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Prolonged Cholestasis due to Hepatitis A Virus Infection

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We present a 12-year old boy with jaundice for 2 weeks. The child was deeply icteric and had hepatomegaly. IgM antibodies for hepatitis A virus were positive. However this child had prolonged cholestasis and cholestyramine was started. The child responded only after prednisolone was started.

Key words: *Hepatitis A, Clinical features, Cholestasis.*

Acute viral hepatitis due to Hepatitis A virus is usually a self limiting illness in children with complete recovery within two months of onset of symptoms [1]. Occasionally, the clinical syndrome of cholestasis, characterized by intense pruritus and prolonged conjugated hyperbilirubinemia, may persist for several months [2].

CASE REPORT

A 12-year old male child was admitted with complaints of jaundice for 6 weeks and itching for 2 weeks. The onset of jaundice was preceded by low grade fever and nausea for 4-5 days. There was no history of bleeding from any site or any symptoms of encephalopathy. There was no past history of jaundice. On examination, the child was deeply icteric. Scratch marks were present all over the body and there was no pallor. His weight was 39 kg and height was 142 cm. The vital parameters were stable, and examination of cardiovascular, respiratory and neurological systems was unremarkable. Liver was

palpable 5 cms below costal margin with a span of 13.5 cms.

Spleen was not palpable and there was no free fluid. A diagnosis of acute viral hepatitis with cholestasis was made. Hematological workup including prothrombin time were normal. Liver biochemistry revealed AST 56 U/L, ALT 36 U/L, alkaline phosphatase 468 U/L, total serum bilirubin 33.5 mg% with conjugated fraction of 23.9 mg%. Total serum proteins were 7.1 g/dL; serum albumin was 3.6 g/dL. IgM antibodies for Hepatitis A virus were positive. HbsAg, Anti HCV and anti HEV IgM were negative. Child was started on ursodeoxycholic acid (UDCA) but there was no improvement in jaundice or itching. After 10 days, serum bilirubin had further increased to 42.3 mg% (conjugated fraction, 32.1 mg%). Oral cholestyramine was added. Ultrasound abdomen ruled out any extrahepatic obstruction. His G6PD levels, osmotic fragility, and coomb's tests were (direct and indirect) negative. Liver histology showed significant intracanalicular cholestasis, maintained lobular architecture, mild portal triaditis and focal spotty necrosis. Over the next six days, his total bilirubin further rose to 45.6 mg% (conjugated, 34.6 mg%), and AST and ALT were 39 and 26 U/L, respectively.

The child was started on 60 mg of daily oral prednisolone. After 3 days of starting steroids, he showed improvement in symptoms. The total bilirubin declined to 22.3 mg% in 14 days. After 2 weeks of daily prednisolone, the dose of oral steroids was reduced to 30 mg and then tapered off over next 4 weeks. After 4 weeks of prednisolone therapy, total serum bilirubin had declined to 2.6 mg%, and at the end of therapy, child was asymptomatic with serum total bilirubin of 0.8 mg%.

DISCUSSION

Though Hepatitis A is usually a self limiting disease, atypical manifestations including prolonged and relapsing course are well known [3,4]. Prolonged cholestasis for a period of upto five to seven months after infection with Hepatitis A has been reported [4,5]. Response to oral or parenteral steroids in such cases has also been documented earlier [1,6].

The mechanism of intrahepatic cholestasis in acute viral hepatitis is not clear. Recent *in vitro* and animal studies on lymphocyte cultures of patients with alcoholic hepatitis and acute viral hepatitis suggest that cellular or humoral immune phenomena might be involved in the pathogenesis [7]. The other proposed mechanism is from interruption in continuity of bile flow secondary to periportal spotty necrosis [8]. The lympholytic action of corticosteroids may be the reason for their efficacy in cholestasis due to Hepatitis A infection.

To conclude, corticosteroids should be considered for the management of patients with prolonged cholestasis secondary to hepatitis A.

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