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Brief Report

Prevalence of Glucose-6-Phosphate Dehydrogenase Deficiency among Male Donors in Shiraz, Southern Iran

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Abstract

The overall incidence of glucose-6-phosphate dehydrogenase (G6PD) deficiency in Iranian population is estimated around 10%–14.9%. G6PD deficiency is an X-linked disorder and 80% of donors are usually male. At present, donors' blood is not routinely screened for G6PD deficiency in Iran blood bank where for detecting such enzyme deficiency, reliance is placed on pre-donation data. Thus, the G6PD deficient blood may be transfused to G6PD deficient premature neonates, resulting in prolonged or severe hyperbilirubinemia. Fluorescent spot method was used to determine G6PD deficiency in 450 blood samples collected from male donors stored less than 7 days. This was followed by the pre-donation questionnaires for the history of previous diseases or jaundice. Of which 27 (6%) samples were G6PD deficient. None of the donors tested for G6PD deficiency recalled, and stated in their pre-donation questionnaires, any history of previous diseases or jaundice.

Six percent G6PD deficiencies among the male donors in Shiraz blood bank was a noteworthy prevalence, which was independent of the pre-donation questionnaires. Therefore, regardless of the questioners' data, it was recommended to screen the blood bags for this enzyme prior to use for simple or exchange transfusion in premature infants.

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Keywords ● G6PD deficiency ● neonates ● exchange transfusion

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Introduction

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common known human enzyme disease, affecting 10% of the world population which accounts for 200 to 400 million affected people worldwide.[1] Deficiency of G6PD enzyme in the red blood cells, under certain circumstances, may lead to an abnormal rupture of the cell wall with resultant hemolytic anemia.[1] The likelihood of developing hemolysis and its severity is determined by the magnitude of the enzyme deficiency which is consistent with the biochemical characteristics of each G6PD variant.[2] In this context, World Health Organization (WHO) has classified the different G6PD variants.[2] The abnormal gene responsible for this inherited deficiency is located on the X-chromosome. Therefore, the illnesses associated with G6PD deficiency occur more frequently in males than females.[3] With the most prevalent G6PD variants (G6PD A[1] and G6PD Mediterranean), hemolysis is induced by sudden

destruction of the older and more deficient erythrocytes. This occurs after exposure to some drugs of high redox potentials, mothballs, henna, fava beans, or following certain infections and metabolic abnormalities. In G6PD deficient neonates, decreased bilirubin elimination may play an important role in development of jaundice.[4]

The most common variants that cause acquired hemolytic anemia pose little health hazards.[5] Therefore, the donors' blood are not routinely screened for G6PD deficiency in Shiraz blood bank and the detection of such enzyme deficiency is relied on the pre-donation data. Considering the potential hazards of severe hemolysis in neonates and maleness of 80% of the donors being males, the present study was conducted to determine the prevalence of G6PD deficiency in male donors referring to Shiraz blood bank and to evaluate the alleged pre-donation data for detecting G6PD deficient donors.

Materials and Methods

This cross-sectional study was performed on 450 blood samples taken from male donors during April 2003 to April 2004. The samples had been stored for less than 7 days in Shiraz blood bank prior to testing for G6PD enzyme deficiency. G6PD enzyme deficiency test was done by florescent spot method. This semi-quantitative assay was most reliable and highly sensitive which classified a sample simply as "normal", or "deficient" when it showed less than 30% of the normal activity. Activities above 30% were unlikely to be accompanied by clinical manifestations. A false negative result of this test was G6PD deficient females and post-acute hemolytic reaction with many circulating young red blood cells.[6,7] Therefore, the reliability of the results obtained was due to the recruitment of male donors in present investigation.

All the pre-donation questionnaires of the positive or negative samples were examined to evaluate the history of hepatitis, pallor or jaundice, tea-colored urine and hemolysis due to febrile illness or dugs. However, there were not any direct questions about clinical signs or symptoms of G6PD deficiency, like hemolytic reaction after receiving drugs, history of favism, etc in current pre-donation blood bank questionnaires.

Results

As inferred from the pre-donation questionnaires, no history of previous diseases, such as hepatitis and febrile illnesses, were found in 450 donors that might increase the chance of hemolysis in G6PD deficient patients. We also did not find any history of drug-induced tea-colored urine, pallor and jaundice or hemolysis. G6PD deficiency was found in 27 (6%) samples of 450 blood bags. According to pre-donation questionnaires, none of the G6PD deficient donors or their families had any history of previous hemolytic reaction or jaundice due to febrile illnesses or drugs.

Discussion

The first study on Iranian medical staff in 1959 showed an incidence of 9.5% G6PD deficiency.[8] Another study on cord blood reported an incidence of 12% G6PD deficiency in males and 0.9% in females in Fars province, Southern Iran.[9] According to the report of WHO, the overall incidence of G6PD deficiency among the Iranian population was 10%–14.9%.[10] Screening of the donors' blood is not routinely performed, because there are no deleterious consequences in recipients of G6PD deficient blood.[11] Under normal circumstances, G6PD deficiency is harmless for most children. However, it can in particular raise a serious problem in neonates, when they develop an infectious illness or being exposed to certain materials or drugs which increase the amount of oxidative stress on the red blood cell wall.[3]

Transfusion of G6PD deficient red cells to the premature infants has been associated with hemolysis and severe hyper-bilirubinemia requiring exchange transfusion.[12] Massive intravascular hemolysis had also occurred after exchange transfusion with G6PD deficient blood.[13,14] The age of red cells was important, because glutathione-dependent antioxidant systems in erythrocytes and antioxidant defense system in plasma was depleted during blood storage.[15] Previous studies have revealed that 12-day period is a safe storage limit.[15]

The prevalence of G6PD deficiency should be considered reliable and noteworthy, regardless of the pre-donation data. Therefore, it is recommended to screen the blood bags allocated for exchange transfusion of premature infants.

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