Papular acrodermatitis with cytomegalovirus hepatitis

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SUMMARY The syndrome of papular acrodermatitis of childhood with hepatitis (Gianotti-Crosti syndrome) is classically considered to be associated with hepatitis B surface antigen (HBsAg) infection. We report an infant with the syndrome, but with serological evidence of infection by cytomegalovirus.

Case report

A one year old Christian Arab boy was admitted to hospital because of fever, coryza, cough, and loss of appetite for one week; and jaundice with dark urine for one day. Two weeks earlier his mother had had an influenza-like illness. On examination the patient was febrile and looked ill, with slight dyspnoea, grunting, and frequent coughing spells. The skin and conjunctivae were mildly jaundiced. Lymph nodes were enlarged in the cervical, axillary, and inguinal regions. Scattered moist rales were heard over both lungs. Heart rate was 160/minute; heart sounds, normal; no murmurs. The abdomen was slightly distended. The liver was palpable, firm smooth and not tender 5 cm below the costal margin, and the spleen was felt 2 cm below the costal margin.

Laboratory investigations were as follows: the urine was positive for bilirubin + to ++; the sediment contained 10–15 white cells per high power field. Urine cultures were negative. Blood count showed a haemoglobin concentration of 9.3 g/dl, leucocytes 15 000/ μ l (15×10^9 /l) (polymorphonuclear cells 58%, band forms 10%, lymphocytes 15%, monocytes 10%, and eosinophils 7%), and platelets 120 000/ μ l (120×10^9 /l). Serum total bilirubin was 73.5 μ mol/l (4.3 mg/dl); direct bilirubin 47.9 μ mol/l (2.8 mg/dl). The maximum value for serum bilirubin was recorded on the third day of admission—112.8 μ mol/l (6.6 mg/dl), and decreased thereafter. Serum glutamic oxaloacetic transaminase was 90 IU/dl, with a maximum of 103 IU/dl one week later. Serum values of alkaline phosphatase were around 3000 IU/dl for 6 weeks, and decreased gradually over the following month. Prothrombin was 30% of control. Serological tests were negative for hepatitis B surface antigen (HBsAg) and antibody, and for antibodies to hepatitis A, herpesvirus, adenovirus, cytomegalovirus (CMV), Epstein-Barr virus, and toxoplasma.

Salmonella and brucella agglutination tests were negative. Blood cultures were sterile.

Five days after admission the fever abated, the jaundice was barely apparent, the infant felt well, and he was sent home. One week later crops of a maculopapular, non-pruritic pink coloured rash appeared, spreading symmetrically over the face, buttocks, arms, and legs (Figure). The skin lesions were 3 to 10 mm in diameter, were well demarcated, and did not coalesce. The eruption lasted 20 days and then faded gradually. Histopathological examination of a skin biopsy showed hyperkeratosis, acanthosis, and focal spongiosis of the epidermis; with lymphocytic and histiocytic infiltration of the dermis,

(a)

Figure Flat topped, discrete papular exanthem shown on (a) the face and (b) (over page) lower limbs.

Fig. 1. Papular acrodermatitis. (b)

especially around the blood vessels. The dermal capillaries were dilated and there was swelling of the vascular endothelium. These features are consistent with a diagnosis of Gianotti-Crosti syndrome.² Repeated serological examinations over 10 weeks were negative for HBsAg and antibody, as well as repeated serologic screening tests for hepatitis A, Epstein-Barr virus, adenovirus, herpesvirus, and toxoplasma. Complement fixing antibodies against CMV appeared, however, in rising titres 8 days after the onset of the rash, and were 1:160 after three weeks. By immunofluorescent technique it was found that 80% of the antibodies were IgM. Culture material from skin lesions, saliva, and urine failed to grow CMV.

Over three months of follow up the infant was clinically well. The lymphadenopathy and hepatosplenomegaly gradually receded; serum transaminase

and alkaline phosphatase values became normal. Immunological functions were studied and were found to be normal.

Discussion

The disease complex in the infant we describe was clinically and histologically consistent with the Gianotti-Crosti syndrome by the finding of acute hepatitis along with the typical skin lesions.^{1,2} Contrary to the classic requirements for the definition of this entity, however,¹ our patient had no indication of HBs antigenaemia. A search for HBsAg and antibody was repeatedly negative; instead, serum antibodies to CMV, negative at first, rose to a titre of 1:160 on repeat examinations, indicating that the patient manifested the clinical array of the Gianotti-Crosti syndrome in association with CMV infection.

It now seems that an association of HBsAg with the Gianotti-Crosti syndrome is not a strict prerequisite for the diagnosis, as increasing instances are being reported where no evidence of HBsAg could be found.³ Furthermore, the picture of the typical rash of papular acrodermatitis of childhood occurring with hepatitis has recently been observed with Coxsackievirus A-16⁴ and with Epstein-Barr virus infection.^{5,6} By the present report, infection with CMV is added to the list of aetiological agents capable of causing the syndrome of papular acrodermatitis of childhood with hepatitis.

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