CASE REPORTS

EMPLOYMENT OF G6PD DEFICIENT PATIENT IN HIGH ALTITUDE AREA– A CASE STUDY

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INTRODUCTION

Glucose-6-phosphate dehydrogenase deficiency (G6PD deficiency) also known as "favism" is an X-linked recessive genetic condition that predisposes to hemolysis[1] and resultant jaundice in response to triggers, such as hypoxia, certain foods, illness and medication[2]. Employment of G6PD deficient military person in high altitude environment causes a cascade of physiological responses triggered by hypobaric hypoxia[3] and often develops into pathological conditions. G6PD screening is not in vogue in initial induction in army or deployment in high altitude area of Force Command Northern Areas (FCNA).

CASE REPORT

A 27 year old patient was deployed at altitude of 18000 feet in FCNA. Initial acclimatization was uneventful. Patient suddenly felt difficulty in breathings and tachycardia and his problem further aggravated by heaters and smoking of peers in igloo. He was later moved to lesser height 15500 feet but problem of suffocation in closed living persisted. His blood pressure was normal with episodes of pallor, breathlessness and frequent tachycardia. He was moved back to Sialkot and later on he developed sore throat, fever and yellow discoloration of sclera. He suddenly became pale after ingestion of Tab Paracetamol. He was admitted in Combined Military Hospital (CMH) Sialkot with provisional diagnosis of Infectious Hepatitis. Serum HBV and HCV was negative. Serum bilirubin was 51 mg/dL while ALT was 71IU/L and Hemoglobin was 10 gm/dL. He was discharged and after three months again developed sudden paleness, difficulty in breathing and admitted again in CMH Sialkot with Serum Bilirubin 70 and Hemoglobin 9 mg/dL. He was thoroughly investigated including liver biopsy which was normal. There was no family history of thalasemia and sickle cell disease in the family. Later on he was tested for G6PD enzyme and found deficient in G6PD enzyme. He was treated conservatively and with folic acid therapy. Patient was counseled in detail for avoidance of precipitating factors and drugs to prevent future complications.

DISCUSSION

G6PD deficiency is an X-linked congenital enzymopathy[4] being present in more than 400 million people worldwide which increases the vulnerability of erythrocytes to oxidative stress. Majority of patients are asymptomatic. Symptomatic patients are almost exclusively male, but female carriers can be clinically affected due to unfavorable lyonization. Clinical presentations include acute hemolytic anemia, chronic hemolytic anemia, neonatal hyperbilirubinemia, and an absence of clinical symptoms.

When there are sufficient grounds to suspect G6PD, a direct test for G6PD[5] is the " Beutler fluorescent spot test[11]". Other investigations are complete blood count and reticulocyte count. In active G6PD deficiency, Heinz bodies can be seen in red blood cells on a blood film, liver enzymes (to exclude other causes of jaundice) and Lactate dehydrogenase (elevated in hemolysis and a marker of hemolytic severity).

The most effective management of G6PD deficiency is to prevent haemolysis by avoiding

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oxidative stress, drugs, foods and infections. Rarely, anemia may be severe enough to warrant a blood transfusion. Folic acid and iron potentially are useful in hemolysis. Patients with G6PD deficiency can pursue military carrier by avoiding oxidative stresses and factors triggering the symptoms.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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