Hepatitis E Virus Infection Leads To Severe Hemolysis In Glucose-6-Phosphate Dehydrogenase Deficiency Patients

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Abstract

Hepatitis E virus is the commonest causes of acute viral hepatitis in India but usually manifests as a mild self-limiting illness. Viral hepatitis E in the presence of glucose-6-phosphate dehydrogenase (G6PD) deficiency may be associated with complications such as severe anemia, hemolysis, renal failure. The incidence of G6PD deficiency in the general population of northern India is reported to be between 2.2% to 14%. Despite both hepatitis E infection and G6PD deficiency being common, their impact on patient illness has only recently been reported. The present study reports a case of severe hemolysis in a patient with G6PD deficiency due to hepatitis E infection leading to anaemia, renal failure and seizures.

Introduction

Glucose-6-phosphate dehydrogenase deficiency is an X-linked recessive hereditary disease characterised by abnormally low levels of glucose-6-phosphate dehydrogenase (abbreviated G6PD or G6PDH), a metabolic enzyme involved in the pentose phosphate pathway, especially important in red blood cell metabolism. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is found in 2.2% to 14% of the general population in North India (1). Hepatitis E is an enterically transmitted virus and is the most common causes of acute viral hepatitis in India (2). The coexistence of viral hepatitis and G6PD deficiency has been reported to be associated with severe jaundice and other complications (3). Hepatitis E infection with G6PD deficiency has been associated with more severe illness in a previous report (4).

Case Report(s)

A 17-year-old boy with no history of liver disease presented with low grade fever, upper abdominal pain, fatigue and anorexia for 10 days. He had noticed a yellow discoloration of the eyes for three days and dark colored urine for five days. On examination, he was deeply icteric. Abdominal examination revealed a soft, tender liver, palpable 4 cm below the costal margin. There was no splenomegaly and the rest of the physical examination was normal. Laboratory investigations revealed a hemoglobin mass concentration of 5.5 g/dl, a total leucocyte count of 18500/cmm, and a total serum bilirubin of 66 mg/dl with a conjugated fraction of 45.7mg/dl. The serum aspartate aminotransferase (AST) concentration was 472 U/L and the alanine aminotransferase (ALT) concentration was 1147 U/L. Serum creatinine was 9.22 mg/dl and urea was 215 mg/dl. The prothrombin time was 12.8s (control: 11.3s). The serum lactate dehydrogenase concentration was 2446 U/L (normal: 200 to 500 U/L) and serum haptoglobin was undetectable. Direct and indirect Coomb’s tests were negative. Serum for vitamin B12 assay was 1076 (Normal range 211-946). Both the peripheral blood smear and the antigen-test were negative for malaria. Normal serum ceruloplasmin levels and the absence of Keyser-Fleischer rings on slit lamp examination excluded Wilson’s disease. In view of very high bilirubin and hemolysis, the G6PD level was also checked which was low at 110 mU/109 erythrocytes (normal range: 245-299). Immunoglobulin (Ig) M anti-hepatitis A virus, hepatitis B surface antigen, IgM anti-hepatitis B core and anti-hepatitis C virus were negative, while IgM anti-hepatitis E virus (HEV) was positive. A diagnosis of HEV hepatitis with G6PD deficiency was made. The patient was managed conservatively, including avoiding all hepatotoxic, nephrotoxic and oxidant drugs, and maintaining an adequate urine output. Over the next week, the serum bilirubin declined to 46.4mg/dl (conjugated fraction: 34.1mg/dl), AST at 117 U/L, and ALT at 430 U/L. The hemoglobin mass concentration increased to 9.7 gm/dl after transfusions. He also underwent three session of hemodialysis. The metabolic parameters gradually improved over five weeks. The hemoglobin mass concentration increased to 10.7 gm/dl, bilirubin fell to 8.1mg/dl (conjugated fraction: 6.7mg/dl) AST was 36U/L, ALT 65 U/L and reticulocyte count fell to 1.2%. Serum creatinine reduced to 5.37 mg/dl. The patient was discharged and followed in the outpatient department. His serum bilirubin and aminotransferase levels were nearly normal.
Discussion

Mild hemolysis associated with decreased red blood cell survival may be commonly seen with viral hepatitis, but is seldom of clinical significance (5). However, when viral hepatitis occurs in G6PD-deficient patients, hemolysis may be severe (5,6). The patient described in this case had severe intravascular hemolysis as evidenced by a fall in hemoglobin, reticulocytosis, unconjugated hyperbilirubinemia, hemoglobinuria and undetectable serum haptoglobin levels. The presence of severe hyperbilirubinemia in patients with viral hepatitis and G6PD deficiency has been previously reported (7,8). In a case control study, Gotsman et al (9) evaluated the impact of G6PD deficiency on patients with Hepatitis A virus infection. They found that although patients with G6PD deficiency had a more severe initial clinical presentation, the clinical outcome was not affected. Abid et al. (4) recently reported a cohort of five patients from Pakistan with G6PD deficiency and Hepatitis E viral infection. All five patients had severe and protracted illness, and four developed acute renal failure. Profound hemolysis in G6PD-deficient individuals is usually precipitated by exposure to selected drugs. However, as in this case, viral hepatitis may precipitate massive hemolysis even without the intake of such drugs (4,5,8). The mechanism of hemolysis is thought to occur through decreased levels of reduced glutathione in red blood cells. Reduced glutathione levels could result from the accumulation of oxidants due to hepatic dysfunction and lead to increased hemolysis in the presence of G6PD deficiency. Despite the high levels of bilirubin in these patients, the prognosis is mainly related to the severity of hepatic injury (7). Acute renal insufficiency, though uncommon in uncomplicated acute viral hepatitis, can occur as a fatal complication of severe intravascular hemolysis in these patients (3). Excess hematin and bilirubin may result in the obstruction of renal tubules, leading to acute renal insufficiency with increased morbidity. Renal failure may be nonoliguric, therefore, kidney function should be assessed by regularly monitoring blood chemistry, and urinary sodium and osmolarity. HEV infection is transmitted through the feco-oral route but, unlike other enteric agents, does not generally spread from infected persons to their close contacts (10).

Conclusion

In patients with acute viral hepatitis and unexplained anemia with very high serum bilirubin levels, intravascular hemolysis should be considered and investigated. Wilson’s disease may present with jaundice and hemolysis and must be excluded. G-6-PD deficiency should also be considered if there is marked hyperbilirubinemia with evidence of hemolysis in patients with acute hepatitis E virus infection.

Abbreviations(s)

Nil

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