International Journal of Medical Laboratory 2022; 9(2): 81-84.

Short Article

Prevalence of Glucose-6-phosphate Dehydrogenase (G6PD) Deficiency in Jiroft City in Southern Iran

Mohadeseh Kamali [1] M.D., Akbar Mehralizadeh [2] Ph.D., Mehdi Taheri Sarvtin [3*] Ph.D.

[1] Department of Internal Medicine, Faculty of Medicine, Jiroft University of Medical Sciences, Jiroft, Iran
[2] Department of Statistics and Epidemiology, Faculty of Health, Jiroft University of Medical Sciences, Jiroft, Iran
[3] Department of Medical Mycology and Parasitology, Faculty of Medicine, Jiroft University of Medical Sciences, Jiroft, Iran

Article history
Received: 9 May 2021
Accepted: 28 Feb 2022
Available online: 30 Jun 2022

Keywords
Deficiency
G6PD
Jiroft
Prevalence

A B S T R A C T

Background and Aims: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a global problem and the most common cause of jaundice in neonates. Hence, this study was conducted to investigate the prevalence of G6PD deficiency in Jiroft city in southern Kerman.

Materials and Methods: This descriptive cross-sectional study was carried out from 2016 to 2019. Blood samples were taken from all patients referred to Imam Hospital in Jiroft city in southern Iran. The G6PD enzyme activity was evaluated by a fluorescent spot test.

Results and Conclusions: In the present study, a total of 7791 newborns were included. Abnormal activity of G6PD was seen in 779 (10%) subjects. Out of 779 patients, 728 (9.4%) were found to be G6PD deficient, and 49 (0.6%) exhibited partial deficiency. A relatively high percentage of G6PD deficiency was seen in newborns of Jirof city. We strongly recommend screening for G6PD enzyme activity in all newborns in this city.

[*]**Corresponding Author:** Department of Medical Mycology and Parasitology, Faculty of Medicine, Jiroft University of Medical Sciences, Jiroft, Iran. **Tel/ fax:** +983443316379, **Email:** mehditaheri.mt@gmail.com

[Downloaded from ijml.ssu.ac.ir on 2026-11-09]

[DOI: 10.18502/ijml.v9i2.10016]

Introduction

Glucose-6-phosphate dehydrogenase (G6PD) is a key enzyme in the pentose monophosphate pathway that protects the body's cells, especially red blood cells, from oxidative damage by producing nicotinamide adenine dinucleotide hydrogen phosphate (NADPH) [1]. The gene encoding this enzyme is located on the X chromosome and consists of 13 exons and 12 introns and encodes 515 amino acids. G6PD deficiency is one of the most common hemolytic disorders, especially in men, affecting approximately 400 million people worldwide [2]. This enzyme deficiency was first discovered in 1956 due to hemolytic anemia following the use of antimalarial drugs in some patients [3]. The prevalence of deficiency of this enzyme plays an important role in the selection of antimalarial drugs. Primaquine is one of the most important antimalarial drugs widely used in malaria-endemic areas to treat and prevent malaria [4]. However, primaquine can cause mild to severe hemolytic anemia in people with G6PD deficiency via oxidative stress [1]. Numerous studies have investigated the prevalence of G6PD deficiency in Iran and other parts of the world [1-3]. South of Kerman is one of the endemic areas of malaria, especially Plasmodium vivax in Iran. Jiroft is one of the most important cities in the south of Kerman, which has a vital role in preventing, identifying, and treating malaria patients in this area. However, no comprehensive study has been conducted on the prevalence of G6PD deficiency in this city. Lack of knowledge about the prevalence of this enzyme deficiency and uncertainty about the existence of correct methods for G6PD deficiency screening can increase the risk of hemolytic anemia and its complications in patients with malaria. So, this study investigated the prevalence of G6PD deficiency in Jiroft city.

Materials and Methods

This cross-sectional study was approved by the Medical Ethics Committee of Jiroft University of Medical Sciences (Ethics Code: IR.JMU.REC.1400.027). In the present study, neonates with jaundice suspected of G6PD deficiency were studied from 2016 to 2019. Blood samples were taken from 7791 newborns referred to Imam Hospital in Jiroft city in southern Iran, and G6PD enzyme activity was measured qualitatively using Kimia Pajohan Company Kit (Kimia Pajouhan, Iran, Lot: 98605) by spot fluorescence method [3].

Statistical analysis

The collected data were statistically analyzed by SPSS version 24 software using descriptive statistics, binomial test, and chi-square test. In the present study, a p-value of less than 0.05 was considered statistically significant.

Results and discussion

In the present study, a total of 7791 newborns (4225 males and 3566 females) were included. G6PD deficiency was seen in 9.4% of newborns (Table 1). In the study by Castro et

[Downloaded from ijml.ssu.ac.ir on 2026-11-09]

[DOI: 10.18502/ijml.v9i2.10016]

al. [5], G6PD deficiency was seen in 1.4% of newborns. Besides, in the study by Alabdulaali et al. [6], G6PD deficiency was observed in 0.78% of participants. In Nejadaria et al. study, G6PD deficiency was seen in 9.09% of participants. The result of the present study was consistent with the result of Nejadaria et al.'s [2] study, but it was inconsistent with the findings of Castro et al. [5] and Alabdulaali et al.'s [6] studies. The differences in the results of various studies might be attributed to several reasons, including geographical location, genetics of individuals, differences in methods of measuring enzyme activity, and technician's skill in measuring enzyme activity. In the current study, G6PD deficiency was significantly higher in males than females ($p < 0.001$). This result is inconsistent with Norbahksh et al.'s [7] study but is consistent with Nejadaria et al. [2]. The gene encoding the G6PD enzyme is X-dependent gene. Therefore, G6PD deficiency is more common in males than females. A high incidence of G6PD deficiency in females may be due to accidental inactivation of one of the two X chromosomes that, in heterozygous women, can cause symptoms similar to those of males to produce homozygotes [2].

In the present study, 49 (0.6%) partially G6PD-deficient cases were detected among all subjects. Partially G6PD-deficient cases have not been identified in some studies due to using qualitative methods [8, 9]. Individuals with partial G6PD deficiency are usually asymptomatic, and failure to identify them can lead to acute hemolytic anemia and serious injury when exposed to oxidative stress, such as infections and some drugs and foods [9]. Some biochemical changes and depletion in the antioxidant defense system can occur during the storage of G6PD-deficient blood [6]. Furthermore, transfusion with G6PD deficient units can lead to hemolytic complications, especially in neonates and infants [6]. Therefore, almost ten percent of the individuals studied could not properly donate blood in the future. It is hypothesized that the overproduction of reactive oxygen species and excess oxidative damage is responsible for the secretion of the cytokine storm, impaired immunity, and pulmonary dysfunction in response to the Coronavirus disease (COVID-19) infection.

Table 1. Distribution of G6PD activity values in newborns according to gender and year in Jiroft, southern Iran

Year	Sufficiency N (%)		Deficient N (%)		Partially deficiency N (%)		Total N (%)
	Male	Female	Male	Female	Male	Female	
2016	531 (39.6)	674 (50.3)	96 (7.2)	38 (2.8)	2 (0.1)	0 (0)	1341 (100)
2017	1071 (46)	1010 (43.4)	200 (8.6)	38 (1.6)	5 (0.2)	2 (0.1)	2326 (100)
2018	1043 (47.8)	935 (42.9)	132 (6.1)	35 (1.6)	29 (1.3)	6 (0.3)	2180 (100)
2019	944 (48.6)	806 (41.5)	167 (8.6)	22 (1.1)	5 (0.3)	0 (0)	1944 (100)

[Downloaded from ijml.ssu.ac.ir on 2026-11-09]

[DOI: 10.18502/ijml.v9i2.10016]

According to this hypothesis, patients with G6PD deficiency are more sensitive to COVID-19 infection due to impaired neutralization of oxidative stresses [10]. So, in the case of disease, it seems that the complications of COVID-19 infection can be more severe in 10% of the subjects. G6PD deficiency is significantly associated with an increased risk of cardiovascular disease up to 70%, even though the exact underlying mechanism is unknown. The loss of critical protective pathways against oxidative stress may play a significant role [11]. According to this theory, the risk of cardiovascular disease in 779 of our study cases is higher than in others.

Conclusion

This study showed that G6PD deficiency could be observed in a significant percentage of newborns in Jirof city, which was higher than in some studies in other cities of Iran. Therefore, a high percentage of newborns in this city are exposed to hemolytic anemia if they use antimalarial drugs for treatment or prevention.

Conflict of Interest

The authors declare that there is no conflict of interest.

Acknowledgement

I would like to thank the staff of Imam Hospital and all those who contributed to this study.

References

[1]. Lo E, Zhong D, Raya B, Pestana K, Koepfli C, Lee MC, et al. Prevalence and distribution of G6PD deficiency: Implication for the use of primaquine in malaria treatment in Ethiopia. Malar J. 2019; 18(1): 1-10.

[2]. Nejadaria M, Mortazavi SM, Kohansal MH. Prevalence of glucose-6-phosphate dehydrogenase deficiency in neonates hospitalized in Pasteur Hospital of Bam, Iran. mljgoums. 2020; 14(2): 9-12.

[3]. Daliri S, Asadollahi K, Rahimi N, Sayehmiri K. Incidence of glucose-6-phosphate dehydrogenase deficiency in malaria-prone regions of Fars province. Tehran Univ Med. 2017; 75(9): 669-74.

[4]. Shekalaghe S, Mosha D, Hamad A, Mbaga TA, Mihayo M, Bousema T, et al. Optimal timing of primaquine to reduce Plasmodium falciparum gametocyte carriage when co-administered with artemether–lumefantrine. Malar J. 2020; 19(1): 34.

[5]. Castro S, Weber R, Dadalt V, Tavares V, Giugliani R. Prevalence of G6PD deficiency in newborns in the south of Brazil. J Med Screen 2006; 13(2): 85-6.

[6]. Alabdulaali MK, Alayed KM, Alshaikh AF, Almashhadani SA. Prevalence of glucose-6-phosphate dehydrogenase deficiency and sickle cell trait among blood donors in Riyadh. Asian J Transfus Sci. 2010; 4(1): 31-3.

[7]. Norbahksh SMK, Kasiri KA, Jalil A, Hashemzade Chaleshteri M. Determining the frequency of glucose-6-phosphate dehydrogenase deficiency in newborn infants in Shahrekord. JSKUMS. 2013; 15(5): 30-37.

[8]. Zareifar S, Pishva N, Farahmandfar M, Benaei S, Cohan N. Prevalence of G6PD deficiency in neonatal sepsis in Iran. Iran J Pediatr. 2014; 24(1): 115-16.

[9]. Azma RZ, Hidayati N, Farisah NR, Hamidah NH, Ainoon O. G6PD enzyme activity in normal term Malaysian neonates and adults using a OSMMR2000-D kit with Hb normalization. Southeast Asian J Trop Med Public Health 2010; 41(4): 982-88.

[10]. Jain SK, Parsanathan R, Levine SN, Bocchini JA, Holick MF, Vanchiere JA. The potential link between inherited G6PD deficiency, oxidative stress, and vitamin D deficiency and the racial inequities in mortality associated with COVID-19. Free Radic Biol Med. 2020; 161(1): 84-91.

[11]. Pes GM, Parodi G, Dore MP. Glucose-6-phosphate dehydrogenase deficiency and risk of cardiovascular disease: A propensity score-matched study. Atherosclerosis 2019; 282:148-153.

[Downloaded from ijml.ssu.ac.ir on 2026-11-09]

[DOI: 10.18502/ijml.v9i2.10016]