International Journal of Hematological Disorders, 2014, Vol. 1, No. 1, 55-57
Available online at http://pubs.sciepub.com/ijhd/1/1/11

DOI:10.12691/ijhd-1-1-11

Effect of G6PD Status in Malaria Infected Individuals on Haematological Parameters

Esan A.J*

Department of Haematology and Blood Transfusion Science**,** Federal Teaching Hospital, Ido-Ekiti, Nigeria
*Corresponding author: ayodelejacob4u@gmail.com

Received November 24, 2014; Revised December 23, 2014; Accepted December 30, 2014

Abstract The aim of this study was to determine the effect of G6PD status no haematological parameters in Plasmodium falciparum malaria infected and non-malaria infected individuals. The study was conducted at Federal Teaching Hospital, Ido-Ekiti, Nigeria. Malaria infected adult individuals; presented with signs and symptoms of malaria infection was used for the study. Two hundred and two blood samples were collected twice from the same malaria infected individuals; grouped as pre-treatment and post anti-malaria drug treatment. One hundred and two blood samples from apparently healthy individuals were collected for control; 5 ml of blood sample was collected and dispensed into di-potassium ethylenediaminetetracetic acid (K2EDTA) vaccutainer bottles for haemoglobin concentration, packed cell volume and red cell indices were analysed using haematology analyser, thick blood film was made and stained with Giemsa's staining technique for malaria parasite detection, the procedure was described by Monica Cheesbrough. Glucose-6-Phosphate dehydrogenase (G-6-PD) was performed using methaemoglobin reduction method within 6 hours of sample collection. The procedure was as described by Dacie and Lewis. Data obtained was analysed using SPSS version 16. The result of this present study showed that, the mean±SD of Hb, PCV, MCV and MCH were significantly ($P<0.05$) lower in G6PD normal compared to G6PD deficient in pre and post anti-malaria drug treatment while mean±SD of RBC, Hb and PCV were significantly ($P<0.05$) higher in G6PD normal compared to G6PD deficient in control subjects. This present study showed that malaria attack in G6PD normal was observed more intensive compared to G6PD deficient.

Keywords: *malaria parasite, G6PD status and haematological parameters*

Cite This Article: Esan A.J, "Effect of G6PD Status in Malaria Infected Individuals on Haematological Parameters." *International Journal of Hematological Disorders*, vol. 1, no. 1 (2014): 55-57. doi: 10.12691/ijhd-1-1-11.

1. Introduction

Different G6PD deficiency variants have different propensities to cause haemolytic anaemia on exposure to oxidant drugs. Knowledge of the different biochemical and molecular variants of G6PD deficiency is important not only between people in different continents but even between people in different regions of the same country. G6PD deficiency produces a wide spectrum of clinical disorders, whose observable differences can be due to different oxidant drugs, different infections, and even different age groups. G6PD enzyme disorder was discovered in the 1950s when it was found that in some people administration of an anti-malaria drug like premaquine results in hemolytic anaemia. Most of these individuals are otherwise asymptomatic. Similar sort of responses had been reported in cases of a few other drugs, favism and in case of some infections. The link between glucose-6-phosphate dehydrogenase (G6PD) deficiency and malaria is further bolstered by the observation of the global distribution of glucose-6-phosphate dehydrogenase deficiency being parallel with that of malaria. In Africa, the prevalence of G6PD deficiency varied, about 28.0% in Nigeria [1]. This observation led to the suggestion that G6PD deficiency has protective advantage as a defense against malaria. Some studies have shown that *P. falciparum parasite* densities are lower in G6PD deficient individuals than those with normal [2]. A previous study by Awah and Uzoegwu, [3] showed that less severe clinical malaria symptoms were observed more in G6PD deficient when compared to G6PD normal subjects during malaria attack. Therefore inheriting genetic G6PD deficient disorders reduces malaria anaemia, parasitaemia and severe malaria symptoms. Hence, G6PD deficiency provides a natural protection against malaria infection. It has been suggested by Beutler, [4] that although malaria is a disease of high morbidity and mortality, it may, nevertheless, contribute to the balanced polymorphism and relatively high frequency of G6PD deficiency, because of the protective advantage as a defense against malaria. For this reason, and because the first observed incidence of drug related haemolysis occurred after the administration of the anti-malaria drug, many studies on G6PD deficiency are related to malaria. Although any one of a large number of oxidant drugs will cause haemolysis in G6PD deficient patients, because of its wide usage in malaria treatment, remains an important cause of haemolytic anaemia in clinical practice. The aim of this

study was to determine the effect of G_6PD status no haematological parameters in *Plasmodium falciparum* malaria infected and non-malaria infected individuals.

2. Materials and Methods

2.1. Subjects and Study Design

This was a cross sectional study conducted at Federal Teaching Hospital, Ido-Ekiti, Nigeria; between November 2012 and March 2013. *Plasmodium falciparum* malaria infected adult individuals; presented with signs and symptoms of malaria infection without evidence of haemolysis were selected for the study. Two hundred and two (202) blood samples were collected (5 ml) twice from the each malaria infected adult individuals; grouped as pre-treatment and post anti-malaria drug treatment. One hundred and two (102) blood samples from apparently healthy non malaria infected adult individuals were selected for control. Patient's consent was sort for through an informed consent form; also ethical approval was obtained from the hospital. Structured questionnaire was used to obtained demographic characteristic and other relevant information for the study.

2.2. Sample Collection

Five millimeters (5 ml) of blood sample was collected from each subject on the first day of visiting hospital as baseline sample after the patient has been clinically diagnosis for malaria infection, another 5 ml of blood sample was collected on the second or third day after taking anti-malaria drugs; blood sample collected was dispensed into di-potassium ethylenediaminetetracetic acid (K_2EDTA) vaccutainer bottles.

3. Methodology

Five millimetres (5 ml) of blood sample dispensed into di-potassium ethylenediaminetetracetic acid (K_2EDTA) vaccutainer bottles were used for haemoglobin concentration, packed cell volume and red blood cell indices analysed using haematology analyser (sysmex automated haematology analyser model kx-21n, manufactured by sysmex co-operation kobe, Japan). Thick blood film was made and stained with Giemsa's staining technique for malaria parasite detection; observed under microscopy using x100 objective lenses, the procedure was described by Monica Cheesbrough, [5]. Glucose-6-Phosphate dehydrogenase (G_6PD) was performed using methaemoglobin reduction method within 6 hours of sample collection. The procedure was as described by Dacie and Lewis [6].

4. Statistical Analysis

Data obtained were analyzed for mean and standard deviation; significant test was done by student t-test. Level of significance was considered as <0.05.

5. Results

Table 1 show mean ±SD of Hb, PCV and red blood cell indices on G_6PD status in pre, post anti-malaria drug treatment and control. The mean ± SD of RBC, Hb, PCV, MCV, MCH and MCHC 4.40 ± 0.58, 11.73 ± 2.06, 35.28 ± 6.15, 78.09 ± 6.29, 27.51 ± 2.94 and 31.62 ± 1.50 respectively in pre treatment G6PD normal were significantly (P<0.05) lower compared to 4.50±0.34, 11.91±1.55, 35.79 ±4.66, 79.73 ±4.78, 28.31 ±1.94, and 32.37 ±1.24 respectively in pre treatment G6PD deficient. Also, the mean ± SD of RDW 14.14 ±2.06 in pre treatment G6PD normal was higher compared to 13.69±2.04 in pre treatment G6PD deficient. However, the mean ± SD of RBC 4.19 ± 0.57 in post treatment G6PD normal was significantly (P<0.05) higher compared to 4.50 ±0.34 in post treatment G6PD deficient. Also, the mean ± SD of Hb, PCV, MCV, and MCH 10.86 ± 2.33, 32.45 ± 6.89, 75.94 ± 6.30 and 26.20 ± 3.06 respectively in post treatment G6PD normal were significantly (P<0.05) lower compared to 10.96 ± 1.67, 32.95 ± 5.02, 77.27 ± 4.48 and 26.84 ± 2.00 respectively in post treatment G6PD deficient. Also, the mean ± SD of MCHC 30.80 ± 1.48 in post treatment G6PD normal was lower compared to 31.51 ±1.34 in post treatment G6PD deficient; hence, the mean ±SD of RDW 13.50 ± 2.06 in post treatment G6PD normal was higher compared to 12.92 ± 1.86 in post treatment G6PD deficient. Moreover, mean ± SD of RBC, Hb and PCV 4.75 ± 0.59, 13.99 ± 1.15 and 42.10 ± 3.27 respectively in control G6PD normal were significantly (P<0.05) higher compared to 3.78 ± 0.28, 11.67 ± 0.54 and 35.29 ± 1.77 in control G6PD deficient; also mean ± SD of MCH 29.29 ± 1.05 in control G6PD normal was significantly (P<0.05) lower compared to mean ± SD of MCH 29.48 ± 0.93 in control G6PD normal was lower compared to 83.77 ± 2.65 in control G6PD deficient Hence, the mean ± SD of MCHC and RDW 32.63 ± 1.39 and 13.42 ± 1.47 respectively in control G6PD normal were higher compared to 32.51 ± 1.50 and 13.06 ± 1.02 in control G6PD deficient.

Table 1. Means ± Sd Of Hb, Pcv And Rbc Indices on G_6pd Status in Pre Treatment, Post-Antimalaria Drug Treatment in Malaria Infected Subjects and Control

Groups	Pre Treatment			Post Treatment			Control		
Parameters	Normal (N=148)	Deficient (N=54)	P value	Normal (N=148)	Deficient (N=54)	p. value	Normal (N=91)	Deficient (N=11)	p. value
RBCX$10^{9/L}$	4.40±0.58	4.50±0.34	0.00*	4.19±0.57	4.07±0.37	0.00*	4.75±0.59	3.78±0.28	0.00*
HBg/dL	11.73±2.06	11.91±1.55	0.00*	10.86±2.33	10.96±1.67	0.00*	13.99±1.15	11.67±0.54	0.00*
PCV%	35.28±6.15	35.79±4.66	0.01*	32.45±6.89	32.95±5.02	0.00*	42.10±3.27	35.29±1.77	0.00*
MCVFl	78.09±6.29	79.73±4.78	0.01*	75.94±6.30	77.27±4.48	0.00*	81.66±11.58	83.77±2.65	0.43
MCHPg	27.51±2.94	28.31±1.94	0.00*	26.20±3.06	26.84±2.00	0.00*	29.29±1.05	29.48±0.93	0.05*
MCHCg/Dl	31.62±1.50	32.37±1.24	0.05*	30.80±1.48	31.51±1.34	0.15	32.63±1.39	32.51±1.50	0.64
RDW%	14.14±2.06	13.69±2.04	0.80	13.50±2.06	12.92±1.86	0.52	13.42±1.47	13.06±1.02	0.10

P<0.05 Significance, P>0.05 no Significant

6. Discussion

Out of 202 *P. falciparum* malaria patients used in the study, 148 (73.3%) were G6PD normal and 54 (26.7%) were G6PD deficient, among the non malaria infected group, 91 (89.2%) were G6PD normal and 11 (10.8%) were G6PD deficient. Prevalence of G6PD status in this present study was similar to Francis *et al*., 2012 reported that out of four (400) individuals screened for this G6PD deficiency, 347 (86.75%) had normal G6PD levels and 53 (13.25%) were G6PD deficient of which 36 (9.0%) were heterozygous and 17 (4.25%) were homozygous. The high frequency of G6PD deficiency in the study population corroborates the role malaria play in the distribution of G6PD genes in most malaria endemic areas in the world [7]. G6PD deficiency was associated with significant reduction in the risk of severe malaria for both G6PD deficient females and males in accordance with the reports of Awah and Uzoegwu [3]. G6PD deficient parasitised erythrocytes could therefore have been phagocytosised earlier thereby destroying the malaria parasite. Haematology parameters on G6PD status in patients infected with *plasmodium falciparum* showed that mean value of red blood cell, haemoglobin concentration, packed cell volume and red cell indices (MCV, MCH, MCHC) were significantly lower in G6PD normal compared to G6PD deficient in pre and post anti-malaria drug treatment. The study supported the fact that G6PD deficiency has protective advantage as a defense against malaria, it provide a natural protection against malaria infection. Some studies have shown that *P. falciparum parasite* densities are lower in G6PD deficient individuals than those with normal [2]. Mean value of red cell distribution width (RDW) was observed higher in G6PD normal compared to G6PD deficient in pre treatment and post anti-malaria drug treatment; result obtained in the present study was similar to the findings of Francis and Pete [8] stated that G6PD deficient had significantly higher Hb concentration, PCV, RBC count than the G6PD non-deficient. Supporting this study, less severe clinical malaria symptoms were observed more in G6PD deficient when compared to G6PD normal subjects during malaria attack [3,8]. However, in post anti-malaria drug treatment, haematological parameters were observed decrease; this was due to the effect of anti-malaria drug used during treatment; causing haemolytic anaemia due to clearance of parasitized erythrocytes and some of unparasitized erythrocytes in the circulation leading to haemoglobin degradation and manifestation of anaemia. Moreover, haematological parameters in control subjects were within normal range; comparing pre anti-malaria drug treatment with control values showed the extent of infection also comparing post anti-malaria drug treatment with control values showed the extent of recovery from malaria infection.

7. Conclusion

This present study showed that malaria attack in G6PD normal was observed more intensive compared to G6PD deficient; this study support the fact that G6PD deficient has genetic resistance to malaria infection.

References

[1] Beutler E, Duparc S. Glucose-6-Phosphate Dehydrogenase Deficiency and Antimalarial Drug Development. *Am J Trop Med Hyg*; (2007); 77 (4):779-789.

[2] Ruwende C, Khoo SC, Snow RW, Yates SN, Kwiatkowski D, Gupta S. Natural selection of hemi-and heterozygotes for G6PD deficiency in Africa by resistance to severe malaria. *Nature;* (1995); 376: 246-249.

[3] Awah, F.M. & Uzoegwa, P.N. Influence of sickle heterozygous status and glucose-6- phosphate dehydrogenase deficiency on the clinico-haematological profile of Plasmodium falciparum infected children. *Biochemistri* (2006); 18, 89-97.

[4] Beutler E. Glucose-6-phosphate dehydrogenase deficiency: a historical perspective. *Blood;* (2008); 111: 16-24.

[5] Monica Cheesbrough. Discrete Laboratory Practice in Tropical Countries Part 1, Cambridge Second Editions. Published by Press Syndicate of the University of Cambridge (2005), chp. 5, page 247-258.

[6] Dacie and Lewis. Pratical Haematology 10th ed. Churchill Livingstone Elsevier (2006)chap. 10: 219-220

[7] El-Hazmi MAF, Warsy AS. The Frequency of Glucose-6-Phosphate Dehydrogenase Phenotypes and Sickle Cell Genes in Al-Qatif Oasis. *Annals of Saudi Med*. (1994); 14 (6): 491-494.

[8] Francis M. AWAH and Pete N. UZOEGWU. Influence of sickle heterozygous status and glucose-6-phosphate dehydrogenase deficiency on the clinico-haematolgoical profile of *Plasmodium falciparum*-infected children *Biokemistri* (2006); 18 (2):89-97.