Case Report
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Unraveling the Mystery: Late-Life Hemolytic Anemia with Blister Cells on Smear

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ABSTRACT

In red cell disorder, glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzymopathy. This enzymopathy leads to hemolysis of red cells under oxidative stress. This deficiency causes red cell hemolysis when exposed to certain drugs and infections. This Enzymopathy presents with different clinical presentations due to various genetic abnormalities involving the gene. In this case report, we describe the case of a 65-year-old male patient who was brought to the emergency department of our hospital with signs of shock and in a gasping condition. We found blister cells (3+) and Heinz bodies on peripheral smear examination. This case highlights the importance of considering red cell enzyme deficiencies in older populations with unexplained hemolysis.

INTRODUCTION

Glucose phosphate dehydrogenase (G6PD) deficiency is frequently seen among the red cell enzymopathies[1]. Around 400 million people in the world are affected[2], with a higher number of cases detected in the African and Mediterranean regions. A meta-analysis showed a global prevalence of 4.5% G6PD deficiency and indicated an incidence of 1.8% in Pakistan[3].G6PD deficiency is an X-linked genetic disorder with a variety of genetic variations in each case. G6PD deficiency leads to hemolysis of red cells due to oxidative stress, mostly deficiency reports in children and the younger population[4, 5]. This case report will highlight the need for the investigation of G6PD deficiency in the differential diagnosis of unexplained hemolytic anemia in the elderly population.

CASE PRESENTATION

A 65-year-old male with a history of hypertension, diabetes, and ischemic heart disease presented to the emergency department with altered mental status, pallor, and gasping condition. On presentation he was hypoglycemic and 8 amp**o**ules of dextrose were given but no improvement in the condition of the patient. The patient's attendant reported a fever, productive cough, and shortness of breath for 3 days. No history of passing dark color stool or hemorrhoids. He had a history of Coronary artery bypass grafting 8 years back.

On examination, he was pale and altered level of consciousness. He had a heart rate of 70 bpm, blood pressure of 135/100 mmHg, and a temperature of 39°C. His oxygen saturation on the pulse oximeter was 70% in room air. Complete blood count showed a hemoglobin of 7.0 g/dl (12.5-16.5), white blood cell count (WBC) of 21.9× 10^9/L (4.0-11.0), and platelets of 87 × 10^9/L (150-450). Reticulocyte was 14% (0.5-2.5%). Peripheral smear examination showed blister cell (3+), spherocyte, leukoerythroblastic picture with marked polychromasia, circulating nucleated red blood cells consistent with hemolysis as shown in pictures 1 & 2. Reticulocyte preparation showed Heinz bodies as shown in picture 3. His Prothrombin time was 32 seconds with an INR of 3.4. Liver function tests showed Total bilirubin 7.7 mg/dl (<1), direct bilirubin 4.52 mg/dl (0.0-0.25), Gamma GT 101 unit/l (0-37), SGOT: 400unit/l and alkaline phosphate was 742unit/l (46-116). Renal function tests showed urea levels of 149 mg/dl (15-55), creatinine of 3.34 mg/dl (0.7-1.18), sodium of 140 mmol/L (135-145), and potassium of 7.0 mmol/L (3.5-5.1). Serum calcium: 7.7 mg/dl (8.4-10.2), Magnesium: 2.77. Arterial blood gas: pH: 7.38, pCO2: 27.5 mmHg, pO2:391 mmHg, O2 saturation: 99.8% (sampling

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done on oxygen inhalation). Echocardiography showed severely dilated Left Atrium and dilated Left Ventricle with severe generalized systolic dysfunction. Chest X-ray showed few infiltrates. The plan is to send the G6PD level after recovery.

The patient was admitted to the intensive care unit and was put on mechanical ventilation. Intravenous fluid resuscitation with saline. Empirical antibiotics for suspected infection. He was on inotropic support. The patient expired after 1 day of admission.

Figure 1

Smear Shows Numerous Blister Cells on Low Power View

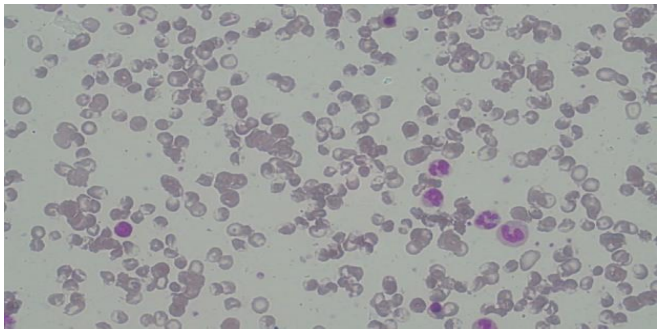


Figure 2

Smear Shows Blister Cells and Nucleated Red Cells on High-Power View

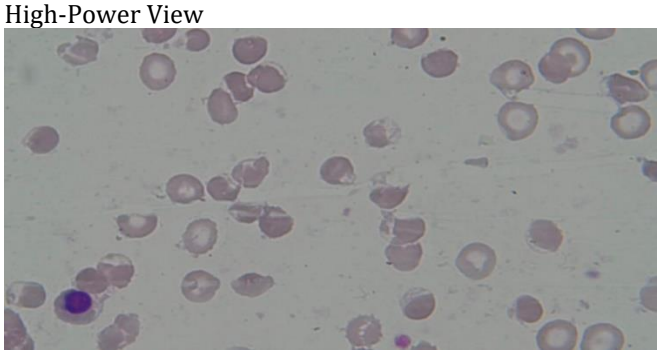
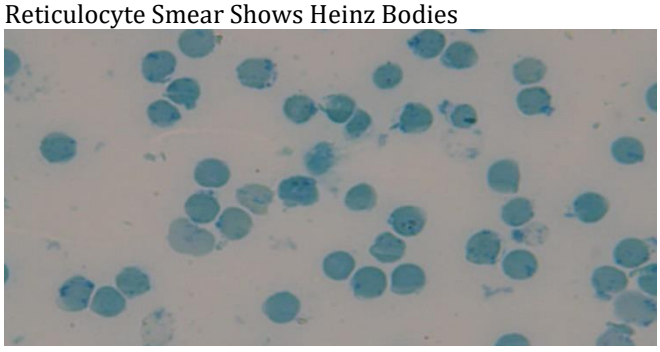


Figure 3

Reticulocyte Smear Shows Heinz Bodies



DISCUSSION

Our patient presented with hemolysis and pallor. A peripheral blood smear showed blister cells and Heinz bodies. He was a suspected case of G6PD deficiency based on the clinical picture and the presence of blister cells on the smear. Unfortunately, we didn't check the G6PD level because the patient was in a gasping condition and had an acute attack of hemolysis. There is no recommendation to check the G6PD level at the time of an acute attack. We had a plan to check after recovery but the patient did not survive. Hemolysis from G6PD deficiency usually manifests 24 - 72 hours after exposure to oxidant stress. It is plausible the metabolic acidosis induced by acute renal failure, acting in combination with an underlying oxidative trigger, precipitated his acute hemolysis. It is important to note that G6PD levels can be falsely normal during an acute hemolytic episode because reticulocytes have higher G6PD activity than mature red blood cells, which necessitates repeat testing after the resolution of hemolysis if there is high clinical suspicion of G6PD deficiency. Morphologically, the anemia is normocytic and normochromic with Reticulocytosis, presence of blister cells & Heinz bodies. Diagnosis requires G6PD assay. Most patients are well-adjusted to their anemia and usually require no treatment except Folic acid supplements. A deficiency of G6PD must be considered in the differential diagnosis of intra- and extravascular hemolysis[6]. Saldanha et al. [7] found an 8% prevalence of G6DP deficiency in individuals of African descent in Sao Paulo, Brazil. However, late presentations of G6PD deficiency have seldom been reported[8].

CONCLUSION

This case highlights the need for physicians to exclude the suspicion of G6PD deficiency in elderly patients who present with hemolysis and shock, especially when there is a recent history of drug exposure and infections. Early identification and management can significantly improve the outcomes in elderly patients.

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