Assessment of Haemoglobin Genotype Variants in Malaria Infected Patients of Two Government Hospitals in Plateau State, North Central Nigeria

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ABSTRACT Genetic factors play a key role in determining resistance and susceptibility to malaria infection. Therefore, a study to assess haemoglobin genotype variants in malaria infected patients of the General Outdoor Patients Departments (GOPD) of Jos University Teaching Hospital and Plateau State Specialist Hospital, Jos, Plateau State was carried out. Thick and thin film were used for the diagnosis of malaria infection. The genotypes were determined by Standard Operating Procedure for electrophoreses. 745 samples were examined from both hospitals, 246 (33.0%) were diagnosed positive while 499 (67.0%) were negative. There was a significant difference in malaria infection in relation to genotypes. HbAA genotype were the most infected with malaria parasites followed by HbAS genotype and the least was HbSS genotype. There was no significant difference in malaria infection in relation to gender. Females were more infected with malaria parasites compared to males. Malaria infection in relation to age groups and genotypes showed a significant difference. Age group 16 to 20 and ≥46 had the highest infection rate. There was a significant difference in trophozoite stage in relation to genotypes. Out of 246 infected patients, 244 (99.2%) were diagnosed at the trophozoite stage, while 2 (0.81%) with the gametocyte stage. There was a significant difference in malaria infection in relation to *Plasmodium* species. 245 (99.56%) were infected with *Plasmodium falciparum* while 1 (0.41%) were infected with *P. malariae.* There was a significant difference in *P. falciparum* infection in relation to genotypes. *P. falciparum* infected more HbAA compared to HbAS and HbSS. This study shows that all haemoglobin genotype variants were susceptible to malaria infection. Therefore, there is a need for government to use media to broadcast the importance of haemoglobin genotype test for each and every individual and make it free for effective treatment of malaria infection.

KEYWORDS: Haemoglobin, Genotype, Malaria, *Anopheles* mosquito, Patients
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INTRODUCTION

Malaria is a life-threatening blood disease caused by *Plasmodium* parasites that are transmitted to humans by female *Anopheles* mosquito (WHO, 2013). The four malaria species that produce human disease are *Plasmodium vivax, P. falciparum, P. malariae,* and *P. ovale*. *P. vivax* is mostly prevalent in the temperate regions (Rasheed *et al*., 2014), *P. falciparum*, the most lethal strain, is the most prevalent species throughout the tropics and subtropics (Institute of Medicine of the National Academies, 2004). *P. malariae* is patchily present over the same range as *P. falciparum*. *P. ovale* is found in tropical Africa (Institute of Medicine of the National Academies, 2004). The effect of malaria infection is most times characterized with particular blood genotype (Patel *et al*., 2017). HbAs is widely known to confer significant protection from severe and uncomplicated malaria (Chinawa *et al*., 2015). Erythrocytes containing HBS or HBC may impede parasite growth and replication relative to normal red cells when subject to low oxygen tensions (Thomas, 2011). The geographical relationship between the transmission intensity of malaria and associated haemoglobin genotype variants remains a burden. Therefore, this study is to

assess the haemoglobin genotype variants in malaria infected patients attending Jos University Teaching Hospital and Plateau State Specialist Hospital, Jos Plateau State.

MATERIALS AND METHODS

The Study Areas

Jos University Teaching Hospital (JUTH) is located on latitude 9° 54′0″N longitude 8° 57′34.7″E in Lamingo Jos East and Plateau State Specialist Hospital, Jos, Nigeria, is located between Latitude 8° 24′N and Longitude 8°32′E in Jos North.

Ethical Clearance

The study protocol was submitted and approved by the Jos University Teaching Hospital (JUTH/DCS/ADM/127/XXV/308) and Plateau State Specialist Hospital (NHREC/09/23/2010b) ethical committee, before the kick-start of the study.

Sampling period

Samples collection was carried out between the periods of June 2017 to October 2018.

Study Population

The population size in respect to this study comprised patients of all ages and sex diagnosed with malaria parasites, attending JUTH and Plateau State Specialist Hospital. The population was found at the General Outdoor Patients Department (GOPD).

Sample Size

A total of 499 blood samples of the population was required using the formula recommended by Cochran (1963).

Sample Method

Patients of the General Outpatient Department (GOPD) were sampled for onward enrolment into the study.

Exclusion criteria: Patients whose blood were not tested for malaria.

Inclusion criteria: Patients who were febrile and tested for malaria using thick and thin blood smear and microscopy.

Sample Collection

The blood samples brought from the GOPD in an EDTA bottle to prevent clotting, for the diagnosis of malaria parasites were collected.

Clinical and Laboratory Diagnosis of Malaria Parasites

Immediately, thick and thin blood films were prepared by making a blood smear with diameter of thick smear 12mm, amount of blood for thick smear was 6µl, area covered by the thick blood smear was 113.14mm^2, Amount of blood for thin smear was 2µl. The blood films were allowed to air dry, placed on a staining rack and flooded with approximately 1ml Giemsa stain for 10 minutes, it was then allowed to stand for 30minutes, washed with water and allowed to air dry. The films were examined under an oil immersion microscope objective (100x). Parasitaemia was determined for febrile patients who tested positive for *Plasmodium*

species by counting the number of parasites (asexual forms only) against 200 white blood cells (WBC). Counting was done using hand tally counters. The number of parasites per microliter of blood was calculated. The genotypes were determined by Standard Operating Procedure for electrophoreses.

Statistical Analysis

Data obtained were expressed in simple percentages and analyzed using R Console software (Version 3.2.2). Pearson's Chi-square test was used to compare the proportion of malaria infection in relation to genotypes, age groups, gender, infective stages as well as *Plasmodium* species. P-values < 0.05 were considered statistically significant**.**

RESULTS AND DISCUSSION

The comparison of malaria infection on haemoglobin genotype variants of infected patients attending Plateau Hospital in relation to age, showed a significant difference ($\chi^2 = 131.68$, df = 2, P < 0.0001) as shown in Table 1. Out of 163 infected patients, 27 individuals representing 16.56% of the age group ≥46 with HbAA genotype represented with 18(15.13%) were the most infected with malaria parasites, followed by age group 16-20 with HbAA genotypes represented with 18 (15.13%) and the least was age group 1-5 with HbAA genotypes represented with 4 (3.36) as shown in Table 1. This may be as a result of gradual decrease of immunity against malaria infection with increasing age upon repeated infection. This contradict with the result of Coker *et al*. (2001) where prevalence of malaria parasitaemia was highest (50.7%) in 16-20 years age group and declined in older groups. The study also established that haemoglobin genotype AA is more susceptible to malaria infection than all other hemoglobin variants. This is because HbAA has normal haemoglobin (both in structure and in quantity) with a higher oxygen binding capacity, thereby enhancing the parasites replication process as reported by Akanbi *et al*. (2010). The findings of this study are slightly lower, in the case of HbAA and HbSS, than previous research (Opara *et al*., 2006) that reported the degree of susceptibility of different genotypes to malaria to be AA (92.3%), AS (5.1%), and SS (2.6%). HbAS is widely known to confer significant protection from severe and uncomplicated malaria (Bougouma *et al*., 2012). However, the protective effect is not applied to people with sickle cell disease who are more vulnerable to malaria.

In this study, the comparison of malaria infection on haemoglobin genotype variants of infected patients attending Plateau Hospital and JUTH showed a significant difference in relation to age. This is in accordance to Schwartz *et al.* (2001), who recorded that different immune responses related to age may be responsible for the different outcomes. The age groups 16-20 and ≥46 with HbAA genotypes respectively were the most infected with malaria parasites, this correlates with the research carried out by Charchuk *et al*. (2015) that reported 68% malaria prevalence in age 5-19 and attributed that to under-nutrition which led to low immunity. The high number of infections recorded at age 16-20 could be because of immunological and hormonal factors (Lalloo *et al*., 2016). In the older patients the prevalence rate can be associated with lowering of immunity with age and the seeking of medical attention only when it is affordable or in severe cases. Irregular prevalence was observed in this study, which is in contrast with the findings of Nwokolo (2017) that reported a progressive increase in prevalence as the age increases. The study is in accordance with the research of Nebe *et al*. (2002) who recorded predominant infection rate in adolescents.

Most of the patients had mild (+) parasite load of 73 (20.4%) and moderate (++) load with 66 (18.4%) whereas severe (+++) parasite load was the least with 24 (6.7%) as displayed in Table 1. This may be attributed to the time this study was carried out which was the dry and cold season with little or no favorable weather condition for the malaria vector and thus transmission of malaria in the study area was low compared to raining season.

The comparison of malaria infection on haemoglobin genotype variants of infected patients attending JUTH in relation to age showed a significant difference ($\chi^2 = 73.542$, df = 2, $P < 0.0001$) as shown in Table2. Out of 83 infected patients, 14 individuals representing 16.87% of the age groups 1-5 and ≥46 with HbAA genotypes represented with 10(15.87) and 14(22.22) respectively were the most infected with malaria parasites, followed by age group 11-15 and 26-30 with HbAA genotypes represented with 8(12.70%) and the least was age group 1-5 with HbAA genotypes represented with 1(1.59) as shown in Table 2. Most patients had moderate (++) parasite load 39 (27.7%), followed by mild (+) parasite load with 29 (20.6%) and those with severe (+++) parasite load were the least with 15 (10.6%) as showed in Table 2. This may have been influenced by division of labour, leisure patterns, and sleeping arrangements leading to different patterns of mosquito exposure for men and women. However, females with HbAA, HbAS, and HbSS genotypes were more infected with malaria parasites than males with HbAA, HbAS and HbSS genotypes respectively. This may be related to the habit of exposing their body and thus making it easy for mosquito parasitic activity (Akanbi *et al*., 2010).

Malaria infection on haemoglobin genotype variants of infected patients attending Plateau Hospital in relation to gender showed a significant difference ($\chi^2 = 74.343$, df = 2, $P < 0.0001$). The breakdown of result revealed that females with HbAA, HbAS, and HbSS genotypes were more infected with malaria parasites than males with HbAA, HbAS and HbSS genotypes respectively as shown in Table 3. Similarly, more females were found to be infected with mild, moderate and severe parasitic load compared with males (Table 3). However, the disease prevalence was slightly higher in females with 46% prevalence rate than males 44%. This is in accordance with the work of Nwokolo (2017) who reported more females being infected than males, but contradicts Dawaki *et al*. (2016) who reported prevalence rate to be higher in males than in females. The study also contradicts that of Williams *et al*. (2005) where males had a higher malaria parasite infection rate than females.

The high prevalence rate of malaria infection reported in this study as relating to female can be associated with the role female gender play in the society, women who get up before dawn to perform household chores are exposed to mosquitoes and consequently to malaria infection. Similarly, more females were found to be infected with mild, moderate and severe parasitic load compared with males in the two hospitals. This can be associated with their socio-economical behavior as it relates to poor hygiene, exposure of the body, delay in treatment, severity of the illness at admission (coma, shock, high fever etc.).

On the other hand, the comparison of malaria infection on haemoglobin genotype variants of infected patients attending JUTH in relation to gender showed a significant difference ($\chi^2 = 67.797$, df = 2, $P < 0.0001$) as shown in Table 4. The result also revealed that females with HbAA, HbAS, and HbSS genotypes were more infected with malaria parasites than males with HbAA, HbAS and HbSS genotypes respectively as shown in Table 4. Similarly, more females were found also found to be infected with mild, moderate and severe parasitic load compared with males (Table 4). Most of the patients had mild (+) parasite load, 73 (44.8%), followed by moderate (++) parasitic load, 66 (40.5%) and those with severe (+++) parasite load were the least

24 (14.7%). The high rate of parasite load in mild and moderate observed in this study could be due to the season the study was carried out, that is within the months of October and November when there was very little or no rain, therefore absence of numerous mosquito breeding sites and thus reduction in their parasitic activity. The cases of malaria can be attributed to stress, irrigation, congestion and man-made water bodies that create breeding sites for malaria vectors.

The results of this study show 58.9% patients were infected with malaria parasites, of which the HbAA genotype were the most infected, 75.9%, followed by HbAS genotype, 22.9% and the least was HbSS genotype 1.2%. The normal daily production of red blood cells (RBC) in a healthy adult is about 0.25 mL/kg and the average lifespan of the cells is about 120 days (Liumbruno *et al.*, 2009). The lifespan of the normal haemoglobin (HbAA) provides adequate time for the multiplication and replication of malaria parasites which takes 48-72hours to replicate and the presence of iron which supplies the parasites nutrients hence increasing it surveillance, this explains the susceptibility of the hemoglobin AA.

There was no record of gametocyte stage in the patients attending Plateau Hospital but rather all 163 individuals infected were diagnosed at the trophozoite stage. The absence of gametocyte stage in the patients attending Plateau Hospital may be attributed to the time taken in the trophozoite stage (asexual stage). Asexual replication continues with repeated release of newly formed merozoites for over 1-3 days, resulting in thousands of parasite-infected cells in the blood stream. These blood stage parasites cause the illness and symptoms associated with malaria that can last for months if not treated (Miller *et al.*, 2002).

The comparison of malaria infection on haemoglobin genotype variants in relation to trophozoite stage in the patients attending Plateau Hospital showed a significant difference (χ^2 = 131.68, df = 2, P < 0.0001) as shown in Table 5. The comparison of malaria infection on haemoglobin genotype variants in relation to trophozoite stage showed a significant difference in the two hospitals. Trophozoite stage, referred to as the erythrocytic stage (blood infection) infect all blood types with variations in the parasite load. A high number of patients with HbAA genotypes were diagnosed with trophozoite stages of the infection, followed by HbAS and HbSS genotypes in the two hospitals. This may be associated with the very efficient splenic retention of such non-adherent infected RBCs which is expected to result in a slower rise of *P. falciparum* parasitaemia in sickle-cell trait carriers (Sedina*et al.*, 2016). Most patients in Plateau hospital had mild trophozoite load, followed by moderate and severe in contrast to patients in JUTH which were observed to have moderate trophozoite load, followed by mild and the severe. The study area may be a contributing factor, with little or no mosquito breeding sites. Another factor could be that the patients who attended Plateau Hospital sought for medical attention immediately symptoms emerged.

High numbers of patients with HbAA genotypes were diagnosed with trophozoite stages of the infection, followed by HbAS and HbSS genotypes (Table 5). There was a significant difference (χ^2 = 15.901, df = 2, P = 0.0003524) in relation to trophozoite stage load. Most patients had mild trophozoite load, followed by moderate trophozoite load and severe trophozoite load as the least (Table 5).

The comparison of malaria infection on haemoglobin genotype variants in relation to infective stages in the patients attending JUTH showed a significant difference (χ^2 = 75.193, df = 1, P < 0.0001). Two patients with HbAA genotypes were diagnosed with gametocyte stage while trophozoite stage was recorded in 81 patients with HbAA genotypes having the highest

followed by HbAS and HbSS genotypes (Table 6). There was a significant difference ($\chi^2 = 15.753$, df = 2, P = 0.0003795) in relation to trophozoite stage load in infected patients attending JUTH (Table 6). Most patients were observed to have moderate trophozoite load, followed by mild trophozoite load and the severe trophozoite load was the least (Table 6). There was re-occurrence of malaria infection in patients around November even after treatment which may be attributed to the inefficiency of anti-malarial drugs to clear the gametocytes stage of infection by *Plasmodium* sp. This helps in completing the malaria cycle and thus continuous transmission of malaria parasites. This finding correlates with the research carried out by Bousema and Draukeley (2011). Gametocytes are cleared relatively slowly from the blood so they accumulate with respect to asexual parasites and can predominate in chronic infections. The gametocytes of *P. falciparum* malaria are relatively insensitive to most anti-malarial drugs whereas the gametocytes of the other human malaria parasites are considered as drug sensitive as their asexual counterparts.

The comparison of malaria infection on haemoglobin genotype variants in relation to *Plasmodium* species showed a significant difference ($\chi^2 = 295.23$, df = 3, P < 0.0001) in patients attending Plateau Hospital as shown in Table 7. The result revealed that *P. falciparum* infected all haemoglobin genotypes but mostly the HbAA genotypes followed by HbAS and the least was HbSS genotypes. *P. falciparum* load in the patients were mostly mild (+) followed by moderate (++) and severe (+++) as shown in Table 7. *P. malariae*only infected patients with HbAA genotypes with moderate (++) load (Table 7). Similarly, the comparison of malaria infection on haemoglobin genotype variants in relation to *Plasmodium* species showed a significant difference ($\chi^2 = 46.81$, df = 2, P < 0.0001) in patients attending JUTH as shown in Table 8. The breakdown of the result showed that *P. falciparum* infected all haemoglobin genotypes but mostly the HbAA genotypes followed by HbAS and the least was HbSS genotypes. The comparison of malaria infection on haemoglobin genotype variants in relation to *Plasmodium* species showed a significant difference in the two hospitals. There are different species of *Plasmodium* responsible for malaria disease and *P. falciparum* is known to causes 85% of malaria cases with an infection rate that occurs principally in tropical areas worldwide (Fadel, 2014). *P. falciparum* was found to infect all haemoglobin genotypes but mostly HbAA genotypes in patients of the two hospitals. The above result is in accordance with the findings of LaMonte *et al*. (2012) which reported that individuals have three microRNAs (miR-223, miR-451, let-7i) that are effective in reducing *P. falciparum* growth and replication, and the latter two are increased in HbAS and HbSS individuals when compared to HbAA individuals. The microRNAs are transformed into the parasite and are inserted into its mRNA in a method similar to trans-splicing or splice-leader trans-splicing, inhibiting translation and reducing *P. falciparum* growth. Therefore, HbAS and HbSS individuals have a genetic advantage over HbAA individuals. Infection with *P. falciparum* is more serious than infections with other malarial species owing to the high frequency of severe complications associated with it (Mangal *et al*., 2017). Severe aneamia is the most common complication of *P. falciparum* infection (WHO, 2014). In this study, *P. falciparum* was responsible for the highest malaria infections in Plateau Specialist Hospital (Table 7). This is in accordance with the findings of Brooks *et al*. (2004), Tidi *et al*. (2013) and Sule *et al*. (2014). It could be due to the availability of mosquito vectors for the transmission of infection and suitable environmental conditions as well as within the mosquito for the multiplication of *P. falciparum*. This finding agrees with the research carried out by Abdul *et al*. (2014). The selection of blood type by *Plasmodium* species explains why there is no equal distribution with the different human species in malaria infection. *P. vivax* (Pv) infects the Duffy antigen that serves as the obligate trans-membrane receptor for infection of red blood cells by the parasite during the 1970s. *P. vivax* is not common in Africa since they rarely express the Duffy antigen and therefore are resistant to

its infection (Howes *et al*., 2015). *P. malariae* and *P. ovale* prefers leucocytes and young red blood cells than the old blood cells (Snow *et al*., 2005). However, there was a case of *P. malariae* in October which indicates that the weather was suitable for its parasitic activity and was detected in the blood type AA which could have been in its new form. *P. falciparum* infected all genotypes but mostly the AA genotypes, 118 (72.8%), followed by AS, 43 (26.5%) and the least infected was patient with SS genotype, 1 (0.6%).

P. falciparum load in the patients were mostly moderate (++) followed by mild (+) and severe (+++) as shown in Table 8. In this study *P. falciparum* load in the patients was mostly mild (+) followed by moderate (++) level and its severity (+++) was low. *P. malariae* only infected patients with AA genotype and its load was at moderate (++) level. Current epidemiological data overwhelmingly indicates that *P. falciparum* is the predominant malaria pathogen across most of sub-Saharan Africa (WHO, 2014). This correlates with the result in this study from Jos University Hospital in Plateau State that recorded *P. falciparum* as being responsible for 100% malaria infection. In this study, *P. falciparum* infected all genotypes but mostly the HbAA genotypes 63(75.9%), followed by the HbAS 19 (22.9%) and the least infected was patient with SS genotype 1(1.2%). *P. falciparum* load in the patients was mostly moderate (++) level followed by mild (+) level and its severity (+++) was low.

Table 1. Malaria Infection in General Outdoor Patients Department of Plateau Hospital in Relation to Age Groups and Genotypes

Age group	Parasite Load			No. Examined			No. Infected (%)			Total No. Examined	Total No. Infected (%)
	+	++	+++	AA	AS	SS	AA	AS	SS		
1-5	3	1	1	5	4	0	4(3.36)	1(2.33)	0(0)	9	**5(3.07)**
6-10	5	2	3	10	4	0	8(6.72)	2(4.65)	0(0)	14	**10(6.13)**
11-15	9	10	3	17	8	0	14(11.76)	8(18.60)	0(0)	25	**22(13.50)**
16-20	10	12	2	27	21	0	18(15.13)	6(13.95)	0(0)	48	**24(14.72)**
21-25	11	8	3	25	20	0	17(14.29)	5(11.63)	0(0)	45	**22(13.50)**
26-30	11	2	5	36	29	0	15(12.61)	3(6.98)	0(0)	65	**18(11.04)**
31-35	7	5	1	17	17	1	8(6.72)	4(9.30)	1(100)	35	**13(7.98)**
36-40	5	8	0	16	14	0	10(8.40)	3(6.98)	0(0)	30	**13(7.98)**
40-45	3	6	0	12	11	0	7(5.88)	2(4.65)	0(0)	23	**9(5.52)**
≥46	9	12	6	37	27	0	18(15.13)	9(20.93)	0(0)	64	**27(16.56)**
Total (%)	**73**	**66**	**24**	**202**	**155**	**1**	**119(100)**	**43(100)**	**1(100)**	**358**	**163(100)**

Table 2. Malaria Infection in Patients Visiting JUTH in Relation to Age Groups and Genotypes

Age group	Parasite Load			No. Examined			No. Infected (%)			Total No. Examined	Total No. Infected (%)
	+	++	+++	AA	AS	SS	AA	AS	SS		
1-5	0	1	1	2	3	1	1(1.59)	0(0)	1(100)	6	**2(2.41)**
6-10	4	8	2	10	6	0	10(15.87)	4(21.06)	0(0)	16	**14(16.87)**
11-15	4	6	0	12	5	0	8(12.70)	2(10.52)	0(0)	17	**10(12.05)**
16-20	2	6	0	6	10	0	5(7.94)	3(15.79)	0(0)	16	**8(9.64)**
21-25	3	2	3	8	5	0	7(11.11)	1(5.26)	0(0)	13	**8(9.64)**
26-30	4	1	4	11	3	0	8(12.70)	1(5.26)	0(0)	14	**9(10.84)**
31-35	4	2	1	7	6	0	3(4.76)	4(21.06)	0(0)	13	**7(8.43)**
36-40	3	0	0	5	4	0	2(3.18)	1(5.26)	0(0)	9	**3(3.61)**
40-45	3	4	1	8	4	0	5(7.93)	3(15.79)	0(0)	12	**8(9.64)**
≥46	2	9	3	20	5	0	14(22.22)	0(0)	0(0)	25	**14(16.87)**
Total (%)	**29**	**39**	**15**	**89**	**51**	**1**	**63(100)**	**19(100)**	**1(100)**	**141**	**83(100)**

Table 3. Malaria Infection in Patients Visiting Plateau Hospital in Relation to Gender and Genotypes

Gender	Parasite Load			No. Examined			No. Infected (%)			Total No. Examined	Total No. Infected (%)
	+	++	+++	AA	AS	SS	AA	AS	SS		
Female	32	64	8	127	96	1	71(59.66)	32(74.42)	1(100)	224	**104(63.80)**
Male	43	10	6	75	59	0	48(40.34)	11(25.58)	0(0)	134	**59(36.20)**
Total (%)	**75**	**74**	**14**	**202**	**155**	**1**	**119(100)**	**43(100)**	**1(100)**	**358**	**163(100)**

Table 4. Malaria Infection in Patients Visiting JUTH in relation to Gender and Genotypes

Gender	Parasite Load			No. Examined			No. Infected (%)			Total Examined	Total No. Infected (%)
	+	++	+++	AA	AS	SS	AA	AS	SS		
Female	27	22	10	61	30	1	50(71.43)	8(61.54)	1(100)	92	**59(70.24)**
Male	16	6	3	28	21	0	20(28.57)	5(38.46)	0(0)	49	**25(29.76)**
Total (%)	**45**	**28**	**13**	**89**	**51**	**1**	**70(100)**	**13(100)**	**1(100)**	**141**	**84(100)**

Table 5. Malaria Infection in Patients Visiting Plateau Hospital in Relation to Infective Stages and Genotypes

Infective Stage	Parasite Load			No. Infected (%)			Total No. Examined (%)
	+	++	+++	AA	AS	SS	
Gametocyte	0	0	0	0(0)	0(0)	0(0)	**0(0)**
Trophozoite	73	66	24	119 (100)	43(100)	1(100)	**163(100)**
Total (%)				**119 (100)**	**43(100)**	**1(100)**	**163(100)**

Table 6. Malaria Infection in Patients Visiting JUTH in Relation to Infective Stages and Genotypes

Infective Stage	Parasite Load			No. Infected (%)			Total No. Examined (%)
	+	++	+++	AA	AS	SS	
Gametocyte	0	0	2	2(3.2)	0(0)	0(0)	**2(2.41)**
Trophozoite	29	39	13	61(96.8)	19(100)	1(100)	**81(97.59)**
Total (%)				**63(100)**	**43(100)**	**1(100)**	**83(100)**

Table 7. Malaria Infection in Patients Visiting Plateau Hospital in Relation to *Plasmodium* species and Genotypes

Parasites	Parasite Load			No. Infected (%)			Total No. Examined (%)
	+	++	+++	AA	AS	SS	
P. falciparum	73	65	24	118(99.16)	43(100)	1(100)	162(99.39)
P. malariae	0	1	0	1(0.84)	0(0)	0(0)	1(0.61)
P. ovale	0	0	0	0(0)	0(0)	0(0)	0(0)
P. vivax	0	0	0	0(0)	0(0)	0(0)	0(0)
Total (%)	73	66	24	**119(100)**	**43(100)**	**1(100)**	**163(100)**

Table 8. Malaria Infection in Patients Visiting JUTH in relation to *Plasmodium* species and Genotypes

Parasites	Parasite Load			No. Infected (%)			Total No. Examined (%)
	+	++	+++	AA	AS	SS	
P. falciparum	29	40	14	63 (100)	19(100)	1(100)	83(100)
P. malariae	0	0	0	0(0)	0(0)	0(0)	0(0)
P. ovale	0	0	0	0(0)	0(0)	0(0)	0(0)
P. vivax	0	0	0	0(0)	0(0)	0(0)	0(0)
Total (%)	29	40	14	**63(100)**	**19(100)**	**1(100)**	**83(100)**

CONCLUSION

The high prevalence of malaria parasites observed in haemoglobin genotype variant AA shows that they are susceptible to malaria infection which can be attributed to the presence of normal hemoglobin both in structure and quantity. As it is rich in iron, it enhances the replication of malaria parasites, with predominant infection rate in adolescents and old aged group. Females were rated with the highest number of infections in this study, which relates to the habit of exposing their body and thus making it easy for mosquito parasitic activity. In this light, Government and Non-Governmental Organizations should enlighten the public to know their blood genotype which brings them to the knowledge of their status and susceptibility to malaria parasitaemia. The World

health Organization and other health sectors should make effort in improving the efficacy of drugs to treat the gametocyte stage and at an affordable price, if malaria is to be knocked out with time. The public should also be encouraged on the importance of sanitizing their environment and the use of Insecticide Treated Nets (ITNs). Self-medication must be strongly discouraged and patients should be promptly treated immediately after confirmation of parasitaemia.

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