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ORIGINAL ARTICLE

Glucose-6-Phosphate Dehydrogenase Deficiency among Newborns in Sana'a City, Yemen

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ABSTRACT

Background: Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common inborn enzymatic defect in the world. Determining the prevalence of G6PD deficiency is an essential step towards evaluating its impact on the health of a population. Therefore, this study aimed to determine the prevalence and factors associated with G6PD deficiency among newborns in Sana'a city, Yemen.

Subjects and methods: This cross-sectional study included 397 newborns from eight public and private hospitals in Sana'a city in the period from January to March 2020. Data about gender and the factors possibly associated with G6PD deficiency were collected using a pre-designed questionnaire. G6PD deficiency was qualitatively detected in fresh blood using rapid diagnostic tests (RDTs). Data were analyzed using SPSS software, and differences were considered statistically significant at *p*-value <0.05.

Results: Of 397 screened newborns (217 males and 180 females), 19 newborns (14 male and 5 female newborns) were G6PD-deficient, with an overall prevalence of 4.8% and a male: female ratio of 2.8:1. G6PD deficiency was significantly associated with consanguinity. However, it was not significantly associated with the gender, mode of delivery, gestational age or birth weight of the newborns.

Conclusions: G6PD deficiency is prevalent among as low as approximately 5.0% of newborns in the hospitals of Sana'a city. It is almost comparable to those reported from other countries in the region. It is more prevalent among males than females, with no statistically significant difference. G6PD deficiency was not associated with the gender, mode of delivery, gestational age and birth weight of the newborns.

Keywords: Newborns; G6PD deficiency; Rapid diagnostic test; Yemen

INTRODUCTION

Glucose-6-phosphate-dehydrogenase (G6PD) deficiency is the most important disorder of the pentose phosphate pathway (PPP) in erythrocyte metabolism [1]. The PPP provides a reducing power to all cells in the reduced form of nicotinamide adenine dinucleotide phosphate (NADPH) [2]. NADPH enables cells to balance the oxidative stress triggered by several oxidant agents and to preserve the reduced form of glutathione and other sulfhydryl groups essential for reducing hydrogen peroxide and reactive oxygen species and maintaining hemoglobin and other red blood cell (RBC) proteins in the reduced state. Since RBCs lack mitochondria, the PPP is their only source of NADPH, and defence against oxidative damage is dependent on G6PD activity [3].

The gene encoding G6PD is located on the X-chromosome, band Xq28. Because the disorder of G6PD deficiency has an X-linked recessive pattern of inheritance, males are more frequently affected [4, 5]. This disorder predisposes to hemolysis and methemoglobinemia in individuals using substances with oxidative properties such as raw beans, some medications, or having infections causing oxidative stress [6, 7].

G6PD deficiency can lead to acute or chronic hemolytic anemia, and hyperbilirubinemia appears in newborns in severe cases. Kernicterus resulting from the accumulation of unconjugated bilirubin in the brain cells at the first month of birth may lead to neonatal death and a number of complications among those who survive, such as mental retardation, disability and vestibule auditory disorders, seizures, hearing loss and speech disorders [1, 8]. Parents of G6PD-deficient newborns should be educated about the risks of jaundice and the importance of avoiding offending agents [9]. Moreover, treatment for G6PD deficiency is simple and inexpensive [10], and can be started before symptoms appear [5, 11].

G6PD deficiency affects approximately 10% of the world's population [12], particularly African populations, Black Americans, Southeast Asian populations and populations around the Mediterranean and the Far East [13, 14]. In Yemen, G6PD deficiency was reported among 5.94% of Yemeni migrants in the United Arab Emirates [15] and 22.6% (7/31) of patients with sickle cell disorder in Taiz [16]. In 2012, G6PD deficiency was reported to be prevalent among 7.1% (36/508) of male blood donors in Sana'a city [17]. Recently, G6PD deficiency was found to be prevalent among 12.0% and 2.3% of male and female children in malaria-endemic areas of Hodeidah governorate, respectively [18]. However, no previous studies had been published on the prevalence of G6PD deficiency among newborns in Yemen so far. Therefore, the present study aimed to determine the prevalence and factors associated with G6PD deficiency among newborns in Sana'a city, Yemen.

SUBJECTS AND METHODS

Study design, setting and population

A cross-sectional study was conducted among Yemeni newborns in four public hospitals (Kuwait University Hospital, Al Gumhouri Teaching Hospital, Al-Thawra Hospital and Al Sabaeen Maternal Hospital) and four private hospitals (University of Science and Technology Hospital, Saudi German Hospital, Cplas Hospital and Mother Hospital) in Sana'a city in the period from January to March 2020.

The study protocol was approved by the Research Ethics Committee of the University of Science and Technology - Sana'a and was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans. Informed consent was obtained from the parents or guardians of the newborns after explaining the purpose of the study. A minimum sample size of 161 was calculated based on an expected prevalence of G6PD deficiency of 12.0% [18] at a confidence level of 95.0% and an accepted marginal error of 5.0%. However, 379 newborns were included in the present study.

Data and sample collection

Data on gender, birth weight, mode of delivery, gestational age and degree of consanguinity between parents were collected using a pre-designed, structured questionnaire through interviews. Fresh blood samples were collected from the newborns of Yemeni mothers (217 males and 180 females) delivered in the selected hospitals.

G6PD screening

Qualitative screening for G6PD deficiency was performed on fresh venous blood using CareStart™ G6PD rapid diagnostic tests (RDTs) (AccessBio, New Jersey, USA) according to the manufacturer's instructions. Briefly, two microliters of blood were added into the sample well and two drops of buffer into the buffer well. Test results were read visually after 10 minutes. Samples with normal G6PD activity produce a distinct purple color

background in the result window while no color change was observed for samples with deficient G6PD activity. Samples with a pale purple color background were classified as normal.

Statistical analysis

Data was entered and analyzed using IBM SPSS Statistics, version 23.0 (IBM Corp., Armonk, NY, USA). Qualitative variables were expressed as frequency and percentage. Normally-distributed quantitative variables were expressed as mean and standard deviation (SD) and non- normally distributed quantitative variables were expressed as median and interquartile range (IQR). Odds ratios (ORs) and 95% confidence intervals (CIs) were also calculated. Chi-square with Yates's correction test was used to show the significance of association between G6PD deficiency and the factors associated with it, which was further analyzed using multiple logistic regressions. Associations at *p*-value <0.05 were considered statistically significant.

RESULTS

General characteristics of the newborns

Of 397 newborns, 217 were males and 180 were female, with a male: female ratio of 1.2:1 and a median age (IQR) of 1.0 (5.0) days. More than half of newborns were born in public hospitals, 36.5% were born to consanguineous parents and 51.1% were delivered by cesarean section. Of the studied newborns, 24.9% were preterm (<37 weeks), 54.4% had a birth weight less than 2.5 Kg, with a mean birth weight of 2.4 ± 0.6 kg. G6PD deficiency was detected among 4.8% of the newborns (Table 1).

Factors associated with G6PD deficiency

G6PD deficiency was 2.4-fold higher in males than females (6.5% *vs.* 2.8%, respectively) with a male: female ratio of 2.8:1, but the difference was not statistically significant (OR = 2.4; 95 % CI: 0.85–6.84, $p = 0.088$. On the other hand, consanguinity between the parents was significantly associated with G6PD deficiency, where newborns to consanguineous parents showed a 2.5-fold higher risk of being deficient compared to their counterparts (OR = 2.5; 95 % CI: 0.98 – 6.38, $p = 0.047$); however, it was not a significant predictor of G6PD deficiency using the multiple logistic regression analysis. On the other hand, mode of delivery, gestational age and birth weight were not significantly associated with G6PD deficiency (Table 2).

Tables

Table 1. Characteristics of the newborns included in the study (2020)*

Variable		*n*	(%)
Age (days)			
	≤ 1	203	(51.1)
	> 1	194	(48.9)
Median ± IQR: 1.0 ± 5.0			
Gender			
	Male	217	(54.7)
	Female	180	(45.3)
Hospital			
	Public	227	(57.2)
	Private	170	(42.8)
Consanguinity between parents			
	Yes	145	(36.5)
	No	252	(63.5)
Mode of delivery**			
	Vaginal	160	(48.9)

Variable		*n*	(%)
	Cesarean section	167	(51.1)
Gestational age			
	Preterm	99	(24.9)
	Full-term	298	(75.1)
Birth weight (kg)			
	<2.5	216	(54.4)
	2.5 - 3.5	171	(43.1)
	>3.5	10	(2.5)
Mean ± SD: 2.4 ± 0.6			
G6PD status			
	Deficient	19	(4.8)
	Normal	378	(95.2)

*Total number of newborns was 397; ** Some data were missing.

Table 2. Factors associated with G6PD deficiency among newborns in Sana'a city, Yemen (2020)

Variable		Total	Deficient		OR	(95% CI)	*p*-value
		N	*n*	(%)			
Gender							
	Male	217	14	(6.5)	2.41	(0.85-6.84)	0.088
	Female	180	5	(2.8)			
Hospital							
	Public	227	15	(6.6)	2.94	(0.96-9.01)	0.049
	Private	170	4	(2.4)			
Consanguinity between parents							
	Yes	145	11	(7.6)	2.50	(0.98-6.38)	0.047
	No	252	8	(3.2)			
Mode of delivery							
	Vaginal	160	9	(5.6)	1.19	(0.45-3.15)	0.734
	Caesarean section	167	8	(4.8)			
Gestational age							
	Preterm	99	6	(6.1)	1.41	(0.52-3.83)	0.679
	Full term	298	13	(4.4)			
Birth weight (kg)							
	<2.50	216	12	(5.6)	1.46	(0.56-3.8)	0.433
	≥2.50	181	7	(3.9)			

OR, odds ratio; CI, confidence interval.

DISCUSSION

Up to the best of our knowledge, this is the first study to report on G6PD deficiency among Yemeni newborns in Sana'a city. The prevalence of G6PD deficiency among newborns delivered in Sana'a hospitals (4.8%) is consistent with those reported among Egyptian newborns (4.3%; 119/2782) and Iranian icteric newborns (4.4%; 12/272) [19, 20]. However, it was slightly higher than those reported from Yanbu, Saudi Arabia (2.0%), Tehran, Iran (2.1%) and Turkey (3.8%) [21-23], but it was lower than those reported from Isfahan, Iran (7.5%), South Brazil (7.9%) and Niger (38.2%) [24-26].

The higher G6PD deficiency rate among males in the present study could be attributed to the fact that the disorder is recessively X-linked, leading to its greater expression in males as they have only one X-chromosome with no ability to suppress the expression of the defective gene. However, the heterozygous females having one defective gene and one normal gene may express as normal or mild deficient and may not be detected by usual screening tests or even enzyme assays [27, 28]. The lack of statistically significant association the gender of the newborn and G6PD deficiency in the present study is consistent with the findings from Brazil and Niger [25, 26]. In contrast, G6PD deficiency was significantly higher among male compared with female neonates in Egypt (6.2% *vs.* 2.1%, respectively) with a male: female ratio 3.2: 1 [19]. The significant association between gender and G6PD deficiency was also reported in Turkey [23] and Shirvan, Iran [29].

Because G6PD deficiency is a hereditary disease, the type of marriage between the parents could be a determinant in the inheritance of such a disorder. Against this background, more than half of the newborns who had G6PD deficiency in the present study were born from a consanguineous marriage, with a statistically significant association between consanguinity and G6PD deficiency. This is consistent with studies from Yemen [18], Egypt [30] and Qatar [31]. It is noteworthy that consanguineous marriages may account for over 50% of total marriages in the Eastern Mediterranean region, affecting the community control of genetic disorders [32]. Therefore, the frequency of homozygous genotypes increases while that of heterozygous genotypes decreases in the population, leading to an increased risk for recessively inherited disorders in the population [33, 34]. In Yemen, consanguinity is reported among 40.0% of total marriages [35]. In Sana'a city, the incidence of consanguinity is relatively high, representing 44·7% of total marriages with first-cousin marriages constituting over 70·0% of consanguineous marriages and 32.0% of all marriages [36]. Nevertheless, consanguinity was not an independent risk factor for G6PD deficiency in the present study, indicating the possible effect of other factors. In contrast, it was found to be a significant independent predictor of G6PD deficiency among children residing in malaria-endemic areas of Hodeidah [18]. The finding of the present study is also in contrast to those reported from Benin [37] and Tunisia [38], where consanguinity was an independent predictor of G6PD deficiency. In contrast to the present study, no significant association was found between first-cousin consanguinity and G6PD deficiency among Saudi children at the community level [39]. Therefore, there is a need for further large-scale studies to study the association between consanguinity and neonatal G6PD deficiency among Yemeni children through community-based studies.

The lack of significant association between the mode of delivery of the newborns and G6PD deficiency in the present study is in line with those reported from Fars, Iran [40] and Egypt [41], while the lack of significant association of G6PD deficiency among newborns and prematurity is consistent with findings from Fars and Isfahan in Iran [40, 42] and India [43]. Some studies suggest that premature infants can even have a higher G6PD activity compared to near term infants, and prematurity does not interfere with the diagnosis of G6PD deficiency [44, 45]. On the

other hand, the lack of significant association between the low birth weight (LBW) of newborns and G6PD deficiency in the present study could be partly attributed to the high prevalence of LBW among Yemeni neonates, which has been aggravated by the humanitarian crisis as a result of the ongoing war. In 2012, the prevalence of LBW was reported among about one-third of infants [46], and the situation is even worse at present. In a longitudinal study, Herz et al. [44] found that LBW infants and prematurely delivered infants have a higher G6PD activity than normal ones. The lack of association between G6PD deficiency and LBW was also reported from Iran [29] and India [43].

This study is limited by the fact that it was hospital-based, and its findings may not be generalizable to the newborns at the community level. However, its findings highlight the necessity of screening newborns for G6PD deficiency using easy-to-use point-of-care diagnostics. Although this study adopted RDTs as a diagnostic tool for screening newborns for G6PD deficiency, previous studies have shown that the CareStart™ G6PD RDT has a high diagnostic accuracy [18, 47]. In Yemen, it showed 100% sensitivity, 100% negative predictive value, 95.4% specificity and 66.0% positive predictive value for screening G6PD deficiency at the cut-off value of ≤20% of the normal activity compared to the reference enzymatic method [18]. The diagnostic reliability of the CareStart™ G6PD RDT for screening G6PD deficiency in male and female newborns with less than 60% residual enzymatic activity was also reported from China [47].

CONCLUSIONS

G6PD deficiency is prevalent among as low as approximately 5.0% of newborns in the hospitals of Sana'a city. It is almost comparable to those reported from other countries in the region. Besides, G6PD deficiency is more prevalent among males than females, but the difference is not statistically significant. Gender, mode of delivery, gestational age and birth weight are not risk factors for G6PD deficiency among Yemeni newborns in Sana'a city.

Declaration of interest

The authors report no conflicts of interest.

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