

Hemolytic Anemia in a Patient with G6PD Deficiency and Acute Viral Hepatitis

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Citation

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Abstract

We report a case of acute viral hepatitis leading a hemolytic episode in a patient with underlying glucose 6 phosphate deficiency.

CASE REPORT

A 51-year old African American male with a past medical history significant for hypertension and chronic hepatitis B virus infection presented with a chief complaint of vomiting and crampy right upper quadrant abdominal pain for two days. The patient also reported a mild yellowing of his skin, orange-colored urine, and light-colored stools for one week. The patient reported a long history of intravenous drug abuse; his most recent use of heroin being three days prior to presentation.

On physical exam the patient had icteric sclera, tenderness to palpation in the right upper quadrant without rebound or guarding, and hepatomegaly with a liver span of 17 centimeters. His initial laboratories were significant for an AST of 2583, ALT of 1948, direct bilirubin of 7.9, and total bilirubin of 13.9.

At this point the differential diagnosis included acute viral hepatitis, autoimmune hepatitis, ischemia, Wilson's disease, and ingestion of a toxic substance. Ischemia was ruled out by a lack of acidosis and normal serum lactate. A Tylenol level was less than 1, an ANA was negative, an anti-LKM1 was negative, and a ceruloplasmin level was normal. These effectively excluded Tylenol overdose, autoimmune hepatitis, and Wilson's disease. Hepatitis serologies were obtained and revealed a positive hepatitis B surface antigen, negative hepatitis B surface antibody, negative hepatitis B core IgM, negative hepatitis A IgM, positive hepatitis C antibody, and positive hepatitis D antibody.

Thus, the patient was diagnosed with an acute viral hepatitis. The possibilities included chronic hepatitis B infection with either acute hepatitis C infection, hepatitis D co-infection, or

hepatitis D superinfection. Hepatitis C is the cause of 20% of acute viral hepatitis in the United States. However, the majority of acutely infected patients are asymptomatic and have a clinically mild course; jaundice is present in fewer than 25 percent. Thus, the patient's clinical presentation made acute hepatitis C unlikely.

Hepatitis D is a defective virus that requires the simultaneous presence of HBV for complete virion assembly and secretion. Coinfection of HBV and HDV in an individual susceptible to HBV infection (ie, anti-HBs-negative) results in acute hepatitis B and D. This entity is clinically indistinguishable from classical acute hepatitis B and is usually transient and self-limited. On the other hand, HDV superinfection of a chronic HBSAg carrier may present as a severe acute hepatitis in a previously unrecognized HBV carrier or as an exacerbation of pre-existing chronic hepatitis B, as in this case. Thus, hepatitis D superinfection represents the most likely cause of this patient's acute hepatitis.

Over the course of the patient's hospitalization his AST and ALT peaked at 3317 and 2475, respectively, and then began to decrease. However, his total and direct bilirubin continued to rise and reached levels of 44.9 and 23.2, respectively. Simultaneously, the patient's hemoglobin and hematocrit, which had been 14.5 and 44.5 on admission, decreased precipitously over the course of four days to 8.2 and 25.3. The patient showed no clinical evidence of bleeding, however hemolysis labs were notable for a low haptoglobin of 11 consistent with a hemolytic anemia. A peripheral blood smear revealed bite cells consistent with glucose 6 phosphate deficiency.

Briefly, glucose 6 phosphate dehydrogenase catalyzes the initial step in the hexose monophosphate shunt, oxidizing glucose 6 phosphate to 6-phosphogluconolactone and reducing NADP to NADPH. The hexose monophosphate shunt is the only red cell source of NADPH, a cofactor that is important in glutathione metabolism. Glutathione is a molecule which protects red blood cells against oxidative damage and thus when absent, predisposes red blood cells to hemolysis.

SUMMARY

In this case, an acute viral hepatitis was the precipitant of a hemolytic episode in a patient with underlying glucose 6 phosphate deficiency. A low glucose 6 phosphate level performed six weeks after the hemolytic episode confirmed the diagnosis.

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