of confirmed Hodgkin's disease. These comprised seven each of: lymphocyte predominant, nodular sclerosing type I and type 2, mixed cellularity, and lymphocyte depletion Rye subtypes. The antibodies have been shown to be highly specific for granulocyte series cells of maturation stages from promyelocytes onwards, including some myeloblasts.5 Activity of cathepsin G has not been observed in other tissue types, and leucocyte elastase has only otherwise been seen in ileal epithelium.3

The antisera were applied to paraffin sections using standard indirect peroxidase, streptavidin-biotin, and immunogold-silver (IGSS) labelling methods.5 Mature granulocytes were intensely and consistently stained, but only very occasional Reed-Sternberg and Hodgkin's cells reacted; when this occurred, the staining was very weak, even with the IGSS method.

In view of the high specificity of the antisera for granulocyte series cells and the high sensitivity of the IGSS method the findings suggested that if indeed Reed-Sternberg and Hodgkin's cells are related to granulocytes, then they share features only in terms of minor epitopes such as 3FNa, which, themselves, are expressed only on cells from the promyelocytic stage of differentiation.

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References


Aplastic crisis in haemolytic anaemia not associated with human parvovirus infection

Human parvovirus (HPV) infection causes aplastic crises in children with chronic haemolytic anaemia, but cases of transient erythroblastopenia not associated with HPV infection in previously healthy children have also been described.2 The aetiological agent is not thought to be the same in transient erythroblastopenia of childhood and aplastic crises in haemolytic anaemias.

During a period of 23 months from March 1984 to January 1986, we saw 24 patients with haemolytic anaemias presenting with an aplastic crisis (nine with hereditary spherocytosis, six sickle cell anaemias, five thalassaemias, one haemolytic anaemia with dyserythropoiesis, and three autoimmune haemolytic anaemias. HPV was isolated by couterimmune-electrophoresis in one patient with sickle cell anaemia, and serological evidence of recent HPV infection was confirmed by the presence of specific anti-HPV IgM (radioimmunoassay) in 18 others. In the remaining five patients (three with sickle cell disease, two with autoimmune haemolytic anaemia), no marker of HPV infection was found. Other infections excluded were infectious mononucleosis, cytomegalovirus, toxoplasmosis, hepatitis A and B, mumps and rubella. Folic acid concentration was normal in all five.

Our experience confirms that HPV is the major aetiologial agent of aplastic crises in patients with chronic haemolytic anaemias (79.2% of our series). The absence of a previous crisis in all 24 patients superficially suggests that the virus might be the only one responsible for such events, but failure to find HPV or specific IgM in five patients implicates other agents as well. Of course, we do not know if the aetiologial agent in transient erythroblastopenia of childhood and in aplastic crisis of haemolytic anaemias not associated with HPV infection is the same.

Letters to the Editor

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Immune thrombocytopenia induced by cephalosporins specific for thiomethyltetrazole side chain

Cephalosporins have only rarely been reported as a cause of thrombocytopenia. Previous reports having been associated with cephalothin1–3; in only one of these was the presence of antibody associated with the drug shown directly.1 Specific structures with a role in the antigen-antibody interaction have not been identified previously. No report has been found selectively implicating second and third generation cephalosporins as a cause of immune thrombocytopenia.

A 69 year old man was admitted to hospital in November 1984 with chronic staphylococcal cellulitis of the leg of three months duration. He had long standing rheumatoid arthritis and ischaemic heart disease. Previous adverse drug effects included necrotic syndrome following penicillin and gastritis after naproxen and indomethacin. The cellulitis had persisted despite treatment with erythromycin, sodium fusidate, cindamycin, rifampicin, fluoxacillin and gentamycin.

In December 1984, the fluoxacillin and gentamycin were stopped and cephalomandole initiated intravenously 1 g five times...