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Prevalence of Glucose -6- Phosphate Dehydrogenase (G6PD) Deficiency (Favism) in Thamar Province-Republic of Yemen

Background: Glucose-6-Phosphate dehydrogenase (G6PD) deficiency is an X-linked recessive disorder expressed mostly in males. Prevalence of G6PD deficiency varies in different parts of the world's according to ethnic variation. The incidence varies among different countries in the world and surveys report rates of less than 1% to 35%. The prevalence of G6PD deficiency in the Arab world has variously been reported range from 1 to 65 %.

Objectives: To study the prevalence of glucose-6-phosphate dehydrogenase deficiency among adult individuals and the severity of the enzyme deficiency according to the WHO classification.

Methodology: The study included 90 adult individuals (68 males & 22 females) included in the study. All sample tested for the complete blood cells count (CBC). Quantitative estimation of G6PD enzyme activity was performed by using a commercially available kits.

Results: A 13/90 subjects studied (14.5%) have G6PD deficiency and 7/ 13 (53.8 %) have WHO class-II enzyme variant, 6 / 13 (46.2%) were of class-III variant and no class-V variant detected.

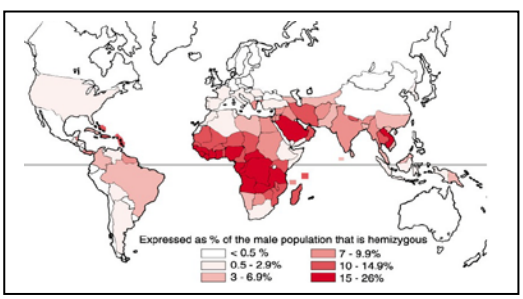
Conclusions: Glucose-6-Phosphate Dehydrogenase deficiency is a commonly occurred type of the inherited red blood cell enzymopathy with predominance of the Mediterranean variant (WHO class-II).

Key Word: Glucose-6-Phosphate dehydrogenase deficiency, prevalence, WHO classification, Arab countries.

INTRODUCTION

Glucose-6-Phosphate dehydrogenase (G6PD) deficiency is an X-linked recessive disorder expressed mostly in males. Since G6PD deficiency discovery by Alving et al, in 1956, G6PD deficiency causes a spectrum of diseases conditions including neonatal hyperbilirubinaemia (The most common clinical manifestation), acute hemolysis (Favism) and chronic hemolysis {1}.

Prevalence of G6PD deficiency varies in different parts of the world's according to ethnic variation. G6PD deficiency is the most prevalent enzyme deficiency worldwide with an estimated 400 million people affected worldwide {2-6}. The incidence varies among different countries and surveys report rates of less than 1% to 35%. According to the WHO, 7.5-10% of the world populations carry one or two genes for G6PD deficiency and 2.9 % are G6PD deficient {2-9}.



WHO Working Group [1989] Glucose-6-phosphate dehydrogenase deficiency. Bull WHO 67:601

G6PD deficiency is quite prevalent in the Africa, Asia, Middle East and Mediterranean and present in several forms throughout the world. The prevalence of G6PD deficiency has variously been reported as in the Arab world ranging from1-65 % {7, 10}.

Populations studied in the Middle East have shown remarkable variation in the prevalence rates of G6PD deficiency. There are considerable differences in the literatures regarding G6PD deficiency prevalence in Arab populations {11}.

Several epidemiological surveys to determine the frequency of G6PD deficiency in different countries in the region have been conducted. The results confirm that the problem is a common genetic disorder. Not all countries in the region have been covered to the same extent in these surveys, in some countries, several extensive accurate studies were performed, while in other only limited non standardized methodologies were used {12}.

The highest frequency (65%) of G6PD deficiency has been reported from KSA (Qatif oasis) of Eastern Saudi Arabia (In the eastern province, both male & female have the highest frequency of G6PD deficiency followed by those in the southwestern provinces){10,13-15 }.

The frequency is high in Bahrain reach 26.4% among blood donors and 28% in neonates. It reach 45% in the region of Sitra & 36% in the western area (Figure: 1) {10,16-18, 19}.

The prevalence of G6PD deficiency in Oman is 25% in male and 10% in female; it reached 29% in Dakhiliya province {10, 20, 21}. The frequency in Iraq is ranging from 6 to 13 % {22-25} and it is 8% for UAE (However in Abu Dhabi, UAE hospital-based study of consecutive patients in the late 1970s revealed a G6PD deficiency frequency of 25%) {10, 26, 27}. Kuwait prevalence of G6PD deficiency figure is 20.4% {28}. The low prevalence's (less than 5%) found in Syria, Egypt, Libya, Lebanon, Tunisia & Algeria courtiers(Figure:1) {10-12,23, 29-33} .

Yemen lies in a geographic situation where the prevalence of this disease is high in the close localities (Oman & KSA). Moreover, high rate of consanguinity reported in Yemen including second cousin relationship (40%) {34}, However, A recent study shows no statistically significant effect of parental consanguity on G6PD deficiency prevalence {35).

In the Middle East, malaria is still present in some area, including defined regions in Iraq, Oman, Saudi Arabia, Syria and Yemen. Most G6PD deficient individuals in the Middle East have the G6PD Mediterranean variant, although G6PD A- is also found in some areas {36-39}. It is suggested that the level above sea (Up to 600-850 meter) seems to have a significant role on the prevalence of malaria & subsequently, the G6PD deficiency that follow the malaria endemicity.

SUBJECT AND METHODS

Subjects: During the period of the study (from March 2010 to May 2010), blood samples (Through venipuncture procedure) were collected from 90 adult individuals (68 male & 22 female) included in the study. Every effort was taken to avoid blood relatives up to first cousin. Individuals data were collected in a study form designed by the author for the purpose of this study including special emphasis on history of fava bean consumption, any history of hemolytic episode following such consumption, whether they were ever diagnosed as having favism of G6PD deficiency and if they had any family history of this condition. The main source of the patients data studied was collected from students in Thamar University (Faculty of Education & Faculty of Management Sciences).

Methods:

1: Basic Haematological Parameters & G6PD enzyme assay: Venous blood samples were collected in EDTA tube (2 ml.) and kept at 2-6 ^{o}C and processed with 48 hours after collection. All samples were first tested for the full blood cells count (FBC) using automated blood cell analyzer (Sysmex KX 21N). Quantitative estimation of G6PD enzyme activity was performed by the oxidation of glucose-6 phosphate (G6P) to 6-phosphogluconate (6-PG), and the concomitant reduction of NADP+ to NADPH. These reactions occur in the presence of G-6-P DH, and the enzyme activity is determined by measurement of the rate of absorbance change at 340 nm due to reduction of NADP (The same tube could be kept for at least one hour and then be viewed under ultraviolet light).

NADPH activity was determined in a narrow-width spectrophotometer (RA-50 / Bayer) and the G-6-PD activity calculated in relation to Hemoglobin concentration. Commercially available kit (Cat. No. 97089, BIOLABO, France & RANDOX Laboratories Ltd, United Kingdom. Cat. No. PD 410) were used and the technique done with careful adherence to the manufacturer instructions. Results were interpreted as the percentage of normal G6PD. Enzyme activity less than 10% was classified as severe deficiency, whereas, the activity between 10 & 60 % of the normal activity was classified as moderate deficiency (The World Health Organization classification of G6PD variants). Reference rang according to manufacturer was 10.1-14 U/g Hemoglobin.

2: Statistical Analysis: The mean ± standard deviation (SD) calculation and two sample "t" tests were employed for statistical evaluation of the results using standard methods by computer programs: Excel & Word program under windows 2007 and SPSS programs. P-value of less than 0.05 was considered significant.

RESULTS

A total 90 subjects were included in this study. Table 1, present the distribution of the individuals studied according to the age and sex and the WHO G6PD variant classification. Out of the total subjects studied: (13/ 90) (14.5%) were G6PD deficient. 68/90 (75.5%) were males & 22/90 (24.5%) were females. 12/13 deficient (92.3%) were males & (1/13) (7.7 %) were females with total M: F ratio (3.5:1). The mean age of the male and female subjects studied is 22 years. The WHO classification of the enzyme activity variant in the subjects studied shows: 7/ 13 (53.8 %) were class-II variant, 6 / 13 (46.2%) were of class-III variant. No class-V variant in the studied subjects. 77/total (85.5 %) shows normal enzyme activity.

The questionnaire results is shown in table 2 with 2/13 subjects (15.4%) had positive past history of hemolysis. 1/13 subject (7.7 %) had positive family history of hemolysis (red urine and jaundice) when exposed to fava beans, mothballs, aspirin or other drugs. No subject had a diagnosed G6PD deficient member in the family. 1/13 subject (7.7 %) had positive history of neonatal jaundice.

DISCUSSION

White et al. (1986) analyzed: a total of 219 Emigrant Yemenis sample in UAE (146 adult male and 73 male

newborn cord blood) on routine EDTA blood samples and determined the frequency of glucose-6-phosphate dehydrogenase deficiency in Yemen to be 6% {40}.

Thamar province located at 500-2400 meters above sea level, which is higher than most other provinces in Yemen. It is inhabited by about 1,330,108 (2004) peoples.

The estimated prevalence of G6PD deficiency in the Present study (Table: 1) is higher than the previous study of White et al. This difference might attribute to {12,41}: demographic distributions, the high consanguinity rate (40% in Yemen) which increase the risk of recessively inherited disease, the high frequency of G6PD deficiency might attribute to the selective advantage of carriers against falciparum malaria and the general lack of public health measures directed at prevention of genetic diseases with little genetic services available. In addition: the White et al subjects were older adult age group and the decreasing deficiency with advancing age could be explained in that during aging, a system develop that might accomplish a full capacity for normal G6PD activity {2,42}.

The results of this study (Table 1) shows that the Mediterranean mutation variant predominate in the subjects studied. This finding agrees with most of the published studies in the Arabian areas and pointing that this variant is the main type in Arab populations {14, 16, 18, 19, 37, 48}. No class-V variant detected, which agree with the universally accepted concept of this type being very rare {6, 7, 37}.

Because G6PD deficiency is a sex-linked disorder, it shows full expression in heterozygous male and homozygous female. Heterozygous female shows variation in the expression depending on the degree of X- chromosome inactivation. Thus, heterozygous female may have undetectable, intermediate or normal enzyme levels in their erythrocytes. The incidence of severe form of the enzyme deficiency in female is considerably lower, with most of the cases being of the mild form of the disease {44}. In the present study, 1/22 (4.5%) studied females show enzyme deficiency (Table 1).

Enzyme activity in the one deficient female studied was of class-III variant which agree with the globally accepted concept that mild form of the disease occur in female (because of the lyonization effect (Mosaic phenomenon) which indicate that females has two X-chromosome, if one is affected and it is inactive, the enzyme level will be normal and if the active X-chromosome has the defective gene, the disease will manifest itself {16, 36}.

WHO recommend screening all newborns in population with prevalence of 3 to 5 percent or more in male {45}. The complication of G6PD deficiency can be prevented by establishing screening programs combined with health education. Other preventive measures include education of patients and doctors, and the provision of a list of agents to be avoided. However, such a genetic trait does not warrant prenatal diagnosis or the advice for termination of pregnancy.

The finding of the Questionnaire results (Table 3) is consistent with that of White et al who conclude in his study that none of the 100 hospital patients who were found to be G6PD deficient had, or gave any history of hemolytic anaemia {46}.

Therefore, addition of past history and family history of hemolysis shows poor predictive value in detection of the G6PD deficiency subject. This is in contrary to the results given by other study {47} and suggest that other preventive measures including education of patients and doctors is essential in predicting and disease control together with the provision of a list of agents to be avoided{18}.

Recently, A group of predictors (Including male gender, younger age group, fever, vomiting (abdominal pain and gastric pain) and negative family history) of severe hemolysis in patients with G6PD deficiency following exposure to oxidant stresses is published {44}.

No statistically significant differences in the basic hematological parameters detected between those with laboratory evidence of G6PD deficiency and those without. This indicate that the affected subjects with Class-II & III variant shows no hematological parameters defect (Subclinical enzyme deficiency) (Table:2) under steady states and only a triggering oxidant stress required for the disease to be clinically overt {4,5, 37}.

Using PCR base technique, four mutations were found accounting for more than 90 % of G6PD deficiency cases among Chinese neonate in Malaysia {47}. So, it is recommended to use molecular methods for detection of G6PD deficiency variant especially in neonates with high or prolonged jaundice. However, such a genetic trait does not warrant prenatal diagnosis or the advice for termination of pregnancy {18}.

Once G6PD deficiency is confirmed, close monitoring, health education regarding triggers and proper follow-up for hemolysis can prevent severe hemolysis, thus decreasing morbidity and mortality in patients with G6PD deficiency {44}.

CONCLUSION

Glucose-6-Phosphate Dehydrogenase (G6PD) deficiency is a commonly occurred type of the inherited red blood cell enzymopathy with predominance of the Mediterranean variant (WHO class-II). Laboratory evidence of G6PD deficiency requires particular attention to the non-defected basic hematological parameters in deficient subjects. Addition of past history and family history of hemolysis has a poor predictive value in

detection of the G6PD deficiency subject. Due to lyonization, many of carriers female cannot be identified as mild form of the disease occur in female. It cannot be claimed our study population was representative of the Yemenis populations.

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Table I. Distribution of the subjects studied according to the age, sex and WHO G6P DH variant classification.

Enzyme activity	No. (%)	Sex	No. (%)	WHO G6P DH variant classes		No. (%)
Normal G6P DH activity	77 (85.5)	M	56 (72.7)	Class-IV		77/90 (85.5)
		F	21 (27.3)			
Increased G6P DH activity				Class-V		None
Deficient G6P DH activity	13 (14.5)	M: 12 (92.3) F: 1 (7.7)		Class- I	(G6P DH: <10 %) with Chronic hemolysis	None
				Class- II	(G6P DH: <10 %) without Chronic hemolysis	7/13 (53.8)
				Class-III	(G6P DH: 10-60 %)	6/13 (46.2)
Total	90 (100%)	M	68 (75.5)	M:F Ratio 3.5:1		
		F	22 (24.5)			

12/13 G6P DH deficient (92.3%) were males and 1/13 (7.7 %) was female with total M: F ratio (3.5:1). 7/ 13 (53.8 %) subjects show class-II variant with 6 / 13 (46.2%) were of class-III variant.

Table II. Questionnaire Results

No.	Questionnaire	Positive response / Total	%
1	Do you have any history of red urine or jaundice (hemolysis) after ingesting fava beans, aspirin or other drugs?	**2/13**	**15.4**
2	Does any member of your family have a history of red urine or jaundice (hemolysis) after ingesting fava beans, aspirin or other drugs?	**1/13**	**7.7**
3	Is there any history of favism in your family members (Diagnosed)?	**0/13**	**0**
4	Do you have any history of neonatal jaundice (incubator or Hospital admission)?	**1/13**	**7.7**
5	Do you have any history of exchange transfusion for neonatal jaundice?	**0/13**	**0**
Note: The individual is not a relative of any cases included in this study.		**Total**	**4/13** **30 %** **9/13** **70%**

No subject had a diagnosed G6P DH deficient member in the family.

Table III. The relationship between G6P DH deficient activity and haematological parameters of the subjects studied.

Parameters	G6P DH enzyme activity		P value
	Normal	Deficient	
	Mean±SD.	Mean±SD.	
G6P DH activity	12.8 ± 2.3	1.8 ± 1.5	**< 0.05**
Hemoglobin (g/dl)	16.4 ± 2.0	16.2 ±1.7	NS
Total WBC count ($\times 10^9$/L)	9.7 ± 3.2	5.9 ± 2.2	
Platelet count ($\times 10^9$/L)	281 ± 69.1	317 ± 66.7	

Data presented as mean ±SD, P-value using t-test for two samples, statistically significant change (<0.05). NS: Not significant

No statistically significant differences in the basic hematological parameters detected between those with laboratory evidence of G6P DH deficiency and those without.

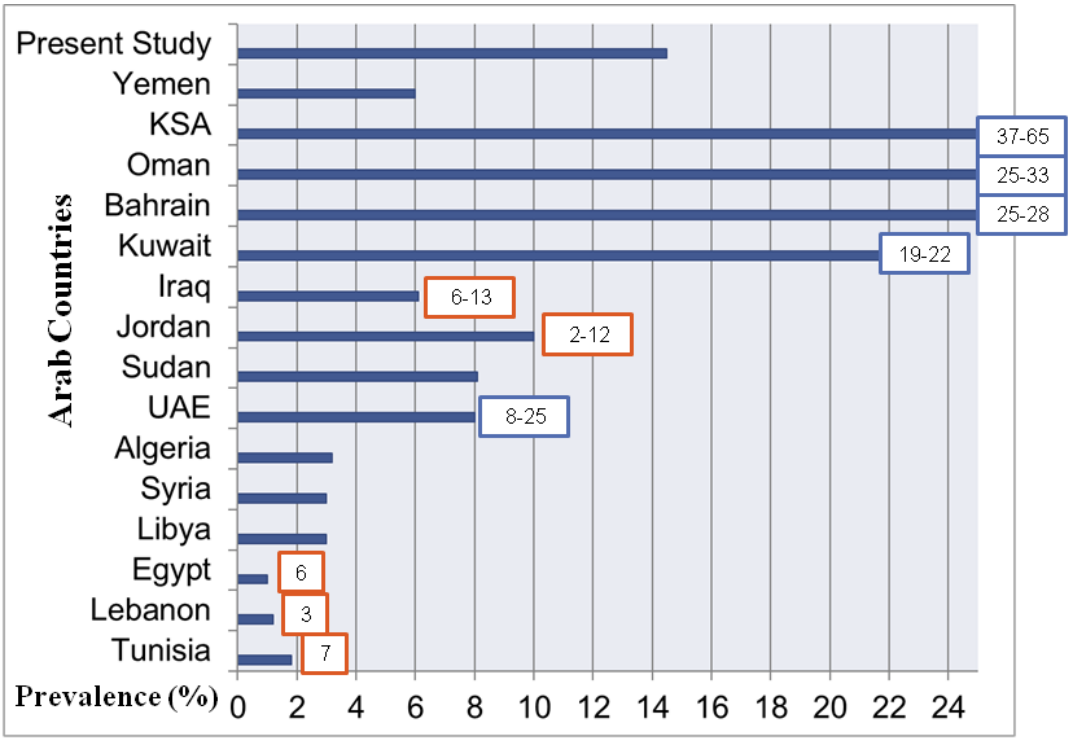

Figure I: Prevalence of G6P DH Deficiency among Arab Populations.