Prevalence of glucose-6-phosphosphate dehydrogenase deficiency in the newborns in Sanandaj, Iran

Nahid Ghotbi¹, Nadia Adnani²

¹ Associate Professor, Kurdistan Research Center for Social Determinants of Health, Kurdistan University of Medical Sciences, Sanandaj, Iran
² Resident, Department of Pediatrics, School of Medicine, Kurdistan University of Medical Sciences, Sanandaj, Iran

Abstract

BACKGROUND: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic disorder and assessment of newborns with or without this deficiency is an essential component in public health evaluation in different countries. Hence, this study was aimed to assess the prevalence of G6PD deficiency in the newborn population in Sanandaj, Iran.

METHODS: This is a cross-sectional study on 2016 newborns in Besat Hospital in Sanandaj, Iran, in the year 2006. Three drops of blood were collected from the infants’ heels using sterile needles. Then fluorescent spot test was utilized to study the activity of G6PD enzyme.

RESULTS: The results of the present study conducted on 2016 neonates showed that 48.80% (984) of them were males and 51.20% (1032) were females. Prevalence of G6PD deficiency in boys and girls were 7.62% and 2.52%, respectively with a male to female ratio of 3:1.

CONCLUSION: G6PD deficiency is a gender related condition with a higher frequency among boys’ population.

KEYWORDS: Glucose-6-Phosphate Dehydrogenase Deficiency, Fluorescent Spot Test, Sex Related

Introduction

Glucose-6-phosphate dehydrogenase (G6PD) is a universally common hereditary disorder. According to World Health Organization (WHO) reports, 2.9% of the world population and 10-15% of the Iranians have G6PD deficiency.¹,² This disease is more prevalent in Africa, Asia, the Mediterranean, and the Middle East, and approximately 200-400 million people suffer from it all over the world.³⁴

G6PD enzyme exists in all cells and has a crucial role in providing cell protection during oxidative stress.⁵ In individuals with G6PD deficiency, lifetime of these cells is lower than normal due to oxidation of red blood cell membrane; therefore, hemolysis occurs. Moreover, excessive red cell hemolysis and increased catabolism raise blood bilirubin and jaundice.⁶

Main symptoms of defect in G6PD enzyme include acute hemolytic anemia and classic appearance of favism, neonatal jaundice, and non-spherocytic hemolytic anemia.⁷ Neonatal jaundice is one of the most important and remarkable symptoms of this deficiency.⁸ An important problem of hyperbilirubinemia is irreversible neurological complications and profound mental retardation in the newborns, which are highly prevalent in Greek, Nigeria, Saudi Arabia, and Southern Iran.⁴⁹ Individuals

Date of submission: 26 Dec 2013, Date of acceptance: 09 Feb 2014


Corresponding Author: Nahid Ghotbi
Email: ghotbinahid@yahoo.com
with this deficiency cannot be blood donors. Moreover, prolonged contact with mild cell hemolytic individuals can cause chronic anemia.\textsuperscript{10}

Identifying neonates with this enzyme deficiency is highly significant. Different studies from around the world have considered screening of neonates regarding G6PD enzyme as a very effective factor in decreasing Kernicterus. Recent studies on health education in ethnic Kurds revealed a high prevalence of this medical and health risk factors.\textsuperscript{11-13}

However, few studies have been conducted on G6PD in ethnic Kurds as one of the Iranian ethnicities and prevalence of the disease in Sanandaj, Iran, has not been identified yet; therefore, detection of the disease would help reduction of side-effects by consuming food and medication containing antioxidants. Hence, the present study was aimed at identifying prevalence of G6PD dehydrogenase deficiency in neonates in Sanandaj.

**Materials and Methods**

This is a cross-sectional study conducted on 2016 neonates born in Besat Hospital in Sanandaj from 2006 to 2007. Permissions were obtained from neonates’ parents. Then three drops of blood collected from the infants’ heels using sterile needles after recording of the infants’ characteristics.

Fluorescent spot test was utilized to study the activity of G6PD enzyme. For this purpose, 100 µl of reagent put in a small container and 10 µl of whole blood containing ethylenediaminetetraacetic acid were taken from the neonates’ feet which was added and mixed afterwards. Subsequently, the solution was kept at the room temperature for 20 min, a drop was taken from it using a 20-µl sampler and put on the filter paper. Then the paper was left out to dry. The resulted spot was put under fluorescent light with the wavelength of 365 µm and its reflected light studied, then the collected data were registered. Samples with G6PD enzyme had the ability to catalyze the chemical reaction, reflect the fluorescent light, and turned into green; however, those with G6PD enzyme deficiency turned into black and could not reflect the light. Data were analyzed in SPSS for Windows (version 16.0, SPSS Inc., Chicago, IL, USA) using chi-square and odds ratio (OR).

### Results

The present study conducted on 2016 neonates including 984 (48.80%) males, and 1032 (51.20%) females. Prevalence of G6PD dehydrogenase deficiency was 5.00%, 7.62% in male infants and 2.52% in female. The ratio of male to female in this particular enzyme was 3.02, in addition, chi-square test proved a statistically significant relationship between G6PD dehydrogenase deficiency and neonates’ sex (P = 0.0001). The OR was 3.19 (confidence interval 1.98-5.17) (Table 1).

<table>
<thead>
<tr>
<th>Sex</th>
<th>With G6PD deficiency, n (%)</th>
<th>Without G6PD deficiency, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>75 (74.3)</td>
<td>909 (47.5)</td>
</tr>
<tr>
<td>Female</td>
<td>26 (25.7)</td>
<td>1006 (52.5)</td>
</tr>
<tr>
<td>Total</td>
<td>101 (100)</td>
<td>1915 (100)</td>
</tr>
</tbody>
</table>

**Discussion**

G6PD dehydrogenase deficiency is a sex-related disorder and has different prevalence rates in different countries. In the present study, prevalence of G6PD dehydrogenase deficiency was 5.0%. Its level of prevalence varies in different regions of the country. It has been reported to be 19.3% in Southeastern Iran,\textsuperscript{14} 10.9% in Mazandaran,\textsuperscript{15} 7.5% in Isfahan,\textsuperscript{16,17} 3.2% in Tehran,\textsuperscript{18} and 2.1% in Zanjan.\textsuperscript{19} In a similar study conducted in Rafsanjan by Alidalaki, G6PD prevalence was 5.0%.\textsuperscript{10} Prevalence of G6PD dehydrogenase deficiency was 25.0% in Oman,\textsuperscript{20} and 22.0% in Nigeria;\textsuperscript{21} despite a lower rate of prevalence of 5.0% and 3.0% in Canada and UK respectively.\textsuperscript{22,23} Therefore, G6PD
prevalence in Sanandaj compared to other countries in the world is in the middle. In
addition, due to differences in level of prevalence in different parts of Iran from one side and
different countries on the other side, it could be
concluded that factors such as race, geographical
conditions, and weather are responsible for the
differences in the number of affected cases.

Prevalence of G6PD dehydrogenase
deficiency in boys and girls were 7.62% and
2.52%, respectively with a male to female ratio of
3:1. In Kazemi et al. study, female population
outnumbered the disease cases.\textsuperscript{18} In a study
conducted by Khalesy et al., boys suffered from
G6PD dehydrogenase deficiency 5 times more
than girls.\textsuperscript{24} In Alidalaki et al. study, there was no
significant difference between boys and girls
regarding the incidence of the disease.\textsuperscript{10} In a
study conducted in Mazandaran by Ahmadi and
Ghazizadeh, it was reported that the incidence
of the disease in boys were 3 times more than
girls.\textsuperscript{25}

Due to sex-related nature of G6PD enzyme
deficiency, in different parts of Iran, especially
where the disease incidence among girls and boys
is the same, differences in levels of prevalence in
the two sexes is likely to be related to regional and
racial factors. However, it is necessary to conduct
related studies further on a larger scale targeting
all over Iran. Therefore, it is recommended to
screen, especially cord blood of male infants right
after birth. It is also recommended that further
national studies on the prevalence of G6PD
enzyme deficiency be conducted.

**Conclusion**

Prevalence of G6PD dehydrogenase deficiency
was 5.00%, 7.62% in male infants and 2.52% in
female in Sanandaj. Therefore, G6PD
dehydrogenase deficiency is a gender related
condition with a higher frequency among boys’
population.

**Conflict of Interests**

Authors have no conflict of interests.

**Acknowledgments**

This article was adopted from dissertation of Nadia Adnani. Here, the researchers would like
to appreciate all those who provided us with
their sincere help and attention particularly
personnel of delivery ward and operation room
of Besat Hospital, Sanandaj, Iran.

**References**

1. Kaplan M, Hammerman C. Glucose-6-phosphate
dehydrogenase deficiency and severe neonatal
hyperbilirubinemia: a complexity of interactions
between genes and environment. Semin Fetal Neonatal
2. Kaplan M, Hammerman C. Glucose-6-phosphate
dehydrogenase deficiency: a potential source of severe
neonatal hyperbilirubinemia and kernicterus. Semin
3. Nkhoma ET, Poole C, Vannappagari V, Hall SA,
Beutler E. The global prevalence of glucose-6-
phosphate dehydrogenase deficiency: a systematic
review and meta-analysis. Blood Cells Mol Dis 2009;
42(3): 267-78.
4. Segal GB. Enzymatic defects. In: Kliegman RM,
Behrman RE, Jenson HB, Editors. Nelson Textbook of
Pediatrics. 18\textsuperscript{th} ed. Philadelphia, PA: Elsevier Science
Health Science Division; 2007. p. 2039-42.
5. Abolghasemi H, Mehrani H, Amid A. An update on
the prevalence of glucose-6-phosphate dehydrogenase
deficiency and neonatal jaundice in Tehran neonates.
6. Glucose-6-phosphate dehydrogenase deficiency. WHO
7. Luzzatto L. Glucose 6-Phosphate Dehydrogenase
and Oski's Hematology of Infancy and Childhood. 6\textsuperscript{th}
8. Usanga EA, Ameen R. Glucose-6-phosphate
dehydrogenase deficiency in Kuwait, Syria, Egypt,
Iran, Jordan and Lebanon. Hum Hered 2000; 50(3):
158-61.
Necessity of glucose-6-phosphate dehydrogenase
activity determination in hyper bilirubinemia
[In Persian].
10. Alidalaki S, Negahban T, Halakooei M, Sayadi AR,
Ahmadi Kohnali J, Esmaeilzadeh JM, et al.
Investigation of Glucose-6-phosphate Dehydrogenase
Deficiency in Rafsanjan, Autumn 2004. J Rafsanjan