Viral Hepatitis with Acute Hemoglobinuria

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Introduction

Acute viral hepatitis A is widely prevalent in India.¹ Viral hepatitis is the leading cause of acute hepatitis in children in our country, and hepatitis A is the most common type of viral hepatitis. Hepatitis A does not commonly present with severe anemia unless it is complicated by fulminant hepatic failure (FHF) and significant blood loss. However, in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency, the most common inherited enzyme deficiency worldwide,² hepatitis A may cause severe anemia in absence of major blood loss. This chance association has been reported earlier by Sarkar et al.³ The coincidence of acute hepatitis A and G6PD deficiency may cause acute intravascular hemolysis, manifest as pallor, icterus, hepatosplenomegaly, and dark-colored urine. The pathogenetic mechanism of hemolytic anemia in hepatitis A infection is ill-understood, but it has been reported to be autoimmune mediated in at least some cases.⁴,⁵ Here, we report a case of acute hepatitis A complicated by FHF and acute intravascular hemolysis associated with G6PD deficiency.

Case Report

A 7-year-old male child born to a nonconsanguineous parent presented with moderate degree fever, yellowish discoloration of eyes, and urine for the past 5 days before admission. There was no history of hematemesis, melena, or any other bleeding, swelling of body, convulsion, and drug ingestion just before present illness or any previous blood transfusion. His urine volume was satisfactory. On examination, the child was deeply icteric, severely anemic, and drowsy. Abdominal examination revealed soft hepatomegaly of 6.5 cm below right costal arch on right midclavicular line and enlarged spleen of approximately 3 cm below the left costal arch. There was no ascites. Except for drowsiness he had no neurological deficit; his plantar reflexes were flexor. Slit lamp examination of the eye did not reveal Keyser–Fleischer rings. On the day following admission, his urine color changed to dark brown or blackish.

Laboratory testing on admission showed a normocytic, normochromic anemia (hemoglobin 4.5 g/dL), reticulocyte count 14.1%, and total leukocyte count 20,000/mm³ (neutrophils 80%, lymphocytes 17%, monocytes 2%, and eosinophils 1%). The platelet count was 350 × 10³/L. Prothrombin time was 15.9 seconds (control, 10.2 seconds), giving an international normalized ratio of 1.3 (normal, 1–1.2), and activated partial thromboplastin time was 23.5 seconds (normal, 22.6–35 seconds). The total serum bilirubin was 61.2 mg/dL (conjugated, 32.9 mg/dL; unconjugated, 28.3 mg/dL); serum total protein was 5.9 g/dL, and albumin and globulin were 3.6 and 2.3 g/dL, respectively, giving a albumin:globulin ratio of...
Liver enzymes were elevated as follows: aspartate aminotransferase 1,474 U/L (normal, 0–40 U/L), alanine aminotransferase 680 U/L (normal, 0–41 U/L) and alkaline phosphatase 354 U/L (normal, 100–250 U/L). Hemolytic anemia and severe hyperbilirubinemia. In particular, G6PD deficiency, with only a few conditions, for example, autoimmune hemolytic anemia and G6PD deficiency, but in their case, administration of vitamin K was considered to have been a contributory factor.

Autoimmune hemolytic anemia was excluded in our case on the basis of negative autoimmune antibody and direct and indirect Coombs test results. Autoimmune hepatitis was ruled out by negative serological studies and by application of an objective scoring system. Despite acute hemoglobinuria, this patient did not develop acute renal failure, contrary to previous reports. G6PD catalyses the initial step in the hexose monophosphate shunt, oxidizing glucose-6-phosphate to 6-phosphogluconolactone, and reducing NADP to NADPH. The hexose monophosphate shunt pathway of glucose metabolism is the only red cell source of NADPH. NADPH acts as a cofactor for conversion of glutathione to reduced glutathione, which is essential to metabolize oxidant products within RBC. Thus, in the absence of reduced glutathione, RBC is vulnerable to hemolysis.

In conclusion, it is suggested that all patients with acute viral hepatitis A, marked hyperbilirubinemia and severe pallor should be assessed for acute intravascular hemolysis and G6PD deficiency. Universal vaccination against hepatitis A, or routine newborn screening for G6PD deficiency in Asian countries like India, would help avoid hepatitis A–associated acute intravascular hemolysis. Evidence in the form of large clinical- and cost-effectiveness studies is required to support a change in national policy.

Conflict of Interest
None.

Source of Funding
None.

Statement Regarding Informed Consent
The parents were informed about the suitability of the case for publication in a medical journal. They gave verbal consent in this regard.

References