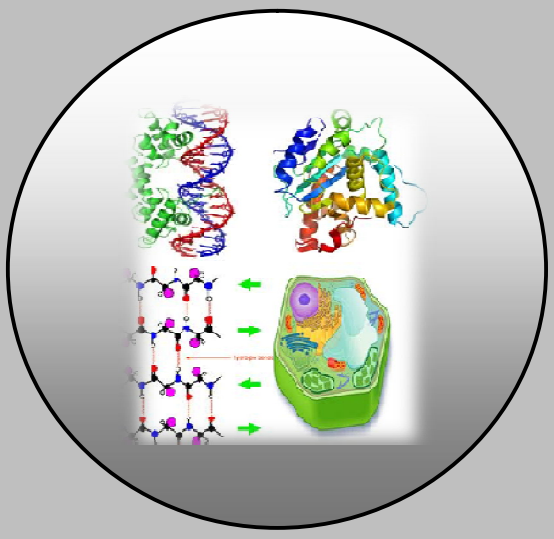
Haematologic and Biochemical Indices of *Plasmodium falciparum* Infected Inhabitants of Owerri, Imo State, Nigeria

J.O. Akaninwor, E.B. Essien, P.C. Chikezie and R.T. Okpara

ISSN 0970-4973 (Print)
ISSN 2319-3077 (Online/Electronic)



J. Biol. Chem. Research
Volume 30 (2) 2013 Pages No. 682-694

Journal of Biological and Chemical Research
(An International Journal of Life Sciences and Chemistry)

Published by Society for Advancement of Sciences®

J. Biol. Chem. Research. Vol. 30, No. 2: 682-694 (2013)
(An International Journal of Life Sciences and Chemistry)

ISSN 0970-4973 (Print)
ISSN 2319-3077 (Online/Electronic)
Published by Society for Advancement of Science®

http:// www.jbcr.in
jbiolchemres@gmail.com
info@jbcr.in

RESEARCH PAPER

Received: 17/06/2013 Revised: 19/08/2013 Accepted: 25/08/2013

Haematologic and Biochemical Indices of *Plasmodium falciparum* Infected Inhabitants of Owerri, Imo State, Nigeria

J.O. Akaninwor*, E.B. Essien*, P.C. Chikezie and R.T. Okpara***

***Department of Biochemistry, University of Port-Harcourt, Port-Harcourt, Nigeria**
****Department of Biochemistry, Imo State University, Owerri, Nigeria.**

ABSTRACT

The present study seeks to investigate changes 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 11-20 and 21-31 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; $ESR_{M;\ 21\text{-}31\ years}$ = 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) excerpt with $WBC\times10^3{}_{M;\ 21\text{-}30\ years}$ = 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 $[Albumin]_{M;\ 21\text{-}31\ years}$ = 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 (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±1.25mg/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.

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

INTRODUCTION

Four species of intracellular protozoa of the genus *Plasmodium* cause malaria in humans. They include *Plasmodium falciparum, P. vivax, P. ovale,* and *P. malariae* (Krotoski*et al*., 1982; Joseph *et al.,* 2011). *P. falciparum* and *P. vivax* cause the most serious forms of the disease (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 80-90% 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 changes occur in malaria infected blood and there are common complications associated with this disease. Haematologic changes 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). Changes 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 e*t al.,* 2004). Therefore, well-informed changes 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 of infected inhabitants of Owerri Municipality have been poorly documented and not widely reported in this regard. Therefore, the present study seeks to investigate changes 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-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 fasting male patients attending clinics at the Federal Medical Center (FMC), St. John Clinic/Medical Diagnostic Laboratories, Avigram Medical Diagnostic Laboratories, and Qualitech Medical Diagnostic Laboratories enrolled for this study. These centers are located in Owerri, Imo State, Nigeria. Age matched asymptomatic/non-malarious fasting male subjects (*n* = 15) constituted the control subjects. The patients were in the following categories- adults (*n* = 11) of 21-31 years old and adolescent (*n* = 10) of 11-20 years old. Exclusion criteria include; gastrointestinal tract infection, protein energy malnutrition, renal diseases, cirrhosis, hepatitis, obstructive jaundice, cancer, diabetes mellitus, hypertension, obesity, smoking, alcoholism, persons living with HIV, patients taking anti-malaria drugs and vitamin supplements, patients who have 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.

Collection and preparation of blood specimen: Blood specimen was collected by venipuncture from fasting subjects using 5.0 mL capacity disposable syringes. Three milliliter (3.0 mL) of the blood samples were transferred into plain bottles to allow for coagulation, whereas the remaining 2.0 mL was transferred into EDTA bottles for malaria parasite tests and haematological studies. The coagulated blood samples were centrifuged 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 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 e*t 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 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 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 versions (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 11-20 years and 21-31 years. Haemoglobin concentrations of malarious subjects within age brackets of 11-20 and 21-31 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 ($p > 0.05$). 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 ($p< 0.05$).

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

Parameters	NM		M		Reference Intervals
	11-20 yrs.	21-31 yrs.	11-20 yrs.	21-31 yrs.	
[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}.

ESR of malarious subjects were above the reference intervals of ESR = 0-15 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±0.74 mm/h; $ESR_{M;\ 21\text{-}31\ years}$ = 26.51±1.42 mm/h.

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; $p< 0.05$, with values below the reference interval: PCV% = 40-54. Serum WBC was raised in malarious subjects compared to control groups ($p < 0.05$) and within reference interval (WBC $\times10^3$ = 4.5-11.0), excerpt with $WBC\times10^3{}_{M;\ 21\text{-}30\ years}$ = 13.77±3.95; $p< 0.05$.

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

Parameters (mg/dL)	NM		M		Reference Intervals[8]
	11-20 yrs.	21-31 yrs.	11-20 yrs.	21-31 yrs.	
Albumin $\times10^3$	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.

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. Likewise, serum albumin and creatinine concentrations were not significantly different ($p< 0.05$) between the non-malarious and malarious subjects. In addition, marginal changes 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 21-31 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±1.25mg/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 changes 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 e*t al.,* 2004; Maina *et al.,* 2010). The present study reported haematologic and biochemical changes 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 e*t 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 the same pattern as described above. Moderate infection caused reduction in haemoglobin concentration, which was more pronounced in adolescents between the age brackets of 11-20 years than their adult counterparts of age brackets between 21-30 years ($p< 0.05$) (Table 1). According to reported 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 e*t 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 e*t al.,* 2004).

Therefore, the interplay of these multifactorial etiologies of anaemia in malaria infection, as described above, was responsible for 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 monitor 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 separate reports. 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) andSumbele *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 reliable biochemical marker for establishing sever 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 experimental subjects, 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 in-fusion 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 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 of 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 changes 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 changes in blood profile of malaria infected individuals habitat in Owerri Municipality.

ACKNOWLDGEMENT

We thank Professor A. A. Uwakwe, Professor of Medical Biochemistry, University of Port Harcourt, for providing us with useful information for the preparation of this article. We also appreciate the efforts of our undergraduate students.

REFERENCES

Adesina, K.T., Balogun, O.R., Babatunde, A.S., Sanni, M.A., Fadeyi A. and Aderibigbe, S. 2009. Impact of malaria parasitaemia on haematologic parameters in pregnant women at booking in Ilorin, Nigeria.*Trends in Medical Research. 4,* 84-90.

Afolabi, B.M. 2001. Malaria: The global scourge. *Nigeria Clinic*. 5, 9-12.

Ali, H.A., Ahmed, M.S.A. Jawahar, L.G. Abdulla, M.U., Nadeem, J.Y. and Hina, S.H. 2009.Hematological and biochemical changes in typhoid fever. *Pakistan Journal of Medical Science.* 25, 166-171.

Ali, M.S.M and Kadaru, A.G.M. 2005.*in vitro* processing of donor blood with sulphadoxine/pyrimethamine for eradication of transfusion induced malaria. *America Journal of Tropical Medicine and Hygiene.* 73(6), 1119 – 1123.

al-Yaman. F., Awburn, M.M. and Clark, I.A.1997.Serum creatinine levels and reactive nitrogen intermediates in children with cerebral malaria in Papua New Guinea. *Transactions of the Royal Society of Tropical Medicine and Hygiene.* 91(3), 303-305.

Amah, U.K., Ahaneku, J.E., Usoro, C.A.O., Ezeoke, A.C.J., Okwara, J.E., Amah, A.K., Etukudo, M.H., Okwara, E.C., Amah, B.C. Comparative study of C-reactive protein and other biochemical parameters in patients with hepatitis B and malaria in Calabar, Nigeria.*Nigeria Journal of Physiological Science.*26, 109 – 112.

Bartels, H., Bohmer, M. and Heierli, C. 1972.Serum creatinine determination without protein precipitation*. Clinica Chimica Acta*, 37, 193-197.

Baure, J.D. 1980.Laboratory investigation of hemoglobin. In: *Gradwohl's Clinical Laboratory Methods and Diagnosis*, (Editors) Sonnenwirth, A.C. and Jarett, L. St. Louis, MO: Mosby.

Beutler, B. and Cerami, A. 1987.Cachectio: More than a tumor necrosis factor. *New England Journal of Medicine*.379-85.

Bidaki, Z.M. and Dalimi, A.A. 2003. Biochemical and hematological alteration in *Vivax* malaria in Kahnouj city*. Journal of Rafsanjan University Medical Science*. 3, 17-24.

Binh, T.Q., Davis, T.M., Johnston, W., Thu, L.T., Boston, R., Danh, P.T. and Anh, T.K. 1997. Glucose metabolism in severe malaria: minimal model analysis of the intravenous glucose tolerance test incorporating a stable glucose label. *Metabolism*. 46(12), 1435-40.

Bottiger, L.E. and Svedberg, C.A. 1967.Normal erythrocyte sedimentation rate and age.*British Medical Journal*. 2, 85–87.

Chandra, S. and Chandra, H. 2013.Role of haematological parameters as an indicator of acute malarial infection in Uttarakhand State of India. Mediterranean *Journal of Hematology and Infectious Diseases.* 5(1): e2013009, DOI 10.4084/MJHID.2013.009.

Cheesbrough, M. 1998. *District laboratory practice in tropical countries*. Cambridge University Press, Cambridge.

Cheung, K. and Hchman, P.E. 1996. Methods of Analysis of Albumin: Liver Function. In: *Clinical chemistry, theory, analysis and correlation*. Kaplan, L.A. and Pesce, A.J. (Eds.). 3rd Edn, Mosby, London.

Chikezie, P.C. 2009. Comparative methaemoglobin concentrations of three erythrocyte genotypes (HbAA, HbAS and HbSS) of male participants administered with five antimalarial drugs. *African Journal of Biochemistry Research.* 3(6), 266-271.

Crawly J. 2004. Reducing the burden of malaria in infants and young children in malaria endemic countries of Africa: from evidence to action. *America Journal of Tropical Medicine and Hygiene*.71 (2 Suppl), 25 -34.

Das, B.S., Nanda, N.K., Rath, P.K., Satapathy, R.N. and Das, D.B. 1999. Anaemia in acute, *Plasmodium falciparum* malaria in children from Orissa state, India. *Annals of Tropical Medicine and Parasitology.*93,109–119.

Das, B.S., Thurnham, D.J. and Das, D.B. 1997. Influence of malaria on markers of iron status in children: implications for interpreting iron status in malaria–endemic communities. *British Journal of Nutrition*. 78, 751-760.

Doumas, B.T., Watson, W.A. and Biggs, H.G. 1971. Albumin standard and measurement of serum albumin with bromocresol green. *Clinica Chimica Acta*. 31, 87-96.

Dreyer, S.J. and Boden, S.D. 2003.Laboratory evaluation in neck pain.*Physical Medicine and Rehabilitation Clinics of North America.* 14, 589–604.

Egwunyenga, A.O., Isamah, G. and Nmorsi, O.P. 2004. Lipid peroxidation and ascorbic acid levels in Nigeria children with acute falciparum malaria.African Journal of Biotechnology. 3, 560-563.

Eiam-Omg, S. and Sitprija, V. 1998. Falciparum malaria and the kidney: a model of inflammation. *American Journal of Kidney Diseases* 32, 361–375.

Emian-Ong, S. 2002.Current knowledge of falciparum malaria-induced acute renal failure.*Journal of Medical Association of Thailand*. Suppl., 16, 24.

Erhart, L.M., Yingyuen, K., Chuanak, N., Buathong, N., Laoboonchai, A., Miller, R.S., Meshnick, S.R., Gasser, Jr R.A. and Wongsrichanalai, C. 2004. Hematologic and clinical indices of malaria in a semi-immune population of western Thailand.*America Journal of Tropical Medicine and Hygiene*. 70(1): 8–14.

Ezechukwu, C., Ekejindu, E., Ugochukwu, E. and Oguatu, M. 2004. Congenitally acquired malaria in hyperendemic area. A Cohort study: *Tropical Journal of Medical Research*. 8(2), 44-48.

Facer, C.A. 1994.Hematological aspects of malaria. In: *Infection and Hematology*. Oxford: Butterworth Heinemann Ltd.

Fawcett, J.K. and Scott, J.E. 1960. A rapid and precise method for the determination of urea.*Journal of Clinical Pathology*. 13, 156-159.

Gu¨nther, A. Burchard, G. D. Slevogt, H. Abel, W. and Grobusch, M. P. 2002. Renal dysfunction in falciparum–malaria is detected more often when assessed by serum concentration of cystatin C instead of creatinine. *Tropical Medicine and International Health*. 7(11), 931–934.

Idonije, O.B., Festus, O., Okhiai, O. and Akpamu, U. 2011. Comparative study of the status of a biomarker of lipid peroxidation (malondialdehyde) in patients with *Plasmodium falciparum* and *Plasmodium vivax* malaria infection.*Asian Journal of Biological Sciences*. 4, 506-513.

Ikekpeazu, E.J., Neboh, E.E. Maduka, I.C. Nwagbara, I.J. and Nwobodo, M.W. 2010.Type-2 diabetes mellitus and malaria parasitemia: Effect on liver function tests. *Asian Journal of Medical Sciences.* 2(5), 214-217.

Joseph, V., Varma, M., Vidhyasagar, S. and Mathew, A. 2011. Comparison of the clinical profile and complications of mixed malarial infections of *Plasmodium falciparum* and *Plasmodium vivax* versus *Plasmodium falciparum* mono-infection. *Sultan Qaboos University Medical Journal.* 11(3), 377–382.

Kayode, O.T. Kayode, A.A.A. and Awonuga, O.O. 2011. Status of selected hematological and biochemical parameters in malaria and malaria-typhoid co-infection. *Journal of Biological Sciences.*11,367-373.

Kazmierczack, S.C., 1996. *Methods of analysis of creatinine, urea, total protein, inorganic phosphate, calcium and pH.: renal function*. In: Clinical Chemistry, Theory, Analysis and Correlation, Kaplan, L.A. and A.J. Pesce, (Eds.). 3rd Edn. Mosby, London.

Krotoski, W.A., W.E. Collins, R.S. Bray, P.C. Garnham and F.B. Cogswell. 1982. Demonstration of hypozoites in sporozoite transmitted *Plasmodium vivax* infection. *America Journal of Tropical Medicine and Hygiene.* 31, 1291-1293.

Kwena, A., Wakhisi, J. and Mambo, F. 2012. Possible Biochemical Markers in *Plasmodium falciparum* Malaria Infected Children with or without Malnutrition at Webuye and Eldoret, Western Kenya. *Advances in Bioresearch.*3(2), 49 – 54.

Kwiatkoski, D. 1989. TNF production in falciparum malaria and its association with schizont rupture. Clinical and Experimental Immunology. 77, 391-396.

Maina, R.N, Walsh, D., Gaddy, C., Hongo, G., Waitumbi, J., Otieno, L., Jones, D. and Ogutu, B.R. 2010.Impact of *Plasmodium falciparum* infection on haematological parameters in children living in Western Kenya. *Malaria Journal.*9, S4. http://dx.doi.org/10.1186/1475-2875-9-S3-S4.

Malcolm, L. and Brigden, M.D. 1999.Clinical utility of the erythrocyte sedimentation rate.*American Family Physician.*60(5), 1443-1450.

Martin, D.W. 1983. Blood plasma and clotting. In: Martin, D.W., Mayes, P. A. and Rodwell, V.W. editors. *Harper's Review of Biochemistry*. 9th ed. California: Lange. Medical Publications.

Menendez, C., Fleming, A.F., Alonso, P.L. 2000.Malaria-related anaemia.*Parasitology Today.*16,469–476.

Mishra, S.K., Mohaptra, S., Mohantu, S., Patel, N.C. and Mohaptra, D.N. 2002. Acute renal failure in *falciparum* malaria.*Journal, Indian Academy of Clinical Medicine.* 3, 141-147.

Mizushima, Y., Kato, H., Ohmae, H., Tanaka, T., Bobogare, A. and Ishii, A. 1994.Prevalence of malaria and its relationship to anaemia, blood glucose levels and serum somatomedin C (IGF-1) levels in the Solomon Islands. *ActaTropica.* 58(3-4), 207-210.

Mockenhaupt, F., Ehrhardt, S., Burkhardt, J., Bosomtive, S., Laryea, S., Anemana, S., Otchwemah, R., Cramer, J., Dietz, E., Gellert, S. and Bienzle, U. 2004. Manifestation and outcome of severe malaria in children in Northern Ghana. *America Journal of Tropical Medicine and Hygiene*. 71(2): 167-172.

Mönig, H., Marquardt, D., Arendt, T. and Kloehn, S. 2002 Limited value of elevated erythrocyte sedimentation rate as an indicator of malignancy. *Family Practice*. (5), 436-438.

National Committee for Clinical Standards. 1993. *Reference procedure for erythrocyte sedimentation rate (ESR) test*, 3rd ed. H2-A3. Villanova, Pa. NCCLS.

Ogbodo, S. O., Okeke, A. C., Obu, H. A, Shu, E.N. and Chukwurah E.F. 2010.Nutritional status of parasitemic children from malaria endemic rural communities in eastern Nigeria. *Current Pediatric Research.*14(2), 131-135.

Onyesom, I. and Agho, J.E. 2011.Changes in serum glucose and triacylglycerol levels induced by the co-administration of two different types of antimalarial drugs among some malarial patients in Edo-Delta Region of Nigeria.*Asian Journal of Science Research.* 4, 78-83.

Onyesom, I. and Onyemakonor, N. 2011.Levels of parasitaemia and changes in some liver enzymes among malarial infected patients in Edo-Delta Region of Nigeria. *Current Research Journal of Biological Sciences* 3(2), 78-81.

Onyesom, I., Ekeanyanwu, R.C. and Achuka, N. 2010.Correlation between moderate *Plasmodium falciparum* malarial parasitaemia and antioxidant vitamins in serum of infected children in South Eastern Nigeria. *African Journal of Biochemistry Research.* 4(12), 261-264.

Perrin, L.H., Mackey, L.J. and Miescher, P.A. 1982. The hematology of malaria in man.*Semin Hematology.* 19,70–82.

Phillips, R.E. and Pasvo, G. 1992. Anaemia of *Plasmodium falciparum* malaria.*Baillieres Clinical Haematology* 5,315–330.

Price, R.N., Simpson, J.A., Nosten, F., Luxemburger, C., Hkirjaroen, L., Kuile, F., Chongsuphajaisiddhi, T. and White, N.J. 2001.Factors contributing to anaemia after uncomplicated falciparum malaria. *America Journal of Tropical Medicine and Hygiene*.65,614–622.

Richards, M.W., Behrens, R.H. and Doherty, J.F. 1998. Short report: Hematologic changes in acute, imported *Plasmodium falciparum* malaria. *America Journal of Tropical Medicine and Hygiene*.59, 859.

Rojanasthien, S., Surakamolleart, V., Boonpucknavig, S. and Isarangkura, P. 1992. Hematological and coagulation studies in malaria.*Journal of Medical Association of Thailand. 75,* 190–194.

Scuderi, P., Sterling, K., Lamks, S. 1986. Raised serum levels of TNF in parasitic infections.Lancet. 2, 1364-1365.

Selvam, R. and Baskaran, G. 1996. Hematological impairments in recurrent *Plasmodium vivax* infected patients. *Japan Journal of Medical Science and Biology*. 49,151–165.

Sitprija, V. 1988.Nephrology in *Falciparum* malaria.*Kidney International.* 34, 867-877.

Strickland, G.T. 1991. *Life cycle of malaria parasite*. In: Tropical Medicine. 7th Edn, W.B. Saunders, USA, 586-601.

Sumbele, I.U.N., Theresa, N.A. Samje, M. Ndzeize, T. Ngwa, E.M. and Titanji, V.P.K. 2010. Hematological changes and recovery associated with untreated and treated plasmodium falciparum infection in children in the mount Cameroon region. *Journal of Clinical Medical Research.*2, 143-151.

Supcharoen, O., Widjaja, H., Ali, K.B., Kitayaporn, D., Pukrittayakamee, S., Wilairatana, P Sathawarawong, W.1992.A study of erythrocyte sedimentation rate in malaria.*Journal of Infectious Disease and Anti microbrobial Agents.*9(4), 193-199.

Tracy, J.W. and Webster, L.T. 2001. Drugs used in the chemotherapy of protozoan infections. In: (Adam, J.G., Limbird, L.E. and Gilman, A.G. (Eds).*Goodman and Gilman's Pharmacological Basis of Therapeutics*.10th Edition, McGraw-Hill, U.S.A.

Udosen, E.O. 2003. Malaria treatment using oral Medkafin: Changes in biochemical and hematological parameters in Nigerian children with uncomplicated falciparum malaria. *Orient Journal of Medicine.* 15, 12-22.

Van den Broek, N.R. and Letsky, E.A. 2008. Pregnancy and the erythrocyte sedimentation rate.*BJOG: An International Journal of Obstetrics and Gynaecology*. 108(11), 1164–1167. DOI: 10.1111/j.1471-0528.2003.00267.x.

WHO 2005. Susceptibility of *Plasmodium falciparum* to antimalarial drugs: report on global monitoring, 1996–2004.WHO/HTM/MAL/2005.1103.

WHO 2008.Severe *P. falciparum* malaria.*Transaction of the Royal Society of Tropical Medicine and Hygiene*. 94, 51-59.

Wickramasinghe, S.N. and Abdalla, S.H. 2000.Blood and bone marrow changes in malaria.*Baillieres Best Pract Research Clinical Haematology.* 13, 277–299.

W.M.A. 2000.World medical association declaration of Helsinki ethical principles for medical research involving human subjects.52nd WMA General Assembly, Edinburgh, Scotland.

Corresponding author: P.C. Chikezie,Department of Biochemistry, Imo State University, Owerri, Nigeria.
E-mail:p_chikezie@yahoo.com; **Phone:** +2348038935327.