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Plasmodium falciparum parasite density in symptomatic and asymptomatic malaria among residents of Abuja, Nigeria

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ABSTRACT

Objective: *Plasmodium falciparum* is prevalent in sub-Saharan Africa and constitutes the greatest public health burden relative to the four species of the parasite that infect humans. World Health Organization (WHO) uses parasite density to describe malaria severity, where 250,000 parasites/μL and 500 parasites/μL are set as the cutoff points for hyperparasitemia in low and high *P. falciparum* endemic regions, respectively. This classification may not be universal as different factors influence parasite density. This study evaluated the parasite densities in symptomatic and asymptomatic persons in Abuja with a view to understanding host tolerance to *P. falciparum* in an endemic condition.

Materials and Methods: This study was carried out using 246 blood samples each from symptomatic and asymptomatic volunteers, from two area councils in Abuja following WHO standard methods for Malaria microscopy.

Results: The result revealed symptomatic and asymptomatic groups with median axillary temperature that differed significantly ($P = 0.012$), parasite densities both lower that WHO cut off mark, and did not differ significantly between symptomatic and asymptomatic, among different age groups and catchment areas ($P > 0.05$).

Conclusion: Parasite density does not differ in symptomatic and asymptomatic subjects in *P. falciparum*, which is endemic in Abuja and is lower than WHO set values.

Keywords: asymptomatic, immunity, parasitemia, parasite density, symptomatic.

INTRODUCTION

Plasmodium falciparum is one of the five species of plasmodium that infect humans to cause malaria. Like other species, it is transmitted through the bite of infected female anopheline mosquito during a blood meal.[1] Malaria is one of the major public health concerns in many parts of the world with total global morbidity and mortality of >80% and 90% mortality in sub-Saharan Africa, including Nigeria.[2]

Amongst the five *Plasmodium* species, *P. falciparum* exerts the severest burden and is very common in most of sub-Saharan Africa and is the most prevalent species in Nigeria.[1] *P. falciparum* has evolved different pathogenic armories and efficient adaptive strategies that enhance its pathogenesis and survival, thus supporting its endemicity in different parts of the world. In general, malaria is an immunological disease that manifests from the immunologic "warfare" between the host immune system and the parasite upon the expression of the plasmodial antigens and toxins in the host system.[3] These immune responses involve interplay between innate (cellular) and acquired (humoral) arms of immune response. The cellular activities aid in limiting the parasite proliferation, and stimulating humoral responses,[4] with consequent expression of pro-inflammatory cytokines. Although the immunological responses are aimed at controlling the invading parasites, excess production of pro-inflammatory cytokines leads to an array of malaria symptoms, which are influenced by host genetic factors, age, exposure to parasite and parasite endemicity/transmission, among other factors.[5] Nonimmune

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persons, persons with suppressed immune system, and immune persons returning to *P. falciparum* endemic region after a protracted period are worst hit by *P. falciparum* disease. Severity of the disease varies with the immune status of an individual ranging from mild levels of pyrexia to complications that could present cerebral malaria, acute renal failure, acute malarial hepatitis, hypoglycemia, hyperpyrexia, noncardiogenic pulmonary edema, adult respiratory distress syndrome, adrenal insufficiency-like syndrome, hyperparasitemia, Blackwater fever, cardiac arrhythmias, and gastrointestinal syndromes. [6] It therefore follows that the parasite density and progress of the disease will depend greatly on the immune status of the subject. *P. falciparum* infects red blood cells of all ages; as a result, it is associated with very high parasite density upon infection.[6] Severe *P. falciparum* infestation is usually associated with parasites of different growth stages and forms: early trophozoites, growing trophozoites, and mature trophozoites in circulating blood.

P. falciparum density shows a positive correlation with disease severity and mortality that it forms one of the WHO criteria for classifying the degree of severity of *P. falciparum* malaria.[7] In a work in India by Mangal *et al.*,[8] all uncomplicated *P. falciparum* malaria had parasite density less than 250,000 parasites/μL which according to WHO is low parasite density in a malaria endemic place; meanwhile all complicated *P. falciparum* cases were associated with parasite densities greater than 500,000 parasites/μL. The same work also showed that high *P. falciparum* density was associated with poor clinical outcomes and 58.33% mortality. In a similar manner, parasite density also affects antimalarial activity as it influences the parasite clearance time of the drugs and the possibility of recrudesce after treatment. According to Borrmann *et al.*,[9] *P. falciparum* density greater than 100,000 parasites/μL is associated with recrudescence and higher clearance time across all ages.

Although *P. falciparum* density positively correlates with pathophysiology and disease fatality, some persons living in endemic regions could have very high *P. falciparum* density without coming down with clinical symptoms, thus establishing different statuses of *P. falciparum malaria*: symptomatic and asymptomatic in a *P. falciparum* endemic place. This variation in tolerance of *P. falciparum* in endemic places however influences *P. falciparum* density as a criterion in determining the severity of the disease as there is evidence of people in different endemic places tolerating the parasite differently. A research on *P. falciparum* density in relation to disease severity in a *P. falciparum* endemic region of Thailand reveals that people present symptoms at a lower threshold than the WHO criteria. [7]

Abuja in Nigeria is a *P. falciparum* endemic area with a perennial and high transmission status, thus it provides a study area for *P. falciparum* density status. This work, therefore, studied *P. falciparum* density pattern and tolerance of people in this region to the parasite.

MATERIALS AND METHODS

The study was conducted in Federal Capital Territory (FCT), Abuja, Nigeria with a tropical climate of rainy and dry seasons. The dry season occurs from October to March, with high ambient temperature and cool, dry, dusty Harmattan wind, mostly in December and January. The wet season occurs from April to September following a hot and sunny period usually in February and March. The temperature in Abuja oscillates between 25°C and 40°C, and average annual rainfall is 999.9 mm.[10] This region is divided into six area councils, namely, Abaji, Abuja Municipal Area Council (AMAC), Bwari, Gwagwalada, Kuje, and Kwali. The AMAC has the highest population among the area councils, followed by Bwari Area Council with populations of 776,298 (55.2%) and 229,274 (16.3%), respectively.

Malaria transmission in Abuja is perennial, and it peaks at the beginning and the end of the rainy season.[11] The region is considered a malaria hyperendemic place, though a recent report shows that the area is one of the three places with a hypoendemic transmission status, but with endemicity of 22%,[12] basically attributed to *P. falciparum*. The study areas for this work, AMAC and Bwari, were thus carefully chosen to give a representation of Abuja considering the population of the areas, location at the central part of Abuja, and availability of institutions/activities that attract people from peripheral parts of Abuja.

The study population is composed of those who are aged 6 years and above and who have lived in Abuja for 5 years or more. A total of 246 participants each for the symptomatic and asymptomatic groups, respectively, were selected for the study. Symptomatic volunteers were recruited from people seeking medical care in Defence Head Quarters Medical Centre (DHQMC), Asokoro, under AMAC and Kubwa General Hospital (KGH) under Bwari Area Council. The DHQMC is a tertiary health facility accessed by military personnel and civilians from various parts of Abuja, and it also handles referral cases, while KGH is also a tertiary health facility accessed by people from all nook and cranny of Abuja and receives referred medical cases. The asymptomatic group was also recruited in these area councils via advocacies in marketplaces and churches for free malaria tests and through the use of banners in open places imploring people to check their malaria status.

Participants included in the study are those aged 6 years and above; for the symptomatic group, those who tested positive to *P. falciparum* via microscopy at the time of sample

collection and showed one or more of following signs, namely, headache, malaise, lassitude, fatigue, abdominal discomfort, muscle and joint pains, fever (axillary temperature ≥ 37.5°C), chills, perspiration, anorexia, or vomiting (Misch and Hawn, 2008) were included; for the asymptomatic group, those who tested positive to *P. falciparum* via microscopy but did not present with signs associated with symptomatic malaria at the time of blood collection were selected for the study. Those who did not test positive to *P. falciparum* or had the following conditions, namely, pregnancy, inflammatory disease, immunodeficiency/immunosuppressive disease(s), were on immune suppressive drugs, or were less than 6 years of age were excluded from the study.

The participants were selected after obtaining their consent and administration of questionnaire by screening volunteers for *P. falciparum* parasitemia, and microscopic examination of thick blood film made from finger prick and stained with 10% Giemsa stain (Rapid staining).[20] After screening, thick and thin blood films were made from each participant, stained with 3% Giemsa stain for 60 min and read according to WHO standard for Giemsa stained malaria microscopy,[20] by two readers and a third reader (level 1 WHO certified) who served as a tie-breaker in discordant results from the two readers for parasite detection, speciation, and quantification. The parasite densities were determined and expressed per microliter of blood according to WHO standard method and formula: Parasite Density (per μL) = parasite count/200 WBC × 8 × 10^6. The results (parasite densities, axillary temperature, age, and gender of the participants) were tabulated.

The results (parasite densities, axillary temperature, age, and gender of the participants) were tabulated and compared according to different parameters: disease status (symptomatic/asymptomatic), catchment areas of the study (AMAC and Bwari Area Council), and age groups. Statistical analysis was performed using IBM SPSS and R software. Descriptive statistics were done to determine the normality and the different assumptions, as the data sets were not normally distributed, nonparametric test was used to determine the relationship, Mann–Whitney U test was used to compare the median parasite density between symptomatic and asymptomatic malaria groups, while Kruskal–Wallis test was used to determine the median parasite density across the age groups in the different catchment areas. Gggplot2 package in R was used for visualizations. In all cases, $P < 0.05$ was statistically significant at 95% CI while $p > 0.05$ was statistically insignificant at 95% CI.

RESULTS

The study population comprises 48% males (n = 238) within which 50.4% (n = 120) fall within the symptomatic group and 49.6% (n = 118) in the asymptomatic group while 51.2% (n = 254) of the overall population is female within which 49.6% (n = 126) was symptomatic and 50.4% (n = 128) was asymptomatic. The age of the population ranged between 10 and –55 years.

The median parasite densities were grouped according to malaria cases (symptomatic and asymptomatic) and stratified according to age (10–20 years, 21–39 years, and 40+ years), segregated by catchment areas of the study (AMAC and Bwari) [Tables 1 and 2, Figures 1 and 2]. The median of the axillary temperature, which was one of the criteria for selection into symptomatic and asymptomatic cases, was found to be 37.80°C for the symptomatic group and 36.90°C for the asymptomatic group while the overall median parasite densities of the two groups were found to be 200/μL and 160/μL respectively. As expected, the median axillary temperatures of the symptomatic and asymptomatic groups differed significantly ($p = 0.012$) but the median parasite densities of the symptomatic and asymptomatic groups did not differ significantly ($p > 0.05$) in the overall population and when segregated according to age groups (10–20 years, 21–39 years, and 40^+ years) in AMAC and Bwari Area Council catchment areas.

Table 1: Overall median parasite density segregated by malaria case.

Parameters	Symptomatic	Asymptomatic	*P*-value
Overall Axillary Temp (°C)	37.80	36.90	0.012
Overall Median Parasite Density (/μL)	200.00	160.00	0.71

Table 2: Parasite density segregated by age, malaria cases, and catchment area.

Age	AMAC		BWARI	
	Axillary Temp (°C)	Parasite Median Density (/μL)	Axillary Temp (°C)	Parasite Median Density (/μL)
10–20 years	Sym/Asym 37.80/36.90	Sym/Asym 240.00/80.00 ($P = 0.13$)	Sym/Asym 37.90/36.90	Sym/Asym 120.00/120.00 ($P = 0.37$)
21–39 years	37.80/36.90	200.00/160.00 ($P = 0.7$)	37.90/36.90	120.00/200.00 ($P = 0.092$)
40 years+	37.80/36.90	4300.00/120.00 ($P = 0.62$)	37.90/36.90	5260.00/4600.00 ($P = 0.77$)

Sym: Symptomatic Cases, Asym: Asymptomatic Cases, AMAC: Abuja Municipal Area Council

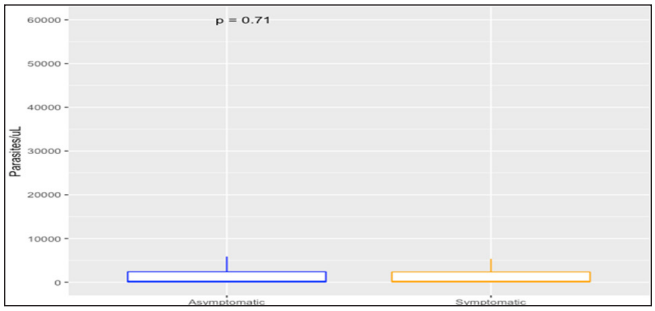

Figure 1: Median parasite density in symptomatic and asymptomatic malaria groups.

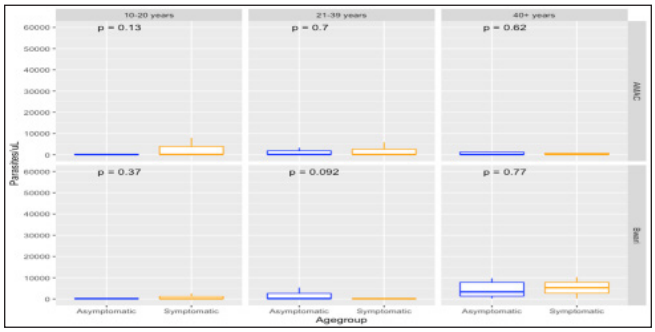

Figure 2: Median parasite density in symptomatic and asymptomatic malaria segregated by age groups and catchment areas.

DISCUSSION

In this study, parasite density, which is one the critical factors in *P. falciparum* pathogenesis, was studied in *P. falciparum* exposed persons grouped into symptomatic and asymptomatic persons. Apparently, the two categories of malaria cases (symptomatic and asymptomatic) showed low parasite densities (median density 200/μL and 160/μL, respectively). The findings in this work revealed a manifestation of malaria symptoms at low parasite density in symptomatic persons whose median parasite density does not differ significantly from median parasite density of asymptomatic cases in the same environment ($P > 0.05$) [Table 1, Figure 1]. In addition, there were no statistically significant differences in the parasite densities of symptomatic and asymptomatic cases across different age groups and the catchment areas [Table 2, Figure 2]. These findings are contrary to the work by Sondo *et al.*[13] in Nanoro, Burkina Faso, which showed that malaria symptoms significantly correlated with high parasite density. Similarly, Ali *et al.*[6] demonstrated that high parasite density is associated with severe clinical illness, complications, and mortality. More so because parasite density has been shown to correlate with age where older people are known to hold lower parasite densities than younger ones. According to Felger *et al.*,[14] acquired immunity to malaria is age-dependent, thus malaria among older people in endemic places occur with low parasite density. These earlier findings are however contrary to the findings in this work as the measured median parasite densities of the symptomatic and asymptomatic groups did not differ significantly both across different age groups and catchment areas.

Notwithstanding, some malaria works in Africa support the observations in this work. In a work in Ghana, low-density parasitemia conditions have been published in symptomatic and asymptomatic malaria cases, where asymptomatic and symptomatic mean *P. falciparum* densities of 160/μL and 451/μL, respectively, were documented. [15] This is quite in line with the observation in this work suggesting that *P. falciparum* density in endemic places may not correlate positively with symptoms, thus it is difficult to define malaria severity with parasite density. In addition, the works by Ali *et al.*,[6], Wogu *et al.*,[16] and Mac *et al.*,[1] showed that parasite density in *P. falciparum* endemic places does not correlate with age greater than 5 years, gender, or pyrexia. This, therefore, suggests that in *P. falciparum* endemic regions, host–*P. falciparum* interaction with respect to disease manifestation may be unique. This could be attributed to acquired immunity following constant exposure to the parasites. The result of this work typically demonstrated what acquisition of immunity against *P. falciparum* in an endemic place could be. As has been established, natural immunity helps in containing parasite proliferation while acquired immunity emerges to clear the parasite amidst survival efforts of the parasite to evade the immune responses. It thus appears that in *P. falciparum* endemic places, individuals acquire immunity which may not eliminate the parasite but strikes a balance, as the parasites cannot multiply easily, but in some instances, symptoms may manifest at such low densities. In view of this, coexistence of symptomatic and asymptomatic cases with similar parasite density is expected in *P. falciparum* endemic places. According to Kimenyi *et al.*,[17] symptomatic and asymptomatic malaria without clear distinction with respect to parasite density can occur owing to possible transition from asymptomatic to symptomatic status in an endemic area. This transition may be due to immune suppression or loss of immunity following treatment of asymptomatic conditions[17] and has been linked to some factors, including parasite, host, and transmission dynamics factors.

In *P. falciparum* endemic places with a high transmission rate, incessant mutation leading to antigenic shift and antigenic variation is common, resulting in the emergence of plasmodial antigens alien to the host immune system or co-existence of different *P. falciparum* strains in an earlier immune host. This could lead to the manifestation of clinical symptoms in individuals with low parasite density due to novel antigens from the mutant strains. In addition, mixed infection by *P. falciparum* strains could also lead to

competitive suppression among the strains, thus resulting in suppressed proliferation and low parasite density. According to Sondo *et al.*,[13] subjects with mixed infection of K1 and MAD20 allelic families of *P. falciparum* had low parasite density against high densities observed in mono-infection of the strains, suggesting competitive suppression between the parasite strains.

Apart from the immunological confrontation of the parasites that limit their proliferation, exogenous factors, such as the use of antimalarial drugs, could also influence the parasite density in endemic places. According to Wilairatana *et al.*,[7] artemisinin-based combination therapy (ACTs) are very potent antimalaria drugs that readily clear the asexual stage of the parasite in a very short time. As a result, the acquaintance period of the parasite and immune system is shortened, thus weakening the acquired immune responses to the parasite and the immune memory, which could make people come down with symptoms at low parasite density. More so when the parasite develops resistance against the ACTs, some asexual stages of *P. falciparum* could become dormant temporarily after drug administration and may regrow after drug clearance, manifesting as low-density parasitemia.[18] With waned *P. falciparum*-specific immunity following regular use of ACTs and manifestation of low-density parasitemia in drug-resistance cases, subjects are highly prone to clinical symptoms at low-density. A variety of antimalarial drugs including ACTs abound in Abuja and are often sold over the counter, thereby allowing easy access and use at any suspected malaria sign. In addition, these drugs are also regularly prescribed in most pyretic conditions by the health professionals even without parasite-based case confirmation. These phenomena may contribute to low-density *P. falciparum* cases observed in this work.

As the median parasite densities did not differ significantly between the symptomatic and asymptomatic cases among different age groups and in different catchment areas, this may suggest similar demography and exposure patterns among the study populations from AMAC and Bwari Area Council. Although the AMAC and Bwari Area Council were chosen to represent the Abuja city center and the satellite towns, respectively, referrals are made to the different clinics that were used, and economic/daily activities take people from one location to the other. Thus, participants may not have been confined to a given geographic area and may have similar exposure to the parasites. With common endemicity and possible exposure pattern, people in Abuja may show similar immunological response to *P. falciparum*, which will influence the way they contain the parasite and thereby the parasite density.

In view of the pathogenesis of *P. falciparum* malaria, the parasite density is a critical factor as it is related to the number of red blood cells invaded, which impacts on the hemoglobin level and cascades into other pathological consequences. According to Ali *et al.*,[6] high parasite density is associated with severe clinical illness, complications, and mortality. It is in view of this, that the WHO established parasite density as one of the cardinal criteria in defining the severity of *P. falciparum* malaria,[19] thus establishing cutoff values for low, moderate, and hyperparasitemia.[7] According to this criterion, *P. falciparum* density of ≥100,000 parasites/μL and 250,000 parasites/μL are considered hyperparasitemia in areas of low and high transmission/endemicity, respectively.[20] Following this criterion, *P. falciparum* density of 250,000 parasites/μL and above is expected to be considered as hyperparasitemia in Abuja, and associated with clinical symptoms. On the contrary, maximum median parasite density of 200 parasites/μL was observed in the symptomatic group and 160 parasites/μL in the asymptomatic group in the overall study group and catchment areas in this study. This, therefore, shows a low parasite density precipitating clinical symptoms in Abuja. It, therefore, follows that the WHO classification of *P. falciparum* hyperparasitemia and by inference disease severity is not well defined for a *P. falciparum* endemic area like Abuja with median density of 200 parasites/μL. Contrary to this criterion too, both the symptomatic and asymptomatic groups share similar density distribution; both tend to be skewed to the right and thus there is more distribution around low parasite density. This, therefore, suggests that people in Abuja, though a *P. falciparum* endemic area, come down with clinical malaria at low parasite density; consequently, if the WHO criterion for hyperparasitemia is used in this area, wrong decisions could be made. In addition, this work reveals that low parasite density abounds in *P. falciparum* endemic areas and is of great epidemiological concern as it impacts negatively on the parasite detection rate, thus enhancing the spread of the disease.

CONCLUSIONS

The findings in this work demonstrated a shift from what was known earlier and expected in *P. falciparum* pathophysiology. *P. falciparum* malaria manifests symptomatically and asymptomatically at lower parasite densities described for malaria-endemic areas. The low parasite densities observed in Abuja being an endemic area might have been caused by different factors ranging from host endogenous and exogenous factors to parasite factors. It is possible that the level of acquired immunity of people living in endemic places does not allow for parasite proliferation upon infection, but pro-inflammatory cytokines being part of acquired immunity could lead to disease manifestation even at the prevailing

low parasite density. The WHO recommended ACTs are ubiquitous in Abuja and Nigeria at large in different brands and combinations without restriction on their use; therefore, they may have contributed to symptomatic conditions at low parasite density since ACT use could lead to sterile *P. falciparum* immunity and consequent disease manifestation at low parasite density. This condition is also suggestive of possible emergence of ACT resistance strains of *P. falciparum* in the region as low-density parasitemia is prevalent in the area amidst incessant use of ACT. With the low *P. falciparum* density and no significant difference in the parasite densities of symptomatic and asymptomatic cases observed in this work, the current WHO classification of hyperparasitemia and low parasitemia and their use in the definition of *P. falciparum* severity may be misleading, necessitating a review.

Ethical approval

Ethical approval for this research was obtained from the Federal Capital Territory Health Research Ethical Committee (FCT HREC), with the number: FHREC/2018/01/15/12-02-18. Informed consent of the clients was obtained in writing (signed/thumb printed) from the volunteers or parents/guardians in the case of dependents. Appropriate questionnaire that captured demographic and health status relevant to the study was also administered to the volunteers before enrolment into the study.

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Declaration of patients consent

The authors certify that they have obtained all appropriate patient consent.

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None.

Conflict of interest

None.

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