

Original Research Article**EFFECTS OF CHILDHOOD MALARIA ON THE BIOCHEMICAL AND
HAEMATOLOGICAL PROFILES OF INFECTED CHILDREN IN ANAMBRA STATE,
NIGERIA****ABSTRACT**

This study was carried out to establish data on the effect of childhood malaria on plasma levels of some haematological and biochemical parameters in malaria infected children of Anambra State, Nigeria. The levels of sodium ion (Na^+), bilirubin, alkaline phosphatase (ALP), serum glutamate oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), total iron concentration (TIC), haemoglobin (Hb), packed cell volume (PCV) and white blood cell (WBC), in blood samples of malaria parasitaemic children and control were evaluated using standard methods. Chi-square, Fisher least significance difference, t-test, analysis of variance and multiple regressions were employed to test significant differences among the variables. For all determination, the significant difference was set at $p < 0.05$. The female children infected with malaria parasite had significantly ($p < 0.05$) higher mean PCV than the male in both communities (31.73 ± 4.58 to 28.25 ± 3.75) and hospitals (31.12 ± 11.78 to 28.27 ± 5.56) surveyed, respectively. Also the female infected children had a significantly ($p < 0.05$) higher WBC count than males. Serum levels of WBC in malaria infection were gametocyte count dependant. Gametocyte count of 1 – 10 and 11 – 100 in both communities and hospitals surveyed respectively had significant ($p < 0.05$) different WBC count. In the age group comparison, children 0 – 4.9 years had significant higher ($p < 0.05$) mean serum level of bilirubin (0.55 ± 0.25 to 0.35 ± 0.14) ($p < 0.05$). Children aged 10 – 14.9 years old had significantly ($p < 0.05$) higher mean level of Hb (10.30 ± 0.29 to 10.90 ± 0.26) than other age groups in the survey. Comparing the parameters with age showed that the PCV and Hb of the malaria infected children aged 5 – 9.9 years were significantly ($p < 0.05$) lower than the control. Sex and age may be a determining factor for low PCV since the male infected children had a significant lower PCV in both community and hospital surveys. Hypernatraemia was generally observed in malaria infection amongst children in Anambra State.

INTRODUCTION

Malaria affects billions of people, or half of the world's population, in 106 countries and territories. It is a major public health problem and cause of morbidity and mortality particularly among children in tropical and subtropical regions of the world (United States Embassy in Nigeria. (2011). Malaria parasites target red blood cells (RBCs), biochemical and haematological changes are one of the most common complications and thus play a major role in the outcomes of the disease. Malaria is caused by a protozoan parasite of the genus: *Plasmodium*. *P. falciparum*, *P. ovale*, *P. vivax*, and *P. malariae* are the most common species, with *P. falciparum* being the most virulent. Haematological alterations that are thought to characterize malaria may be related to the overt biochemical changes that occur during the asexual stage of the life cycle of the malaria parasite. In Anambra State and other documented endemic sites in Nigeria, malaria is holoendemic and the number of asymptomatic cases in children and adults are on the increase (Ogunledun *et al.*, 1990; Iwalokun *et al.*, 2001). It is a common knowledge that malaria infection provokes morphological, ultra structural and biochemical changes in erythrocyte membrane to enable compartmentalization, growth, cell differentiation, increased nutrient uptake and survival of the parasite (Sherman and Greenan, 1984; Gero and Kirk, 1994; Iwalokun, *et al.*, 2002). Malaria pathogenesis is based mainly on extensive changes in biochemical and haematological parameters (Bidaki and Dalimi, 2003; Adeosun *et al.*, 2007). WHO's criteria acknowledge that some biochemical and haematological features should raise the suspicion of severe malaria (WHO, 2000). Ogunledun *et al* (1990) reported that diagnosis of malaria parasite in the blood film could be made with certainty on identification in blood films along with signs and symptoms associated with the disease.

Entry of *P. falciparum* into erythrocytes usually leads to a marked increase in secretion of inflammatory cytokines (TNF α , IL-1, IL-10, and IFN γ), endothelial cell activation (due to over expression of cell adhesion molecules; ICAM-1, VCAM-1), activation of the coagulation cascade (due to platelet consumption and endothelial damage), and sequestration of parasitized RBCs (due to over expression of cell adhesion molecules, pfEMP, and iNOS). These along with other mechanisms set in motion events that ultimately result in morphological and numerical changes in the different blood cell lines (Haruna *et al.*, 2013).

Significant alterations in both biochemical and haematological variables have being reported during malaria infection (Wareel *et al.*, 1990; WHO, 2000; Bidaki and Dalimi, 2003; Mishra and Mohanty, 2003). However, few studies have been conducted to explore the possible biochemical

and haematological changes in children with the asymptomatic (communities) and symptomatic (hospitals) malaria parasites living in malaria-endemic area of Anambra State, Nigeria. Therefore, this work was carried out to investigate and compare the possible biochemical and haematological changes that may occur in home and hospital managed childhood malaria respectively in Anambra State.

MATERIALS AND METHODS

Study Area

The study area is Anambra State. Anambra is a State in South-eastern Nigeria with a population of about 4 million made up of 2,117,984 males and 2,059,844 females (population estimation of 2006) (Ministry of Lands and Survey, Awka (2010).

Study population

Eighty-two children aged 0 - 14.9 years from the sampled households in the communities and 166 of their counter parts that attended the out patient clinics of the selected General Hospitals in Anambra State were recruited into the study

Sampling Technique

Stratified random sampling was used. All hospitalized or non-hospitalized patients (children 0-14.9 years) with acute febrile illness were randomly sampled two times in a month from April 2012 to March 2013. Fifty homes were randomly sampled by balloting in each of the 13 communities for 12 months.

Ethical Clearance and Exclusion Criteria

Ethical clearance was obtained from the University of Nigeria Teaching Hospital Ituku - Ozalla in Enugu State, with which permission was obtained from the management of the selected General Hospitals for the study. Informed consent was obtained from the mothers/caregivers before the collection of blood samples. Children who have sickle cell haemoglobin were excluded from the study because of the fact that malaria parasites do not grow well in sickle Hb cells.

Collection of blood sample

With the help of the medical team 1ml venous blood were obtained from the children in the communities and hospitals after cleaning the site with spirit and kept in ethylenediamine tetra-acetic disodium acid (EDTA) vacutainers to avoid clotting and ensure preservation of the

samples. Their names, age and sex were recorded against the samples. Blood was sampled from malaria symptomatic and non symptomatic children aged 0 – 14.9 years.

Preparation of thick films blood smears for microscopy

Thick film blood smears were prepared from the blood samples according to Sood (2006). Large drop of blood samples were deposited at one end of the slide and were spread out evenly with the corner of another slide to a diameter of about 20mm. They were put in distilled water for 10 minutes for dehaemoglobinisation, dried in a flat position to ensure even distribution of blood and stained with Giemsa's stain for 20 minutes. The stain was washed out with buffered water of pH 6.8 and stood upright to dry in the air, and viewed under x 100 objective (oil immersion) lens. The thick smears were used to confirm the presence or absence of malaria parasite. The asexual forms of the parasite were counted in 200 leucocytes. The degree of parasitaemia were graded according to the number of parasite per micro litre thus, 1-999 (+), 1000-9999 (++) and >100000 (+++) (Cheesbrough, 2006).

Biochemical analysis from blood sample

Four ml blood samples were obtained from the sampled children from the different communities and hospitals and put in a cleaned dried test tube. The blood samples were centrifuged for 45 minutes and serum separated into plain clean glass tube for biochemical tests. Parameters determined were: Sodium ion (Na^+), Bilirubin, Alkaline Phosphatase (ALP), Serum Glutamate Oxaloacetate Transaminase (SGOT), Serum Glutamate Pyruvate Transaminase (SGPT) and Total iron concentration in blood samples of malaria infected children and control. They were evaluated using the method of Sood (2006).

Data Analysis

T- test was used to determine the haematology, biochemical and parasite density of *Plasmodium* infected and uninfected children, infected males and females, the different *Plasmodium* gametocyte count and control, and between gametocyte count levels for both community and hospital surveys. Analysis of variance (ANOVA) was used to compare the effects of the different factors (sex, gametocyte count and age) on the haematology and biochemical parameters.

RESULTS

Children (asymptomatic and symptomatic) between the ages of 0 -14.9 (4.12 ± 0.27) years were sampled from thirteen communities and eleven General Hospitals in Anambra State. A total of eighty-two children were sampled in the community which include 39 males (47%)

and 43 females (53%). One hundred and sixty six (166) children who attended the out patient clinic of General Hospitals/Comprehensive Health Centres in Anambra State were sampled, comprising of 95 males (57.2%) and 71 females (42.8%).

Gender based investigation of the biochemical and haematological indices, and the parasite density, of malaria infected male and female children in Anambra State, Nigeria (communities and hospitals) showed that there were no significant ($p > 0.05$) differences in the mean values of, ALP, SGOT, SGPT Na^+ , total protein, bilirubin, total iron and Hb of both sexes in the communities. The mean PCV and WBC of the children differed significantly ($p < 0.05$), with females having higher values of PCV (31.73 ± 4.58 to 28.25 ± 3.73 and WBC (5495.45 ± 1197.81 to 4631.25 ± 821.15), female children from the community survey had higher parasite density than male children but the difference in density was insignificant ($p > 0.05$).

Similarly, male infected children in the hospital survey had a higher mean parasite density than their female counter parts but the difference in density was not significant ($p > 0.05$). The mean PCV and SGOT of the children differed significantly ($p < 0.05$). The female infected children had higher mean PCV, while the males had higher SGOT (33.89 ± 32.61 to 23.29 ± 19.71) even though all were within the normal ranges. There were no significant differences ($p > 0.05$) in the mean levels of Hb, WBC, Na^+ total protein, bilirubin, ALP, SGPT and total iron of both sexes (Table 1).

The comparison of the effects of sex on the biochemical and haematological indices, and the parasite density of malaria infected male and female children in Anambra State, Nigeria is summarized in table 2. There was no significant difference ($p > 0.05$) in the ALP of both male and female malaria infected children from the communities and hospitals, however, there was a significant ($p < 0.05$) difference in the SGOT. SGOT of malaria infected males and females in the hospitals did not differ significantly ($p > 0.05$), but they differed significantly ($p < 0.05$) from SGOT of infected males and females from the communities. Similarly, Na^+ of infected males and females in the hospitals differed significantly ($p < 0.05$) from the Na^+ of malaria infected males and females in the communities. Only the infected female in the communities had a significant higher ($p < 0.05$) total protein more than the infected males and females in the communities and hospitals.

Infected males in the communities had significantly higher (515.50 ± 182.92) total iron more than the males and females in hospitals and also females in the communities, however, there was

no significant ($p>0.05$) difference in the level of bilirubin of both sexes in the communities and hospitals.

Also there were no significant differences in the PCV and Hb concentration of both sexes in the communities and hospitals, while there was significant differences ($p<0.05$) in the WBC of malaria infected males and females in the communities and males and females in the hospitals. The parasite density of malaria infected females in the hospitals (123140.91 ± 28706.33) was significantly higher ($p<0.05$) than females and males parasite density in the communities, but there were no differences in the parasite density of males and females in the hospitals.

The result of analyses based on malaria parasite count (Table 3) showed that the serum level of SGOT, SGPT, Na, total protein, bilirubin, and total iron of malaria infected children from the communities with 1-10 gametocytes were slightly elevated more than that of the uninfected children, however, the differences were not significant ($p > 0.05$). The malaria infected children had a higher significant white blood cell count ($p < 0.05$) than their uninfected counter part while the differences in their mean PCV (%) and Hb (g/dl) were not significant ($p> 0.05$). Mean serum levels of the liver enzymes of malaria infected and uninfected children with 1-10 gametocytes from the hospitals differed but the differences were not significant ($p> 0.05$). Also the serum level of ALP, SGOT and SGPT was higher in the malaria uninfected children of 11 – 100 gametocytes from the hospitals but the difference was insignificant ($p> 0.05$), however, there was a significant difference ($p<0.05$) in their WBC. Both malaria infected and uninfected children from hospital had the same mean PCV ($P = 29.33$).

The comparison of the effects of gametocytes count on the biochemical and haematological indices showed that there were no significant differences ($p>0.05$) in the ALP and SGPT of malaria infected children with 1 – 10 (communities), 1 – 10 (hospitals) and 11 – 100 (hospitals) gametocytes count, however, SGOT of children with 1 – 10 and 11 – 100 gametocytes in the hospitals differed significantly ($p<0.05$) from infected children with 1 – 10 gametocytes count in the communities. No significant difference in the SGOT of infected children with 1-10 and 11 – 100 gametocytes in the hospitals. Na^+ of malaria infected children in the hospitals with 1 – 10 and 11 – 100 gametocytes differed significantly ($p < 0.05$) from infected children with 1 – 10 gametocytes in the communities. Also total iron of infected children in the communities with 1 -10 gametocytes was significantly higher than their counter parts in the hospitals

On the other hand, there was no significant difference in the PCV and Hb concentration in the children with different gametocytes count in both communities and hospitals. WBC count of infected children with 1 – 10 and 11- 100 parasites count in the hospitals differed significantly ($p < 0.05$) from 1 – 10 parasites count children in the communities. The parasite density of infected children with 11 – 100 parasites count in the hospitals was significantly higher ($p < 0.05$) than 1- 10 counts in the communities and hospitals respectively, however, 1 -10 parasites count in the communities and hospital did not differ significantly ($p > 0.05$) (Tables 4).

Age – related biochemical and haematological indices, and parasite density of malaria infected and uninfected children showed that the level of the liver enzymes (ALP and SGOT), sodium ion and protein of the malaria infected children of 0.4.9 years from the communities were reduced compared to the uninfected but the differences were not statistically significant ($p > 0.05$). Also there were no significant differences ($p > 0.05$) in the serum levels of the PCV, Hb and WBC of the two groups however, the serum level of bilirubin (0.55 ± 0.25 to 0.35 ± 0.14) and parasite density differed significantly ($p < 0.05$). On the other hand malaria infected children between 5 - 9.9 years also in the communities had a higher level of bilirubin, ALP and SGOT more than the uninfected but the differences were not significant ($p > 0.05$). Total protein, total iron, PCV, Hb and WBC of malaria infected and uninfected children 5-9.9 years did not differed significantly ($p > 0.05$), however, their parasite density differed significantly ($p < 0.05$). An elevated level of serum liver enzymes was observed in the malaria infected children aged 10- 14.9 years in the communities but the differences were not significant ($p > 0.05$). They had higher serum level of sodium ion, lower total protein and total iron but the differences were not significant ($p > 0.05$). However, the mean Hb (10.30 ± 0.29 to 10.90 ± 0.26) of malaria uninfected children was significantly ($p < 0.05$) higher than the infected children who also had significant ($p < 0.05$) higher parasite density.

The malaria uninfected children of 0 – 4.9 years from the hospitals had higher mean serum levels of the liver enzymes, Na^+ , bilirubin, PCV, Hb and WBC but the differences were not significant ($p > 0.05$). Similarly, malaria infected children of 5 – 9.9 years from the hospitals had higher mean serum levels of liver enzymes but without significant ($p > 0.05$) difference. Their mean PCV and Hb differed significantly ($p < 0.05$) (Table 5).

Comparing of age - related biochemical and haematological indices, and parasite density of malaria infected children in the communities and hospitals in Anambra State, Nigeria (Table 6),

there was no significant difference ($p > 0.05$) in the ALP, SGPT, total protein and bilirubin concentration of malaria infected children aged 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years from the communities and hospitals. SGOT concentration of infected children aged 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years in the hospitals did not differ significantly ($p > 0.05$), but they differed significantly from SGOT of children aged 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years in the communities. Similarly SGOT of age groups 0 – 4.9 years and 10 – 14.9 years in the communities did not differ significantly from those aged 5 – 9.9 years. Also Na^+ of infected children aged 0 – 4.9 years and 5 – 9.9 years in the hospitals did not differ significantly ($p > 0.05$), they differed from Na^+ concentration of age groups 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years in the communities. Na^+ of age group 10 – 14.9 years in the hospitals did not differ significantly from that of 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years in the communities. Total iron concentration of infected children aged 5 – 9.9 years (438.87 ± 128.86) in the communities was significantly higher than the other age groups in the communities and hospitals respectively. Total iron of malaria infected children aged 0 – 4.9 years and 10 – 14.9 years in the communities and 10 – 14.9 years in the hospitals did not differ ($p > 0.05$) from those of 0 – 4.9 years and 5 – 9.9 years in the hospitals.

The PCV and Hb concentration in the different age groups of malaria infected children in both communities and hospitals did not differ significantly ($p > 0.05$). WBC of malaria infected children aged 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years from hospitals did not differ significantly ($p > 0.05$), but they differed ($p < 0.05$) from WBC of children aged 0 – 4.9 years, 5 – 9.9 years and 10 – 14.9 years from communities. Also the parasite density of children aged 10 – 14.9 years in the hospitals was ($p < 0.05$) higher than those of the other age groups from the communities and hospitals.

DISCUSSION

The assessment of haematological profile can be used to determine the effect of malaria parasite on the blood of male and female children. The significant decrease in male PCV in the communities showed that the males were slightly anaemic. Also there was a significant difference in the serum level of the WBC of both sexes even though both were within the normal range (4,500 – 10,000) (Sood, 2006). This result is consistent with Laura *et al.* (2004). This may be attributed to increasing breakdown of red blood cells thus resulting in anaemia in malaria which is a major cause of infant death in malaria endemic Africa. The no significant difference

in the serum levels of total protein, bilirubin and total iron between male and female infected and uninfected children respectively in these communities is in disagreement with Ikekpeazu *et al.* 2010. Both sexes had hypernatraemia with out significant difference in the serum level. However, infected females from the communities had significant higher total protein more than infected males and females from the hospitals. Sodium (Na^+) is a major cation of extra cellular fluid that plays a central role in the maintenance of normal distribution of water and osmotic pressure in various fluid compartments, the significant reduction can occur as a result of high temperature and this will led to disorder of water balance. Serum sodium concentration higher than normal can be caused by gastroenteritis, osmotic diuresis and pronounced sweating (Sood, 2006). The asymptomatic state of the infected children may have contributed to the insignificant changes observed in the biochemical parameters.

Based on parasite count, there was no significant difference in the mean serum level of total protein, total iron, ALP, SGOT, bilirubin and SGPT of the infected children with 1-10 gametocytes and the control group. However, in the comparison of the gametocytes count of only infected children in the communities and hospitals there were significant differences in the SGOT, Na^+ and total iron of children with 1 – 10 gametocytes count. This significance raise signifies hepatic dysfunction which is in agreement with Ashok *et al.* (1979). Transient derangement of liver function is a common feature of childhood malaria especially in *P. vivax* malaria. Higher than normal ALP levels may indicate liver disease among others while lower than normal levels may indicate protein deficiency and poor nutrition (Sood, 2006). The significant difference in the mean serum level of the WBC of infected children of 1-10 gametocytes is in line with Ladhani *et al.* (2002) and Pavithran (2005). Leucocytosis is associated with severity and mortality of childhood *falciparum* malaria (Pavithran, 2005).

In the comparison of the effects of malaria infection on the biochemistry and haematology based on age groups 0-4.9 and 5-9.9 years in the communities, all the parameters with the exception of bilirubin did not show any significant difference, but there was a significant difference between the parasite density of the infected and control in all the age groups. The result is in line with Orimadegun *et al.* (2008). High level of bilirubin can be caused by liver diseases, excessive haemolysis/destruction of RBCs (haemolytic jaundice), obstruction of the biliary tract (obstructive jaundice) and drug induced reactions (Sood, 2006). But in the older children (10-14.9 years), there was a significant difference in the Hb and a significant higher parasite density

which is consistent with Ekvall *et al.* (2001). Percentage parasitaemia was related to blood Hb and the levels of Hb were positively associated with age. Haemoglobinuria even though not observed in this study have been a marker of severe haemolysis in childhood malaria. The drop in Hb may be due to selective removal of parasites from infected RBCs by macrophages (pitting) (Ekvall *et al.* 2001).

In the hospital infection there was significant elevation in the serum level of the liver enzyme SGOT in the *Plasmodium* infected male more than their female counterpart. This result is consistent with Garba and Gregory (2006) and Ashok *et al.* (1979). This showed that malaria can bring about the inhibition of certain liver functions even in the absence of normal clinical signs of hepatic insufficiency; however, normal liver function may be restored after termination of the malaria attack. Long continued low-grade malaria infection can cause permanent liver damage (Ashok *et al.*, 1979). Similarly the significant difference between the PCV of the infected female and male in the hospitals is in line with Opara *et al.* (2006) and Jayant and Mani (2010). Generally, mild anaemia is very common in childhood malaria but may vary with Hb <5g/dl or Hct <15% in the presence of parasite count >10,000 (symptomatic and asymptomatic). Anaemia in malaria, however, is associated with combination of haemolysis of parasitized red blood cells, accelerated removal of both parasitized and innocent non parasitized red blood cells, deprived and ineffective erythropoiesis due to tumour necrosis factor and spleen phagocytises or pooling. This haematological assessment showed that sex can be a determinant of anaemia (PCV <15%g/dl). This is in line with Gayawan *et al.* (2014) who reported that other important risk factors to anaemia apart from sex include household wealth index and presence of fever in the last two weeks preceding the survey. Malaria parasitaemia significantly lowers the PCV of infants especially between the first six to ten months of life (Afolabi *et al.*, 1997). Further evaluation of the effects of sex on the haemoglobin concentration of malaria infected children in Anambra State showed no significant difference.

In the hospital assessment of the effects of malaria infection on the biochemical profile of children based on age, there was no significant difference between the infected and control. However, there was significant difference in the SGOT of infected children aged 5 - 9.9 years from the communities and hospitals in Anambra State. This result is in disagreement with Zaki *et al.* (2012) who reported a significant decrease in ALP, SGOT, total protein and total iron of malaria infected children of different age groups compared to the uninfected. The inconsistency

may be attributed to the fact that the children were already receiving treatment, because these parameters were normally found to be at their normal levels six weeks after treatment (Ashok *et al.*, 1979). Age was observed as a determinant of anaemia in childhood malaria in this study. The age group 5 – 9.9 years had a significant low PCV and Hb and this is in line with Opara *et al.*, (2006). Anaemia in childhood malaria is more prevalent in lower age groups of 0-10 months with fatal consequences (Afolabi *et al.*, 1997).

Their WBC serum level showed no significant difference in this study and is in agreement with Opara *et al.* (2006). Divergent views, have been expressed on WBC in malaria, some have reported leucopenia (Laura *et al.*, 2004), while others have reported Leukocytosis (Pavithran, 2005).

Gametocyte count was also used to determine the effects of malaria on the biochemical and haematological indices of malaria infected children and control. No alteration was observed in both the liver enzymes, and some other biochemical and haematological parameters of infected children with gametocyte 1-10 and 11-100 counts. This is in disagreement with Adeosun *et al.* (2007) and Kitua *et al.* (1997). Alteration in haematology (PCV % and Hb gm/dl) was related to parasite count and is very common in the presence of chronic and recurrent malaria (Jayant and Mani, 2010). The hypoproteinaemia reported in other studies may have resulted from occasional transitory acute glomerulonephritis, microscopic haematuria and mild proteinuria of less than 1g/24hrs (Adeosun *et al.*, 2007). Difference in the WBC blood level was also observed in the assessment based on parasite count and is in line with Ladhani *et al.* (2003). This may be associated with concurrent infections and/or poor prognosis. White blood cell count during malaria are generally characterized as being low (Laura *et al.*, 2004) a phenomenon that is widely thought to reflect localization of leucocytes away from the peripheral circulation and to the spleen and other marginal pools rather than actual depletion or stasis.

Conclusion: Malaria parasite infection (symptomatic and asymptomatic) may be associated with anaemia, and haemoglobinuria which may be as a result of increase in breakdown of parasitized and non parasitized red blood cells. Serum level of WBC in malaria infection is gametocyte count dependent. Sex and age may be a determining factor for low PCV since the male infected children had a significant lower PCV in both community and hospital surveys. Hypernatraemia

was generally observed in malaria infection amongst children in Anambra State. It is recommended that sex as determinant of anaemia in children should be further investigated.

REFERENCES

- Adeosun, O. G., Oduola, T., Akanji, B. O., Sunday, A. M., Udoh, S. J. and Bello, I. S. (2007). Biochemical alteration in Nigerian children with acute *falciparum* malaria. *African Journal of Biotechnology*, 6(7): 881 – 885.
- Afolabi, O. M., Salako, L. A. and Mafe, A. G. (1997). *Symptomatology of clinical malaria in the first 6 months of life in urban Nigeria. Abstracts of a two-day national symposium on malaria in Nigeria*. Nigeria Institute of Medical Research, Yaba. 13 – 15.
- Ashok, P., Aneja, S., Berry, A. M. and Ghosh, S. (1979). Hepatic dysfunction in childhood malaria. *Archives of Disease in Childhood*, 54: 139 – 141.
- Bidaki, Z. M. and Dalimi Ashl, A. H. (2003). Biochemical and haematological alteration in *vivax* malaria in Kahnonj city. *Journal of Rafanjan University of Medical Sciences*, 3(11): 17 - 24.
- Ekvall, H., Arese, P., Turrini, F., Ayi, K., Mannu, F., Premji, Z. and Bjorkman, A. (2001). Acute haemolysis in childhood *falciparum* malaria. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 95: 611 – 617.
- Garba, I. and Gregory, U. (2006). AST/ALT ratio in acute uncomplicated malaria infection. *The Internet Journal of Anaesthesiology*, 14: 2.
- Gayawan, E., Ekundayo, D. A. and Samson, B. (2014). Possible determinants of spatial pattern of anaemia among young children in Nigeria: Bayesian semi-parametric modelling. www.ncbi.nlm.nih.gov/pub_med/24486460 Accessed 20th May, 2014.
- Gero, A. and Kirk, K. (1994). Nutrient transport pathways in *Plasmodium* infected erythrocytes: What and where are they? *Parasitology Today*, 10: 395 - 399.
- Haruna, M.1., Sharif, K., Larry, F. S., Abdul, S., and Christine, N. (2013). How reliable are haematological parameters in predicting uncomplicated *Plasmodium falciparum* malaria in an endemic region. <http://dx.doi.org/10.1155/2013/673798>. Accessed 29th July, 2014.
- Ikekpeazu, E. J., Neboh, E., Aguchime, N. C., Maduka, I. C. and Anyanwu, E. G. (2010). Malaria parasitaemia effect on serum sodium and potassium levels. *Biology and Medicine*, 2(2): 20 - 25.
- Iwalokun, B. A. Adeola, S. A. and Hodona, S. O. (2001). The prevalence and significance of parasitaemia, symptomatic and asymptomatic cases of malaria in Lagos, Nigeria. *African Journal of Medical Pharmaceutical Sciences*, 3: 44-51

- Iwalokun, B. A., Ogunledun, A. and Bamiro, S. B. (2002). Serum electrolyte changes in Nigerian children with asymptomatic and symptomatic *falciparum* malaria. *Brazilian Journal of Medical and Biological Research*, 17: 15 – 320.
- Jayant, P. and Mani, B. (2010). Paediatric Infectious Diseases Conference 2010 Mumbai. *Paediatric on Call*, 1: 3 –5
- Kitua, A. Y., Smith, T. A., Alonso, P. L., Urassa, H., Masanja, H., Kimario, J. and Tanner, M. (1997). The Role of low level *Plasmodium falciparum* parasitaemia in anaemia among infants living in an area of intense and perennial transmission. *Tropical Medicine and International Health*, 2: 325 – 333.
- Laura, M., Kritsanai, Y., Niphon, C., Nilawan, B., Anintita. L., Scott, R. M., Steven, R., Meshnick Robert, A., Gasser, J.R. and Chansuda, W. (2004). Haematological and clinical indices of malaria in a semi - immune population of western Thailand. *American Society of Tropical Medicine and Hygiene*, 70(1: 8 - 14.
- Ladhani, S., Lowe, B., Cole, A. O., Kowuondo, K. and Newton, C. R. J. C. (2002). Changes in white blood cells and platelets in children with *falciparum* malaria. *British Journal of Haematology*, 119(3): 839 - 847.
- Ministry of Lands and Survey, Awka (2010). Map of Anambra State, Nigeria.
- Mishra, S. K. and Mohanty, S. (2003). Problems in the management of severe malaria. *The Internet Journal of Tropical Medicine*, 1(1): 1 - 10.
- Ogunledun, A., Kofie, B. A. K., Adetunji, J. A., Fakoya, E. A. O. and Bamgboye, E. A. (1990). Prevalence and significance of symptomatic and asymptomatic malaria parasitaemia in Sagamu, Nigeria. *Nigeria Journal of Parasitology*, 11: 149-158.
- Opara, K. N., Atting, I. A., Ukpoma, I. G., Nwabueze, A. A. and Inokon, I. I. (2006). Susceptibility of genetic indices to *falciparum* malaria in infants and young children in southern Nigeria. *Pakistan Journal of Biological Sciences*, 9: 452 –456.
- Orimedegun, A. E., Amodu, O. K., Olumese, P. E. and Omotade, O. O. (2008). Early home treatment of childhood fevers with in effective anti malarial drug is deleterious in the outcome of severe malaria. *Malaria Journal*, 29(7): 143.
- Pavithran, K. (2005). Haematological changes in malaria. *South East Asian Journal of Tropical Medicine and Public Health*, 19: 25 – 33.
- Sherman, I. W. and Greenan, J. R. T, (1984). Altered red cell membrane fluidity during schizogonic development of malarial parasites (*Plasmodium falciparum* and *P. lophurae*). *Transactions of Royal Society of Tropical Medicine and Hygiene*, 78: 641 – 644.
- Sood, R. (2006). *Textbook of Medical Laboratory Technology*. First Edition. Jaypee Medical Publisher, New Delhi, India.
- United States Embassy in Nigeria. (2011). Nigeria Malaria Fact Sheet. [www.http://nigeria.usembassy.gov](http://nigeria.usembassy.gov) Accessed 6th June, 2013.

Wareell, D. A., Molyneux, M. E. and Beals, P. F. (1990). Severe and complicated malaria. *Transactions of Royal Society of Tropical Medicine and Hygiene*, 84(2): 1 - 65.

World Health Organization, (2000). Severe *falciparum* malaria. *Transactions of Royal Society of Tropical Medicine and Hygiene*, 94(1): 1 – 90.

World Health Organization, (2015). Factsheet on the World Malaria Report 2014. WHO Geneva.

Zaki, H. Y., Abdalla, B. E. and Babkier, H. E. (2012). Biochemical profile of children with severe *Plasmodium falciparum* malaria in Central Sudan: a case control study. *Al NSeelaim Medical Journal*, 3(8): 185 – 627.

Table 1: Biochemical and haematological indices, and parasite density of malaria infected and control subjects in relation to sex in Anambra State, Nigeria

Variables	Infected males	Infected females Communities	Standard range	P – value
Sampled size (n)	16	22	–	0.05*
ALP (iu/l)	51.67 ± 11.21	54 .03 ± 13.61	20 - 140	0.574 ^{ns}
SGOT (µ /l)	5.72 ± 4.21	7.36 ± 8.23	8 – 40	0.472 ^{ns}
SGPT (µ /l)	4.84 ± 3.0	7.20 ± 12.09	5 – 35	0.452 ^{ns}
Na ⁺ (mmol/l)	155.25 ± 45.63	152.75 ± 67.40	133 – 155	0.8886 ^{ns}
Total protein (g/dl)	6.49 ± .23.38	6.62 ± 0.22	6.0 – 8.0	0.696 ^{ns}
Bilirubin	0.57 ± 33	0.47 ± 0.18	0.0 2 -10	0.239 ^{ns}

(mg/dl)				
Total iron (µg/dl)	475.12 ± 745.33	327.45 ± 80.46	6 – 150	0.360 ^{ns}
PCV (%)	28.25 ± 3.75	31.73 ± 4.58	25	0.018*
Hb (gm/dl)	9.88 ± 1.27	10.45 ± 1.79	10 – 13	0.28 ^{ns}
WBC (mcl)	4631.25 ± 821.15	5495.45 ± 1197.81	4,500 – 10,000	0.018*
Parasite density	1.24 x 10 ⁴ ± 1.34 x 10 ³	1.54 x 10 ⁴ ± 1.38 x 10 ³		0.511 ^{ns}
<u>Hospitals</u>				
Sampled size (n)	90	67	–	0.05*
ALP (iu/l)	63.95 ± 66.77	1.028 ± 7822.51	20 - 140	0.244 ^{ns}
SGOT (µ /l)	33.89 ± 32.61	23.29 ± 19.71	8 – 40	0.020*
SGTP (µ /l)	21.15 ± 56.94	14.29 ± 25.19	5 – 35	0.359 ^{ns}
Na⁺ (mmol/l)	244.50 ± 78.87	256.82 ± 88.34	133 - 155	0.403 ^{ns}
Total protein (g/dl)	8.66 ± 11.10	9.26 ± 9.56	6.0 – 8.0	0.734 ^{ns}
Bilirubin (mg/dl)	2.27 ± 13.41	0.80 ± 0.35	0.02 – 10	0.371 ^{ns}
Total iron (µg/dl)	223.22 ± 148.99	217.69 147.30	6 – 150	0.817 ^{ns}
PCV (%)	28.27 ± 5.56	31.12 ± 11.78	25	0.046*
Hb (gm/dl)	10.52 ± 9.93	9.87 ± 2.04	10 – 13	0.595 ^{ns}
WBC (mcl)	8377.78 ± 2587.48	8812.68 ± 2356.14	4,500 – 10,000	0.281 ^{ns}
Parasite density	7.21 x 10 ⁴ ± 1.94 x 10 ³	1.21 x 10 ⁵ ± 2.31 x 10 ³		0.078 ^{ns}

Table 2: Comparison of sex - related biochemical and haematological indices, and the parasite density of malaria infected male and female children in Anambra State, Nigeria

Parameters	Community	Hospital	Community	Hospital
ALP (iu/l)	54.44 ± 3.83 ^a	63.95 ± 7.04 ^a	52.00 ± 2.29 ^a	1042.37 ± 970.15 ^a
SGOT (µ /l)	4.38 ± 0.48 ^b	33.87 ± 3.44 ^a	8.34 ± 1.90 ^b	23.49 ± 2.44 ^a
SGPT (µ /l)	7.36 ± 3.56 ^a	21.15 ± 6.04 ^a	5.41 ± 0.65 ^a	14.42 ± 3.12 ^a
Na⁺(mmol/l)	145.59 ± 15.37 ^b	244.50 ± 8.42 ^a	161.24 ± 9.87 ^b	255.15 ± 10.94 ^a
Total protein (g/dl)	6.61 ± 0.15 ^b	8.66 ± 1.2 ^b	38.60 ± 32.07 ^a	9.26 ± 1.19 ^b
Bilirubin (mg/dl)	0.49 ± 0.3 ^a	2.27 ± 1.41 ^a	0.54 ± 0.70 ^a	0.81 ± 0.4 ^a
Total iron (µg/dl)	515.50 ± 182.92 ^a	223.22 ± 15.71 ^b	292.38 ± 21.12 ^b	219.30 ± 18.20 ^b

PCV (%)	30.94 ± 1.15 ^a	28.27 ± 0.59 ^a	29.71 ± 1.02 ^a	31.20 ± 1.46 ^a
Hb (gm/dl)	10.72 ± 1.15 ^a	10.52 ± 1.05 ^a	9.82 ± 0.39 ^a	9.88 ± 0.25 ^a
WBC (mcl)	5143.75 ± 330.27 ^b	8315.62 ± 286.59 ^a	5142.86 ± 222.49 ^b	8865.91 ± 287.20 ^a
Parasite density	11981.25 ± 1955.19 ^b	71989.11542.04 ^{ab}	15957.14 ± 3781.49 ^b	123140.91 ± 28706.33 ^a

N/B: Mean values in a row with different alphabets as superscript are significantly different (p <0.05)

Table 3: Biochemical and haematological indices of malaria infected and uninfected children with different gametocytes count in thick blood film in Anambra State, Nigeria (Communities and hospitals)

Variables	Malaria infected children	Uninfected children Communities 1-10 gametocytes	Standard range	P – value
Sampled size (n)	38	38	-	1.00
ALP (iu/l)	53.03 ± 12.55	55.18 ± 12.312	20 - 140	0.437 ^{ns}
SGOT (μ /l)	6.67 ± 6.18	6.28 ± 9.55	8 - 40	0.838 ^{ns}
SGPT (μ /l)	6.21 ± 9.38	5.09 ± 2.31	5 – 35	0.447 ⁿ

Na⁺(mmol/l)	153.80 ± 52.12	145.41 ± 25.60	133 – 155	0.347 ^{ns}
Total protein (g/dl)	6.49 ± 109.25	6.23 ± 11.27	6.0 – 8.0	0.366 ⁿ
Bilirubin (mg/dl)	0.51 ± 0.25	0.448 ± 0.16	0.02 – 10	0.163 ^{ns}
Total iron (µ g/dl)	389.63 ± 484.09	292. 43 ± 70.99	6 – 150	0.192 ^{ns}
PCV (%)	30.26 ± 454	30.14 ± 5.00	25	0.905 ^{ns}
Hb (gm/dl)	10.21 ± 1.60	10.33 ± 1.56	10 – 13	0.719 ^{ns}
WBC (mcl)	5131.58±1129.01	4551.14±1005.66	4,500 – 10,000	0.016 [*]
Hospitals				
<u>1 – 10 gametocytes</u>				
Sampled size (n)	104	90	-	0.128 ^{ns}
ALP (iu/l)	61.20 ± 60.65	75.78 ± 39.29	20 - 140	0.48 ^{ns}
SGOT (µ /l)	26.59 ± 29.32	23.44 ± 20.71	8 - 40	0.754 ^{ns}
SGPT (µ /l)	17.62 ± 50.17	17.93 ± 17.54	5 – 35	0.985 ^{ns}
Na⁺ (mmol/l)	255.91 ± 90.03	222.56 ± 90.34	133 – 155	0.289 ^{ns}
Total protein (g/dl)	7.87 ± 7.76	12.70 ± 17.49	6.0 – 8.0	0.118 ^{ns}
Bilirubin (mg/dl)	1.95 ± 12.49	1.14 ± 0.74	0.02 – 10	0.846 ^{ns}
Total iron (µ g/dl)	217.36 ± 152.66	248.76 ± 150.14	6 – 150	0.555 ^{ns}
PCV (%)	29.33 ± 10.186	29.33 ± 5.17	25	0.999 ^{-ns}
Hb (g/dl)	10.35 ± 9.29	9.89 ± 1.84	10 – 13	0.909 ^{ns}
WBC (mcl)	8204.8 ± 2619.51	7355.56 ± 4578.51	4,500 – 10,000	0.386 ^{ns}
<u>11-100 gametocytes</u>				
Sampled size (n)	53	9	-	0.044 [*]
ALP (iu/l)	1.29 ± 879.15	75.78 ± 39.29	20 140	0.683 ^{ns}
SGOT (µ /l)	34.78 ± 25.48	23.44 ± 20.71	8 - 40	0.211 ^{ns}
SGPT (µ /l)	19.01 ± 36.69	17.93 ± 17.54	5 – 35	0.932 ^{ns}
Na⁺ (mmol/l)	236.42 ± 67.87	222.56 ± 90.34	133 – 155	0.592 ^{ns}
Total protein	10.98 ± 15.38	12.70 ± 17.49	6.0 – 8.0	0.762 ^{ns}

(g/dl)				
Bilirubin	1.04 ± 0.43	1.14 ± 0.74	0.02 – 10	0.590 ^{ns}
(mg/dl)				
Total iron	227.72 ± 139.01	248.76 ± 150.14	6 – 150	0.680 ^{ns}
(µg/dl)				
PCV (%)	29.81 ± 5.41	29.33 ± 5.17	25	0.808 ^{ns}
Hb (gm/dl)	10.04 ± 1.80	9.10 ± 1.84	10 – 13	0.938 ^{ns}
WBC (mcl)	9266.98 ± 2071.46	7355.56 ± 4578.51	4,500 – 10,000	0.042*

Table 4: Comparison of malaria gametocytes count - related biochemical and haematological indices, and the parasite density of malaria infected children from communities and hospitals in Anambra State, Nigeria

Variables	Communities	Hospitals	
	1 – 10	1 -10	11- 100
Gametocytes count			
ALP (iu/l)	53.05 ± 2.09 ^a	65.07 ± 5.36 ^a	1280.18 ± 1208.13 ^a
SGOT (µ /l)	6.63 ± 1.13 ^b	29.11 ± 30.34 ^a	30.19 ± 24.16 ^a
SGPT (µ /l)	6.25 ± 1.56 ^a	18.65 ± 5.24 ^a	17.59 ± 4.09 ^a
Na+(mmol/l)	154.47 ± 8.66 ^b	255.74 ± 8.81 ^a	230.88 ± 6.26 ^a
Total protein (g/dl)	24. 77 ± 18.20 ^a	8.17 ± 0.99 ^a	10.36 ± 1.74 ^a
Bilirubin (mg/dl)	0.52 ± 0.42 ^a	2.07 ± 1.24 ^a	0.84 ± 0.41 ^a
Total iron (µ g/dl)	388.86 ± 80.76 ^a	211.56 ± 15.53 ^b	241.00 ± 17.37 ^b
PCV (%)	30.24 ± 0.76 ^a	30.04 ± 1.04 ^a	28.49 ± 3.80 ^a
Hb (gm/dl)	10.21 ± 0.27 ^a	9.70 ± 0.20 ^a	11.33 ± 1.76 ^a
WBC (mcl)	5143.24 ± 187.79 ^b	8703.45 ± 273.81 ^a	8247.17 ± 287.85 ^a
Parasite density	14237.84 ± 2303.59 ^b	66221.90 ± 4753.24 ^b	146896.23 ± 39208.35 ^a

N/B: Mean values in a row with different alphabets as superscript are significantly different (p <0.05)

Table 5: Biochemical and haematological indices of malaria infected and uninfected children of different age groups from the communities and hospitals in Anambra State, Nigeria

Variables	Infected children	Uninfected children	Standard range	P –value
	Communities			
	0 – 4.9 years			

Sampled size (n)	11	15	-	0.514 ^{ns}
ALP (iu/l)	57.03 ± 14.57	62.36 ± 11.87	20 – 140	0.314 ^{ns}
SGOT (u/l)	9.72 ± 11.29	4.05 ± 1.97	8 – 40	0.067 ^{ns}
SGPT (u/l)	4.65 ± 3.32	6.27 ± 2.19	3 – 35	0.571 ^{ns}
Sodium (mmol/l)	157.82 ± 47.11	144.67 ± 10.97	133 – 155	0.304 ^{ns}
Total protein (g/dl)	6.37 ± 0.60	6.46 ± 0.63	6.0 – 8.0	0.703 ^{ns}
Bilirubin (mg/dl)	0.55 ± 0.25	0.35 ± 0.14	0.02 – 10	0.016*
Total iron (ug/dl)	328.82 ± 80.55	283.07 ± 50.22	6 – 150	0.087 ^{ns}
PCV (%)	29.91 ± 4.70	29.07 ± 6.49	25	0.718 ^{ns}
Hb (gm/dl)	9.92 ± 1.83	9.93 ± 2.02	10 – 13	0.99 ^{ns}
WBC(mcl)	5745.45 ± 997.36	4880.00 ± 120 to 46	4,500 – 10,000	0.64 ^{ns}
<u>5 – 9.9 years</u>				
Sampled size (n)	23	24	–	0.982 ^{ns}
ALP (iu/l)	50.20 ± 9.97	52.53 ± 11.81	20 – 140	0.470 ^{ns}
SGOT (u/l)	5.00 ± 3.31	7.85 ± 12.72	8 – 40	0.305 ^{ns}
SGPT (u/l)	7.09 ± 11.85	5.08 ± 2.62	5 – 35	0.423 ^{ns}
Sodium (mmol/l)	146.98 ± 41.49	147.96 ± 33.47	133 – 155	0.929 ^{ns}
Bilirubin (mg/dl)	0.45 ± 0.17	0.48 ± 0.15	0.02 – 10	0.548 ^{ns}
Total protein (g/dl)	36.02 ± 140.39	11.44 ± 15.03	6.0 – 8.0	0.398 ^{ns}
Total iron (ug/dl)	438.87 ± 617.98	284.50 ± 79.68	6 – 150	0.231 ^{ns}
PCV (%)	30.39 ± 4.92	30.50 ± 4.41	25	0.939 ^{ns}
Hb (gm/dl)	10.33 ± 1.65	10.471 ± 1.37	10 – 13	0.752 ^{ns}
WBC(mcl)	4782.61 ± 1021.43	4316.67 ± 899.76	4,500 – 10,000	0.102 ^{ns}
<u>10 – 14.9 years</u>				
Sampled size (n)	4	5	-	0.989 ^{ns}
ALP (iu/l)	58.33 ± 18.69	46.40 ± 1.67	20 – 140	0.192 ^{ns}
SGOT (u/l)	7.82 ± 3.02	5.48 ± 1.21	8 – 40	0.152 ^{ns}
SGPT (u/l)	5.43 ± 1.77	4.58 ± 0.89	5 – 35 u/l	0.377 ^{ns}
Sodium (mmol/l)	182.00 ± 109.526	135.40 ± 3.578	133 – 155	0.365 ^{ns}
Bilirubin (mg/dl)	0.78 ± 0.49	0.60 ± 0.53	0.02 – 10	0.439 ^{ns}
Total protein (g/dl)	6.10 ± 1.54	6.90 ± 0.43	6.0 – 8.0	0.293 ^{ns}
Total iron (ug/dl)	273.75 ± 134.25	358.60 ± 48.58	6 – 150	0.226 ^{ns}
PCV (%)	30.50 ± 1.73	31.6 ± 1.14	25	0.287 ^{ns}
Hb (gm/dl)	10.30 ± 0.29	10.90 ± 0.26	10 – 13	0.013*
WBC(mcl)	5450.0 ± 1517.67	4690.0 ± 712.74	4,500 – 10,000	0.350 ^{ns}
<u>Hospitals</u>				
<u>0 – 4.9years</u>				
Sampled size (n)	93	6		
ALP (iu/l)	65.77 ± 56.85	81.83 ± 47.52	20 – 140	0.501 ^{ns}
SGOT (μ /l)	26.88 ± 30.21	30.42 ± 22.60	8 – 40	0.779 ^{ns}
SGPT (μ /l)	18.50 ± 55.22	24.68 ± 18.10	5 – 35	0.786 ^{ns}
Na⁺(mmol/l)	255.24 ± 90.53	188.33 ± 93.90	133 – 155	0.83 ^{ns}
Total protein	8.17 ± 10.58	15.90 ± 21.19	6.0 – 8.0	0.110 ^{ns}

(g/dl)					
Bilirubin		2.21 ± 13.10	1.37 ± 0.82	0.02 – 10.0	0.876 ^{ns}
(mg/dl)					
Total	iron	211.22 ± 163.69	325.80 ± 120.48	6 – 150	0.096 ^{ns}
(µg/dl)					
PCV (%)		29.90 ± 10.98	27.00 ± 3.46	25	0.522 ^{ns}
Hb (gm/dl)		9.61 ± 2.08	9.15 ± 1.32	10 – 13	0.598 ^{ns}
WBC (mcl)		8909.14 ± 2544.13	8383.33 ± 5330.45	4,500-10,000	0.652 ^{ns}
<u>5 – 9.9 years</u>					
Sampled size (n)		41	3	-	0.01*
ALP (iu/l)		16.33 ± 9999.93	63.67 ± 14.15	20 – 140	0.789 ^{ns}
SGOT (µ /l)		31.53 ± 18.49	4.73 ± 1.37	8 – 40 µ /l	0.330 ^{ns}
Na⁺(mmol/l)		252.5 ± 74.62	291.00 ± 7.94	133 - 155	0.382 ^{ns}
Total protein		10.65 ± 13.61	6.30 ± 2.86	6.0 – 8.0	0.588 ^{ns}
(g/dl)					
Bilirubin		0.75 ± 0.28	0.68 ± 0.19	0.02 – 10	0.710 ^{ns}
(mg/dl)					
Total	iron	204.25 ± 114.69	94.67 ± 2.136	6 – 150	0.109 ^{ns}
(µg/dl)					
PCV (%)		29.00 ± 4.03	34.00 ± 5.29	25	0.048*
Hb (gm/dl)		9.90 ± 1.32	11.67 ± 1.67	10 – 13	0.032*
WBC (mcl)		8029.27 ± 2482.36	5300.00 ± 1819.341	4,500 - 10,000	0.070 ^{ns}

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522 Table 6: Comparison of age - related biochemical and haematological indices, and parasite

523 density of malaria infected children in the communities and hospitals in Anambra State, Nigeria

Variables		Age Groups (Years)			
		0 – 4.9		5 – 9.9	
		Communities	Hospitals	Communities	Hospitals
		10 – 14.9		Communities	Hospitals

ALP (iu/l)	57.30 ± 4.78 ^a	66.67 ± 5.93 ^a	50.26 ± 2.07 ^a	1632 ± 1561.76 ^a	58.50 ± 9.43 ^a	64.89 ± 2.97 ^a
SGOT μ/l)	9.88 ± 3.76 ^{bc}	27.15 ± 3.16 ^{ab}	5.00 ± 0.69 ^c	31.39 ± 3.6 ^a	7.83 ± 1.51 ^{bc}	35.36 ± 5.85 ^a
SGPT (μl)	4.65 ± 1.11 ^a	18.93 ± 5.85 ^a	7.09 ± 2.47 ^a	14.86 ± 2.90 ^a	5.43 ± 0.88 ^a	21.84 ± 8.25 ^a
Na+(mmol/l)	160.70 ± 15.38 ^b	258.35 ± 9.65 ^a	146.98 ± 8.65 ^b	248.10 ± 10.56 ^a	182.00 ± 54.77 ^b	213.26 ± 12.94 ^{ab}
Total protein (g/dl)	6.35 ± 0.20 ^a	8.22 ± 1.11 ^a	36.02 ± 29.27 ^a	10.46 ± 2.13 ^a	6.10 ± 0.77 ^a	8.91 ± 1.35 ^a
Bilirubin (mg/dl)	0.57 ± 0.82 ^a	2.23 ± 1.38 ^a	0.45 ± 0.34 ^a	0.75 ± 0.44 ^a	0.78 ± 0.26 ^a	0.96 ± 0.11 ^a
Total iron (μ g/dl)	319.90 ± 24.97 ^{ab}	212.94 ± 17.06 ^b	438.87 ± 128.86 ^a	202.64 ± 18.15 ^b	273.75 ± 67.13 ^{ab}	289.44 ± 23.92 ^{ab}
PCV (%)	29.80 ± 1.56 ^a	29.88 ± 1.15 ^a	30.39 ± 1.04 ^a	29.12 ± 0.63 ^a	30.50 ± 0.87 ^a	28.74 ± 0.92 ^a
Hb (gm/dl)	9.89 ± 0.61 ^a	9.61 ± 0.22 ^a	10.33 ± 0.39 ^a	9.90 ± 0.21 ^a	10.30 ± 0.15 ^a	13.45 ± 4.05 ^a
WBC (mcl)	5850.00 ± 311.72 ^b	8873.43 ± 282.22 ^a	4782.61 ± 212.98 ^b	8007.32 ± 382.36 ^a	5450.00 ± 758.84 ^b	8213.04 ± 427.30 ^a
Parasite density	15870.00 ± 4525.11 ^b	66513.00 ± 4858.88 ^b	14021.74 ± 3127 ^b	59258.54 ± 8110.37 ^b	11400.00 ± 4248.14 ^b	263252.17 ± 84566.84 ^a

524 N/B: Mean values in a row with different alphabets as superscript are

525 significantly different (p <0.05)

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