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A cholestatic type of hepatitis A in a child

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ABSTRACT This paper reports a case of cholestatic type hepatitis A, a rare clinical manifestation of hepatitis A in children. The diagnosis was based on the presence of IgM Anti HAV, symptoms longer than 8 weeks, bilirubin concentration exceeding 10g/dl, and absence of substantial hepatocellular and biliary damage. Corticosteroid therapy resulted in complete recovery. **[Paediatr Indones 2001; 41:308-310]**

***Keywords**: hepatitis A, cholestasis, corticosteroid*

HEPATITIS A IS AN ACUTE, USUALLY SELF LIMITING necroinflamatory disease of the liver caused by enterically transmitted hepatitis A virus infection (HAV).[1] This disease is endemic in developing countries, where infection is predominant in childhood.[2] The diagnosis is based on the presence of IgM anti-HAV. Symptomatic patients have four main clinical presentations,i.e. classical, relapsing, cholestatic, and fulminant.[3] Cholestatic hepatitis A, a specific form, shows persistent jaundice, chemical evidence of intrahepatic cholestasis and absence of substantial hepatocellular disease.[4] This variant is rare in children, it is usually found in elderly patients, mainly in women.[5] The purpose of this report is to present a case of cholestatic type of hepatitis A in a child.

Case Report

A 9-year-old girl was admitted to Dr. Soetomo Hospital Surabaya on August 7, 2000 because of jaundice, malaise, headache, and fever. She was referred from Hajji Hospital Surabaya and had been hospitalized for 25 days with chronic hepatitis and cholelithiasis. Her history revealed that she had jaundice and other symptoms since 7 weeks before admission. The jaundice was progressive. She also suffered from pruritus, lack of appetite and loss of weight. Muscle pain was not noted. The urine was dark. Defecation was normal. There was no history of drug injection, blood transfusion, or contact with patient with liver diseases. She was not immunized against hepatitis A or B.

Physical examination on admission revealed a 9 year old weak girl, bodyweight 33 kg and height 135 cm. Her temperature was 37.5°C, blood pressure 110/70 mmHg, pulse rate was 100 /min and respiratory rate 24/ min. She was severely jaundiced without anemia, cyanosis or dyspnea. The heart and lungs were normal. The liver was palpated 3 cm below the costal margin, smooth, normal consistency, and tender. The spleen was not palpable. The extremities were normal. There were no clinical signs of chronic liver disease.

The previous laboratory data from Hajji Hospital Surabaya were as follows: June 30: bilirubin: direct 7.41 mg/dl, total 7.51 mg/dl, AST 50 IU/L, ALT 115 IU/L, negative HbsAg. July 27: direct bilirubin 7.73 mg/dl, total bilirubin 8.52 mg/dl, AST 424 IU/

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L,ALT 516 IU/L, alkaline phosphatase 560 IU. August 3rd: direct bilirubin 14.38 mg/dl, total bilirubin 18.38 mg/dl, AST 690 IU/L, ALT 510 IU/L, alkaline phosphatase 439 IU.

The first abdominal ultrasonography was performed on July 17 demonstrating normal liver size, mild increase of echoparenchym, no dilatation of intra or extrahepatic bile ducts (Figure 1). Gallbladder showed thickened wall and sludge. The other organs were normal. Repeat ultrasound was done on July 28 which showed increased heterogenousity echoparenchym, thickening of the gallbladder wall and a gall stone sized about 0.8 cm (Figure 2). Based on those data, our diagnosis on admission was hepatitis and suspected gallstone. We did repeated blood count, liver function tests, serology marker for hepatitis A and C, urinalysis, and repeat ultrasound. Treatment was started with cholestyramine, ursodeoxycholic acid and multivitamins.

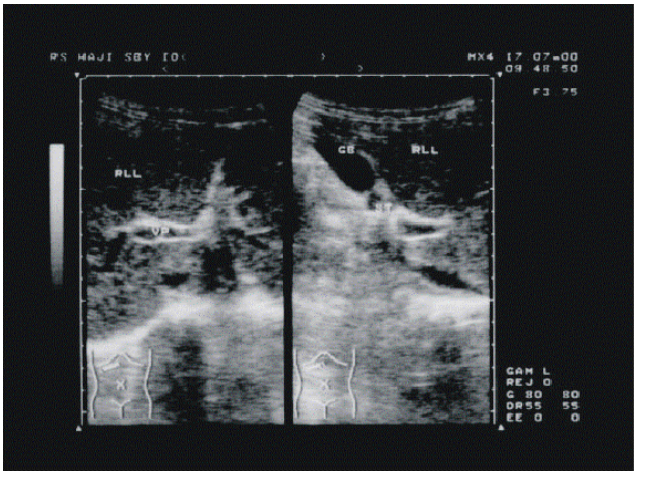

Figure 1. USG (July 17th): Increased liver echoparenchym with thickening and sludge of gallbladder wall and suspected gallstone (arrow).

The laboratory results on August 8, 2000 showed Hb 12.4 g/dl, WBC 8.800/µl, platelets 340.000/µl, ESR 17/hour, with normal differential count. The total protein 7.5 g /dl, albumin 3.8 g/dl, direct bilirubin 7.68 mg/dl, total bilirubin 12.04 mg/dl, AST 167 IU, ALT 337 IU. Serology for IgM anti HAV was positive, and anti HCV was negative. Ultrasound examination on the third day of hospitalization revealed hepatomegaly with decreased intensity of echoparenchym. Intrahepatic and extrahepatic bile ducts were not dilated. Gallbladder showed a wall thickening and sludge with no gallstone. Therefore our diagnosis was acute cholestatic type of hepatitis A and corticosteroid was commenced. This was followed by clinical improvement, nausea abated on the 5th day, urine was normal on the 10th day, and on the 13rd day, the appetite improved. Corticosteroid was discontinued on day 14th and the patient was discharged on the 16th day. She was still subicteric and her liver was palpable 2 cm below the costal margin. On the 15th day after discharge, she came in good condition, her weight increased to 35 kg, she had no complaint, no jaundice, the liver was 2x1 cm below costal margin. On examination, laboratory data revealed direct bilirubin 0.96 mg/dl, total bilirubin 1.82 mg/dl, AST 43 IU, ALT 49 IU.

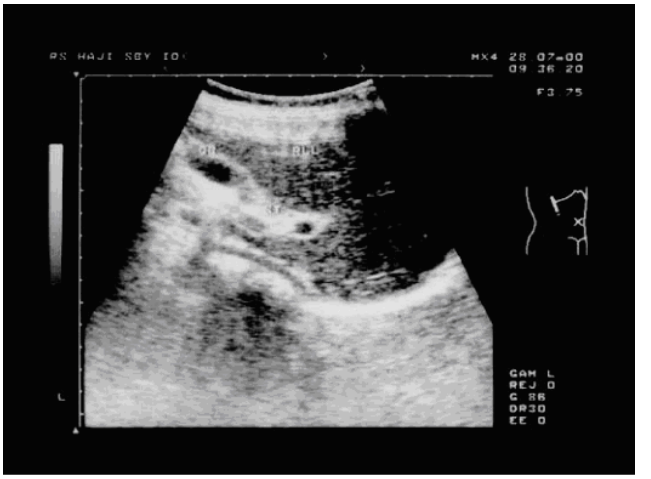

Figure 2. USG (July 28th): Increased liver echoparenchym, thick gallbladder wall, suspicion of stone (arrow).

Discussion

A case of hepatitis A viral infection (HAV) with unusual clinical manifestations was misdiagnosed as chronic hepatitis with cholelithiasis. The diagnosis was based on the prolonged clinical course (classical HAV infection are self limiting and lasts 8 weeks.[2,3,6]) and ultrasound findings. Since our evaluation for serologic markers was positive only for IgM anti HAV and we could exclude the possibilities of cholestatic drugs jaundice, [3,4,7] then the diagnosis of viral hepatitis A infection was definite.

The first abdominal ultrasound showed a mild increase of echoparenchym with thickened wall and sludge in the gall bladder while the second ultrasound found the presence of a gall stone. These findings were not supported by the clinical features of

gall stone such as severe abdominal pain of sudden onset or persistent vomiting. The third ultrasound detected no gall stone. The presence of stone in the second examination might be caused by an echogenic bile, which is usually seen in bile stasis.[8]

Cholestatic hepatitis A viral infection occurs in 10% of patients with symptomatic disease.[3,4] It is characterized by a prolonged fever, pruritus and jaundice for at least 8 weeks, bilirubin concentration increasing more than 10 mg/dl, absence of other hepatotrophic viral markers, no evidence of previous liver disease or previous exposure to hepatotoxic substances, and normal biliary tree in the abdominal ultrasound.[1,5,7,9] All of these was found in our patient.

The pathogenesis of cholestatic hepatitis A is not understood, however a factor isolated from serum and lymphocytes causing cholestasis in rats suggested an immunological mediated response.[3] Based on this pathogenesis, a short course corticosteroid to minimize immunological reaction[3,10] was given as an adjunct besides ursodeoxycholic acid for treatment of symptomatic hepatitis.[5,11] Our patient showed a good response, and the prognosis was good.

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