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Full Length Research Paper

An investigation on the haematologic and biochemical indices of moderately *P. falciparum* infected male inhabitants of Owerri, Imo State, Nigeria

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In spite of threats malaria pose to public health, specific records on alterations in some haematologic and biochemical indices of inhabitants of Owerri Municipality infected with *P. falciparum* malaria have not received wide reports and documentation. Accordingly, the present study sought to investigate alterations in haematologic and biochemical indices of moderately *P. falciparum* infected male inhabitants of Owerri Municipality. Haematologic and biochemical indices were estimated by spectrophotometric methods. Haemoglobin concentrations of malarious subjects within age brackets of 11 to 20 and 21 to 31 years were below reference interval; $[Hb]_{M;11\text{-}21\ years}$ = 10.53 ± 0.23 g/dl (p < 0.05); $[Hb]_{M;\ 21\text{-}31\ years}$ = 11.51 ± 1.10 g/dl (p < 0.05). There was no significant difference (p > 0.05) in erythrocyte sedimentation rate (ESR) between the two malarious groups; $ESR_{M;\ 11\text{-}20\ years}$ = 29.80 ± 0.74 mm/h; $ESR_{M;}$ = 26.51 ± 1.42 mm/h. Packed cell volume (PCV) of malarious subject gave the following values: $PCV\%_{M;\ 11\text{-}20\ years}$ = 26.82 ± 0.78; $PCV\%_{M;\ 21\text{-}31\ years}$ = 25.82 ± 0.78; p > 0.05. Serum white blood cell count (WBC) was raised in malarious subjects compared to control groups (p > 0.05) except with $WBC \times 10^3{}_{M;\ 21\text{-}30\ years}$ = 13.77 ± 3.95; p > 0.05. Serum albumin was lower in malarious subjects; $[Albumin]_{M;\ 11\text{-}20\ years}$ = 4.70 ± 0.05 mg/dl and $[Albumin]_{M;\ 1\text{-}21\text{-}31\ years\ 31\ years}$ = 4.31 ± 0.09 mg/dl; p > 0.05, whereas, serum creatinine concentrations of malarious subjects gave higher values: $[Creatinine]_{M;\ 11\text{-}20\ years}$ = 0.88 ± 0.71 mg/dl and $[Creatinine]_{M;\ 21\text{-}31\ years}$ = 1.14 ± 0.42 mg/dl; p > 0.05. Serum urea concentrations of malarious subjects were significantly (p < 0.05) higher than the corresponding non-malarious age group. Serum fasting blood sugar (FBS) was significantly (p < 0.05) lower in malarious groups compared to corresponding non-malarious subjects. Specifically, $[FBS]_{M;\ 11\text{-}20\ years}$ = 63.34 ± 1.66 mg/dl and $[FBS]_{M;\ 21\text{-}31\ years}$ = 69.45 ± 1.25 mg/dl; p < 0.05. Subjects with moderate malaria infection showed symptoms of anaemia, alterations in nitrogen and carbohydrate metabolism, exemplified by raised serum level of urea and low level of FBS.

Key words: Haemoglobin, packed cell volume, erythrocyte sedimentation rate, fasting blood sugar, malaria, *Plasmodium falciparum.*

INTRODUCTION

Several species of intracellular protozoa of the genus *Plasmodium* cause malaria in humans. They include *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, Plasmodium malariae* (Krotoski et al., 1982;

Joseph et al., 2011) and more recently, *P. knowlesi* (Figtree et al., 2010; Lee et al., 2011; Marchand et al., 2011). *P. falciparum* and *P. vivax* cause the most serious forms of the disease (World Health Organization (WHO), 2005; Idonije et al., 2011; Joseph et al., 2011). Sporozoites from bite of female mosquitoes (genius Anopheles) infect humans and are the progenitor of the disease condition. The parasites have a complicated life cycle that requires a vertebrate host for the asexual cycle and female *Anopheles* mosquitoes for completion of the sexual cycle. Malaria poses a threat to public health with 80 to 90% of morbidity and mortality occurring in Africa, afflicting both young and old (Afolabi, 2001; Ikekpeazu et al., 2010; Ogbodo et al., 2010). In addition, reports showed that malaria could be transmitted by transfusion of infected blood (Strickland, 1991; Ali and Kadaru, 2005), sharing needles (Tracy and Webster, 2001) and congenital transmission (Ezechukwu et al., 2004).

Blood is a tissue that circulates in a virtually closed system of blood vessels. It is composed of solid elements-red, white blood cells, and platelets, suspended liquid medium-plasma. Therefore, the plasma is an extracellular fluid confined within the vascular system. The water and electrolyte composition of plasma is particularly the same as that of intracellular fluid, made up of water, electrolytes, metabolites, nutrients, proteins and hormones.

Physicochemical properties of the blood are constant but may undergo slight variations under normal physiologic conditions. However, the relative constancy in the internal environment of the blood system exhibits wide and profound perturbation and distortions under clinically defined pathophysiologic states. Some of these conditions include malignancy, genetic defects, malnutrition, parasitic infections etc. Studies have revealed that haematologic and biochemical alterations occur in malaria infected blood and there are common complications associated with this disease. Haematologic alterations that are associated with malaria infection include anaemia, thrombocytopenia, and disseminated intravascular coagulation (Facer, 1994; Perrin et al., 1982; Maina et al., 2010; Chandra and Chandra, 2013). Alterations in physicochemical parameters of *P. falciparum* infested blood may vary with level of malaria endemicity, presence of haemoglobinopathies, nutritional status, demographic factors and level of malaria immunity (Price et al., 2001; Erhart et al., 2004). Therefore, well-informed alterations in blood parameters in malaria infection enable the clinician to establish reliable diagnosis and therapeutic interventions.

Although haematologic and biochemical indices of *P. falciparum* infected individuals of Nigerian origin have been widely reported (Udesen, 2003; Egwunyenga et al., 2004; Adesina et al., 2009; Kayode et al., 2011), specific records on blood chemistry of infected inhabitants of Owerri Municipality have been poorly documented and not widely reported in this regard. Nevertheless, there are reports on high prevalence of *P. falciparum* malaria amongst inhabitants of South Eastern Nigeria (Udesen, 2003; Ibekwe et al., 2004). Therefore, the present study seeks to investigate alterations in haematologic and biochemical indices of moderately *P. falciparum* infected male inhabitants of Owerri Municipality.

MATERIALS AND METHODS

Study area

The study was conducted between May, 2011 and August, 2011 in Owerri Municipality, Imo State, Nigeria, which lies on rainforest belt (Latitude 5.485°N and Longitude 7.035°E). The wet season is within the period of March to September, when breeding of *Anopheles* mosquitoes is at its peak and bites are prevalent. Twenty-one (21) clinically confirmed (WHO, 2008) and randomly selected malarious and 12-h fasting male out-patients attending clinics at the Federal Medical Center (FMC), St. John Clinic/Medical Diagnostic Labora-tories, Avigram Medical Diagnostic and Research Laboratories, and Qualitech Medical Diagnostic Laboratories enrolled for this study. All laboratory investigations were carried out in Avigram Medical Diagnostic and Research Laboratories. These centers are located in Owerri, Imo State, Nigeria. Age matched asymptomatic/non-malarious fasting male subjects (n = 15) constituted the control subjects, who also are residents of Owerri Municipality. The patients were in the following categories- adults (n = 11) of 21 to 31 years old and adolescent (n = 10) of 11 to 20 years old. Exclusion criteria for both patients and control subjects included; gastro-intestinal tract infection, protein energy malnutrition, renal diseases, cirrhosis, hepatitis, obstructive jaundice, cancer, diabetes mellitus, hypertension, obesity, smoking, alcoholism, persons living with human immunodeficiency virus (HIV), patients taking anti-malaria drugs and vitamin supplements, patients who had treated malaria in the past 2 months (Onyesom and Onyemakonor, 2011; Idonije et al., 2011) and patients with low or high parasitaemia.

Ethics

The Ethical Committee of University of Port Harcourt, Port Harcourt, Nigeria, approved the study in compliance with the Declaration on the Right of the Patient (WMA, 2000). Before enrolment for the study, the patients/subjects involved signed an informed consent form. Guardian/Parent signed the consent form on behalf of participants below the age of 15 years old.

Collection and preparation of blood specimen

Blood specimen was collected by venipuncture from 12-h fasting subjects using 5.0 ml capacity disposable syringes. Three milliliter (3.0 ml) of the blood samples were transferred into plain bottles to

Table 1. Some haematological indices of non-malarious and malarious subjects.

Parameter	NM		M		Reference intervals
	11-20 years	21-31 years	11-20 years	21-31 years	
[Hb] g/dl	15.67±0.20^{a}	16.70±0.96^{a,b}	10.53±0.23^{c}	11.51±1.10^{c,d}	13.5-18.0*
ESR mm/h	16.30±1.08^{a}	15.2±0.60^{a,b}	29.80±0.74^{c}	26.51±1.42^{c,d}	0-15†
PCV %	33.94±0.64^{a}	33.94±0.61^{a,b}	26.82±0.78^{c}	25.82±0.78^{d}	40-54
WBC ×10^{3}	6.39±6.98^{a}	7.53±2.26^{a,b}	10.13±4.75^{a,b,c}	13.77±3.95^{a,b,c,d}	4.5-11.0‡

*Richards et al. ,(1998); ‡Erhart et al. ,(2004); †Bottiger and Svedberg (1967); Means in the row with the same letter are not significantly different at p > 0.05 according to LSD. NM: Non-malarious; M: Malarious. WBC: cell/µl/mm^{3}.

allow for coagulation, whereas the remaining 2.0 ml was transferred into ethylenediaminetetraacetic acid (EDTA) bottles for malaria parasite tests and haematological studies. The coagulated blood samples were centrifuged {(LC-412) - China Chemical Centrifuge, Lab Centrifuge} at 3000 rpm for 10 min, the serum transferred into Bijou bottle and stored frozen until required for biochemical analyses (Onyesom et al., 2010).

Malaria parasite density test

Measurement of parasite density of peripheral blood smear was by Giemsa stained techniques. The films were examined microscopically (Meiji Techno MT4210/4310 Phase Contrast Microscope) using × 100 objective under oil immersion (Cheesbrough, 1998) as reported by Sumbele et al. (2010). Level of parasitaemia was in microliter (µl) of blood thick film preparation (Erhart et al., 2004). According to WHO (2005), level of parasitaemia was graded as low+ (1 to 999/µl), moderate++ (1000 to 9999/µl) and severe+++ (> 10,000/µl).

Haematological studies

The modified method (Baure, 1980), based on cyanomethaemoglobin reaction was used for the determination of haemoglobin concentration (Chikezie, 2009). Packed cell volume (PCV) was measured using whole blood mixed in a 10 µl mark capillary pipette. The set up was centrifuged at 3000 rpm for 30 min. The hematocrit was removed from the centrifuge (Orbital 260 Micro-centrifuge/Micro-haematocrit - CLEMENTS CENTRIFUGE) and the volume of packed red cell column was read off and expressed as percentage of whole blood volume. Estimation of white blood cell count (WBC) was according to methods of National Committee for Clinical Laboratory Standards (NCCLS) (1993). Estimation of erythrocyte sedimentation rate (ESR) was according to the Westergreen's methods as described by Supcharoen et al. (1992).

Biochemical studies

Fasting blood sugar (FBS) was measured by standard methods as reported by Kazmierczack (1996). Serum urea level was determined according to the method described by Fawcett and Scott (1960) and reported by Kayode et al. (2011). Creatinine level in the blood was determined according to the methods described by Bartels et al. (1972). Albumin concentration in the blood was measured by the method of Doumas et al. (1971) and as described by Cheung and Hchman (1996).

Statistical analyses

The experiments were designed in a completely randomized method, and data collected were analyzed by the analysis of variance procedure while treatment means were separated by the least significance difference (LSD) incorporated in the statistical analysis system (SAS) package of 9.1 version (2006). The correlation coefficients between the results were determined with Microsoft Office Excel, 2010 version.

RESULTS

Table 1 showed that haematological indices of non-malarious subjects were within reference intervals and there was no significant difference ($p > 0.05$) between the age brackets of 11 to 20 years and 21 to 31 years. Haemoglobin concentrations of malarious subjects within age brackets of 11 to 20 and 21 to 31 years were below reference interval; $[Hb]_{M;11\text{-}21\ years} = 10.53 \pm 0.23$ g/dl ($p < 0.05$); $[Hb]_{M;\ 21\text{-}31\ years} = 11.51 \pm 1.10$ g/dl ($p < 0.05$). These values represented 22.97 and 31.08% drop in serum haemoglobin concentrations compared to corresponding non-malarious subjects. Serum haemoglobin concentrations between the two malarious groups were not significantly different ($p > 0.05$).

ESR of malarious subjects were above the reference intervals of ESR = 0 to 15 mm/h (Table 1) and was significantly different ($p < 0.05$) compared to the control subjects. However, there was no significant difference ($p > 0.05$) in ESR between the two malarious groups; $ESR_{M;\ 11\text{-}20\ years} = 29.80 \pm 0.74$ mm/h; $ESR_{M;\ 21\text{-}31\ years} = 26.51 \pm 1.42$ mm/h. PCV of malarious subject gave the following values: $PCV\%_{M;\ 11\text{-}20\ years} = 26.82 \pm 0.78$; $PCV\%_{M;\ 21\text{-}31\ years} = 25.82 \pm 0.78$; $p > 0.05$, with values below the reference interval: PCV% = 40 to 54. Serum WBC was raised in malarious subjects compared to control groups ($p > 0.05$) and within reference interval ($WBC \times 10^{3}$ = 4.5 to 11.0), except with $WBC \times 10^{3}{}_{M;\ 21\text{-}30\ years} = 13.77 \pm 3.95$; $p > 0.05$.

Table 2 showed that there was no significant difference ($p > 0.05$) between the two non-malarious groups in connection to the four experimental biochemical indices.

Table 2. Some biochemical indices of non-malarious and malarious subjects.

Parameter (mg/dl)	NM		M		Reference Intervals*
	11-20 years	21-31 years	11-20 years	21-31 years	
Albumin ×10^3	5.18±0.29^{a}	4.46±0.05^{a,b}	4.70±0.05^{a,b,c}	4.31±0.09^{a,b,c,d}	3.5-5.5
Creatinine	0.62±0.27^{a}	0.94±0.51^{a,b}	0.88±0.71^{a,b,c}	1.14±0.42^{a,b,c,d}	0.7-1.5
Urea	10.70±0.94^{a}	12.72±0.51^{a,b}	17.10±0.74^{c}	26.14±0.98^{d}	8-20
FBS	89.42±0.64^{a}	87.47±1.06^{a,b}	63.34±1.66^{c}	69.45±1.25^{d}	60-100

*Martin, (1983): Means in the row with the same letter are not significantly different at $p > 0.05$ according to LSD. NM: Non-malarious; M: Malarious.

Likewise, serum albumin and creatinine concentrations were not significantly different ($p > 0.05$) between the non-malarious and malarious subjects. In addition, marginal alterations in serum albumin and creatinine concentrations in non-malarious and malarious subjects were within reference intervals (Table 2). Specifically, serum albumin was lower in malarious subjects; $[Albumin]_{M;\ 11\text{-}20\ years}$ = 4.70 ± 0.05 mg/dl and $[Albumin]_{M;\ 21\text{-}31\ years}$ = 4.31 ± 0.09 mg/dl; $p > 0.05$, whereas, serum creatinine concentrations of malarious subjects gave higher values: $[Creatinine]_{M;\ 11\text{-}20\ years}$ = 0.88 ± 0.71 mg/dl and $[Creatinine]_{M;\ 21\text{-}31\ years}$ = 1.14 ± 0.42 mg/dl; $p > 0.05$. Serum urea concentrations of malarious subjects were significantly higher than the corresponding non-malarious age group. Serum urea concentration of malarious subjects between the age brackets of 21 to 31 years was above the reference interval; $[Urea]_{M;\ 21\text{-}31\ years}$ = 26.14 ± 0.98 mg/dl; $p < 0.05$. Serum FBS concentration was significantly ($p < 0.05$) lower in malarious groups compared to corresponding non-malarious subjects. Specifically, $[FBS]_{M;\ 11\text{-}20\ years}$ = 63.34 ± 1.66 mg/dl and $[FBS]_{M;\ 21\text{-}31\ years}$ = 69.45 ± 1.25 mg/dl; $p < 0.05$. These values represented 29.17 and 20.60% drop in serum FBS concentrations compared to their corresponding non-malarious age group.

DISCUSSION

Haematologic alterations associated with malaria infection are well recognized and have been widely reported (Das et al., 1999; Mishra et al., 2002; Udosen, 2003; Bidaki and Dalimi, 2003; Erhart et al., 2004; Maina et al., 2010). The present study reported haematologic and biochemical alterations associated with moderate malaria infection in male subjects. The decreased haemoglobin concentrations in malarious subjects (Table 1) were predictive as had been reported by several authors (Das et al., 1999; Mishra et al., 2002; Udosen, 2003; Bidaki and Dalimi, 2003; Erhart et al., 2004; Maina et al., 2010). Earlier reports had posited that malaria-related anaemia is often more severe in areas of intense malaria transmission and affects younger children rather than older children or adults (Phillips and Pasvol, 1992; Menendez et al., 2000). Also, the present study showed that moderate malaria infection among male subjects in Owerri Municipality exhibited alterations in haemoglobin concentrations as previously reported elsewhere. Thus, moderate *P. falciparum* infection caused reduction in haemoglobin concentration, which was more pronounced in adolescents between the age brackets of 11 to 20 years than their adult counterparts of age brackets between 21 to 30 years ($p > 0.05$) (Table 1).

According to reports by Maina et al., (2010) as contained in the National Guidelines for Diagnosis, Treatment and Prevention of Malaria for Health Workers in Kenya, anaemia is defined as [Hb] < 10 g/dl for both males and females. Furthermore, severe malaria anaemia is defined as [Hb] < 5 g/dl in the presence of hyperparasitaemia (> 200,000 parasites/µl). Therefore, the drop in haemoglobin concentrations in the malarious subjects (Table 1) approximately connoted mild anaemia. The decreased PCV levels were also expected from previous reports (Adesina et al., 2009; Ogbodo et al. 2010; Kayode et al., 2011).

Furthermore, the drop in PCV values in the two malarious groups confirmed symptoms of anaemia in these study groups. Two striking factors are responsible for the development and presentation of anaemia in malaria infections.

1. Rapid rate of haemolysis associated with the pathophysiology of the disease condition (Phillips and Pasvol, 1992; Selvam and Baskaram, 1996; Erhart et al., 2004).

2. Reduced rate of haemoglobin biosynthesis, which is often connected to level of immunity and nutritional status of infected individuals (Das et al., 1999; Price et al., 2001; Wickramasinghe and Abdalla, 2000; Erhart et al., 2004).

Therefore, the interplay of these multifactorial etiologies of anaemia in malaria infection, as described above, may have contributed significantly to the drop in haemoglobin concentrations in the malarious groups by 22.97 and 31.08% (Table 1). In concord with the present findings,

studies among non-immune or semi-immune populations outside Africa have also shown statistically significant levels of mild anaemia in falciparum malaria patients (Rojanasthien et al., 1992; Das et al., 1999).

Elevation of ESR have been reported in acute and chronic infections (Kwiatkoski et al., 1989), chronic inflammatory disorders (Kwiatkoski et al., 1989; Supcharoen et al., 1992; Dreyer and Boden, 2003) malignancies especially Hodgkin's disease (Malcolm and Brigden, 1999; Mönig et al., 2002; Dreyer and Boden, 2003), tissue necrosis (Scuderi, 1986; Beutler and Cerami, 1987) and pregnancy (van den Broek and Letsky, 2008). Supcharoen et al. (1992) used ESR as basis for the diagnosis and monitoring of therapeutic intervention of malaria. They suggested that ESR was elevated during acute malaria infection and declined with recovery. Thus, the present findings as presented in Table 1 were in agreement with the reports of Supcharoen et al. (1992). However, measurement of ESR is often used as a non-specific test for acute illness and may reflect the acute process of the disease.

Erhart et al. (2004) stated that semi-immune persons in Western Thailand with parasitaemia tended to have significantly lower white blood cell. Perrin et al. (1982) and Rojanasthien et al. (1992) reported contrary findings during malaria infection in man. The non-significant ($p > 0.05$) increase in serum WBC in the present study contradicts these two previous separate reports mentioned above. Nevertheless, the present study showed that the malarious subjects did not exhibit leukocytosis, which was defined as total WBC > 17,000/µl, frequently seen in 8% malarious individuals as against 3% non-malarious children living in Western Kenya (Maina et al., 2010).

In another study, Kayode et al. (2011) indicated significant increase ($p > 0.05$) in WBC of malaria and malaria typhoid co-infected patients, which they posited could have been elicited by increased production of leukocytes at the onset of the infection to wade off malaria parasite and typhoid pathogens. Similarly, increase in WBC in pregnant and non-pregnant malaria patients has been reported by Adesina et al. (2009) and Sumbele et al. (2010). However, the works of Ali et al. (2009) noted both increased and decreased WBC in the blood of typhoid patients examined in Dubai. From these indications, the use of serum level of WBC as an index for diagnosis may not be very reliable. Therefore, WBC should always be thoroughly re-evaluated for malaria for reproducibility and reliability.

Studies carried out by Amah et al. (2011) showed significant reduction in serum levels of albumin in malaria patients in endemic regions of Calabar, Nigeria. Many authors have proposed the use of serum albumin levels as a reliable biochemical marker for establishing severe pathologic conditions such as malnutrition and infectious diseases (Das et al., 1997; Kwena et al., 2012). Malaria infections are accompanied with significant decrease in plasma albumin concentrations (Kwena et al., 2012) as well as in malnutrition and pregnancy. However, the prevailing plasma albumin concentration in malaria infection is dependent on the nutritional status of the affected individual and hepatic functionality (Crawly, 2004; Ogbodo et al., 2010). Probably, based on the nutritional and hepatic status of the study subjects/participants in the study groups, the report presented here showed non-significant ($p > 0.05$) reduction in serum albumin levels in malarious subjects compared to non-malarious groups (Table 2). Contrary to these observations, Ogbodo et al. (2010) showed that there was initial significant ($p < 0.05$) increase in serum levels of albumin in low and moderate malaria infections, but decreased as the malaria density increased. Based on these observations, they recommended the use of albumin infusion in place of other colloidal solutions as a good intervention in severe malaria.

Creatinine and urea are nitrogenous low threshold substances with immense clinical application in ascertaining renal function. Impairment of renal function during severe falciparum malaria is common (al-Yaman et al., 1997; Eiam-Omg and Sitprija, 1988; Gu¨nther et al., 2002; Mockenhaupt et al., 2004). Table 2 shows that the malarious groups presented marginal increases in serum creatinine concentration ($p > 0.05$). Paradoxically, serum levels of urea were significantly ($p < 0.05$) raised in the same malarious subjects under investigation. But elevation of serum urea concentration could also connote evidence of dehydration, consumption of proteinous meals and tissue catabolism. Nevertheless, this was an obvious indication that moderately malaria infected subjects exhibited alteration in nitrogen metabolism with underlying compromised renal function. According to Sitprija (1988), raised blood urea concentration reflected gradual progression towards renal dysfunction. Specifically, serum levels of urea had been observed to increase more rapidly than serum creatinine concentration in individuals with renal dysfunction (Emian-Ong, 2002).

Blood sugar levels in malaria infection have received the attention of several researchers. Studies by Kayode et al. (2011) indicated hypoglycemia in both malaria and typhoid co-infected patients. They posited that the level of hypoglycemia correlated with severity of infection, which was elicited by hyper-secretion of insulin. Their report corroborates the studies by Onyesom and Agho (2011), who noted the incidence of hypoglycemia in malaria patients in Edo-Delta state. The role of low serum insulin-like growth factor-1 (IGF-1) and low blood glucose levels in malaria infection was reported by Mizushima et al. (1994). They noted that *P. falciparum* infected children with low IGF-1 levels (< 50 ng/ml) presented hypoglycemia compared to other study groups. In another study,

Binh et al. (1997) reported the relative contribution of insulin-mediated and non-insulin-mediated plasma glucose levels in severe malaria. The report stated that there was a corresponding increase risk of hypoglycemia as infection progressed because host glucose production becomes insufficient for host/parasite demand. The study also revealed that basal plasma glucose increased in uncomplicated malaria because of peripheral insulin resistance. Moderate malaria infection caused significant reduction in serum FBS levels (Table 2) with blood sugar levels tending towards hypoglycemia ([FBS] < 60 mg/dl). Accordingly, the present report supports the findings of previous authors (Mizushima et al., 1994; Binh et al., 1997; Kayode et al., 2011; Onyesom and Agho 2011).

Finally, although these alterations in haematologic and biochemical indices in association with malaria infection are not novel, our findings have added more information, hitherto the limited knowledge and sparsely reports on alterations in blood profile of malaria infected individuals habitat in Owerri Municipality .

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