A case of vertical transmission of hepatitis A virus infection

T Erkan, T Kutlu, F Çullu and GT Tümay
Paediatric Gastroenterology Unit, Cerrahpaşa Faculty of Medicine, University of Istanbul, Istanbul, Turkey

We present a case of hepatitis A infection in a 2.5-month-old male who became icteric after 18 d of birth. The diagnosis of hepatitis A was made by compatible clinical symptoms, laboratory results and liver biopsy showing evidence of hepatitis, and confirmed by detection of anti-HAV IgM antibodies. Because the mother had an acute icteric hepatitis A 1 week before delivery, and the viraemic phase of hepatitis A infection is very short, approximately 7 d, we suggest that the infant was infected by his mother, before birth.

Cholestasis, hepatitis A, vertical transmission

Although there is not sufficient evidence, it is thought that vertical transmission of hepatitis A virus (HAV) is uncommon, because there is a brief viraemic phase early in the incubation period (1, 2). Cases of post-transfusion hepatitis A or nosocomial cases of hepatitis A via asymptomatic infected nurses in a neonatal intensive care unit have been reported (3–6). Cases of prolonged intrahepatic cholestasis due to hepatitis A have also been described (7). This form may be result of the host factors or of the origin of the virus. Complete remission is achieved and chronic hepatitis does not occur.

Vertical transmission has been documented on one previous occasion (8). Therefore, we aimed to present this case, in which a pregnant woman with hepatitis A transmitted HAV to her offspring.

Case report

OT, a 2.5-month-old boy was admitted to hospital with a history of prolonged jaundice and convulsion. The patient was born at 33 weeks gestational age by vaginal delivery, with a birthweight of 2100 g—the first pregnancy of the mother. The postnatal period had been reported as uneventful. Because the mother had an acute icteric hepatitis A 1 week before delivery, she did not suckle her offspring during the first 15 postnatal d. The patient was jaundiced 3 d after delivery and the jaundice resolved in the next 10 d of life. However, he became icteric 3 d after suckling; he presented dark urine, but not acholic stool. Just 1 d before admission, three convulsions were observed in the patient.

The patient did not receive any vaccine. Parental consanguinity was denied and any metabolic or hereditary disorder was not defined in the family.

Physical examination revealed the jaundice, his course has been marked by normal growth (height 10th centile for age, weight 10–25th centile) with fine general condition. Abdominal examination revealed soft hepatomegaly (2 cm on the right and 1 cm on the left lobes) and soft splenomegaly of 1 cm.

Initial investigations showed hemoglobin 9.1 g/dl, haematocrit 26%, white blood cell count 7900/μl, platelets 37 3000/μl. The total bilirubin was 6.4 mg/dl (direct 3.4). Aspartate aminotransferase was 161 U/l (normal 15–18 U/l), alanine aminotransferase 100 U/l (normal 17–22 U/l), alkaline phosphatase 1169 U/l (normal 100–600 U/l) and γ-glutamyl transpeptidase 100 U/l (normal 4–28 U/l). The prothrombin time was 15.9 s (activity 52.6%). C-reactive protein level was normal. Serum calcium level was very low (5.3 mg/dl; normal 8.8–10.8 mg/dl), phosphorus 7 mg/dl (normal 3.5–6.5 mg/dl), magnesium 0.7 mg/dl (normal 1.2–1.8 mg/dl). The serum parathormone level was normal. The patient recovered rapidly by calcium and vitamin D supplementation. The serum and urine aminoacid chromatography results were normal. Also sweat chloride test and α1-antitrypsin level were normal.

The serology for anti-toxoplasma IgM, IgG, anti-herpes virus IgM, IgG, anti-HIV-1, -2, anti-rubella IgM, anti-cytomegalovirus IgM, HBs Ag, anti-HBe IgM, anti-HBs were negative and for anti-rubella IgG, anti-cytomegalovirus IgG and anti-HAV IgM were positive.

Abdominal ultrasound showed a hyperechoic liver and bile ducts were observed. A needle liver biopsy revealed portal areas containing rare inflammatory cells, rare giant cells and intracytoplasmic bile pigments (Fig. 1) and bile plugs in canalicular spaces.

Chest and vertebrae X-rays were normal. Ophthalmologic examination was noted to be normal.

The investigations after 2 weeks revealed only aspartate aminotransferase 2 times of normal and a total bilirubin of 2.4 mg/dl (direct 1.3). Alanine aminotransferase, alkaline

© Scandinavian University Press 1998. ISSN 0803-5253
phosphatase, γ-glutamyl transpeptidase, serum calcium and magnesium levels returned to normal. Finally, the control laboratory work-up revealed normal results at the first month of follow-up.

Discussion

An initial diagnosis of cholestatic hepatitis was made with increased direct bilirubin, presence of bilirubin in the urine, increased microsomal and cholestatic enzymes. Her stool was not acholic and the abdominal ultrasound showed the presence of extrahepatic bile ducts; finally the needle liver biopsy confirmed the diagnosis of cholestatic hepatitis. The history of acute HAV infection in the mother and the positive serology for anti-HAV IgM in the patient were decisive for the diagnosis of acute HAV infection.

Although the diagnosis was certain, other causes of cholestasis were also investigated. The diagnosis of cystic fibrosis, α-1-antitrypsin deficiency and tyrosinemia were ruled out by the normal results of sweat chloride test, α-1-antitrypsin level and aminoacide chromatography respectively. The positive serology of anti-rubella IgG and anti-cytomegalovirus IgG were suggested as passive transmission of these antibodies from the mother. The diagnosis of syndromic or non syndromic intrahepatic ductal paucity was ruled out by the absence of acholic stool, normal vertebra roentgenogram, absence of embryotoxon at ophthalmologic examination and the histologic findings of liver biopsy. Hypocalcemic convulsions have taken place, because the patient did not receive any vitamin D preparation during the period of prolonged cholestasis. The control investigations after 2 weeks revealed normal results, except for slightly elevated total bilirubin and aspartate aminotransferase levels.

There were no transfused blood products, staff, visitors or any infected family member which can be implicated as a source of infant’s infection. Although hepatitis A is easily spread from person to person, any other infected case was not defined in the family, and source of infection was not identified for mother. The father had detectable anti-HAV IgG but anti-HAV IgM was negative.

Because the viraemic phase of hepatitis A infection is very short, as approximately 7 d before the icteric phase, we suggest that the infant was infected by his mother before birth. In our case, the history of maternal icteric hepatitis A infection aided the diagnosis. Transmission at delivery by contamination with maternal faeces and transmission by breast milk are the other possibilities of transmission. However, peak shedding of virus in the faeces occurs during the clinical prodrome and before biochemical evidence of hepatitis. So this mode is not possible for transmission, because the mother did not carry the virus in the faeces at the time of delivery. Breastfeeding only commenced 3 d before the baby became jaundiced, do could not have been the route because the incubation period of hepatitis A is 2–6 weeks.

In conclusion, HAV infection is encountered in the spectrum of intrahepatic cholestatic disorders thus we support the recommendation that all infants with cholestasis without any cause, should be evaluated for HAV infection with the consideration of vertical transmission from infected and anicteric mother. Also, we recommend that babies born to women with hepatitis A around term should be given normal immunoglobulin prophylaxis.

References


Received Dec. 29, 1997. Accepted in revised form May 12, 1998