Screening for G6PD Deficiency in Blood Donor Population

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Received: 25 June 2009 / Accepted: 26 April 2010 / Published online: 19 October 2010
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Dear Editor,

Glucose-6-phosphate dehydrogenase deficiency is the most common enzyme deficiency worldwide. Approximately 400 million people are said to be affected worldwide. According to world health organization 7.5% of world population are carriers of glucose-6-phosphate dehydrogenase deficiency and 2.9% were G6PD deficient [1]. It causes spectrum of diseases including neonatal hyperbilirubinemia, acute hemolytic and chronic hemolytic anemia. Usually the affected individuals are asymptomatic unless they are provoked by oxidative stress [2]. This enzyme deficiency is very prevalent in individuals of Africa, America, Mediterranean, and East Asia. In India the incidence of G6PD has been variably reported as 0–37% in different castes and communities. Higher incidence of G6PD deficiency is seen in north and west India (15%) as compared to south India (1–2%) [3]. It implies that this inherited metabolic disorder is an important health problem in India and it is necessary to carryout large scale screening in the whole population. Severe manifestations of G6PD deficiency can be prevented if necessary precautions are taken, hence this study was done to determine the incidence of G6PD deficiency in asymptomatic population

represented by healthy blood donors and to characterize the demographic profile and laboratory profile of such group.

The study was conducted on 2005 healthy blood donor samples. This screening program included 2000 male and 5 female blood donors who came to blood bank for donation of blood. These donors were from different part of India. The G6PD activity was assessed in their red cells using anticoagulated (EDTA) venous blood.

Sixteen blood donors out of 2005 screened by methaemoglobin reduction test were found to be G6PD deficient. All of them were male. These individuals were clinically normal and had no significant past history. The incidence of G6PD deficiency was 0.8% in the sampled population.

Among this 16 G6PD deficient subjects two blood donors were from West Bengal, one from Kerala and the rest of the 13 subjects were from Karnataka.

The mean Haemoglobin values of the normal donor population vs. G6PD deficient donor population were compared using unpaired *t* test. The difference in the mean Hb value was not statistically significant (*P* value > 0.5).

Number of donors screened	2005
Number of male donors	2000
Number of female donors	5
Number of G6PD deficient donors	16
Mean Hb of normal male donors	15.8 gm/dl
Mean Hb of G6PD deficient donors	14.8 gm/dl

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Sixteen units of blood were transfused to patients. There was two [12.5%] transfusion related reaction among the patients who received the G6PD deficient blood.

One of the patients, who was transfused following upper Gastro intestinal bleeding, had itching and rashes all over the body after transfusion of 50 ml blood. The reaction

workup done ruled out haemolytic reaction. A probability of allergic reaction was considered. As in this patient transfusion was stopped and he did not receive further transfusions for next 48 h, his Hb fell from 9.0 to 7.7 gm/dl.

The second patient was a neonate undergoing surgery for a ruptured meningocele. The blood was transfused during surgery following which the baby developed mottling and passed red colored urine. A hemolytic transfusion was suspected. The transfusion reaction workup showed haemolysis in the post transfusion sample and hemoglobinuria. But blood bag showed no hemolysis. However as pretransfusion sample also showed haemolysis, a hemolytic reaction could not be conclusively reported. Hemoglobin dropped from 12.2 to 8.0 gm/dl.

The beneficial effects of blood transfusion, for the typical 70 kg human each unit of whole blood should increase the hematocrit by 3–5% or hemoglobin by 1–1.5 gm/dl [4]. Testing for G6PD deficiency is not routinely practiced for donors units [2]. Various studies have reported a mild increase in bilirubin, LDH, and decrease in hemoglobin following the transfusion of G6PD deficient blood. This is especially in neonates (preterm). Transfusion of G6PD deficient red cells to the premature infants has been associated with hemolytic and severe hyperbilirubinemia requiring exchange transfusion [5]. In our study also one neonate developed suspected hemolytic transfusion reaction following transfusion of G6PD deficient blood. Under normal circumstances the transfusion of G6PD deficient blood is harmless for most of the individuals until they are exposed to certain oxidative drugs.

This study was an attempt to find out the prevalence of G6PD deficiency in our population. This will help us to quantitate the magnitude of the problem in the population and initiate measures to screen the population or a selected group in the population. The prevalence of G6PD being very low in this study (0.8%) and as many people remain asymptomatic unless challenged with oxidative drugs or infection, screening the whole population for G6PD deficiency is not warranted. As one of the neonates receiving G6PD deficient blood developed suspected hemolytic reaction, screening of blood to be transfused for select population like neonates is advisable.

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