

Case Report

Severe Hemolytic anemia in Hepatitis A Virus Infection: An Unusual and rare entity

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Abstract: Hemolytic anemia is rare and unusual feature of Hepatitis A viral infection. The case is 10 year male presented with viral hepatitis but had prolonged and high serum bilirubin levels. The cause found to be hemolytic anemia on investigations. The outcome of the child was managed conservatively and steroids and doing well. Prolonged and severe jaundice in viral hepatitis is rare and hemolysis should be kept in mind as cause.

Keywords: Hemolytic anemia, hepatitis.

INTRODUCTION:

Viral hepatitis continues to be a major public health problem in both developing as well as developed countries. It is caused by at least 5 pathogenic hepatotropic viruses recognized to date; hepatitis A(HAV), B (HBV), C (HCV), D (HDV), and E (HEV). HAV infection is the most prevalent hepatotropic virus and causes acute hepatitis only. The anicteric illness often present with clinical symptoms indistinguishable from other forms of viral gastroenteritis, particularly in young children. In older adolescents it characteristically presents with an acute febrile illness with abrupt onset of anorexia, nausea, malaise, vomiting, and jaundice. The typical duration of illness is 7-14 days. Other rare manifestations include bone marrow suppression, villous changes, gastrointestinal ulceration, pancreatitis, myocarditis [1]. Hemolytic anemia is rare manifestation with viral hepatitis A.

CASE REPORT

10 years male presented with high grade fever, non-bilious vomiting for 2 days and yellowish discoloration of eyes for one day. There was no history of any diarrhea, bladder trouble, rash, blood transfusion, drug intake and jaundice in past. Clinical examination revealed icterus and 4 cm tender hepatomegaly. Ancillary investigations revealed initial hemoglobin of 12.6 g/dL, liver enzymes (SGOT/SGPT 6990/6100) and serum bilirubin of 13.6 mg/dL. Prothrombin time was normal, IgM anti Hepatitis A antibodies were positive. We managed the child conservatively with antipyretics, intravenous fluids. On day 3 of admission the child

became sleepy but arousable on verbal commands and yellowish discoloration of body deepened. The child was shifted to intensive care unit in view of hepatic encephalopathy and managed. The repeat investigations showed a fall in Hb to 9g/dL, SGOT/PT decreased to 2010/2830 but serum bilirubin raised to 43.8 mg/dL. The child showed clinical improvement over next 2-3 days except worsening of jaundice. But his Hb continued to fall 5.8 g/dl, serum bilirubin was rising (48, 43.9 mg/dl) and liver enzymes were decreasing (SGOT/PT 656/1190, 317/809, 137/560) gradually. In view of above clinical picture a possibility of hemolytic anemia was kept and workup was done. Peripheral smear showed polychromasia, anisopoikilocytosis, nucleated RBCs, reticulocyte count was 12%, DCT, G6PD, ANA were negative. The child was started on steroids keeping a possibility of autoimmune hemolytic anemia after hematologist consult. The child showed a dramatic response and his serum bilirubin fell from 43.9 to 32.2 mg/dL within 24 hours and Hb did not fall further. After 72 hours his serum bilirubin further decreased to 17 mg/dL, reticulocyte count dropped to 9% and peripheral smear did not show any evidence of hemolysis. The patient improved over next 4 days and discharged. Steroids were tapered and stopped in two weeks. After one month the liver enzymes and serum bilirubin returned to normal and child was asymptomatic.

DISCUSSION:

The extrahepatic manifestations of HAV infection include hemolytic anemia, aplastic anemia,

acute renal failure, and acute reactive arthritis [2]. Chau and colleagues found incidence of 4 % in their study of 434 patients of hepatitis. 53% of the patients with hemolysis were G6PD deficient [3]. Kanematsu *et al.*; reported largest series of hemolytic anemia from Japan for a period of 26 years. They reported 48 patients who had hemolytic anemia complicated with viral hepatitis, 25 with acute hepatitis A, 10 with acute hepatitis B, 10 with some other viral hepatitis, and 3 with chronic hepatitis. Four mechanisms were suggested for hemolysis in the patients with viral hepatitis. First in individuals who has predisposition to hemolytic anemia, viral infection accelerates the red cell destruction and hemolysis become visible. Viral injury to the red cell membrane and liver failure, hypersplenism induce the hemolysis were other mechanism. Autoimmune hemolytic anemia induced by immunological disturbances caused by viral infection was the fourth mechanism suggested [4].

Hosnut *et al.*; reported two children presenting with HAV infection with G6PD deficiency who had hemolysis on first day of admission with hemoglobin falling from 8.5 to 5.1, 9.8 to 6.1 g/dl. They extensively investigated both cases for the etiology of hemolysis. Both kids were G6PD deficient, auto antibodies were positive (ANA, SMA, anti-Ds DNA), reticulocyte count were raised but DCT was negative in both cases. Both cases improved with supportive care and steroids were not used in either [5]. Similarly, in our case we found evidence of hemolysis on peripheral smear, reticulocyte count was raised but both G6PD and DCT were normal.

Many other authors reported hemolytic anemia in HAV infections mostly in adult patients but we did not find any reports in children except for above. JA reported case of acute hepatitis A which resulted in a fall in hemoglobin concentration from 14.6 to 4.5 g/dl due to an acute hemolytic anemia with an associated rise in bilirubin from 149 to 960 mmol/L [6].

Ibe M reported a case of massive acute hemolysis associated with an acute hepatitis A in a 39-year-old woman in the absence of G6PD-deficiency. Although tests for autoimmune hemolytic anemia were negative the patient made a dramatic recovery after treatment with corticosteroids similar to our patient, suggesting an immune mediated mechanism nonetheless [7]. Urganci NA reported a case of autoimmune hepatitis and autoimmune hemolytic anemia following hepatitis A infection [8].

CONCLUSION:

Hepatitis A is common cause of viral hepatitis which sometimes present with unusual manifestation like hemolytic anemia that should be recognized early to prevent morbidity and mortality. A short course of steroids may hasten recovery in autoimmune hemolytic

anemia caused due to HAV infection and Hepatitis A vaccination may be useful for prevention.

REFERENCES:

1. Centers for Disease Control and Prevention: Update: prevention of hepatitis A after exposure to hepatitis A virus and in international travelers. Updated recommendations of the advisory committee on immunization practices (ACIP), MMWR MorbMortal Wkly Rep 2007; 56:1080-84.
2. Song KS, Kim MJ, Jang CS, Jung HS, Lee HH, Kwon OS *et al.*; Clinical features of acute viral hepatitis A complicated with acute renal failure. Korean J Hepatol, 2007; 13:166-73.
3. Chau TN, Lai ST, Lai JY, Yuen H; Hemolysis complicating acute viral hepatitis in patients with normal or deficient glucose-6-phosphate dehydrogenase activity. Scand J Infect Dis. 1997; 29:551-53.
4. Kanematsu T, Nomura T, Higashi K, Ito M; Hemolytic anemia in association with viral hepatitis. Nihon Rinsho. 1996; 54:2539-44.
5. Hosnut FO, Ozcay F, Bayrakci US, Avci Z, Özbek N; Etiology of hemolysis in two patients with hepatitis A infection: glucose-6-phosphate dehydrogenase deficiency or autoimmune hemolytic anemia. Eur J Pediatr. 2008; 167:1435-39.
6. Tibble JA, Ireland A, Duncan JR; Acute autoimmune haemolytic anaemia secondary to hepatitis A infection. Clin Lab Haematol. 1997; 19:73-77.
7. Ibe M, Rüde B, Gerken G, Meyer zum,Büschel KH, Lohse AW; Coombs-negative severe hemolysis associated with hepatitis A. Z Gastroenterol. 1997; 35:567-75.
8. Urganci N, Akyildiz B, Yildirmak Y, Ozbay G; A case of autoimmune hepatitis and autoimmune hemolytic anemia following hepatitis A infection. Turk J Gastroenterol. 2003; 14:204-210.