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RESEARCH ARTICLE

Prevalence of Glucose-6-Phosphate Dehydrogenase deficiency among Hausa and Fulani ethnics in Kano, North-West Nigeria

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ABSTRACT

Background: Glucose-6-phosphate dehydrogenase catalyzes the rate-determining step in the pentose phosphate pathway. Its activity is a key determinant of the reduced nicotinamide adenine dinucleotide phosphate to oxidized nicotinamide adenine dinucleotide phosphate ($NADPH$-to-$NADP^+$) ratio in the cytoplasm and thus contributes to the replenishment of the antioxidant glutathione system. Glucose-6-phosphate dehydrogenase deficiency is a common X-linked human enzyme defect of red blood cells. Individuals with this gene defect appear normal until exposed to oxidative stress which induces haemolysis but it is infrequently taken into consideration in health practices.

Aim: The present work was designed to determine the frequency of occurrence of glucose-6-phosphate dehydrogenase (G-6-PD) deficiency among Hausa and Fulani ethnics.

Material and methods: Cross-sectional comparative study in which 5 mL of venous blood samples were collected from 1000 individuals (596 males and 404 females) all of whom gave informed consent were screened for G-6-PD-deficiency by qualitative methaemoglobin reduction and quantitative enzyme activity measurement techniques.

Results: Glucose-6-phoshate dehydrogenase deficiency was found in 194 (19.4%) of participants studied. Of these 10.3% were Fulani and 22.2% were Hausa with mean G-6-PD enzyme activity of 2.5 IU/gHb in a range from 1.4 to 2.4 IU/gHb. G-6-PD deficiency was found in 121 of 596 (20.3%) males and 73 of 404 (18.1%) females screened.

Conclusions: These results suggest high occurrence of phenotypic G-6-PD deficiency in Hausa ethnics of Kano with lower enzyme status among males living with G-6-PD deficiency.

Keywords: G-6-PD deficiency, Hausa, Fulani, Kano-Nigeria

Introduction

Glucose-6-phosphate dehydrogenase (G-6-PD) gene provides instructions for making an enzyme glucose-6-phosphate dehydrogenase (G-6-PD, EC 1.1.1.49). This enzyme, which is active in virtually all type of cells, is involved in the normal processing of carbohydrates and plays a critical role in red blood cells. In the hexose-mono phosphate shunt, G-6-PD catalyses the production of NADPH, which plays a major role in protecting cells from potentially harmful reactive oxygen species[1].

G-6-PD deficiency affects some 400 million people worldwide and is the most common human enzymopathy[2]. G6PD deficiency is common throughout sub-Saharan Africa, regions in the Mediterranean, and parts of Southeast Asia and is thought to be preserved in these populations because it results in few gross complications and it confers a selective advantage against malaria[3].
The study of Ella *et al.* [4] has reported global phenotypic estimation of G-6-PD deficiency as 4.9%. Of these 3.4% was reported from America, 4.7% in Asia, 3.9% in Europe, 6.0% in Middle East, 2.9% in Pacific and 7.5% in Africa.

Luzzatto and Gordon-Smith [5] reported the prevalence of G-6-PD deficiency in Nigeria ranging from 4 – 26% with the male population having about 20 – 26%. Oduola *et al.* [6] reported 26.7% as prevalence of G-6-PD deficiency in Ile-Ife. Akanni *et al.* [7] reported a prevalence rate of 19.5% among prospective and suitable blood donors in Osogbo with G-6-PD deficiency. In Jos, 20% of the male individuals studied had G-6-PD enzyme activity below the normal reference range and so are G-6-PD deficient [8]. Mockenhaupt *et al.* [9] reported 24.1% as prevalence of G-6-PD deficiency in Nigeria. The study by May *et al.* [10] reported 20.3% in Ibadan. Omisakin *et al.* [11] reported 25.5% as prevalence of G-6-PD deficiency among blood donors in Nigerian population of Ekiti State. In Sokoto, 37.6% G-6-PD deficient subjects were reported by Jelani *et al.* [12]. In Enugu, Ogbodo *et al.* [13] reported 10.5% prevalence.

The clinical manifestations of G-6-PD deficient subjects are highly variable from neonatal jaundice and severe acute haemolytic anaemia following the ingestion of certain drugs, during some infection and notably through eating fava beans [14-15]. G-6-PD deficiency has been implicated in the pathogenesis of a number of common diseases including cardiovascular diseases, hypertension, diabetes mellitus, cardiac dysfunction and atherosclerosis [16-17].

Nigeria is endemic for malaria and antimalarial drugs are routinely prescribed for most cases. The rationale to screen for glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is their increased likelihood to receive oxidant drugs and subsequent potential of severe haemolytic events to a level that could warrant blood transfusion and even death through kinecterus [18].

Thus, the knowledge of G-6-PD status of patients will contribute in better management of patients with G-6-PD deficiency, planning programs to improve neonatal health and in the distribution of various medications, especially antimalarial drugs, as G-6-PD deficiency is most prevalent in malaria-endemic areas.

Materials and methods

STUDY AREA

The study area was the metropolitan city of Kano which comprised of eight local government areas (n=8 LGAs) including: Dala, Fagge, Gwale, Kano Municipal, Kumbotso Nassarawa, Tarauni and Ungogo LGAs in Kano State. Kano is located at the extreme part of North Western Nigeria between longitude 3° and 7° east and between latitude 10° and 14° north of the equator. It shares borders with Jigawa State to the North- East, Katsina State to the North-West, Kaduna State to the South-West and Bauchi State to the South-East [19].

SUBJECTS

The study was approved by the ethical committees of Kano State Ministry of Health (NHREC/ 17/03//2018) and permissions were obtained from various study sites. Following an advocacy outreach to create awareness on Nasara FM (98.5 Hz) and Vision FM (98.5 Hz and 92.5 Hz) radio station in Kano metropolis. Study subjects were informed about the study, their right and responsibility of the research was explained through an informed consent and assent form which was duly signed by all participants that were recruited into the study. A close ended questionnaire was administered to screen for eligibility.

One thousand apparently healthy volunteers of the study population were recruited for the study to establish the prevalence of G-6-PD deficiency in Kano. Individuals transfused with whole blood in the last 3 months, sign of jaundice, cigarette smoking were excluded. .

STUDY DESIGN

Cross-sectional comparative design

BLOOD COLLECTION

Blood specimen (5 mL) was collected from the cubital vein of the forearm of 1000 apparently healthy volunteer of Kano residents and dispensed into EDTA bottle. Blood specimen were processed and subjected to full blood count (FBC) and G-6-PD deficiency screening.

METHODS

Two analytical methods were used for G-6-PD deficiency screening; Methaemoglobin reduction test method [20] and G-6-PD assay activity [21] using Trinity G-6-PDH test kits to detect and investigate the deficient samples. Full blood count (FBC) analysis using Genesis™ HA 6000 Auto Haematology Analyser (Nortek company, Japan). The screening and Haemoglobin estimation was carried out on the day of blood collection.

STATISTICAL ANALYSIS

GraphPad InStat Software was used to check for typographic errors, outliers and to carry out normality testing for continuous data. Statistical analysis was performed using SPSS (version 26). The descriptive statistical tool was applied to establish prevalence of G-6-PD status. Mann-Whitney U statistics test was performed to compare of G-6-PD activity between deficient and non-deficient participants. Kruskal-Wallis test was performed to compare G-6-PD activity with respect to three categories of participants. Descriptive data were expressed as percentages and continuous data were expressed as Mean ± SD.

Results

SOCIO DEMOGRAPHIC CHARACTERISTICS OF THE STUDY PARTICIPANTS

A total of 1000 volunteer were enrolled for the study without violating the eligibility criteria. During the study period, more male subjects than female subjects participated in the study. Most subjects were within the age of 21-30 years. Most of the study populations were unmarried (single). Among the ethnic groups represented in the study population (Hausa, Fulani, Yoruba, Igbo, others), the proportion of Hausa ethnics in the study population was higher. More participation in the study was recorded from people residing in Ungogo local government in the metropolitan city of Kano (Table 1).

Table 1: Socio-demographic characteristics of study participants

Demographic features		n	% (percentage)
Gender			
	Male	596	59.6
	Female	404	40.4
Age group (years)			
	1-10	23	2.3
	11-20	121	12.1
	21-30	661	66.1
	31-40	157	15.7
	41-50	28	2.8
	51-60	10	1.0
Marital status			
	Married	251	25.1
	Single	749	74.9
Ethnic group			
	Hausa	671	67.1
	Fulani	29	2.9
	Yoruba	100	10.0
	Igbo	28	2.8
	Others	172	17.2
Residence			
	DAL	79	7.9
	FGE	150	15.0
	GWL	85	8.5
	KBT	106	10.6
	KMC	57	5.7
	NSR	69	6.9
	TRN	91	9.1
	UGG	363	36.3

n=number

%=percentage

DAL= Dala

FGE= Fagge

GWL= Gwale

KBT=Kumbosto

KBT=Kano Municipal

NSR= Nassarawa

TRN=Tarauni

UGG-=Ungogo

PREVALENCE OF G-6-PD DEFICIENCY IN KANO RESIDENT'S INDIVIDUALS

Among 1000 screened study participants, 194 (19.4%) were phenotypically deficient for G-6-PD using the methaemoglobin reduction test while the remaining 806 (80.6%) were non-deficient volunteers (Table 2).

Table 2: Prevalence of Glucose-6-Phosphate Dehydrogenase deficiency in Kano

Subjects	Number	% (percentage)
G-6-PD Normal	806	80.6
G-6-PD Deficient	194	19.4
Total	1000	100

% = percentage, G-6-PD= glucose-6-phosphate dehydrogenase

PREVALENCE OF GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY IN KANO ACROSS GENDER

The G-6-PD deficiency was found In 121 of 596 (20.3%) males and 73 of 404 (18.1%) females screened (Table 3).]

Table 3: Gender-wise distribution of glucose-6-phosphate dehydrogenase deficiency

Gender	Screened subjects	Deficient subjects	% (percentage)
Male	596	121	20.3
Female	404	73	18.1
Total	1000	194	19.4

% = percentage, G-6-PD= glucose-6-phosphate dehydrogenase

PREVALENCE OF G-6-PD DEFICIENCY IN KANO ACCORDING TO ETHNICITY

The frequency of G-6-PD deficiency by ethnic origin of the study participants indicted that Ebira tribe had (13.1%), Fulani (10.3%), Hausa (22.2%), Igala (20.5%), Igbo (21.4%), Idoma (8.2%), Kanuri (14.3%), Nupe (37.5%) whilst those of Yoruba ethnics were 19.0% (Table 4).

Table 4: Distribution of glucose-6-phosphate dehydrogenase deficiency by ethnicity

Ethnicity	Screened subjects	Deficient subjects	% (percentage)
Ebira	61	8	13.1
Fulani	29	3	10.3
Hausa	671	149	22.2
Idoma	49	4	8.2
Igala	5	1	20.5
Igbo	28	6	21.4
Kanuri	7	1	14.3
Nupe	8	3	37.5
Yoruba	100	19	19.0
Total	1000	194	19.4

% = percentage, G-6-PD= glucose-6-phosphate dehydrogenase.

ASSESSMENT OF G-6-PD ACTIVITY AMONGST HAUSA AND FULANI ETHNICS

Semi-quantification of G-6-PD activity performed amongst Hausa and Fulani ethnics of the study participants. It indicates the highest activity for those with normal G-6-PD subjects while the deficient participants had the least G-6-PD enzyme activity. However, G-6-PD enzyme activity in deficient subjects were significantly lower than the non-deficient participants ($p<0.001$). In contrast, there was no statistically significant difference among those with intermediate enzyme activity in comparison with deficient participants ($p>0.05$) (Table 5).

Table 5: Glucose-6-phosphate dehydrogenase enzyme activity in Hausa and Fulani ethnics

G-6-PD Status	Activity (IU/gHb)	Range
Normal (n=48)	11.9 ± 3.3[b]	6.1-19.9
Intermediate (n=11)	3.8 ± 0.3[a]	3.7- 4.9
Deficient (n=141)	2.5 ± 0.5[a]	1.4-3.4

Mean differences between three groups were compared (using Kruskal-Wallis test). n= group sample size. Values are expressed as Mean ± SD. Differences in mean values with the same superscript in the same column are statistically insignificant (P>0.05) and those with different values in the same column are statistically significant (P<0.05).

G-6-PD ACTIVITY AMONGST HAUSA AND FULANI ETHNICS ACROSS GENDER

Enzyme activity was observed to decrease in deficient males compared to females, though not statistically significant (p>0.05). Consequently, enzyme activity was significantly higher in male than female among non-deficient individuals (Table 6).

Table 6: Gender-wise distribution of glucose-6-phosphate dehydrogenase activity

	Activity (IU/gHb)	
G-6-PD Status	Males (n=50)	Females (n=50)
Normal	12.17 ± 3.03[c]	11.69 ± 3.62[b]
Deficient	2.42 ± 0.48[a]	2.56 ± 0.54[a]

n= Sample size. Values are expressed as Mean ± SD using MannWhitney U test statistical tool for analysis. Alphabetical values (a,b, c,d) with the same superscript in the same column are statistically insignificant (P>0.05) and those with different values in the same column are statistically significant (P<0.05). IU=international unit. g=gram, Hb=haemoglobin

Discussion

We report the results of a comprehensive epidemiological study investigating glucose -6-phosphate dehydrogenase (G-6-PD) deficiency among people residing in Kano metropolis. Global prevalence of G-6-PD enzyme is widely variable according to the geographical region and the analytical method employed [22]. Vast documentary evidenced from a study has indicated lower prevalence of G-6-PD deficiency to be 3.9% in Europe and 3.4% in America when compared to 7.5% in Africa [4]. Our results showed that the prevalence of phenotypic G-6-PD deficiency using methaemoglobin reduction method is 19.4% which is comparable to the study that reported 19.5% in Osogbo, Osun State, Nigeria [7], but however lower when compared to other studies showing 20.4% [23], 22.5% [24], 46.6% [25] in Kano, Nigeria. Our finding was higher in comparison to various studies showing 14.17% in Iran, Middle East [26], 13% in Kafanchan, North- Western Nigeria [27] 9% in Ogoni and 13% in Etche ethnic groups in Rivers State, South- South Nigeria [28]. So it is evident from our observation that G-6-PD deficiency is quite high in people residing in Kano metropolitan city.

In this study, more male subjects than female subjects participated. The results indicate a higher prevalence of G-6-PD deficiency among male (20.3%) than female (18.1%). Our findings with regards to the sexes corroborates with various studies reported from other parts of the country, such as Ibadan (29.3%) in males and (4.6%) in females [29], Kano with a higher proportion of males' deficient neonates when compared to females in a proportion of 3:1 [25] and Oshogbo (89.7%) in male subjects and (10.3%) in females [7]. Furthermore, Uzoegwu and Awah [30] reported the frequency of G-6-PD deficiency (9.21%) in males and (1.20%) in females participant in North West province of Cameroon and that of the South West province (10.85%) in males and (1.46%) in females participant reveals comparable results with the current study. A similar observation was reported in Thailand by Nuchprayoon *et al* [31] who found that the deficiency in males and females was 11.1 and 5.8 %.

On the contrary, Mombo *et al*. [32] reported higher prevalence of G-6-PD deficiency amongst (27.9%) in females compared to (17.8%) in males in a Central African country (Gabon). In addition, in Ilorin, North-Central Nigeria, equal occurrence of G-6-PD deficiency was reported among neonates of both sexes [33]. The divergent findings on this subject matter as observed by earlier researchers may be due to sample size disparity across the studies. In addition, the pattern of inheritance of the mutant gene which is an X-linked recessive trait predominantly a disease of males, thus making the,

illness associated with G-6-PD deficiency to be more expressed in erythrocytes of the affected male and few homozygous females with the mutant gene (s) on both X-chromosomes also manifests the condition [34, 18]. This result may however suggest that male subjects with G-6-PD deficiency are under risk for certain diseases.

In this study, the majority of the ethnic group was Hausa. Our findings have equally shown a prevalence of G-6-PD deficiency of 22·2% for Hausas and (10.3%) for Fulani, 19% in Yoruba, 37.5% in Nupe, 14.3% in Kanuri, 21.4% in Igbo, 20.5% in Igala, 8.2% in Idoma and 13.1% in Ebira people were living with G-6-PD deficiency and this results was concordant with the study of Adedemy *et al.* (35) who reported (1/5) 20% occurrence of G-6-PD deficiency among Fulani, (3/7) 42.9% among Yoruba, in Benin and it reflect the diverse spread of G-6-PD deficiency among Nigerian ethnicity. Data of the Hausa and Fulani ethnics is scarce among the north western Nigeria for comparison. However, our findings are in tandem with earlier studies that reported 23% and 39% of the population in Sub-Saharan regions [22], 28.1% [10] and 28.7% [29] in Lagos, Nigeria, 26.7% in Oshogbo, Osun State [6] whereas the study of Williams *et al.* [36] found lower occurrence of 16.9% in Yoruba, 10.1% in Igbo and, 10.5% in Igede people, 5.0% in Tiiv people on the contrary.

It has been evidenced that the frequency of G-6-PD deficiency varies worldwide among different ethnic groups. However, these divergent findings are mostly related to bias in patients selection, methods discrepancies, malarial endemicity, sample size, and different contributions of various ethnic groups to which over-representation of major ethnics in Kano community might be a reason for higher incidence of G-6-PD deficiency. On the contrary, under representation of other ethnics might not signify that they are free from G-6-PD deficiency.

The mean G-6-PD activity of 2.50 found in the present study is below the normal range (6.97 to 20.5 U/g Hb) indicated by the manufacturer of the test kit used (Randox Kits). These findings corroborates with studies reported by Ondei *et al.* [37] and Pereira *et al.* [38] in Brazil, in Kenya by Shah *et al.* [39] and in Venezuala (America) by Vizzi *et al.* [40]. This significantly lower G-6-PD activity in the test subjects is not unexpected as G-6-PD activity have been accepted as an indication for deficiency that has been implicated in the pathogenesis for many disease such as diabetes angiopathy, hypertension, retinopathy, cardiac dysfunction in people living with G-6-PD deficiency [41]. This may possibly due to non-random inactivation of X-chromosome or preferential selection of the defective clone can variably lower enzyme level producing different phenotypes [42]. Hence, red cells of carriers can exhibit normal, intermediate or grossly deficient G-6-PD activity.

Conflict of interest

None

Conclusion

This study has demonstrated that, the occurrence of G-6-PD deficiency among participants of Hausa and Fulani ethnics is relatively high. It is recommended that awareness of community wise occurrence of G-6-PD deficiency can encourage screening beforehand as this will assist the management of any possible drug associated symptoms, improve neonatal health by reducing the risk of children with neonatal jaundice which may be a possibility for kinecterus and high death-rates. Since with this high prevalence, blood products are not still checked for G6PD deficiency. So, they may be used for transfusion in neonates with jaundice or for patients using oxidants.

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