

Prevalence of Glucose-6-phosphate Dehydrogenase Deficiency in People Visiting Health Care Center, KFU, Al-Hasa

Glucose-6-phosphate Dehydrogenase Deficiency

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ABSTRACT

Objective: To determine the prevalence of G6PD deficiency in people visiting Health Care Center of King Faisal University (KFU), Al-Hasa.

Study Design: Observational / Descriptive study.

Place and Duration of Study: This study was conducted at the Health Care Center, KFU, Al-Hasa, from August 2014 to April 2015.

Materials and Methods: Patients presenting with weakness and anemia were included in the study. Total 214 patients, consisting of 116 children (age 3 to 14 years) and 98 adults (age 15 to 50 years), were screened. The blood samples were analyzed by using "G6P-DH Fluorescence Screening Test". This kit detects fluorescence under U/V lamp if the sample has G6PD activity.

Results: Total 36 (16.8%) of the 214 patients, tested, were deficient for G6PD. Among those deficient patients, 15 (7%) were children (11 males and 4 females), and 21 (9.8%) were adults (13 males and 8 females). The highest prevalence of G6PD-deficiency occurred among adults, particularly males.

Key Words: G6PD deficiency, Red blood cells, Enzymopathy, Anemia.

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INTRODUCTION

Glucose-6-phosphate dehydrogenase (G6PD) is the major enzyme for red blood cells (RBCs) to get the energy from glucose¹. It produces reduced nicotinamide adenine dinucleotide phosphate and reduced glutathione phosphate that play an important role in protecting cells from oxidative damage. Deficiency of G6PD is an enzymopathological disorder of RBCs in humans which is caused due to mutations in G6PD gene, located on X-chromosome². Therefore, this deficiency is most common among males³. G6PD deficiency is one of the most common enzyme disorders, affecting more than 4 million people worldwide⁴. It has high prevalence in Mediterranean, Asian, and African people⁵. In the Middle East it has high range that varies from 3% to 29%^{6,7}. In Asia, the prevalence ranges from 6.0% to 15.8%^{8,9} and in Africa, from 3.6% to 28%^{9,10}.

Lots of individuals with G6PD-deficiency usually do not experience any signs or symptoms, only some individuals present with hemolytic anemia. A wide range of hemolytic syndromes is included in the clinical

manifestations of G6PD deficiency. G6PD deficiency is usually associated with acute hemolytic anemia and neonatal jaundice, which is usually a result of external oxidant agents¹¹. Some oxidant agents such as fava beans¹²⁻¹⁴, topical application of henna^{15,16} and infections¹⁷⁻¹⁹ have been reported to trigger hemolytic anemia in G6PD-deficient individuals. This disorder had been also shown to be common in our region Al-Hasa several years ago (in 1998)²⁰. However, there is a paucity of current prevalence and awareness of G6PD deficiency among people visiting Health Care Center of KFU. Therefore, this study was specifically aimed to determine the prevalence of G6PD deficiency among people visiting the Health Care Center of KFU.

MATERIALS AND METHODS

Subjects and samples: The patients presenting with weakness and anemia were screened for G6PD. The study contained 214 patients, consisting of 116 children (age 2 to 14 years), including males 49 (42.2%) and females 67 (57.8%), and 98 adults (age 15 to 50 years old), including males 25 (25.5%) and females 73 (74.5%). The peripheral blood sample (2-3ml) was collected and analyzed for G6PD screening.

Material and Procedure: For screening of G6PD, the kit "G6P-DH Fluorescence Screening Test" (supplied by UDi Dammam company) was used. 5 micro liters of whole blood were collected in a suitable anticoagulant and added into a test tube with 100 micro liters of working reagent, provided in the kit. This mixed tube

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was put on Filter Paper and allowed to air-dry for 30 min. After the Filter Paper was completely dry, it was observed under a long wave UV-lamp in a darkened room. The positive and negative controls, provided in the kit, were run in parallel. The samples which did not fluoresce were labelled as deficient for G6PD.

RESULTS

Out of 214 patients, 36 (16.8%) were deficient for G6PD. Amongst those G6PD deficient patients, 15 (7%) were children, including 11 (73.3%) males and 4 (26.7%) females, and 21 (9.8%) were adults, including 13 (61.9%) males and 8 (38.1%) females. Figure 1 describes the number of patients, deficient for G6PD-deficiency in individual group. Figure 2 shows the percentage of the patients, deficient for G6PD in individual group. On the basis of individual groups of the subjects, we had 4 groups, i.e., female children, female adults, male children and male adults. The highest prevalence of G6PD-deficiency was found among male adults which was 13 of 25 (52%). The second most common group was male children, which was 11 of 49 (22.4%). The third common group was female adults, which was 8 of 73 (10.9%), and the fourth group was female children which was 4 of 67 (5.9%).

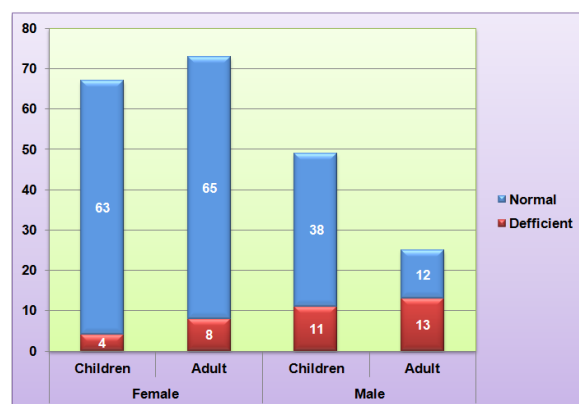


Figure No.1. Number of patients with G6PD deficiency (red box) among individual group.

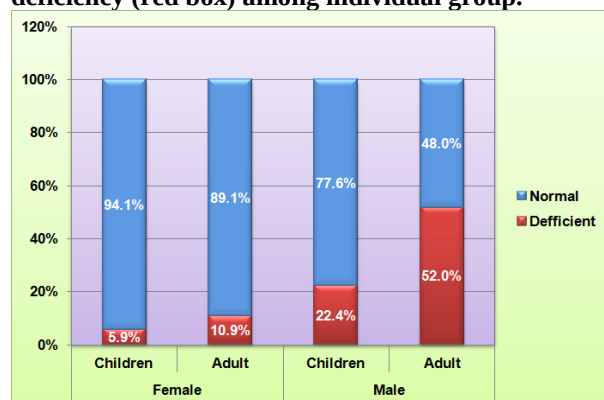


Figure No.2. Percentage of patients with G6PD deficiency (red box) among individual group.

DISCUSSION

G6PD deficiency has high prevalence in Mediterranean, Asian and African people⁵. The prevalence of G6PD deficiency in all over the Middle East was reported to have high range variation from 3% to 29%^{6,7}. In Asia, the prevalence ranges from 6.0% to 15.8%^{8,9} and in Africa between 3.6% to 28.0%⁹. Saudi Arabia, which is in the Middle East, is a large country with around 30 million people living in an area of 2,149,690 km², and the research has reported variable incidence of G6PD deficiency among Saudi populations in its various regions²¹. In our current study, we investigated the prevalence of G6PD deficiency in people who visited Health Care Center of KFUPM Al-Hasa with the clinical presentation of weakness and anemia. We investigated a significant prevalence of this disorder, which was 16.8%. In comparison to the other studies, we have unique findings as we investigated the patients who were or belonged to the literate members of King Faisal University AL-Hasa. The highest prevalence of G6PD deficiency was found among adult males. The second most common group was male children. Our findings justify the prevalence of G6PD deficiency among males as it is an X-linked disease which most commonly affects this group^{2,3}. Other groups in KSA have investigated the prevalence of this disease in general population which include both literate and illiterate people. For example, previous reports on G6PD deficiency showed the prevalence of 45.9% in Al-Qatif and 36.5% in Al-Hasa Saudi Arabia²². Alabdulaali et al. reported an incidence of G6PD deficiency of 1.13% in blood donors in the capital city Riyadh²³, while in the rural city of Al-Kharj it was found to be 1.91%²⁴. Other studies in the big cities, such as, Najran, Riyadh, Bisha, Al-Ula and Makkah²⁵⁻²⁷ showed the range from 3.5 to 6.7%. The frequencies in Al Hofuf, Khubar and Jazan²⁵, and Al-Baha²⁸, and Al Qunfudhah²⁹ have been reported to be between 11.6 and 18%. Alharbi and Khan showed the prevalence of 4.75 in Taif city³⁰. These results vary from region to region, being common in certain regions and less common in other regions of KSA.

Though our results are consistent with the previous studies to have the prevalence of around 17% of this disease, our findings differ from other groups as we chose a restricted group of patients, belonging to literate people of KFUPM, who visited the health care center of KFUPM with the clinical presentation of weakness and anemia. Therefore, in our study the patients who were not deficient for G6PD, might be suffering from other hematological disorders. This is an alarming sign that there is a lack of awareness of G6PD deficiency disorder among even the KFUPM people. Furthermore screening and genetic counseling programs for not only G6PD deficiency but also for other genetically transmitted hematological disorders need to be conducted throughout Al-Hasa region on big

platform including educational institutes and public places.

Therefore, there is a need to arrange the awareness programs on G6PD deficiency and its screening and genetic counseling throughout Al-Hasa province.

CONCLUSION

This study has shown a significant prevalence of G6PD deficiency among KFU population and their families. Further health programs on G6PD deficiency need to be launched throughout Al-Hasa region in order to avoid the factors which cause hemolysis in these patients and to prevent the transmission of this disease to the next generations through genetic counseling.

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Conflict of Interest: The study has no conflict of interest to declare by any author.

REFERENCES

1. Frank JE. Diagnosis and management of G6PD deficiency. *Am Fam Physician* 2005;72 (7): 1277–82.
2. Mason PJ, Bautista JM, Gilsanz F. G6PD deficiency: the genotype-phenotype association. *Blood Rev* 2007;21:267-283.
3. Ademowo OG, Falusi AG. Molecular epidemiology and activity of erythrocyte G6PD variants in a homogeneous Nigerian population. *East Afr Med J* 2002;79(1):42–44.
4. Cappellini MD, Fiorelli G. Glucose-6-phosphate dehydrogenase deficiency. *Lancet* 2008;371 (9606):64–74.
5. 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.
6. Al-Riyami A, Ebrahim GJ. Genetic blood disorders survey in the sultanate of Oman. *J Trop Pediatr* 2003;49 Suppl 1:i1–i20.
7. Usanga EA, Ameen R. Glucose-6-phosphate dehydrogenase deficiency in Kuwait, Syria, Egypt, Iran, Jordan and Lebanon. *Hum Hered* 2000;50(3): 158–161.
8. Iwai K, Hirono A, Matsuoka H, et al. Distribution of glucose-6-phosphate dehydrogenase mutations in Southeast Asia. *Hum Genet* 2001;108(6):445–449.
9. Louicharoen C, Nuchprayoon I. G6PD Viangchan (871G.A) is the most common G6PD-deficient variant in the Cambodian population. *J Hum Genet* 2005;50(9):448–452.
10. De Araujo C, Migot-Nabias F, Guitard J, Pelleau S, Vulliamy T, Ducrocq R. The role of the G6PD Aeth376G/968C allele in glucose-6-phosphate dehydrogenase deficiency in the Seerer population of Senegal. *Haematologica* 2006;91(2):262–263.
11. Cappellini MD, Fiorelli G (2008) Glucose-6-phosphate dehydrogenase deficiency. *Lancet* 371: 64-74.
12. Arese P, Mannuzzu L, Turrini F. Pathophysiology of favism. *Folia Haematol Int Mag Klin Morphol Blutforsch* 1989;116:745-752.
13. Arese P, Turrini F, Fasler S, Lutz HU. Recent advances in the biochemistry of favism. *Biomed Biochim Acta* 1990;49:S284-S288.
14. Gaetani GF, Mareni C, Salvidio E, Galiano S, Meloni T, Arese P. Favism: erythrocyte metabolism during haemolysis and reticulocytosis. *Br J Haematol* 1979;43:39-48.
15. Kok AN, Ertekin MV, Ertekin V, Avci B. Henna (*Lawsonia inermis* Linn.) induced haemolytic anaemia in siblings. *Int J Clin Pract* 2004;58: 530-532.
16. Zinkham WH, Oski FA. Henna: a potential cause of oxidative hemolysis and neonatal hyperbilirubinemia. *Pediatrics* 1996;97:707-709.11.
17. Pamuk GE, Dogan Celik A, Uyanik MS. Brucellosis triggering hemolytic anemia in glucose-6-phosphate dehydrogenase deficiency. *Med Princ Pract* 2009;18:329-331.
18. Schulze Zur Wiesch J, Wichmann D, Hofer A, van Lunzen J, Burchard GD, Schmiedel S. Primary HIV infection presenting as haemolytic crisis in a patient with previously undiagnosed glucose 6-phosphate dehydrogenase deficiency. *AIDS* 2008; 22:1886-1888.
19. Siddiqui T, Khan AH. Hepatitis A and cytomegalovirus infection precipitating acute hemolysis in glucose-6-phosphate dehydrogenase deficiency. *Mil Med* 1998;163: 434-435
20. Neonatal screening for sickle cell disease, glucose-6-phosphate dehydrogenase deficiency and a-thalassemia in Qatif and Al-Hasa. *Ann Saudi Med* 1998;18(4):289-92.
21. Muzaffer MA. Neonatal screening of glucose-6-phosphate dehydrogenase deficiency in Yanbu, Saudi Arabia. *J Med Screen* 2005; 12: 170–171.
22. Al-Ali AK. Common G6PD variant from Saudi population and its prevalence. *Ann Saudi Med* 1996; 16: 654–656.
23. Alabdulaali MK, Alayed KM, Alshaikh AF, et al. Prevalence of glucose-6-phosphate dehydrogenase deficiency and sickle cell trait among blood donors in Riyadh. *Asian J Transfus Sci* 2010; 4: 31–33.
24. Saleem TH, Mendis BS and Osanyintuyi SO. Glucose-6-phosphate dehydrogenase deficiency in

- a rural Saudi population. *J Trop Med Hyg* 1991; 94: 327–328.
25. el-Hazmi MA, Al-Swailem AR, Al-Faleh FZ, et al. Frequency of glucose-6-phosphate dehydrogenase, pyruvate kinase and hexokinase deficiency in the Saudi population. *Hum Hered* 1986; 36: 45–49.
26. el-Hazmi MA and Warsy AS. Glucose-6-phosphate dehydrogenase deficiency in Saudi Arabia. A study in Al-Ula. *Hum Hered* 1988; 38: 317–322.
27. el-Hazmi MA, Warsy AS, Bahakim HH, et al. Glucose-6-phosphate dehydrogenase deficiency and the sickle cell gene in Makkah, Saudi Arabia. *J Trop Pediatr* 1994; 40: 12–16.
28. el-Hazmi MA and Warsy AS. Frequency of glucose-6-phosphate dehydrogenase phenotypes and deficiency in Al-Baha. *Hum Hered* 1989;39: 313–317.
29. el-Hazmi MA and Warsy AS. Glucose-6-phosphate dehydrogenase variants and sickle cell genes in Al-Qunfuda, Saudi Arabia. *Trop Geogr Med* 1991;43: 174–179.
30. Alharbi KK, Khan IA. Prevalence of glucose-6-phosphate dehydrogenase deficiency and the role of the A- variant in a Saudi population. *J Int Med Res* 2014;42(5):1161-7.