

Short reports

HBsAg-positive giant cell hepatitis with cirrhosis in a 10-month-old infant

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SUMMARY A Japanese boy, who was born to an asymptomatic HBsAg and HBeAg carrier mother, developed acute type B hepatitis at age 6 months. Hepatosplenomegaly and abnormal liver function tests persisted. Liver biopsy at 10 months showed giant cell hepatitis with cirrhosis. He was given alternate-day therapy with prednisolone for 7 months. HBsAg was detectable from ages 6 to 19 months, but not after. Anti-HBs and anti-HBe were not present at any time.

Asymptomatic HBsAg carriers are common in Japan and in other parts of Asia.¹ It is estimated that about 2.5% of Japan's population are asymptomatic HBsAg carriers. In Japan antigenaemia has been shown to be common in infants born to asymptomatic carrier mothers. Vertical transmission of HBsAg appears to be rare in the USA and other Western countries. Liver dysfunction in such cases is generally slight.

We report a case of HBsAg-positive giant cell hepatitis with cirrhosis in an infant born to an asymptomatic carrier mother.

Case report

A 6-month-old boy with jaundice and hepatosplenomegaly was admitted to the Department of Paediatrics, Teikyo University. His condition had been normal at birth, with a birthweight of 3.5 kg, and he had thrived and developed normally for the first 4 months. He was not breast fed. His mother noticed the scleral icterus when he was 5½ months old, and hepatosplenomegaly and moderate jaundice were found at a health clinic a fortnight later. On admission, his liver was palpated 7 cm below the right costal margin, and the spleen could be felt 2 cm below the left costal margin. His growth and development were normal.

Laboratory data on first admission showed total serum bilirubin 5.3 mg/100 ml (91 µmol/l) with a direct fraction of 4.4 mg/100 ml, serum aspartate

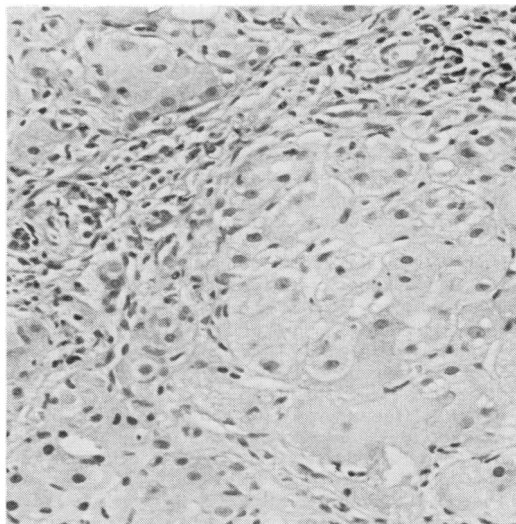


Fig. 1 *Incomplete pseudolobular formation. Fibrous septi from the periportal tract surround the parenchymas which consist of multinuclear giant liver cells with up to 10 nuclei. Vacuolation of the cytoplasm is noticed here and there. The periportal tract contains a slight cell infiltration mainly composed of lymphocytes. (H and E × 160).*

transaminase (AST) 378 U/l, serum alanine transaminase (ALT) 306 U/l, and lactate dehydrogenase 546 U/l. Prothrombin time was 18.4 seconds, partial thromboplastin time 42.4 seconds. Total protein 7.5 g/100 ml (75 g/l), albumin/globulin ratio 1.1; alkaline phosphatase, haemoglobin, white blood cell count, and erythrocyte sedimentation rate were normal. Serum immunoglobulin levels (IgG and IgM) were higher than normal. Serological tests to rubella, cytomegalovirus, toxoplasmosis, and syphilis gave negative results. Serum HBsAg was positive until our patient was 18 months old, and thereafter was not. Anti-HBs was not found at any time. HBeAg and anti-HBe could not be detected.

On second admission, at age 10 months, a liver biopsy was performed. The hepatic lobular architecture was greatly disturbed or even lost, because of irregular fibrous extension from periportal tracts into the parenchyma along sinusoids. There was a tendency towards subdivision of hepatic lobuli, but complete pseudolobuli were not formed. The hepatic parenchyma was composed mainly of multinuclear giant cells, containing up to 10 nuclei. A varying degree of vacuolar degeneration in the cytoplasm was noticed in scattered areas. Pigmentation suggesting iron deposition could not be confirmed. No extramedullary hematopoiesis was observed. Bile thrombi were sparse. In periportal tracts, there was a moderate degree of cell infiltrate composed of neutrophils, eosinophils, lymphocytes, and plasma cells (Fig. 1).

The patient was given alternate-day therapy with prednisolone for 7 months. The levels of serum bilirubin, AST, and ALT returned to normal, but the hepatomegaly persisted (Fig. 2).

This had been the mother's first pregnancy and she was 25 years old. She had no history of blood transfusions or hepatitis, and her liver function tests were normal. HBsAg and HBeAg levels were positive in her serum at all times.

Discussion

Transmission of hepatitis B virus from HBsAg carrier mothers to their infants is common in Japan. It has been reported that the hepatic dysfunction of infants and children who acquire the infection by

vertical transmission of hepatitis B virus from mothers is generally slight.

Chronic liver disease in infants with antigenaemia appears rare, and there have been few reports that associate HBsAg-positive childhood cirrhosis with chronic active hepatitis. Wright *et al.*² reported a 4-month-old girl who developed acute HBsAg-positive hepatitis, which became chronic with persistent hepatosplenomegaly. Liver biopsy at age one year showed active cirrhosis. She had apparently acquired the hepatitis virus from her mother who had been jaundiced at the end of pregnancy. Bancroft *et al.*³ reported a 5½-month-old boy with giant cell hepatitis. Three of the 4 other members of his family were asymptomatic HBsAg-positive carriers. McCarthy's case⁴ was an 8-month-old girl. Her mother had received a blood transfusion 6 years before her pregnancy. The cases of two Japanese patients⁵⁻⁶ were similar. The infant reported here is consistent with other cases. The use of treatment with corticosteroids in our patient had little effect on the hepatomegaly.

Careful follow-up of infants born to hepatitis B-positive carrier mothers is essential. More must be learned about the mode of infection of such infants to make prevention a possibility.

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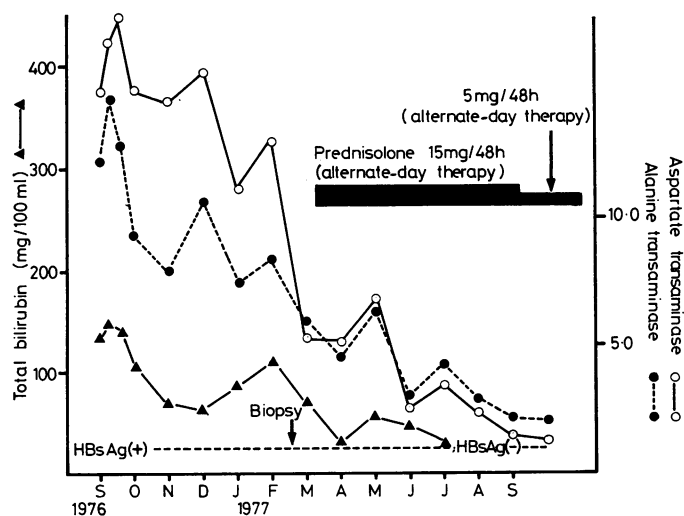


Fig. 2 Clinical course of the patient.

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Disseminated eosinophilic infiltration of a newborn infant, with perforation of the terminal ileum and bile duct obstruction

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SUMMARY A preterm boy died 4 days after delivery from septicaemia which at necropsy was found to be due to perforation of an eosinophilic lesion of the terminal ileum. Eosinophilic infiltration was also found in kidney, lymph node, bone marrow, portal tracts of liver, gall bladder, and bile duct with associated obstruction of the cystic duct and mucocele of the gall bladder. No allergic cause for the infiltrate was found in either the infant or his mother. Eosinophilic infiltration of neonatal spleen, lymph node, intestinal mucosa, epicardium, thymus, pancreas, portal tracts of the liver, and skin has been reported but the aggressive behaviour of the infiltrate in this patient bears more resemblance to the eosinophilic gastroenteritis that has been described in older children and adults.

Eosinophilic infiltration of the alimentary tract of the adult occurs in two forms—namely eosinophilic gastroenteritis and inflammatory fibroid polyp of the gastrointestinal tract—each has been comprehensively reviewed by Johnstone and Morson.¹⁻² The condition is described much less often in children and in two reviews³⁻⁴ only 13 cases were recorded in children under 12 years, the youngest being a 2-year-old boy. Eosinophilic infiltration of the gastrointestinal tract has also been described in neonatal necrotising enterocolitis⁵ but the significance of this infiltrate is unknown. No reference to disseminated eosinophilic infiltration in the neonate followed by intestinal perforation has been found in a search of the recent literature.

Case report

A white boy, weighing 1490 g, was born by spontaneous vertex delivery at 35 weeks' gestation to a healthy 19-year-old rubella-immune group A Rh-positive mother, who had a severe antepartum haemorrhage due to placenta praevia. The pregnancy had been uneventful, apart from vaginal bleeding at 13 and 18 weeks which had settled without treatment on each occasion. No drugs had been taken during the pregnancy.

There was no birth asphyxia, regular respirations were established within 1 minute and, apart from being preterm, there were no abnormalities on clinical examination. 1-mg of vitamin K₁ was given intramuscularly. Intermittent intragastric tube feeding with low solute milk was started and, apart from occasional regurgitation of small amounts of feed, the infant made satisfactory progress. Meconium was first passed at age 8 hours.

At age 48 hours the infant's condition deteriorated suddenly with the clinical picture of peripheral circulatory failure. There was no significant abdominal distention. Cerebrospinal fluid showed only 1 lymphocyte/mm³ and no organisms were found on microscopical examination or culture. The haemoglobin was 10.9 g/dl, packed cell volume 33.9%, and white blood count 1000 × 10⁹/l. The scanty nucleated cells seen in the blood film were either lymphocytes or normoblasts with no increase in eosinophils.

Septicaemia was diagnosed and treatment with intravenous gentamicin and cloxacillin was started.