Hemolysis in Acute Alcoholic Hepatitis: Zieve’s Syndrome

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Abstract

A 45-year-old man presented with acute alcoholic hepatitis, jaundice, and anemia on admission. There was no history of bleeding or any evidence of gastrointestinal blood loss. Lab studies revealed hemolysis as the cause of anemia. The patient was diagnosed with Zieve’s syndrome and managed with supportive measures. He recovered well and was discharged to a detoxification unit in a stable condition. Zieve’s syndrome has been described in literature, mostly in non-English language case studies, but is largely under-recognized and under-reported. Diagnosis should be made quickly to avoid unnecessary invasive diagnostic interventions.

Introduction

Zieve’s syndrome is an uncommonly recognized form of acute hemolytic anemia that manifests as a triad of jaundice, hyperlipidemia, and alcoholic steatohepatitis.1 The syndrome was first described in 1957 by Dr. Leslie Zieve. While there are many etiologies of anemia in alcoholics, Zieve’s syndrome is distinct in that it is an acute hemolytic anemia.1,2

Case Report

A 45-year-old man presented for voluntary admission to the alcohol detoxification unit. His alcohol history included consuming 4 hard lemonades in the morning, at least 1 pint of vodka during the day, and a “few” beers at night. He started consuming alcohol at about age 20 years, and since then, had been drinking heavily every day. At presentation, he complained of nausea, vomiting, weakness, and dark urine over the prior 2 weeks. He denied abdominal pain, fevers, chills, hematemesis, coffee ground vomitus, or melena. His past medical history was significant for hypertension and alcoholic liver disease. There was no history of any illicit drug use.

Physical examination was remarkable for scleral icterus, conjunctival pallor, and a dry oral mucosa. Laboratory tests showed hemoglobin 6.5 g/dL, hematocrit 19%, mean corpuscular volume 115 fl, red cell distribution width 17%, total bilirubin 16 mg/dL, direct bilirubin 6.3 mg/dL, alkaline phosphatase 47 U/L, aspartate aminotransferase 47 U/L, lactate dehydrogenase 326 U/L, albumin 3.5, and an international normalized ratio of 1:1. The patient’s peripheral smear showed polychromasia, macrocytosis, tear drop cells, ovalocytes, spur cells, and schistocytes (Figure 1). A stool occult blood test was negative. Upper endoscopy was normal with no varices. Additional laboratory tests were consistent with hemolytic anemia, including reticulocytosis (12%), elevated LDH level, and an undetectable haptoglobin level. The patient was diagnosed with Zieve’s syndrome, and was provided supportive management with intravenous hydration, supplementation of thiamine and folate, and management of alcohol withdrawal. He recovered rapidly during his short stay. His bilirubin trended down within a few days and hemoglobin remained stable 2 weeks after initial transfusion at 9.7 g/dL. He was discharged to a detoxification unit in a stable condition.

ACG Case Rep J 2015;2(4):250-251. doi:10.14309/crj.2015.75. Published online: July 9, 2015.

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Patients with Zieve’s syndrome may be recurrent, and the definitive treatment is alcohol cessation. \(^7,8\) Patients with Zieve’s syndrome may have other associations that need timely recognition and treatment, such as intracranial hemorrhage, acute renal failure, and myalgias. \(^9-11\) The challenging task remains identification of the syndrome to prevent unnecessary invasive testing or referrals and provide definitive therapy such as alcohol cessation counseling and enrollment into programs that would prevent relapse of alcoholism. Zieve’s syndrome should be suspected whenever there is anemia and elevation of unconjugated bilirubin in the setting of acute alcohol intake with no obvious sign of gastrointestinal bleeding.

**Disclosures**

Author contributions: S. Shukla obtained the clinical data, wrote the manuscript, and is the article guarantor. M. Sitrin reviewed the manuscript.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received: January 20, 2015; Accepted: May 14, 2015

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