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Prevalence of Glucose-6-Phosphate Dehydrogenase Deficiency in Malaria Patients Attending the Muhammadu Abdullahi Wase Specialist Hospital Kano Nigeria

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Abstract

Glucose-6-Phosphate Dehydrogenase (G-6-PD) deficiency is the most important disorder of the pentose phosphate pathway in erythrocyte metabolism resulting in decreased activity of the enzyme. G-6-PD deficiency has been reported to be common in populations living in malaria endemic areas. Certain drugs that are used in the treatment of malaria and other conditions are known to induce hemolysis in G-6-PD deficient individuals. This study was carried out to determine the prevalence of G-6-PD deficiency in individuals with malaria infection. One hundred subjects were recruited in the study with 50 individuals positive for malaria infection as test subjects and 50 negatives for malaria infection serving as controls. A G-6-PD deficiency prevalence of 14% was found in the study, 86% of the G-6-PD deficient individuals were from the control group while 14% were from the malaria positive group. A significant difference ($p \leq 0.01$) in G-6-PD deficiency was found between the malaria group and the control group. Among the G-6-PD deficient individuals 93% were males. There was a significant ($p \leq 0.05$) difference in Packed Cell Volume and hemoglobin levels between the G-6-PD deficient group and the G-6-PD normal group. The lower prevalence of G-6-PD deficiency among the malaria group compared to the control group validates studies linking G-6-PD deficiency with malaria. Also, the higher prevalence seen in control group and males, suggests that gender plays an important role in this enzyme deficiency hence the need for G-6-PD screening in our community given the overuse of agents that may predispose a G-6-PD deficient individual to oxidative stress which could cause some hematological complications such as hemolytic anemia.

Keywords: Glucose-6- phosphate dehydrogenase deficiency, malaria, erythrocytes, pentose phosphate pathway.

INTRODUCTION

Glucose-6-phosphate dehydrogenase (G-6-PD) is an enzyme in the pentose phosphate pathway (PPP) that plays an important role in protecting cells from oxidative damage by producing phosphorylated nicotinamide-adenine dinucleotide (NADPH), and reduced glutathione. In the erythrocyte, which lacks a nucleus, mitochondria and other organelles, PPP is the only biochemical pathway for generating reducing capacity (Carter *et. al.,* 2011). In G-6-PD deficiency, erythrocytes cannot produce enough NADPH, which affects glutathione peroxidase, thioredoxin reductase, and catalase activity. When erythrocytes are exposed to oxidative stress, cells cannot eliminate this stress, causing hemolysis. G-6-PD deficiency is mainly found in Africa, Asia, and Mediterranean Europe, areas where malaria is endemic, or has been endemic (Tishkoff *et al.,* 2001). Four hundred million people worldwide carry one or two deficient G-6-PD genes, making G-6-PD deficiency the most common enzyme pathology. Clinical manifestations of G-6-PD deficiency include neonatal jaundice and drug-induced hemolysis which may follow the ingestion of agents with oxidant properties, such as anti malarials like primaquine, sulphonamides, nitrofurantoin, and several anti-inflammatory agents (Pinna *et. al.,* 2008). Presently malaria is one of the major health challenges faced in Nigeria. Malaria is a public health problem worldwide, and a serious parasitic disease in the developing world,

causing high morbidity and mortality with an annual incidence of 247 million cases and nearly one million deaths, most of which afflict children living in Africa (WHO, 2008). The pathogenenesis of malaria is complex, and the clinical representation of disease range from severe and complicated, to mild and uncomplicated, to asymptomatic malaria (Laishram *et. al*., 2012). Effective treatment of malaria within 24 hours from onset of symptoms, helps prevent life threatening complications. It has been suggested that G-6-PD deficiency offers protection against malaria infection (Luzzatto and Bienzle, 1979). Dapsone, used in combination therapy for the treatment of *Plasmodium falciparum* (World Health Organization 2006), and primaquine, used to eliminate the hypnozoite reservoirs of *Plasmodium vivax* and *Plasmodium ovale,* can induce serious hemolytic events (World Health Organization, 2008; Fanello *et. al.* 2008). Wickramasinghe and Abdalla (2000) postulated that treatment of malaria with primaquine or dapsone and use of sulphonamides in G-6-PD deficient individuals can lead to life threatening acute intravascular hemolysis followed by anemia and acute renal failure. Although primaquine and dapsone have not been frequently used in malaria therapy in past years, White (2008) proposed a role for primaquine in a combination therapy with artemisinin derivatives for the treatment of infection by *Plasmodium falciparum*. This strategy is now becoming standard treatment in many developing countries. This implies that patients treated for malaria with an artemisinin–primaquine combination or even as prophylaxis for malaria may suffer from hemolytic anemia because of oxidative stress in their G-6-PD-deficient erythrocytes. Lack of severe clinical effects in most G-6-PD deficient individuals has led to little interest in this enzyme disorder and hence its concomitant effects. This in turn has led to the high use of drug regimens that could possibly induce some hematological complications in G-6-PD deficient individuals with or without malaria infection.

MATERIALS AND METHODS

Study Area: The sampling was conducted at the Abdullahi Wase Specialist Hospital, Kano, North Western Nigeria.

Ethical Consideration: Approval of the study was sought from the Ethical Committee of the Ministry of Health Kano, Kano State.

Study Population: Subjects comprised individuals diagnosed with malaria infection and apparently healthy individuals from the blood donor unit of the Abdullahi Wase Specialist Hospital, Kano. Informed consent was sought from the subjects.

Inclusion Criteria: Individuals with established malaria infection who agreed to participate were included in the study. Apparently healthy individuals were also included in the study as controls.

Exclusion Criteria: Individuals who declined consent were excluded from the study.

Sampling Techniques: At the sampling site, subjects who satisfied the inclusion criteria were selected. Subjects were categorised into two groups. Group 1**:** 50 individuals with malaria infection and Group 2: 50 apparently healthy individuals. Basic clinical and demographic information of all subjects were obtained from hospital records and through personal interviews from control group.

Sample Collection and Processing: Exactly, 3mls of venous blood from each subject was collected via the antecubital vein and put into an EDTA (Ethylene Diamine Tetra-acetic Acid) treated blood collection tube.

Confirmatory Test for Malaria: Malaria test strips were used to confirm malarial infection in the test group.

Determination of G-6-PD Activity: Samples were screened for G-6-PD activity using the Methaemoglobin Reduction Test as described by Cheesebrough, 2000 (Cheesebrough, 2000) with the underlying principle of the test being that haemoglobin is oxidised to methaemoglobin by sodium nitrite while the redox dye-methylene blue activates the pentose phosphate pathway, resulting in the enzymatic conversion of methaemoglobin back to haemoglobin in those red cells with normal G-6-PD activity. In G-6-PD deficient cells there is no enzymatic re-conversion to haemoglobin.

Determination of Packed Cell Volume: The packed cell volume (PCV) percentage in the anticoagulated blood sample was determined according to the Microhematocrit method (Cheesebrough, 2000) using microhematocrit centrifuge.

Haemoglobin Determination:

Haemoglobin concentration was determined using the Cyanmethaemoglobin method, (Cheesebrough, 2000). This is a type of colorimetric method in which the blood is mixed with a solution containing potassium ferricyanide and potassium cyanide, the potassium ferricyanide oxidizes iron to form methaemoglobin. The potassium cyanide then combines with methaemoglobin to form cyanmethaemoglobin, which is stable color pigment read photo metrically at a wave length of 540nm.

Statistical Analysis:

Data collected along with results of laboratory findings were collated and analyzed using SPSS version 18.0 (SPSS inc). The student t- test was used to test for significant differences in means of various groups, $p \leq 0.05$ was considered statistically significant. Continous variables were described as mean (standard deviation) and categorical variables as frequency (percentage).

RESULTS

Socio Demographic Characteristics:

Seventy two percent of the study population were males while thirty eight percent were females. Gender distribution between groups showed a higher percentage of males (79%) in the control group as compared to the females (21%) within this group. The mean age of the malaria group was 25.94± 8.86 years and that of the control was 22.68± 6.82 years. The highest number of individuals was observed in the 20-30 years age range in both groups.

Variation in G-6-PD Activity:

Fourteen individuals (14%) were found to be G-6-PD deficient while 86 (86%) had normal G-6-PD enzyme activity across the study population (table 1). Of the 14 individuals with G-6-PD deficiency in the study population, 86% (12/14) were from the control group while 14% (2/14) were from the malaria group hence within the malaria group 4% (2/50) were G-6-PD deficient and 24% (12/50) deficient in the control group (figure 1). A significant difference ($p \leq 0.01$) in G-6-PD deficiency was found between the malaria group and the control group.

Table 1: G-6-PD activity of subjects

Subject type	Number of subjects	%
All subjects	100	100
Normal subjects	86	86
G-6-PD deficient subjects	14	14

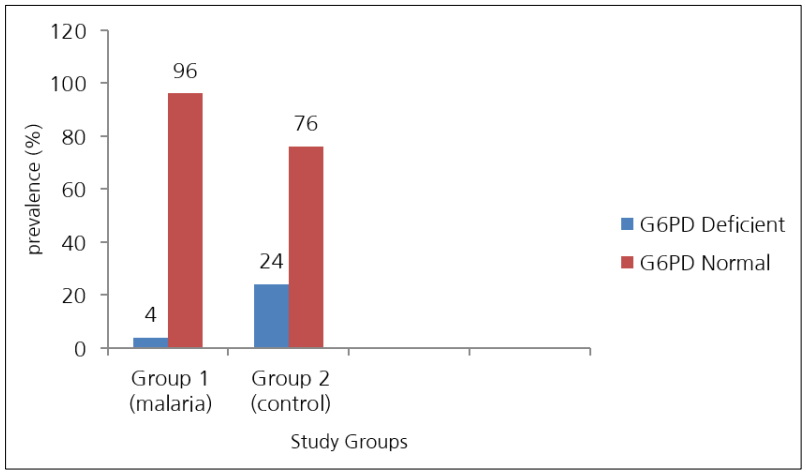

Figure 1: G-6-PD Activity Distribution Across Groups

Figure 2 shows the gender distribution of both the G-6-PD deficient individuals and the individuals found to have normal enzyme activity. Ninety three percent (13/14) of the G-6-PD deficient individuals were males while 7% (1/14) were females. Within the individuals with normal G-6-PD activity, 72% (63/86) were males while 28% (24/86) were females.

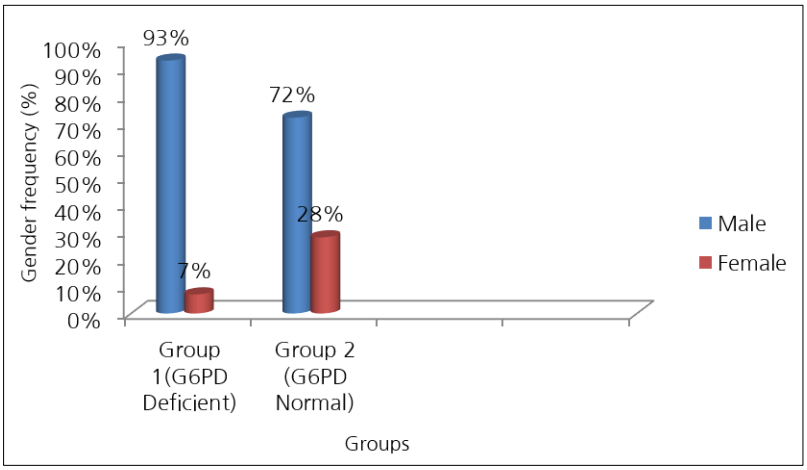

Figure 2: Gender Distribution of G-6-PD Activity in Study Population

Variation in Packed Cell Volume (%) and Hemoglobin (g/dl)

Mean PCV values for the G-6-PD normal group was 38.60±4.38 and for the G-6-PD deficient group mean PCV was 40.14±1.57. The mean haemoglobin value for the G-6-PD normal group was 13.08±1.69 while for the G-6-PD deficient group mean heamoglobin value was 14.39±1.46. There was significant difference ($p \leq 0.05$) between the two G-6-PD groups in both PCV and haemoglobin.

DISCUSSION

The prevalence of G-6-PD deficiency found in the study population falls within the range reported for Nigerians from other parts of the country. This prevalence has been put at 4- 26% and 20-26% among male Nigerians (Beutler, 1994). Much of the work culminating in the report of G-6-PD activity and deficiency was done in the mid 1970s and 1980s from the South Western region of Nigeria; although isolated reports of G-6-PD deficiency in specific clinical conditions were made from the North Eastern region of Nigeria (Tugwell, 1973), there is limited report of G-6-PD activity and deficiency in healthy population in the Northern region of Nigeria as far as the authors are aware. Abdullahi *et.al.* (2017) found a 22.5% G-6-PD deficiency prevalence in their study at the Aminu Kano Teaching Hospital North Western Nigeria among HIV infected individuals and blood donors. The higher percentage of males with G-6-PD deficiency agrees with several reports from Nigeria and other parts of the world. Male gender appears to play an important role in the pathophysiology of G-6-PD deficiency. This study agrees with the work of Egesie *et. al.* (2008) who found a G-6-PD deficiency prevalence of 20% among healthy Nigerian males. Ademowo and Falusi (2002) reported a prevalence of 21% in South Western part of Nigeria in male individuals. It may then be that the prevalence of G-6-PD deficiency among Nigerian males irrespective of their geographical location is relatively constant. Odaburhine (2015) stated that the genes responsible for inheriting G-6-PD are located on the x-chromosomes. Consequently, enzyme deficiency finds full expression in males carrying a variant gene (hemizygotes) and in female homozygous. Thus, the prevalence of G-6-PD deficiency in any population is determined by the number of deficient males. The higher number of males in the G-6-PD normal group could be attributed to a larger number of males present in the total study population. Age did not appear to have a role in the prevalence of G-6-PD enzyme deficiency in both the malaria group and the apparently healthy group. Glucose-6-phosphate dehydrogenase activity in the malaria group was significantly lower ($p \leq 0.01$) compared to the apparently healthy (control subjects) group. This finding is in agreement with the reports of Wang *et al.,* (2009) and Tripathy and Reddy (2007) who reported significantly lower activity of G-6-PD in malaria infected individuals. This result confirms the reports of other workers who have found that few individuals with malaria infection have G-6-PD deficiency. Capellini and Fiorelli (2008) reported that malaria parasites grow very slowly in G-6-PD deficient erythrocytes. The prevalence of the deficiency is correlated with geographical distribution of malaria which leads to the theory that carriers of G-6-PD deficiency may have partial protection against malaria infection (Nkohma *et. al.,* 2009). It has been postulated that G-6-PD deficiency is beneficial as it is known that erythrocytes that are deficient in G-6-PD are resistant to *Plasmodium falciparum* invasion since the parasite requires the enzyme for its normal survival in the host cell (Ruwende 1995 and Tishkoff 2001). This deficiency seems to offer a selective protection against *P.falciparum* malaria. It has, however been reported that some *P.falciparum* parasite strains have been able to synthesize their own G-6-PD enzyme thereby evading the immunity offered by G-6-PD deficiency in such individuals (Usanga and Luzzatto, 1985). Carter *et.al.* (2011) reported that hemizygously-deficient males and homozygously deficient females that have been infected with *Plasmodium falciparum* are less ill than non–G-6-PD-deficient individuals and that the infection is usually not lethal in G-6-PD deficient individuals possibly because *Plasmodium falciparum* can proliferate less efficiently in their erythrocytes. Several workers have postulated that although the exact mechanism of this protection is still unclear, it is possible that erythrocytes with low G-6-PD activity offer a hostile environment for parasite growth and thus advantage to G-6-PD deficient subjects. Moreover, it is known that in the pentose phosphate pathway of erythrocytes, glucose-6 phosphate dehydrogenase enzyme has an important role in production of NADPH which is essential for protection against oxidative stress in erythrocytes. In the case of G-6-PD deficiency, erythrocytes cannot produce enough NADPH, which affects glutathione peroxidase and other enzymes activities (Peters and Van Noorden, 2009). When erythrocytes are exposed to oxidative stress, cells cannot eliminate this stress, causing hemolysis. Haematological parameters, haemoglobin (Hb) and packed cell volume (PCV) of the study subjects were investigated as both malaria infection itself and G-6-PD deficiency could lead to anaemia and haemolytic incidence. Although, no anemia was found in both the G-6-PD normal and G-6-PD deficient individuals, the G-6-PD normal group which comprised mostly individuals with malaria had lower mean PCV and haemglobin levels. This was not surprising as malaria usually causes a decrease in both PCV and haemoglobin levels. The higher PCV and haemoglobin levels seen in the G-6-PD deficient group could be attributed to the higher number of apparently healthy individuals and also high number of males in the study population in general.

CONCLUSION

The lower prevalence of G-6-PD deficiency found in the malaria group validates other studies from different parts of the world linking malaria and G-6-PD deficiency. Given the preponderance of indiscriminate

self administration of drugs particularly over the counter antibiotics, analgesics and antimalarials in our environment which are contraindicated in G-6-PD deficient individuals, treatment using such drugs should be preceded by G-6-PD screening to avoid hemolytic anemia hence the need for G-6-PD screening.

REFERENCES

- Abdullahi, H., Maigatari, U., and Dakata, A.M (2017): Glucose-6-Phosphate Dehydrogenase Deficiency in Individuals Infected with Human Immunodeficiency Virus at the Aminu Kano Teaching Hospital Kano, North Western Nigeria. *IJBCRR.17(2):1-12*
- Ademowo O.G. and Falusi A.G. (2002): Molecular epidemiology and activity of erythrocyte G6PD variants in a homogeneous Nigerian population. *East Afr Med J* .79: 42-44.
- Beutler E. (1994): G-6-PD deficiency. *Blood* 84(11) 3613-3636
- Cappellini, M.D. and Fiorelli, G. (2008): Glucose-6- phosphate dehydrogenase deficiency. *Lancet,* 371, 64–74.
- Carter N., Pamba A., Duparc S., Waitumbi J.N. (2011): Frequency of glucose-6-phosphate dehydrogenase deficiency in malaria patients from six African countries enrolled in two randomized anti-malarial clinical trials. *Malar J*.10:241.
- Cheesbrough M. (2002): Blood Transfusion Practice: Blood donation and Storage of blood, District Laboratory Practice in tropical countries, low-price edition by Cambridge Universal Press pp. 352-353.
- Egesie D., Joseph D.E., Isiguzoro I., and Egesie U.G. (2008): Glucose-6-phosphate dehydrogenase (G6PD) activity and deficiency in a population of Nigerian male residents in Jos. *Niger J Physiol Sci*. 23(1–2):9–11.
- Fanello C.I., Karema C., Avellino P., Bancone G., Uwimana A., Lee S.J. and d'Alessandro U. (2008) High risk of severe anaemia after chlorproguanil-dapsone1artesunate antimalarial treatment in patients with G6PD (A-) deficiency. *PLoS One* 3:e4031.
- Luzzatto L, Bienzle U (1979) The malaria/G.-6-P.D. hypothesis. *Lancet* 1:1183–1184
- Nkhoma, E.T., Poole, C., Vannappagari, V., Hall, S.A. and Beutler, E. (2009): The global prevalence of glucose-6-phosphate dehydrogenase deficiency: a systematic review and meta-analysis. *Blood Cells, Molecules, & Diseases*, 42, 267–278.
- Odaburhine O.E. (2015): Prevalence of glucose-6-phosphate dehydrogenase deficiency among sickle cell patients. *Afr J Med Health Sci* ;14:83-6.
- Peters A.L and Van Noorden C.J. (2009): Glucose-6-Phosphate Dehydrogenase Deficiency and Malaria: Cytochemical Detection of heterozygous G-6-PD Deficiency in Women. *J.Hist.cyt* 57(11):1003-1011
- Pinna A., Solinas G, Masia C, Zinellu A, Carru C, and Carta A.(2008): Glucose-6-phosphate dehydrogenase (G6PD) deficiency in nonarteritic anterior ischemic optic neuropathy in a Sardinian population, Italy. *Invest Opthalmol Vis Sci*.49(4):1328–1232.
- Ruwende C., Khoo S. C., Snow R. W.,Yates S. N., Kwiatkowski D., Gupta S., Warn P., Allsopp C.E., Gilbert S. C., and Peschu N. (1995): Natural selection of hemi- and heterozygotes for G6PD deficiency in Africa by resistance to severe malaria. *Nature.* 376: 246-249.
- Tishkoff S.A., Varkonyi R., Cahinhinan N., Abbes S., Argyropoulos G., Stro-Bisol G., and Drousiotou A.,(2001): Haplotype diversity and linkage disequilibrium at human G6PD: recent origin of alleles that confer malarial resistance. *Science* 293:455–462
- Tripathy V. and Reddy B.M. (2007): Present status of understanding on the G6PD deficiency and natural selection. *J PostgradMed* 53:193–202
- Tugwell P. (1973): Glucose-6-phosphate dehydrogenase deficiency in Nigerians with jaundice associated with lobar pneumonia. *Lancet*. 968-970.
- Usanga E. A. and Luzzatto L. (1985): Adaptation to glucose-6-phosphate dehydrogenase-deficient host red cells by production of parasites encoded enzyme. *Nature*. 313: 793-795.
- Wang F.L., Boo N.Y., Ainoon O., Wong M.K. (2009): Comparison of detection of glucose-6-phosphate dehydrogenase deficiency using fluorescent spot test, enzyme assay and molecular method for prediction of severe neonatal hyperbilirubinaemia. *Singapore Med J*. 50(1):62–67.
- White N.J. (2008): The role of anti-malarial drugs in eliminating malaria. *Malar J 7*(suppl 1):8
- Wickramasinghe S.N., Abdalla S.H. (2000): Blood and bone marrow changes in malaria. *Best Pract Res Clin Haematol* 13:277–299
- World Health Organization (2006): Guidelines for the Treatment of Malaria. Geneva, WHO Press
- World Health Organization (2008): Antimalarial chlorproguanildapsone (LapDap™) withdrawn following demonstration of post-treatment haemolytic anaemia in G6PD deficient patients in a Phase III trial of chlorproguanil-dapsone-artesunate (Dacart™) versus artemether-lumefantrine (CoartemR) and confirmation of findings in a comparative trial of LapDap™ versus Dacart™.
- QSM/MC/IEA 117. World Health Organisation