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Prevalence and Ethnic Variability of G6PD Deficiency Among Newborns in the Northern Emirates: A Three-Year Retrospective Study at Thumbay Hospitals, Ajman, UAE

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ABSTRACT

SUMMARY

BACKGROUND

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is one of the most common enzymatic inherited red cell disorder. This study investigated the prevalence and distribution of G6PD deficiency among newborns in Thumbay hospital and other health care facilities across the Northern Emirates in United Arab Emirates (UAE).

Methods

This was a retrospective cross-sectional study focusing on data of newborns screened for G6PD levels over three years (2020–2022) at Thumbay Laboratory, Ajman.

Resutls

New-borns from Southern Asia exhibited the highest prevalence of G6PD deficiency, with 88 cases (40.3%) out of 3,921 newborns. Western Asia also showed significant prevalence with 75 cases (34.4%) among 729 new-borns. Conversely, regions like Eastern Asia and Europe/America reported negligible or no cases.

Conclusion

The findings of this study indicate a high prevalence of G6PD deficiency in newborns born in the Northern Emirate of the UAE, particularly those from Southern and Western Asia, with Southern Asia showing the highest rate. Males had lower G6PD levels as compared to females, though these differences were not statistically significant

Keywords: *G6PD deficiency, Newborns, anemia, United Arab Emirates*

1. INTRODUCTION

Glucose-6-phsophate dehydrogenase (G6PD) deficiency anemia is a sex-linked hereditary clinical condition characterized by hemolytic anemia, attributable to the inability of red cells to metabolize oxidative agents. This condition arises from over

400 mutations/deletions affecting the *G6PD* gene, situated on the long arm of X chromosome (Xq28), primarily affecting males due to its X-linked recessive nature [1]. Regarded as the most prevalent enzymopathy, it afflicts roughly 400 million individuals globally [2].

Within the hexose monophosphate shunt, G6PD catalyzes the conversion of nicotinamide adenine dinucleotide (NADH) to nicotinamide adenine dinucleotide phosphate (NADPH). NADPH transforms oxidized glutathione (GSSH) into reduced glutathione (GSH). GSH acts to counteract reactive oxygen species (ROS), comprising superoxide, hydrogen peroxide, and hydroxyl radicals, generated from the partial reduction of molecular oxygen. Moreover, GSH maintains the sulfhydryl groups of hemoglobin in reduced state [3]. In case of G6PD deficiency, reduced NADPH production leads to diminished GSH levels. Consequently, the reduced availability of GSH impedes ROS detoxification. Accumulated ROS within red cells inflict damage upon their membranes, precipitating hemolysis. Additionally, ROS induce hemoglobin denaturation, culminating in the formation of Heinz bodies. Heinz bodies are subsequently cleared by reticuloendothelial cells, leading to hemolytic episodes [4].

The pathogenesis of G6PD deficiency-related anemia implicates oxidant drugs, including certain antibiotics (e.g., sulfamethoxazole), antimalarials (e.g., primaquine), and antipyretics (e.g., acetanilide). Infections and the ingestion of fava beans also increases oxidative stress, triggering hemolysis [1,5,6].

G6PD deficiency exhibits a notable prevalence in tropical and subtropical regions, encompassing Africa, Asia, the Mediterranean, and the Middle East. In the Mediterranean locale, the estimated incidence among males' ranges from 1–2%. Its geographic distribution correlates with areas endemic to malaria [6,7]. In Gulf countries, the estimated incidence rate of G6PD deficiency is up to 27% [8,9,18,10–17]. In Saudi Arabia, studies have documented varying incidence rates of G6PD deficiency, spanning from 0–52%, with discrepancies observed across provinces [19–20].

The United Arab Emirates society presents a cosmopolitan landscape, yet indigenous populations retain traditional values, fostering consanguineous marriages as commonplace. Consequently, an increased prevalence of genetic disorders is observed. Despite this, genetic services remain fragmented, with scant studies addressing hereditary disorders in newborns over the past decade. Hence, research our endeavors focus on investigating the prevalence of G6PD deficiency among newborns and examining the factors correlated with the occurrence of the disease, such as gender and nationality among newborns delivered in Thumbay and other healthcare facilities in the Northern Emirates including Ajman, Ras Al Khaimah, Umm Al Quwain, and Fujairah..

2. MATERIAL AND METHODS

This study adopted a laboratory-based retrospective cross-sectional design, focusing on data collected over the three years (2020–2022) from Thumbay Laboratory, Ajman. Newborn blood spot samples meeting proper labeling criteria were included, while those deemed mislabeled, charred, or soiled were excluded. Processing of all newborn samples were carried exclusively at Thumbay Laboratory, Ajman, United Arab Emirates.

Ethical approval was obtained from the Institutional Review Board (IRB) of Gulf Medical University, Ajman, affirming the methodological and ethical integrity of the project. The IRB recognized the minimal risk associated with this non-interventional research, and provided its recommendation for project approval (Ref. No. IRB/COHS/FAC/47/AUG–2022). Informed consent was waived off due to the retrospective nature of the study.

G6PD analysis was conducted using the Standard Biosensor G6PD analyzer (Suwon, South Korea). The methodology involved the application of an enzymatic colorimetric detection system for semi-quantitative measurement of G6PD activity. Descriptive statistic was used for reporting frequency.

3. RESULTS

The distribution of participants across continents for the years 2020, 2021, and 2022 revealed that majority of participants were from Asia while Africa and Europe/America exhibited lower counts over the same period. Tables 1 illustrates the distribution of participants across sub continents for the three years.

In 2020, out of a total population of 3,000 tested samples, 95.0% were classified as normal, while 5.0% exhibited G6PD deficiency. Similarly, in 2021, among 2,016 newborns, 97.2% were normal and 2.8% G6PD deficient. In 2022, out of 977 newborns, 98.8% were normal, with only 1.2% presenting with G6PD deficiency. Table 2 presents the distribution of individuals with normal G6PD status and G6PD deficiency across different regions in the current study.

Overall, the total number of severe cases across the study period amounted to 79. In 2020, there were 47 cases of severe G6PD deficiency, constituting 59.3% of the total. In 2021, 27 cases were reported, accounting for 34.6% of the total. In 2022, 5 cases were identified, representing 6.1% of the total. Distribution of moderate and mild cases of G6PD deficiency is depicted in table 3. Regarding severity levels, the mean G6PD concentration varied across different severity categories during the study period as shown in figure 1.

Fig 1: Comparative Analysis: Mean Abnormal G6PD Concentration (U/g) Across Years and Severity of G6PD Deficiency.

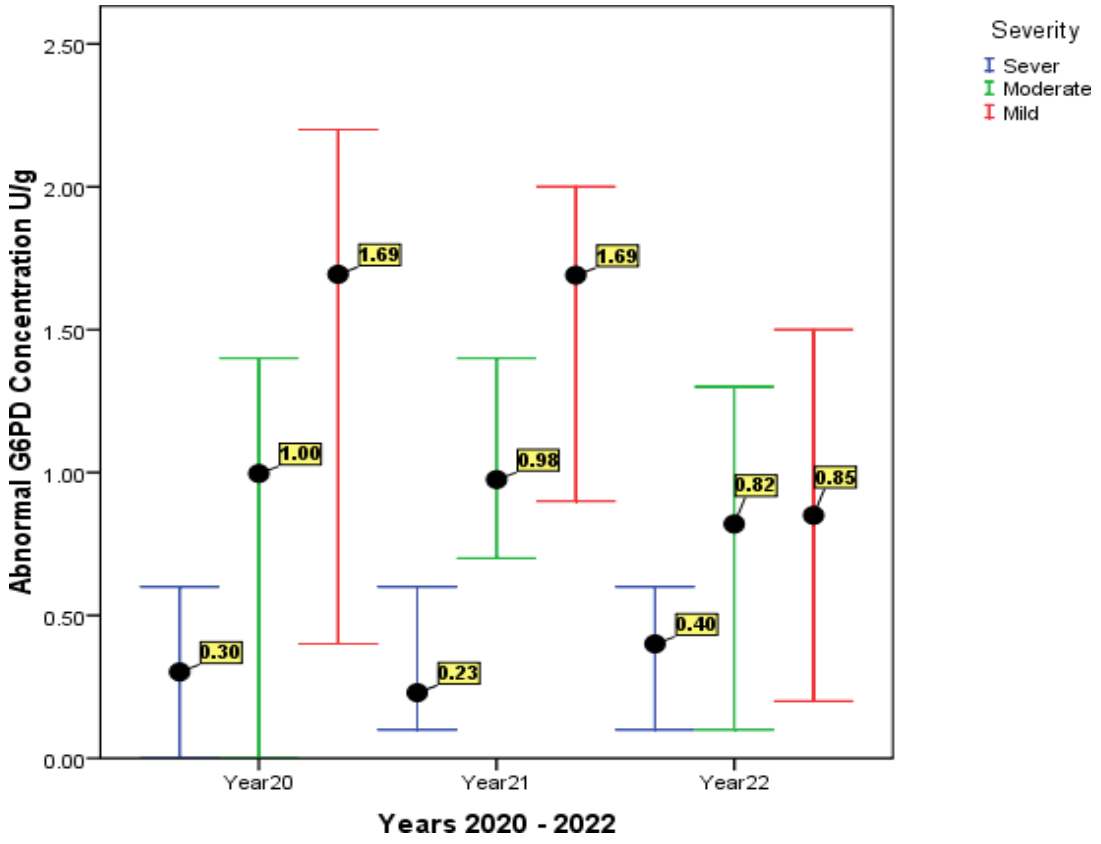

Table 4 delineates gender distribution across regions, revealing notable variations. In Asia, Central Asia records the highest counts for both males (51, 1.63%) and females (60, 2.09%), followed closely by Western Asia with 385 males (12.34%) and 407 females (14.16%). Southern Asia stands out with a significant male predominance, boasting 2102 males (67.41%) compared to 1901 females (66.16%). Conversely, Eastern and Southeast Asia exhibit lower counts for both genders, with minimal representation. In Africa, gender distribution varies modestly across regions, with Northern Africa showcasing slightly higher counts compared to Western, Middle, Eastern, and Southern Africa. These findings underscore gender disparities across regions, with implications for demographic studies and healthcare planning.

Table 5 presents a comparative analysis of mean G6PD concentration (U/g Hb) by gender and G6PD results indicating a significantly higher mean concentration in individuals with normal G6PD levels (5.85 U/g) compared to those with G6PD deficient status (1.69 U/g), with a p-value of 0.002. While males displayed a slightly lower mean concentration (5.74 U/g Hb) compared to females (5.85 U/g Hb), the difference was not statistically significant, with a p-value of 0.056.

Table 1. Population Distribution of Participants by Years and Sub-Continents

Region	Years						Total
	2020		2021		2022		
	N	Frequency (%)	N	Frequency (%)	N	Frequency (%)	
Europe and America	3	0.11	7	0.20	1	0.10	11

Asia	North Asia	5	0.17	9	0.26	1	0.10	15
	Central Asia	57	1.89	84	2.44	6	0.62	147
	Western Asia	557	18.56	382	11.05	78	7.95	1017
	Southern Asia	1833	61.11	2422	70.09	684	70.04	4939
	Eastern Asia	5	0.17	2	0.06	1	0.10	8
	Southeast Asia	53	1.78	247	7.15	67	6.82	367
Africa	Northern Africa	371	12.37	138	3.98	79	8.06	588
	Western Africa	41	1.37	52	1.51	18	1.86	111
	Middle Africa	10	0.34	108	3.14	8	0.83	126
	Eastern Africa	58	1.95	2	0.06	33	3.41	93
	Southern Africa	5	0.17	2	0.06	1	0.10	8

Table 2. Prevalence of G6PD Deficiency in Newborns Across Sub-Continents.

		Normal		G6PD deficiency	
		N	Frequency (%)	N	Frequency (%)
Asia	European and American	9	0.15	0	0
	North Asia	10	0.17	2	0.92
	Central Asia	88	1.52	3	1.38
	Western Asia	729	12.63	75	34.4
	Southern Asia	3,921	67.97	88	40.3
	Eastern Asia	6	0.1	0	0
	Southeast Asia	291	5.04	15	6.88
Africa	Northern Africa	454	7.87	20	9.17
	Western Africa	81	1.4	10	4.59
	Middle Africa	93	1.61	2	0.98
	Eastern Africa	83	1.44	3	1.38
	Southern Africa	6	0.1	0	0

Table 3. Prevalence Distribution of G6PD Deficiency Severity Across Years

Year	Sever		Moderate		Mild		Total
	N	%	N	%	N	%	
2020	47	59.3	56	81.2	47	67.2	150
2021	27	34.6	8	11.6	21	30	56
2022	5	6.1	5	7.2	2	2.8	12
Total	79	100	69	100	70	100	218

Table 4. Distribution of Participants by Gender and Sub-continents

Region	Gender			
	Male		Female	
	N	Frequency (%)	N	Frequency (%)

Europe and America		6	0.19	3	0.1
Asia	North Asia	10	0.32	1	0.03
	Central Asia	51	1.63	60	2.09
	Western Asia	385	12.34	407	14.16
	Southern Asia	2102	67.41	1901	66.16
	Eastern Asia	5	0.16	1	0.03
	Southeast Asia	160	5.13	144	5.01
Africa	Northern Africa	251	8.05	224	7.79
	Western Africa	50	1.6	41	1.43
	Middle Africa	51	1.63	46	1.6
	Eastern Africa	45	1.44	42	1.46
	Southern Africa	3	0.1	4	0.14

Table 5. Comparative Analysis of Mean G6PD levels (U/g) and Gender

Variable		G6PD (U/g Hb)		p value
		Mean	SD	
G6PD Levels	Normal	5.85	1.07	0.002
	Low	1.69	1.02	
Gender	Male	5.74	1.41	0.056
	Female	5.85	1.99	

4. DISCUSSION

G6PD deficiency anemia is the most common enzymatic inherited disorder of red cells across the globe [1]. The disease is highly prevalent in the Mediterranean area and the Middle East, including Saudi Arabia [19–20].

Findings of the current study showed that the prevalence of G6PD deficiency among newborns varies across different subcontinents in the screened population, as evidenced by the data presented in the table 1. In Asia, particularly in Southern Asia, the prevalence of G6PD deficiency was notably high, with 88 cases accounting for 40.3% of the total tested new-born population in this study. Western Asia also exhibited a significant prevalence, with 75 cases representing 34.4% of newborns. Similarly, Southeastern Asia shows a substantial prevalence of 15 cases, constituting 6.88% of new-borns. Conversely, the prevalence of G6PD deficiency in other subcontinents, such as Eastern Asia, was relatively low, with no reported cases in the dataset. Similarly, European and American newborns showed no cases of G6PD deficiency among newborns. Across African subcontinents, while Northern Africa presented a moderate prevalence of 20 cases (9.17%), other regions such as Middle Africa and Southern Africa demonstrated lower prevalence rates, with only 2 and 0 cases reported, respectively. These findings highlight significant regional disparities in the prevalence of G6PD deficiency among newborns in the screened population.

In this study, we investigated the mean G6PD concentration by gender and G6PD levels, shedding light on potential variations and their clinical relevance. Our findings underscored a substantial difference in mean G6PD concentration between individuals with normal and deficient status, emphasizing the importance of early detection and intervention strategies. Furthermore, gender-based analyses revealed nuanced differences, albeit not statistically significant, warranting further exploration. In a recent local study, it was found that neonates admitted to a tertiary care perinatal center for phototherapy had 10.5% prevalence of G6PD deficiency. Furthermore, the deficiency of G6PD was more prevalent in male neonates, which is similar to the findings of this study [22]. For detailed comparison of G6PD deficiency in Arab world please refer to table 6 and a systemic review by Alangari et al [2]. This study concludes that G6PD deficiency is more common in newborns of Southern Asian ancestry

Table 6. Prevalence of G6PD deficiency in some selected Arab countries

Country	Study participants / prevalence (%)	Reference
UAE (2014)	UAE nationals: 7.4 Non-UAE nationals: 3.4 UAE males: 11.6 UAE females: 3.6 Non-UAE male: 5 Non-UAE females: 1.7	[12]
UAE (Al-Ain) 1980	Overall: 11 (UAE nationals	[10]
Saudi Arabia (Riyadh)	Overall: 2 Male: 79 Female: 21	[22]
Saudi Arabia (Al-Ahsa; Eastern region)	Overall: 25 Male: 33.8 Female: 13.5 Newborn patients overall: 18.8 Male: 26 Female: 9.9	[20]
Saudi Arabia (Al-Qassim)	2.9 (100% male patients)	[23]
Qatar	2.51	[24]
Egypt	Overall: 4.3 (Newborns)	[25]
Egypt (2016)	Overall: 8.9 (Jaundiced neonates)	[26]
Iran	Overall: 3.2 Male: 5.1 Female: 1	[27]

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