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Articles

Incidence of Glucose-6-Phosphate Dehydrogenase (G-6-PD) Deficiency in Apparently Healthy Individuals in Some Parts of North Central Nigeria

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ABSTRACT

A community based study of the incidence of G-6-PD deficiency in apparently healthy individuals in Jos South (Plateau State) and Jaba (Kaduna State) Local Government Areas (LGAs) of North Central Nigeria was carried-out. The screening of G-6-PD deficiency was performed on 270 subjects which comprised 120 individuals resident in Jaba and 150 in Jos South LGAs. The results were analysed according to G-6-PD activity, sex, genotype, marital status and age group. The results showed that 56 (20.7%) were G-6-PD deficient with females having higher incidence of 35 (12.0%) than 21(7.8%) in males. The incidence of the deficiency was higher in Jos South with 34(22.7%) than Jaba with 22(18.3%). Twenty six (9.6%) of the total population sampled had reduced G-6-PD activity while 30(11.1%) subjects were totally deficient in the enzyme activity. Within the deficient females, the heterozygotes were twice the number of homozygotes. With respect to marital status, unmarried had a higher incidence of G-6-PD deficiency occurring in 45(16.67%) than the married with 11(4.1%) subjects. The age group of 16-22 years old recorded highest number of deficient subjects of 23. The result therefore suggests a need for periodic screening of populations before marriage to avoid the transfer of this genetic trait.

Keywords: Glucose 6-Phosphate Dehydrogenase, haemolysis, jaundice.

INTRODUCTION

Glucose-6-phosohate dehydrogenase deficiency is one of the most well known human genetic defect (enzymopathy) affecting over 400 million subjects throughout the world (1,2,). This genetic disorder affects various geographic populations (3); occurring both in black and white races (4).

The major clinical consequences of G-6-PD deficiency is haemolytic anaemia (6). This disorder is usually episodic, but the vast majority of people with G-6-PD deficiency have no symptoms until subjected to oxidative challenge (7). The major symptoms of haemolytic anaemia are jaundice, dark urine, abdominal and back pain, lowered red blood cell count and elevated bilirubin (8). Individuals that suffer from severe and chronic form of deficiency in addition, may have gallstones, enlarge spleen, defective white blood cell and cataracts (8).

G-6-PD deficiency is associated with neonatal jaundice, kernicterus and even death (9). This is because attacks of haemolytic anaemia are serious for infants (9). Newborns with G-6-PD deficiency are about 1.5 times as likely to get neonatal jaundice than normal ones (10,11).

In Africa where malaria is endemic, individuals with G-6-PD deficiency suffer more as a result of combined effect on red blood cells due to excess oxidative free radicals (12). It has been recorded that the highest incidence of G-6-PD deficiency is found in those with the great-

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est occurrence of thalassaemia (13) and in areas in which malaria is endemic (7). Thus the deficiency is very common among persons of Africa, Asian and Mediterranean descent. Several reports have been published on this genetic disorder in Greece (14), Romania (15), Algeria (16) the United States (17) and Saudi Arabia (18).

Inspite of concerted efforts in research and published work in other countries, there still appears to be a dearth of information on the incidence of this deficiency particularly in North Central Nigeria. This paper reports attempts to screen some apparently healthy individuals for G-6-PD deficiency in 2 states of North Central Nigeria.

MATERIALS AND METHODS

Study Subjects

A total of 270 apparently healthy volunteers were randomly selected from Jaba and Jos South Local Government Areas (LGAs) of North Central Nigeria. This comprised 120 subjects resident in Jabe and 150 in Jos South LGAs of Kaduna and Plateau States respectively. The selected individuals included 133 males and 137 females with ages ranging from 7 to 80 years.

Sample collection and analysis

The blood samples from apparently healthy individuals were collected through venepuncture from the anticubital vein of the forearm into EDTA containers. Each of the blood samples collected was screened immediately using methaemoglobin reduction test as described by Brewer *et al.* (19).

RESULTS

Table 1 shows the prevalence of G-6-PD in the subject population. Approximately one among every 10 persons was either deficient or had reduced enzyme activity. The relationship between the enzyme deficiency and sex is shown in table 2. The frequency of occurrence of normal subjects was comparable in both sexes. Among the females, the prevalence of heterozygotes was twice the rate homozygote subjects. The distribution of subjects in the two LGAs indicates that both normal and deficient subject distribution patterns are comparable in the Jos and Jaba LGAs (Table 3). Among the single and married subjects, the deficiency state in singles was almost twice the frequency observed in married subjects ($P<0.05$). (Table 4).

The highest number of deficient subjects by age was found among the adolescents and others in early adulthood within the 16 – 22 years age group followed by subjects of 23 – 29 years of age (Table 5).

Table 1: G-6-PD prevalence in the total population screened

G-6-PD	No. (%) of subjects
Normal	214(79.3)
Reduced activity	26(9.6)
Deficient	30(11.1)

Table 2: The relationship between G-6-PD deficiency and sex

Group	No. (%)[a] of subjects by sex	
	Males (n=133)	Females (n=137)
Normal	112(84.2)	102(74.5)
G-6-PD deficient	21(15.8)	35(25.5)
Homozygotes		11(31.4)[b]
Heterozygotes		24(68.6)[b]

[a]Percentage is based on the No. of subjects by sex.
[b]Percentage is based on the No. of deficient females.

Table 3: Compared incidence of G-6-PD deficiency in the 2 LGAs.

G-6-PD status	No. (%)[a] of subjects by LGA	
	Jaba (n=120)	Jos South (n=150)
Normal	98(81.7)	116(77.3)
Deficient	22(18.3)	34(22.7)

[a]Percentage is based on population size

Table 4: The relationship between G-6-PD deficiency and marital status.

G-6-PD status	No. (%)[a] of subjects of status:	
	Single (n=189)	Married (n=81)
Normal	144(76.2)	70(86.4)
Deficient	45(23.8)	11(13.6)

[a]Percentage is based on No. with status

Table 5: The relationship between G-6-PD deficiency and ages of subjects

Age Group (yrs)	No. of Deficient subjects	Z^2
7-15	2	0.05
16-22	23	0.74
23-29	19	0.00
30-36	8	0.66
37-43	2	0.13
44-50	-	2.58
>51	2	1.50
Total	56	5.66

DISCUSSION

The results of all the volunteers screened are presented for total and LGA incidence, sex, marital status, age and genotype in tables I-VI respectively. Data generated were subjected to statistical analysis using percentages and chi-square.

The occurrence of G-6-PD deficiency in this study population from North Central Nigeria was recorded in 56(20.7%) of the 270 subjects who were apparently healthy individuals. This number comprised 26(9.6%) with reduced enzyme activity and 30(11.1%) with complete deficiency. The incidence rate obtained is low when compared to 26.8% of the 272 patients earlier reported from Ahmadu Bello University Teaching Hospital, Zaria (20). This is expected among the sick because deficiency of the enzyme on its own could make an individual more liable to oxidative stress and the onset of disease. The reduced G-6-PD activity could be attributed to the fact that females have two X-chromosomes producing two types of cell populations (21). These constitute the heterozygous female which appear to be entirely normal while others appear to be affected (2). The deficient group consists of hemizygous males and female homozygotes.

Furthermore, the incidence rate of deficiency was observed to be significantly higher in females than in males. The genes responsible for inheriting G-6-PD deficiency are located on the X-chromosomes. Consequently, enzyme deficiency finds full expression in males carrying a variant gene (hemizygotes), and in female homozygotes (22). Thus, the prevalence of G-6-PD deficiency in any given population is determined by the number of deficient males (8). Females are also at risk of haemolysis and jaundice (23). The occurrence of higher rates of deficiency (although not significantly different) of the enzyme in Jos South than in Jaba, can be attributed to the presence of more deficient males in the community since in any given population the prevalence is determined by the number of deficient males (8). In a population, with high prevalence rate, early detection of the enzyme deficiency by neonatal screening can prevent complications such as neonatal jaundice which is usually severe enough to cause death

or permanent neurological damage (7). Early detection can also help to avoid the recourse to permanent support with drugs by deficient individuals.

Our results also revealed a total incidence rate of 25.6% deficiency in females. This is higher than 19.0% reported in Saudi Arabia and 14.0% in Israel recorded by Abbas Al-Omran *et al*. (8) and Kaplan and Abramor (11) respectively. Furthermore, the G-6-PD deficient heterozygote showed higher incidence than homozygote. The high incidence rate in Saudi females has been attributed to high rate of consanguinity in the population, leading to an increased number of female homozygotes (24). This observation is however in contrast to our findings which show the frequency of heterozygous females to be twice that of the homozygotes. The explanation may relate to the fact that consanguinity is not a common practice in Northern Nigeria. With respect to marital status, the incidence rate was observed to be significantly higher in singles than among the married subjects in the population. We also recorded the highest number of deficient subjects within the age interval of 16-22 years old. Since these subjects are of marriageable age, there is a need for the development of appropriate control strategies based on the availability of relevant and adequate information on the frequency and severity of its complication.

In view of the clinical importance of G-6-PD deficiency, the need for early screening to detect its occurrence is apparent. Since this enzyme deficiency in marriage group exists, there is a compelling need for introducing measures such as genetic counseling and public health education as part of the overall health and welfare service in Nigeria.

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