Dear Sir,

Glucose-6-phosphate dehydrogenase (G6PD) deficiency, affecting about 330 million people worldwide, is the most common hereditary enzymopathy. (1) Although most individuals are asymptomatic, this condition can lead to the development of neonatal jaundice, chronic nonspherocytic hemolytic anemia and acute hemolytic anemia induced by certain types of drugs, infections or the consumption of broad beans.(2)

Moreover, blood transfusions from G6PD deficient donors are less effective as the red blood cells of these individuals are deficient in antioxidant defense mechanisms; they are more vulnerable to extra- and intra-vascular hemolysis due to oxidative stress and can have excessive storage degradation with decreased survival.(3)

The overall prevalence of G6PD deficiency is geographically correlated to endemic malaria, hence higher in sub-Saharan Africa, the Middle East, Southeast Asia, Mediterranean Europe and some areas of Latin America.(4) Studies on the Brazilian population have reported a prevalence of G6PD deficiency in about 1% to 10% of the population with the highest rates being found among men of African descent.(4) In the particular case of Rio Grande do Norte, previous studies have shown a prevalence of 3.5% in patients seen at a referral hospital in the city of Natal.(5) However, no study on G6PD deficiency in a provincial population of Rio Grande Norte had been performed previously. Hence, this cross-sectional study was conducted during the period from August 2006 to August 2008, to determine the prevalence of G6PD deficiency in blood donors from the city of Mossoró, the largest provincial city of Rio Grande do Norte.

A total of 714 individuals (aged 18-62 years, mean 30 years) who voluntarily went to the local blood center to donate blood were analyzed; 576 (80.7%) were men and 138 (19.3%) women. 343 (48.0%) said they were white and 371 (52.0%) stated they were mulatto, brown, yellow or black (grouped as nonwhites).

Donors were informed about the nature and importance of the work and were asked about their willingness to participate in the study. Those who agreed were asked to sign informed consent forms and a standard questionnaire designed to obtain demographic data was applied. Subsequently, 5-mL samples of peripheral venous blood were collected in tubes containing 50 µL 10% EDTA for laboratory tests.

The G6PD activity was initially measured by the methaemoglobin reduction test(6) and with confirmation of positive results by the methaemoglobin reduction test modified for semiquantitative evaluation.(7) Individuals were considered deficient if the enzyme activity was below 70%. Statistical analyses of the results were carried out employing the Pearson χ² test using the PEPI program. Statistical significance was set for p-values <0.05.

The protocol of this study was approved by the Research Ethics committee of the Hospital Universitário Onofre Lopes (CAAE # 0395.0.000.294-07).

Twenty-seven (3.8%) cases of G6PD deficiency were identified, a percentage similar to those described for the population of Natal (3.5%) and Bahia (3.2%). Among the G6PD deficient individuals, 21 (77.8%) were male and 6 (22.2%) were female. There were no statistically significant differences in the prevalence in enzyme deficiency between genders (χ² = 0.151, p = 0.698) (Table 01), even though it has characteristically X-linked inheritance. This finding may be attributed to the fact that the deficiency does not cause significant harm and thus reproduction between affected individuals and carriers of G6PD deficiency is relatively common.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Total samples</th>
<th>G6PD activity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>(%)</td>
</tr>
<tr>
<td>Male</td>
<td>576</td>
<td>555 (96.4)</td>
</tr>
<tr>
<td>Female</td>
<td>138</td>
<td>132 (95.7)</td>
</tr>
<tr>
<td>Total</td>
<td>714</td>
<td>687 (96.2)</td>
</tr>
</tbody>
</table>

(Pearson χ² test): χ² = 0.151, p = 0.698

Thirteen (48.1%) of the G6PD deficient individuals stated they were white and 14 (51.9%) stated they were non-whites; no statistical differences were observed between the ethnic groups (χ² = 0.145; p = 0.703) (Table 2).

These relatively high prevalence rates of G6PD deficiency, together with the reduced effectiveness of blood transfusions from...
individuals with this genetic condition, reinforces the need of
G6PD deficiency screening of donors, especially when blood is
transfused in neonates.

Abstract

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most
common human enzymopathy. It affects as many as 330 million
individuals worldwide. This deficiency may determine neonatal
jaundice, chronic nonspherocytic hemolytic anemia and acute
hemolytic anemia induced by drugs, infections and broad bean
ingestion. The efficacy of blood transfusion is decreased when the
donor is G6PD deficient. In this study, we aimed at determining the
prevalence of G6PD deficiency in blood donors of Mossoro, Brazil.
Samples of 714 blood donors (576 men and 138 women; 343 white
and 371 non-white) with ages ranging from 18 to 62 years and that
accepted to participate in the study were analyzed. All participants
answered a standard questionnaire. G6PD activity was analyzed
by the methemoglobin reduction test with deficiency being confirmed
by the semiquantitative test. The overall prevalence of G6PD
deficiency in blood donors was 3.8%, similar to the rate described
for others regions of Brazil. There was no significant statistical
difference in the frequency of G6PD deficiency between men and
women, nor between white and non-white blood donors. This relatively
high frequency of G6PD deficiency highlights a need to screen blood
donors for this condition.

Keywords: Glucosephosphate dehydrogenase deficiency; Blood
donors; Anemia

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