CASE REPORT

Hepatitis C in A Child with Thalassemia

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ABSTRACT We present a case of hepatitis C in a patient with thalassemia major. The transmission seems to be through blood transfusion. The diagnosis of hepatitis C was determined by the presence of anti HCV in serum. Ursodeoxycholic acid therapy gave a satisfactory result and the patient was discharged after showing improvement of both clinical signs and liver function tests. He was suggested for regular examination to evaluate anti HCV and his thalassemia. It is believed that if anti HCV titer is more than 6 months, hepatitis may become chronic hepatitis C. **[Paediatr Indones 1995; 35:47-51]**

Introduction

Hepatitis C is caused by non A non B virus. Transmission of hepatitis C virus occurred via parenteral route, and this disease has been endemic in many parts of the world.[1-4] Recently, sporadic hepatitis C was also found without a history of transfusion or percutaneous injection, and a non-A non-B hepatic disease which is not endemic.[5-8]

It is believed that approximately from 150 000 to 170 000 new cases of hepatitis C occur in the United States annually. The precise prevalence of hepatitis C in Indonesia has not reported so far, but some investigations reported that anti hepatitis C virus incidence was 3-4% in donor's blood, 30-40% in acute hepatitis and 70-89% in liver cirrhosis and cancer. The prevalence of hepatitis C accompanies thalassemia is 58%.[5,8-10] The clinical symptoms of hepatitis C is generally milder than hepatitis B and the diagnosis is based on the presence of and hepatitis C virus in serologic examinations.[7,11,12]

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Case Report

A 7 year-old Indonesian boy who had Javanese mother and Balinese father was admitted to the Department of Child Health, Sanglah General hospital in Den-

pasar on January 27, 1993 with the chief complaint of generalized jaundice. He was diagnosed as having thalassemia major when he was 4 years old (his mother had thalassemia minor and his father is a Hb E carrier). He received seven series of transfusions at the Department of Child Health, Sutomo Hospital in Surabaya, and the latest being On December 20, 1992.

The patient had no jaundice before his present illness and had no family history of similar disease. He had been suffering from this symptom since 3 days before admission. The jaundice was first noticed on his eyes and later spread to the entire skin. Seven days before admission he was subfebrile, with malaise, headache, nausea, anorexia. He also passed dark urine and dark yellow stool.

On physical examination on January 27, 1993 the child was found to be alert. The pulse rate was 100/minute and regular, the blood pressure was 100/70 mmHg, the respiratory rate was 20/minute, the temperature was 37.5° C and body weight was 20 kg. He was well nourished. The sclerae were jaundiced while his conjunctivae were pale. The ENT, heart and lungs were within normal limits. The abdomen was not distended, and the liver was palpated 3 cm below the costal line; it was tender, smooth, sharp, and painless. The spleen was palpated at Schuffner III. The skin was entirely yellow.

Hematologic values showed the hemoglobin content of 7.31 g/dl, WBC 8200/µl, with differential count 2% eosinophils, 62% segments and 36 lymphocytes, the platelets were 310 000/µl. The blood sedimentation rate was 4/16 mm. Bleeding and clotting times were 3 and 6 minutes, respectively. Liver function tests revealed total bilirubin of 32.9 mg/dl, direct bilirubin 22,1 mg/dl, AST 1267 U/l, ALT 867 U/l, ALP 277 U/l, GGT 72 U/l. Serology tests showed positive anti HCV while HBS-Ag, IgM Anti HBC IgM Anti HAV tests gave negative result. Urinalysis showed: pH 7, urobilin + 4, urobilinogen was negative and bilirubin + 3.

The working diagnosis on admission was post-transfusion hepatitis C in a patient with thalassemia major. He was treated with cholestyramine 4x5 g daily and ursodeoxycholic acid 100 mg 2 times daily, and 200 ml of packed red cells transfusion.

On February 3, 1993 the hemoglobin content was 10.0 g/dl, serum iron 190 µg/dl, iron binding capacity 386 µg/dl, total iron binding capacity 576 µg/dl. It was planned to put this patient on desferrioxamine B.

On February 15, 1993 urobilin and bilirubin urine were negative. Liver function tests revealed a fast recovery rate as shown in the following table:

Date	Bilirubin (mg/dl)		AST	ALT	ALP	GGT
	Total	Direct				
2/7/93	10.2	7.4	110	95	215	
2/15/93	5.7	3.7	70	81	190	
2/22/93	4.1	2.7	47	56	157	
3/1/93	3.9	2.3	46	30	150	29

Physical examination on 5th March, 1993 showed that the patient was alert, and was not anemic. The blood pressure was 100/70 mmHg, the pulse rate was 96/minute, the respiration rate was 20/minute, the temperature was 36,5°C and body weight 22 kg. The sclerae were not jaundiced. The abdomen was supple and not distended, the liver was palpated 2 cm below costal angle, tender, smooth, with sharp edge, and on deep palpation there was no visceral pain. The spleen was palpated to Schuffner III and there was no jaundice observed on the skin. The patient was discharged in a good condition with ursodeoxycholic acid therapy and was planned for further evaluation.

Discussion

Until recently the exact pathogenesis of liver damage caused by hepatitis C virus is not clear. Most investigators report that hepatitis C virus is a primary cytopathic virus for liver cells. Immune response factor does not play a major role because liver cell damage is primarily due to the virus it self. Soewignyo[14] stated that there were two groups of chronic hepatitis C, the first group had high and the other had normal interferon level. The first group had a lower ALT value and a relatively better histopathologic features than the second one.[6,12]

Transmission of virus hepatitis C generally occurs via parenteral route, e.g. through exchange of blood or body secretions. The virus has been demonstrated in the blood, semen, saliva, and breast milk. Thus, transmission may occur through intimate contact of any type, homosexuality, as well as vertical transmission. Individuals at high risk include those with frequent exposure to blood or blood products. In addition, hemophiliacs, thalassemics, hemodialysis patients and intravenous drug abusers are at high risk for disease.[3,6]

The incubation period of non-A non-B transfusion-related hepatitis is 2 to 26 weeks (average 7 to 8 weeks), although shorter incubation periods of 1 to 2 weeks have been noted. On the other hand the incubation period can be as long as 26 weeks. Patients with incubation periods of less than 30 days may develop into fulminant hepatitis 35.7% of all cases, while if incubation period is more than 30 days, they generally become chronic hepatitis.[2,6,12,14]

The illness, which often show gastrointestinal symptoms and does not accompanied by jaundice (75%), is characterized by moderately elevated serum aminotransferase levels, often with marked fluctuation of serum ALT values.[6,12]

The severity of the symptoms is influenced by age and transmission process, the older the patient the more severe the disease.

Compared to hepatitis B, viral hepatitis C infection more often leads to a chronic form. Fulminant disease is rare, and generally occurs as a superinfection in chronic hepatitis B patients. The process to chronic condition is more often starting from hepatitis B. Thirty percents of hepatitis C infection will become chronic persistent hepatitis, 40% to chronic active hepatitis, and 20% will end into cirrhosis. Transmission through blood transfusions more often lead to active chronic hepatitis and cirrhosis compared to those who are non parenterally-sporadic infected. Usually, hepatocellular carcinoma in hepatitis C is associated with a chronic hepatitis B process and cirrhosis.

Up to 40% of fulminant hepatitis cases can be ascribed to non-A non-B virus. They appear to have a more severe clinical course than cases ascribed to hepatitis A or B.[2,6,12] Serologic examinations showed that hepatitis C is associated with a positive anti HCV reaction that could be detected between 4 to 24 weeks after the clinical symptoms appear (mean 15 weeks). When antibody against hepatitis virus can detected for more than sixth months, the hepatitis C becomes chronic.[4]

In our case, the transmission seems to be through blood transfusion and six weeks later, jaundice and elevated liver function tests such as bilirubin, AST, ALT, ALP, and GGT were noted.

Hepatitis C therapy is still under further investigation. Recently, physicians give alpha interferon therapy for adults with good results, which is in contrary in children. Treatment with alpha interferon is now accepted for chronic liver disease due to hepatitis C virus infection. In this disease, alpha interferon seems to act primarily as a very effective anti viral agent.[15,16] Ursodeoxycholic acid decrease hepatocellular damage in a patient with chronic hepatitis, Ursodeoxycholic acid is an important factor for hepatitis treatment because of its non-choleretic and non-toxic.

In our case, the patient was treated with ursodeoxycholic acid with good result. Jaundice disappeared and bilirubin, AST, ALT, ALP levels decreased and GGT reached normal value.

The prognosis of hepatitis C is usually poor because 50-75% will become chronic. We plan to evaluate anti HCV on the sixth month to determine the prognosis. The concomitant presence of thalassemia could worsen the hepatitis because of hemosiderosis.

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