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# Effect of Haematological and Biomedical Parameters on Malaria Infected Children

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**ABSTRACT**

This study investigates the effect of haematological and biomedical parameters in malaria infected children. The effect of malaria parasitaemia were measured on some haematological and biochemical parameters in children and the severity of the infection were compared with the changes in these parameters. The same was comparison was done for non- infected and apparently healthy subjects. Our result showed that children of age two and five years are more susceptible to malaria attack. Hypoglycaemia was found amd total protein level, albumin and globulin were reduced in children with malaria infection. Anaemia, thrombocytopenia, and moderate leucocytosis were found in children with malaria parasites infection also.Malaria diagnosis in children should include malaria parasite density analysis in order to assist the clinician on the right management approach so at to reduce child mortality due to malaria infection..

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## 1. Introduction

Malaria is a major global health challenge especially in the tropics. In 2020, the estimated number of deaths by malaria stood at 627,000 out of a total estimate of 241 million cases of malaria infections. In the African region, children aged 5 and below accounted are more affected with an estimated death of 80% of all malaria deaths in the region, with sub-Saharan Africa being the most affected (WHO, 2021, CDC, 2021). Children less than five years (<5 yrs.) are mostly affected every year by the malaria. (WHO, 2021, WHO, 2000). Malaria, which was once widespread, is now mostly restricted to Africa, Asia, and Latin America (WHO, 2000), with tropical Africa accounting for nearly 80% of all cases and deaths worldwide. This is due to the fact that the malaria vectoral system in Africa south of the Sahara is perhaps the most powerful available to human plasmodia everywhere (Campbell et al., 1980).

Malaria is endemic in Nigeria, with P. falciparum being the most common parasite (Uko et al., 1998). It is the most common reason for patient visits to health care facilities and is consistently recognized as one among the top five causes of death, particularly in young children and babies (WHO, 2002). These malaria parasites are microorganisms caused by the protozoan parasite plasmodium. Plasmodium can infect animals such as reptiles, birds, and mammals, and there are over 100 kinds. Plasmodia *falciparum*, *vivax*, *ovale*, and *malariae* are the four Plasmodium species that infect humans. Malaria is spread from person to person by infected female Anopheles mosquitoes. Malaria can also be passed down from parents to children (Bruce-chwatt, 1972). Malaria has a wide spectrum of clinical symptoms and varies in severity. Fatigue, lethargy, headache, vomiting, and dysentery are common symptoms of uncomplicated malaria. Malaria fever is hypothesized to be caused by the rupturing of schizonts, which causes human mononuclear cells to release tumor necrosis factor (TNF) and other proinflammatory cytokines. (Kwiatkowski, *et al*., 1989).
In quasi patients, untreated sickness might last weeks or months, although only P. *falciparum* causes significant illness. According to current estimates, malaria kills 4.8 million children under the age of five in Sub-Saharan Africa (SSA) each year, averaging 9 deaths each minute. The only part of the globe where the number of children dying is growing instead of decreasing is Sub-Saharan Africa (UNSD, 2005). According to a United Nations Statistics Division (UNSD) research, 5.1 million children were projected to have died in 2015 base on trend, with Africa accounting for 57% of these deaths.
Inadequate health infrastructure and poor socioeconomic situations exacerbate malaria control challenges in nations like Africa, Asia, and Latin America. With the rise in anti-malaria drug resistance in recent years, the situation has gotten even more complicated.
The key to good illness management in malaria infected individuals is a timely and accurate diagnosis. Because the clinical presentation of malaria is variable, and it might be difficult to distinguish it from viral fever, enteric fever, or even Leptospirosis in a tropical nation, the most extensively used approach for diagnosing malaria in the tropics is unreliable (WHO, 2000). The use of microscopic diagnostics and haematological parameters from peripheral blood will be beneficial. The pathogenesis of malaria is mostly based on significant alterations in biochemical and haematological markers (Bidaki and Dalimi, 2003). Some haematological and biochemical signs might prompt suspicion of severe malaria, according to the World Health Organization's guidelines (WHO, 2000). Changes in physiological parameters are common in malaria infection. Thrombocytopenia and hypoglycaemia are common finding in Plasmodium *falciparum* infection. Complication in acute P. *falciparum* malaria associated with a high morbidity and mortality in children. (Nyirjesy, 1993). The other biochemical parameters like total protein and albumin were significantly lower in parasitaemic children than in the non parasitaemic children (Adeosun, *et al*., 2006). There are some scientific publications on biochemical and haematological parameters, but none have been reported in Port Harcourt. Therefore, this work needs to be carried out to assess some of the parameters and to apply any changes observed to malaria diagnosis and management as well as understanding malaria pathogenesis.
This study measures the effect of malaria parasitaemia on some haematological and biochemical parameters in children living in Port Harcourt and compare the severity of this infection on the changes in these parameters for both infected and non- infected children.

## 2. Materials and Methods

### 2.1 Study Areas and Population

The study was done in Port Harcourt metropolis in Rivers State, Nigeria. The samples were collected in Faith clinic, Wimpey, Trans Amadi health centre, Rumuigbo Health centre and University of Port Harcourt teaching Hospital, Choba. Children presented with symptoms of malaria (who have not taken any antimalaria drugs for the past 3 days but may be on any pain killer before the time of collection) in the outpatient clinic were chosen as malaria positive subjects while children that came for Pre-school medical check-up without any symptoms (also screened for malaria and tested negative) were used as apparently healthy controls after obtaining permission from the physicians and their parents. Their age ranged less

than 5 years of age. A total of 100 children that were malaria parasite positive were chosen for this study. Fifty (50) apparently healthy age-sex matched children were used as controls. All subjects were 1- 5 years of age. Throughout the period of collection of samples for this research none of age zero year (less than 1 year) was positive for malaria parasites infection.

### 2.2 Laboratory Procedure

#### 2.2.1 Collection and Processing of Samples

Five millilitres of venous blood were collected through vene-puncture using the antecubital vein from both groups of subjects and the apparently healthy children. Two millilitres of blood was dispensed into dipotassium ethylenediamine tetra acetic acid tubes for full blood count and one millilitre into the sodium fluoride containers for glucose estimation: and two millilitres into the lithium heparinized tubes for protein and albumin estimation tests. The little drops remaining in the syringe was dropped on a cleaned slide for thick films. Sample collected each day were taken to the laboratory and the analysis were performed the same day. The blood samples in the sodium fluoride tubes and lithium heparinized tubes were centrifuged immediately after collection, the plasma separated into another plain tube and used for the analysis the same day.

#### 2.2.2. Haematological Parameters

The samples collected into the EDTA bottles from the two categories of the children were subjected to the following haematology analysis: Haemoglobin (Hb), Packed cell volume (PCV), Platelet (Plt) count, White blood cell (WBC) - total count and White blood cell (WBC) – differential count.

**Haemoglobin Estimation by Cyanomethaemoglobin Method (Dacie and Lewis Method 2007)**

**Principle**: The test depends on the oxidation of haemoglobin and its derivative by potassium ferricyanide to methaemoglobin at an alkaline medium. Subsequent reaction with potassium cyanide produced the stable red Cyanomethemoglobin which is measured spectrophotometrically at 540nm.

**Procedure:** 20µl of blood was washed into 4ml of Drabkin reagent (haemoglobin reagent) in the test tubes. The test tube was covered with a rubber bung inverted several times and allowed to stand at room temperature for 5 minutes to ensure complete conversion to cyanomethaemoglobin. The absorbance was read at 540nm wavelength against a blank (4ml of Drabkin reagent only). The absorbance of the standards was read alongside with those of the samples and the control.

$$\text{Haemoglobin g/l} = \frac{\text{Absorbance of test x Concentration of Standard}}{\text{Absorbance of Standard}}$$

**Packed Cell Volume (PCV) Estimation (Brown, 1993)**

**Principle**: The packed cell volume (PCV) or haematocrit is a measurement of the proportion of red cells in whole blood. PCV measurement can be used as a screening tool for anemia.

**Procedure:** Well-mixed anticoagulant tube blood was allowed to enter the microhaematocrit tube by capillary action, leaving about 15mm unfilled. One end of the tube was sealed with plasticine. The capillary tubes were then placed in the radial grooves of the centrifuge (HETTICH, GERMANY) exactly opposite each other with the sealed end away from the centre of the centrifuge. The tubes were centrifuged at approximately 10,000 rpm for 5 minutes using the microhaematocrit centrifuge. The PCV was subsequently determined by measuring the height of the total blood column using a microhaematocrit reader (Brown, 1993).

**White Blood Cell (WBC) Count (Leucocyte Count) - (Dacie and Lewis method, 1998)**

**WBC - Total Count**

**Principle:** White Blood cells diluting fluid (Turke´s solution) contain a weak acid (Glacial acetic acid) to lyse the red blood cells and stain (Gentian violet) to stain the nucleus of the white blood cells.

**Procedure**:

One in twenty (l in 20) dilution of the blood was made using the 2% glacial acetic acid tinged with few drops of gentian violet. By pipetting 50µl of whole blood into 950µl of Turk's solution in the 70x10mm glass tube. The diluted sample was mixed and allowed to stand for 15 minutes for complete destruction of red cells. The sample was loaded into the improved Neubauer counting chamber, and the white cells present in the four corner ($1mm^{2)}$ areas were counted using x10 objective microscope. Calculation was done from the first principle.

**WBC – Differential Count**

**Procedure:** A thin blood smear film was made and labelled. The blood film was allowed to dry and then fixed in methanol for 5 minutes. After, the slide film was stain with 10% Giemsa stain for 30 minutes inside the staining jar. Thereafter the stain was washed off with distilled water and allowed to stand in the rack to drain and air dried. It was observed using the oil immersion objective. The cells were identified by their lobes and granules and counted.

**Platelet Counts (Dacie and Lewis Method 1998)**

One in twenty dilution of the blood was made using ammonium oxalate solution. The suspension was mixed on the mixer for 10 mins. The improved Neubauer counting chamber was then filled with the suspension by using pasture pipette; allowed to settle for some minutes and then counted using x 40 microscopic objective. The platelets appeared under ordinary illumination as small (but not minute) highly refractive particles. Calculation was done using the first principle.

**Preparation of Blood Smear**

Thick films were made from the sample that was left in the syringe after dispensed into the individual anticoagulant tubes (before clotted) and then thin films were made for each patient from the EDTA tubes. Allowed to dry.

**Staining Method and Estimation of Parasite Density (By Giemsa Method)**

Thick smear: - After drying, the slide films were transferred to the staining jar containing 4% of Giemsa stain. Allowed to stain for 30 minutes. Thereafter the films were rinsed with distilled water. Arranged on the rack to drain and air dried. And later observed using the oil immersion objective to detect the parasites and to estimate parasite concentration.

The total Parasites were counted until when the total White Blood Cells reached 200 and Parasites density were calculated (Trape, 1985).

$$ParasiteDensity = \frac{TotalParasiteCounted}{200} X \frac{TotalWBC(foreachpatient)}{1}$$

Thin Smear: - After drying, they were fixed in a jar of methanol for 5 minutes. Then the fixed films were transferred to a staining jar containing 10% of Giemsa stain and allowed to stain for 30 minutes. Then rinsed with distilled water and allowed to stand in the rack to drain and air dried. Then observed by using the oil immersion objective. The species of the Plasmodium was identified.

### 2.2.3 Biochemical Parameters

The samples collected into the sodium fluoride tubes and lithium heparinized tubes from the two categories of the children were subjected to the following biochemical analysis: Glucose, Protein, Albumin and Globulin.

**Glucose Estimation Test was carried using Enzymatic Method.**

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**Principle**: **Glucose Oxidase Method (Enzymatic)**

Glucose is oxidized by glucose oxidase to gluconic acid and hydrogen peroxide. The hydrogen peroxide is broken down to water and oxygen by the enzyme peroxidase in the presence of an oxygen acceptor (4-amino phenazone) which is converted to a colour compound, which is read spectrophotometrically at 520nm and this is proportional to the concentration of the blood sugar (Barham and Trinder, 1972).

**Procedure:** The test tubes were respectively labelled blank, standard, control and sample. 1.0ml (1000µl) of the Oxidase reagent was pipetted into each tube. 0.01 ml (10µl) of the distilled water, standard, control and sample were pipetted into their respective tubes, mixed and incubated at $37^0$C for 5minutes. The content of the tubes were read at 520nm using the content of blank tube to zero the Spectrophotometer. The concentration of glucose was determined using the calculation below.

$$\text{Concentration of sample} = \frac{\text{Absorbance of sample x Concentration of Standard}}{\text{Absorbance of Standard}}$$

**Total Protein Estimation (Using Biuret Method)**
**Principle:** Cupric ions, in alkaline medium interact with protein peptide bonds resulting in the formation of a coloured complex. (Henry, et al., 1974).

**Procedure:** The test tubes were respectively labelled blank, standard, control and sample. 5.0ml of Biuret reagent was pippetted into each tube. 0.1ml of distilled water, standard, control and sample were pipetted into their respective tubes mixed, incubated for 30 minutes at 25ºc.The absorbances were measured against the reagent blank at wavelength of 546nm.The concentration of total protein was determined using the calculation below.

$$\text{Total protein (g/l)} = \frac{\text{Absorbance of sample x Concentration of Standard}}{\text{Absorbance of Standard}}$$

**Albumin estimation using bromocresol green.**

**Principle**: The measurement of serum albumin is based on its quantitative binding to the indictor 3, 3` 5, 5`-tetrabromo-m-cresol sulphonephthalein (bromocresol green BCG). The albumin-BCG complex absorbs maximally at 578nm, the absorbance being directly proportional to the concentration of albumin in the sample. (Douma, *et al*., 1971).

**Procedure:** The test tubes were labelled blank, standard, control and sample. 3.0ml of Bromocresol green reagent was pipetted into each tube. 0.01ml of the distilled water, standard, control and sample was pipetted iinto their respective tube, mixed and incubated at 25ºc for 5 minutes. The absorbances were measured at 578nm against the reagent blank. The concentration of albumin was determined using the calculation below.

$$\text{Albumin (g/l)} = \frac{\text{Absorbance of sample x Concentration of Standard}}{\text{Absorbance of Standard}}$$

**Globulin Estimation**-: Globulin was calculated from the difference between total protein and albumin.

**Statistical Analysis**
Data analysis was undertaken using SPSS. SPSS procedures for unrelated t-test and ANOVA of variance are very useful since they include an option for its calculation when the variances of the samples of scores are significantly different from each other.
Significance level was set at alpha 0.05 ($p < 0.05$).

## 3. Results and Discussion

One hundred and fifty (150) children of age less than 5 years {male and female} were participating in this study. One hundred children {male and female} were malaria positive while fifty were non-malaria positive (control). The results of this study given below.

Table 1 below show the prevalence of infection among children of age 1-5yrs, and the species of the Plasmodium prevalent. The P. *falciparum* was mostly common; ninety-six (96) children had P. *falciparum* while only four (4) children had P. *vivax* infection. Age 1 year had only 6 children positive for malaria and one out of them had specie of P. *vivax* while others had specie P. *falciparum.* Age 2 years recorded 32 children positive for malaria; only one (1) was positive for P. *vivax* while 31 children were positive for P. *falciparum*. Age 3 years recorded only 14 children positive, and 4 years also recorded 14 children positive; all of them had P. *falciparum*, none was positive for P. *vivax*. Age five (5) years had the greatest population that was positive for malaria infection. Thirty-four (34) cases were recorded; only thirty-two (32) were positive for P. *falciparum* while only two were positive for P. *vivax.*

Table 2 shows the total parasite density population with the total number of children of different ages <5 yrs (less than 5 yrs) that were positive in each group of the parasitaemia by intensity of the infection. Children with heavy parasitaemia have the highest percentage. It was found out that children of this age, 1-5 years were prone to heavy and severe parasitaemia. Fifty-four (54) out of hundred (100) had heavy parasitaemia (1000-240,000 µl/bld) and forty-four (44) children had severe parasitaemia of >240,000 µl/bld. Two (2) had moderate (100-999µl/bld); none of them had scanty (1-99µl/bld) parasite densities.

Table 3 displays the mean value of haematological parameters for parasitized and non-parasitized subjects for the whole population. The mean Hb (g/l) was significantly reduced (8.89 ± 1.79 g/dl) for the parasitized infected children and 12.27 ± 0.69 g/dl for the non-infected control children; P (< 0.05). The mean PCV (%) was 28.15 ± 5.37% for the parasitized children and 38.62 ± 2.36% for the control children (p-value < 0.05). This was significantly reduced compared with the control value ($p < 0.05$).

Platelets ($10^9$/l) was observed to be significantly lower in parasitized children (149.3 ± 84.45 x$10^9$/l) than in control (305.74 ± 72.88 x$10^9$/l) ($p<0.05$).

The WBC was 9.16 ± 2.72 x $10^9$/l for parasitized children and 7.476 ± 1.76 x $10^9$/ for non-parasitized. The WBC was higher in malaria children than in control ($p> 0.05$), Neutrophils (%) in parasitized children was 51.95 ± 10.57 and control 43.34 ± 10.65 ($p<0.05$). Lymphocytes (%) in parasitized children was 39.25 ± 11.68 and 48.96 ± 10.33 in control ($p>0.05$). Monocytes (%) was 6.15 ± 2.66 in parasitized children and 2.36 ± 2.16 in non-parasitized ($p>0.05$). Eosinophil (%) was 1.58 ± 0.94 in parasitized children and 3.78 ± 2.28 in non- parasitized children ($p>0.05$). Basophil (%) was 1.19 ± 0.64 parasitized children and 1.70. ± 1.13 in non-parasitized children ($p>0.05$).

Table 4 shows the mean values of biochemical parameters for parasitized and non-parasitized children with the standard deviation and the p-values. Glucose (mmol/l) mean value was reduced in parasitized children 3.25 ± 0.67 compared to non-parasitized children 5.0 ± 0.54 ($p<0.01$). Total protein (g/l) in parasitized children was observed to be 56.13 ± 8.47 and non-parasitized children was 68.12 ± 5.17 ($p<0.05$). It was reduced compared with the control. Albumin (g/l) also was significantly low in parasitized children, it was 35.86 ± 6.65 than in non-parasitized which was 42.20 ± 5.7 ($p<0.05$). Globulin (g/l) in parasitized children was 20.24 ± 4.08 and in non-parasitized was 25.92 ± 5.46 ($p<0.05$).

**Table 1 Prevalence of Malaria among the study population.**

| Year | No. of blood films examined | Malaria positive | P. *f* | Other species (P.*vivax*) |
|---|---|---|---|---|

| | | | | |
|---|---|---|---|---|
| 1 | 6 | 6 | 5 | 1 |
| 2 | 32 | 32 | 31 | 1 |
| 3 | 14 | 14 | 14 | 0 |
| 4 | 14 | 14 | 14 | 0 |
| 5 | 34 | 34 | 32 | 2 |
| Total | 100 | 100 | 96 | 4 |

P. f – Plasmodium *falciparum*
P. *vivax* – Plasmodium *vivax*

**Table 2: Distribution of Parasite Density of the 100 Children with Malaria Infection**

| **Parasitaemia (Parasites/µl)** | **Number of Subjects** |
|---|---|
| 1 – 99 (Scanty) | 0 |
| 100 – 999 (Moderate) | 2 |
| 1000 – 240000 (Heavy) | 54 |
| >240000 (Severe) | 44 |

**Table 3:- Mean Values of Haematological Parameters for Parasitized and Non-Parasitized Subjects**

| Parameters | Parasitized (100) | Non-Parasitized (50) | P-Value |
|---|---|---|---|
| Haemoglobin (g/dl) | 8.89 ± 1.79 | 12.27 ± 0.69 | <0.05 |
| PCV (%) | 28.15 ± 5.37 | 38.62 ± 2.36 | <0.05 |
| WBC ($10^9$/L) | 9.16 ± 2.72 | 7.476 ± 1.76 | >0.05 |
| Platelet ($10^9$/l) | 149.33 ± 84.45 | 305.74 ± 72.88 | <0.05 |
| Neutrophils ($10^9$/l) | 51.95 ± 10.57 | 43.34 ± 10.65 | <0.05 |
| Lymphocytes (%) | 39.25 ± 11.68 | 48.96 ± 10.33 | >0.05 |
| Monocytes (%) | 6.15 ± 2..66 | 2.36 ± 2.16 | >0.05 |
| Eosinophils (%) | 1.58 ± 0.94 | 3.78 ± 2.28 | >0.05 |
| Basophils (%) | 1.19 ± 0.64 | 1.70 ± 1.13 | >0.05 |

*All values given as Mean ± SD*

**Table 4: Mean Values of Biochemical Parameters for Parasitized and Non- Parasitized.**

| Parameters | Parasitized (100) | Non-Parasitized (50) | P-Value |
|---|---|---|---|
| Glucose (mmo/l) | 3.25 ± 0.67 | 5.0 ± 0.54 | P < 0.01 |
| Total protein (g/l) | 56.13 ± 8.47 | 68.12 ± 5.17 | P< 0.05 |
| Albumin (g/l) | 35.86 ± 6.65 | 42.20 ± 5.7 | P< 0.05 |
| Globulin (g/l) | 20.24 ± 4.08 | 25.92 ± 5.46 | P<0.05 |

*All values given as Mean ± SD*

Table 5 displays the parasite density and haematological & biochemical parameters. The children that have heavy parasitaemia (1000-240,000µl/bld) were 54 and their result was compared with the control (N =50). The Hb (g/l) was observed to be reduced in children with heavy parasitaemia which was 9.10 ± 1.8 lower than the control which was 12.3 ± 0.69 (p< 0.05). The PCV (%) was 28.34 ± 6.7 in heavy parasitaemic children and 38.62 ± 2.36 in control (p<0.05). WBC ($10^9$/l) in heavy parasitaemic children was 9.0 ± 2.7

and 7.48 ± 1.76 in control. This was significantly raised compared with the control ($p< 0.05$). Platelet ($10^9$/l) was reduced in heavy parasitaemia it was 153 ± 93.0 x ($10^9$/l) and the control was 305.74 ± 72.68 x $10^9$/l ($p<0.05$).
The mean value for Neutrophils was 51 ± 10.95 % in heavy parasitaemia and 43.34 ± 10.65 % in control ($p>0.05$). It was raised compared with the control. Monocyte was observed to be 6.4 ± 2.96 % in heavy parasitaemia and 2.36 ± 2.16 in control ($p >0.05$). Eosinophil was 1.82 ± 0.94% in heavy parasitaemia and 3.78 ± 2.28 in control. This is not significant ($p>0.05$). Also Basophil was 1.28 ± 0.64% in heavy parasitaemia and 1.70 ± 1.13% in control. It was non- significant $P>0.05$.

Glucose was observed to be reduced in parasitized children 3.35 ± 0.66 mmol/l and 5.0 ± 0.54 mmol/l in controls. ($P<0.01$). Total protein was significantly reduced also in children with 57.42 ± 10.0g/l than in control 68.12 ± 5.12 ($p< 0.05$). Albumin was 37.4 ± 7.32 g/l lower than in control which was 42 20 ± 5.69 ($p<0.05$). Globulin was 20.38 ± 3.10 in density and 25.92 ± 5.46. g/l in control ($p< 0.05$).

Table 6 observed the severe parasite density (>240,000µl/bld) and the haematological and biochemical parameters. Forty-four (44) children were having severe parasite density. Hb was 8.34 ± 1.8 g/l for the severe parasitaemia and was lower than control which was 12.3 ± 0.69 g/l ($p <0.05$). The PCV was 27.30 ± 2.7% in severe PD than control 38.62 ± 2.36 % ($p<0.05$). In severe parasitaemia the platelet was 140.25 ± 70 x$10^9$/l and in control 305.74 ± 72.88 x$10^9$/l ($p<0.05$).
The WBC ($10^9$/l) was 9.5 ± 2.8 in severe PD and 7.48 ± 1.76 in control ($p>0.05$). Neutrophil (%) was 54 ± 10.6 in severe PD and 43.34 ± 10.7 for the control ($p>0.05$). Monocytes in severe PD were 6.4 ± 2.9 and 2.36 ± 2.16 in control ($p>0.05$). Eosinophils was 1.8 ± 0.94% in severe PD and 3.48 ± 2.28 % in control ($p>0.05$). Basophil was 1.0 ± 6.4 %, and 1.70 ± 1.13 in control. ($p>0.05$).
Glucose was significantly reduced in severe PD (3.0±0.68 mmol/l) compared to 5.0 ± 0.54 in control ($p<0.001$). Total protein was significantly reduced also when compared with the heavy parasite density the value was 52.48 ± 5.0 g/l and 68.12 ± 5.12 g/l in control. ($p<0.05$). The Albumin mean value was 32.47 ± 4.40 in severe PD and control was 42.20 ± 5.69 ($p<0.05$). Globulin was 20.01 ± 1.20 in severe PD and 25.12 ± 5.46 in control, $p<0.05$.

Table 4.7 displays the moderate parasites density (100-999µl/bld) with the haematological & biochemical parameters. The Hb (g/dl) was 9.14 ± 1.77 for moderate parasites density and 12.27 ± 0.69 for the control sample. The PCV (%) was 28.8 ± 6.73 in moderate Pd and the control was 38.62 ± 2.36. The WBC ($10^9$/l) was 8.97 ± 2.67 and 7.48 ± 1.76 for the control. The platelet (109/l) was 155 ± 90.34 and 305.74 ± 72.88 for the control. The Neutrophils was 50.86 ± 10.15 and the control was 43.34 ± 10.65. Lymphocyte was 40.74 ± 11.16 for the moderate Pd and 48.96 ± 10.33 for the control. Monocytes recorded a value of 5.64 ± 2.11 and 2.36 ± 2.16 for the control. The Eosinophil was 1.82 ± 0.94 for the moderate Pd and 3.78 ± 2.28 for the control. The Basophils was 1.28 ± 0.64 in moderate Pd and 1.70 ± 1.13 for the control. Glucose (mmol/l) was reduced in moderate Pd, 3.4 ± 0.66 was recorded and 5.0 ± 0.54 was recorded for the control. Total protein (g/l) was reduced: 58.48 ± 10.41 was found and the control was 68.12 ± 5.17. Albumin was 38.06 ± 8.22 in moderate Pd and the control was 42.20 ± 5.69. Globulin in moderate Pd was 20.42 ± 8.04 and the control was 25.92 ± 5.46. It was reduced.

Table 4.8 summarized the total parasites density estimated for different categories of parasitaemia with the total numbers of children found positive in each group and their respective haematological & biochemical parameters. These include Moderate=100-999µl/bld; Heavy=1000-240,000µl/bld; and Severe= >240,000µl/bld; on the studied children (1-5 yrs). Hb, PCV and Platelets were decreased as parasites density increases. Also, the WBC and differential appeared normal. Glucose and total protein reduced as parasites density increases. The albumin and globulin also reduced with increase in parasite density.

**Table 5: Mean Values of Haematological and Biochemical Parameters in Patients with heavy parasite density and in control (mean ± SD)**

| Parameters | 1000 – 240,000µl/bld Heavy n = 54 | Control n = 50 | P-Values |
|---|---|---|---|
| Hb (g/dl) | 9.10 ± 1.8 | 12.3 ± 0.69 | $p < 0.05$ |
| PCV (%) | 28.34 ± 6.7 | 38.62 ± 2.36 | $p < 0.05$ |
| WBC ($10^9$/l) | 9.0 ± 2.7 | 7.48 ± 1.76 | $p > 0.05$ |
| Platelet ($10^9$/) | 153 ± 93.0 | 305.74 ± 72.68 | $p < 0.05$ |
| Neutrophils (%) | 51 ± 10.95 | 43.34 ± 10.65 | $p > 0.05$ |
| Lymphocytes (%) | 39 ± 11.96 | 48.96 ± 10.33 | $p > 0.05$ |
| Monocytes (%) | 6.4 ± 2.96 | 2.36 ± 2.16 | $p > 0.05$ |
| Eosinophils (%) | 1.82 ± 0.94 | 3.78 ± 2.28 | $p > 0.05$ |
| Basophils (%) | 1.28 ± 0.64 | 1.70 ± 1.13 | $p > 0.05$ |
| Glucose (mmo/l) | 3.35±0.66 | 5.0±0.54 | $p < 0.01$ |
| Total Protein (g/l) | 57.42 ± 10.00 | 68.12 ± 5.12 | $p < 0.05$ |
| Albumin (g/l) | 37.4 ± 7.32 | 42.20 ± 5.69 | $p < .0.05$ |
| Globulin (g/l) | 20.38±3.10 | 25.92±5.46 | $p < 0.05$ |

**Table 6: Mean Values of Haematological and Biochemical Parameters in Patients with Severe parasite density and in control (mean ± SD)**

| Parameters | > 2 40,000µl/bld Severe n = 44 | Control n = 50 | P-Values |
|---|---|---|---|
| Hb (g/l) | 8.34 ± 1.8 | 12.3 ± 0.69 | $p < 0.05$ |
| PCV (%) | 27.30 ±2.7 | 38.62 ±2.36 | $p < 0.05$ |
| WBC (10g/l) | 9.5 ± 2.8 | 7.48 ±1.76 | $p < 0.05$ |
| Platelet (10g/l) | 140.25 ± 70.0 | 305.74 ± 72.88 | $p < 0.05$ |
| Neutrophils (%) | 54 ± 10.6 | 43.34 ± 10.7 | $p < 0.05$ |
| Lymphocytes (%) | 38 ± 11.90 | 48.96 ± 10.3 | $p < 0.05$ |
| Monocytes (%) | 6.4 ± 2.9 | 2.36 ± 2.16 | $p < 0.05$ |
| Eosinophils (%) | 1.8 ± 0.94 | 3.48 ± 2.28 | $p > 0.05$ |
| Basophils (%) | 1.0 ± 64 | 1.70 ± 1.13 | $p > 0.05$ |
| Glucose (mmo/l) | 3.0± 0.68 | 5.0 ± 0.54 | $p < 0.01$ |
| Total Protein (g/l) | 52.48 ± 5.0 | 68.12 ± 5.12 | $p > 0.05$ |
| Albumin (g/l) | 32.47 ± 4.40 | 42.20 ±5.69 | $p > 0.05$ |
| Globulin (g/l) | 20.01±1.20 | 25.12±5.46 | $p > 0.05$ |

**Table 7: Mean Value of Haematological & Biochemical Parameters In children with Moderate Parasite Density and in Control**

| Parameters | 100-999 µl/bld | Control |
|---|---|---|

| | Moderate | |
|---|---|---|
| Hb (g/dl) | 9.14 ±1.77 | 12.27±0.69 |
| PCV (%) | 28.8±6.73 | 38.62±2.36 |
| WBC ($10^9$/l) | 8.97±2.67 | 7.476±1.76 |
| Platelets ($10^9$/) | 155.20±90.34 | 305.74±72.88 |
| Neutrophils (%) | 50.86±10.15 | 43.34±10.65 |
| Lymphocytes (%) | 40.74±11.16 | 48.96±10.33 |
| Monocytes (%) | 5.64±2.96 | 2.36±2.16 |
| Eosinophils (%) | 1.82±0.94 | 3.78±2.28 |
| Basophils (%) | 1.28±0.64 | 1.70± 1.13. |
| Glucose (mmo/l) | 3.4±0.66 | 5.0±0.54 |
| Total protein (g/l) | 58.48 ± 10.41 | 68.12 ± 5.17 |
| Albumin (g/l) | 38.06 ± 8.22 | 42.20 ± 5.69 |
| Globulin (g/l) | 20.42 ±8.04 | 25.92 ± 5.46 |

**Table 8: Mean Values of Haematological & Biochemical Parameters in Parasite Densities of 100-999µl/bld, 1000-240,000µl/bld AND >240,000µl/bld**

| PARAMETERS | 100-999 µl/bld N = 2 | 1000-240,000 µl/bld N = 54 | >240,000 µl/bld N = 44 | F VALUES |
|---|---|---|---|---|
| Hb (g/l) | 9.14±1.77 | 9.20+1.8 | 8.34±1.8 | 3.23 |
| PCV (%) | 28.8±6.7 | 28.34±6.7 | 27.30±2.7 | 1.62 |
| WBC ($10^9$/l) | 8.97±2.67 | 9.0±2.7 | 9.5±2.8 | 1.39 |
| Platelets ($10^9$/l) | 155±90.34 | 153 ±93.0 | 140 ± 70.0 | 0.11 |
| Neutrophils (%) | 50.86 ± 10.15 | 51 ± 10.95 | 54 ± 10.6 | 2.66 |
| Lymphocytes (%) | 40.74 ± 11.16 | 39 ±11.96 | 38 ± 11.90 | 2.54 |
| Monocytes (%) | 5.64 ±2.11 | 6.4 ± 2.96 | 6.4±.2.9 | 0.64 |
| Eosinophils (%) | 1.8 2 ±0.94 | 1.82± 0.94 | 1.1 ±0.94 | 1.67 |
| Basophils (%) | 1.28 ± 0.64 | 1.28±0.64 | 1.0 ± 0.64 | 1.19 |
| Glucose (mmoll) | 3.4± 0.66 | 3.35 ± 0.66 | 3.0 ± 0.68 | 1.42 |
| Total protein(gll) | 58.48 ±10.41 | 57.42 ±10.0 | 52 .48 ± 5.0 | 3.80 |
| Albumin (gll) | 38.06 ± 8.22. | 37.04 ± 7.32 | 32.47±4.40 | 3.23 |
| Globulin (gll) | 20.42 ±8.04 | 20.3±3.0 | 20.01±1.20 | 1.52 |

Figure 1 shows malaria positive for P.*falciparum* and P.*vivax* for children of age 1 to 5 years. Peak values of malaria infections occurred for children aged 5 years old and followed by children age 2 years old. Children ages 3 & 4 years old had the same peak while age 1 year old had lowest peak. Figure 2 shows the distribution of malaria parasite density among 100 children. Children with heavy parasite density constitute a total of 54% while severe malaria cases was 44% and moderate parasites densities was 2%.

**mean values of haematological parameters for parasitized and non-parasitized subjects is shown in** Figure 3. The figure shown the variations of haematological parameters for parasitized (patient) and non-parasitized (control) subjects. The mean Hb, PCV, and platelets values for patients was reduced when compared with the control, suggesting that malaria parasite has a direct effect on Hb, PCV, and platelets levels.

Figure 4 shows the mean values of biochemical parameters for parasitized (patient) and non – parasitized (control). Teh figure demonstrates that biochemical parameters for parasitized children are lower compared to the non-parasitized (control) children. Figure 5 and Figure 6 shows thick film of Plasmodium *Falciparum* for high parasite density and scanty parasite density.

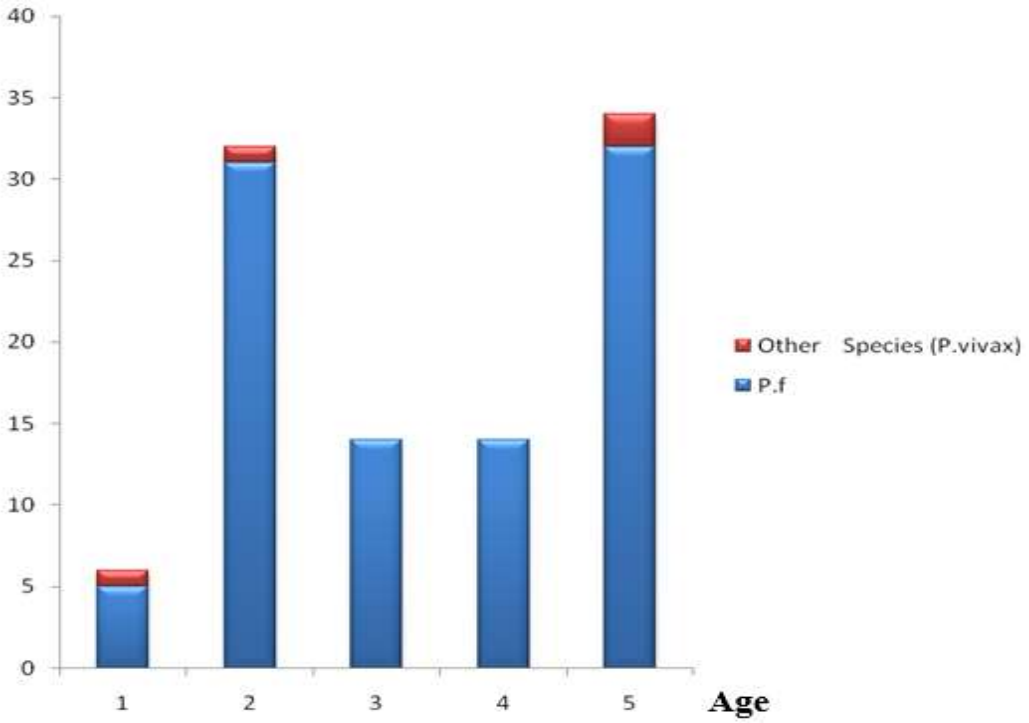


**Figure 1: Malaria Positive for P.*falciparum* and P.*vivax***

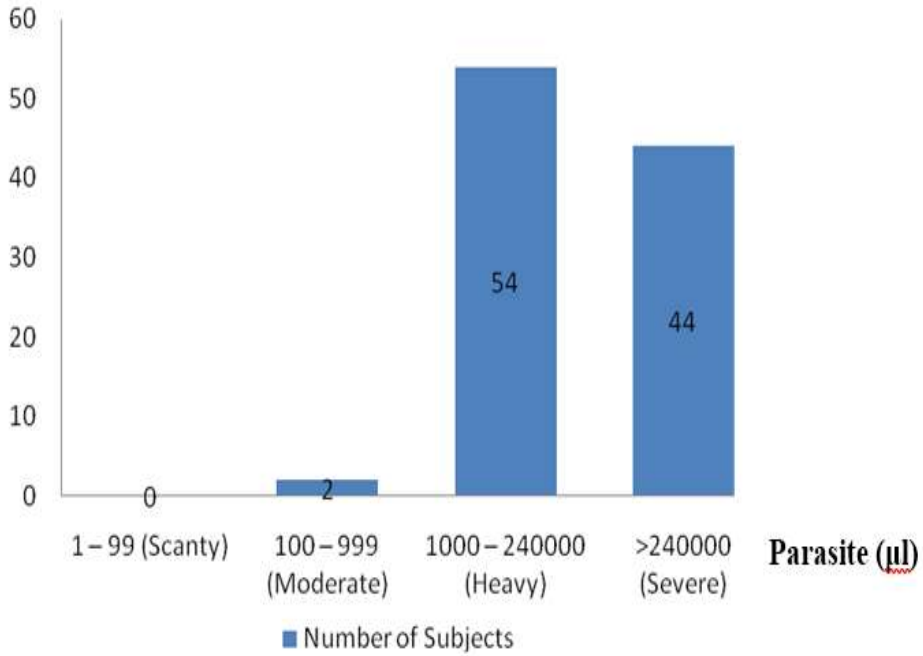


**Figure 2: Distribution of malaria parasite density among the 100 Nigeria children.**

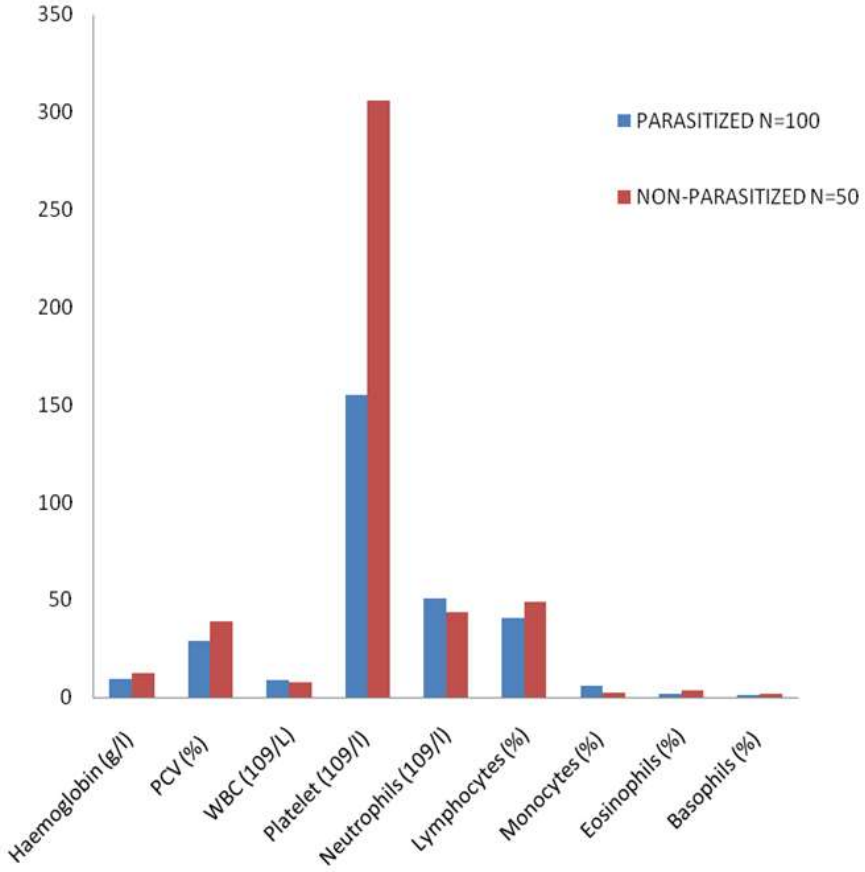


**Figure 3: Mean Values of Haematological Parameters for Parasitized and Non-Parasitized Subjects**

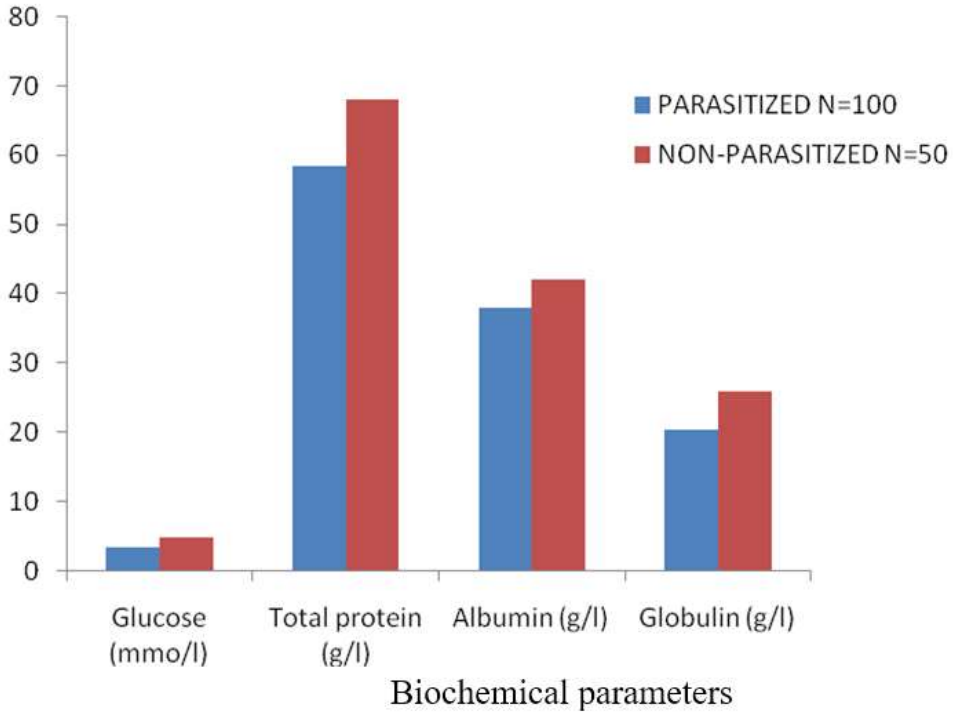


**Figure 4: Mean Values of Biochemical Parameters for Parasitized and Non – Parasitized**

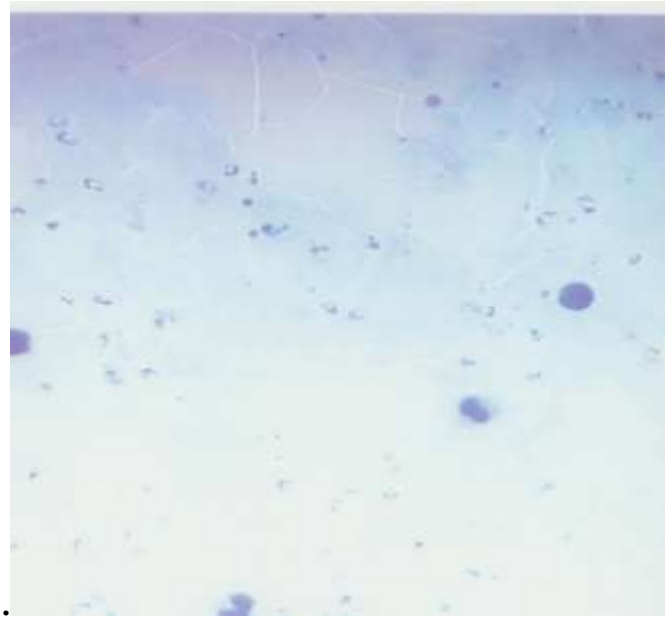

**Figure 5: Thick Film of Plasmodium *Falciparum* with High Parasite Density.**

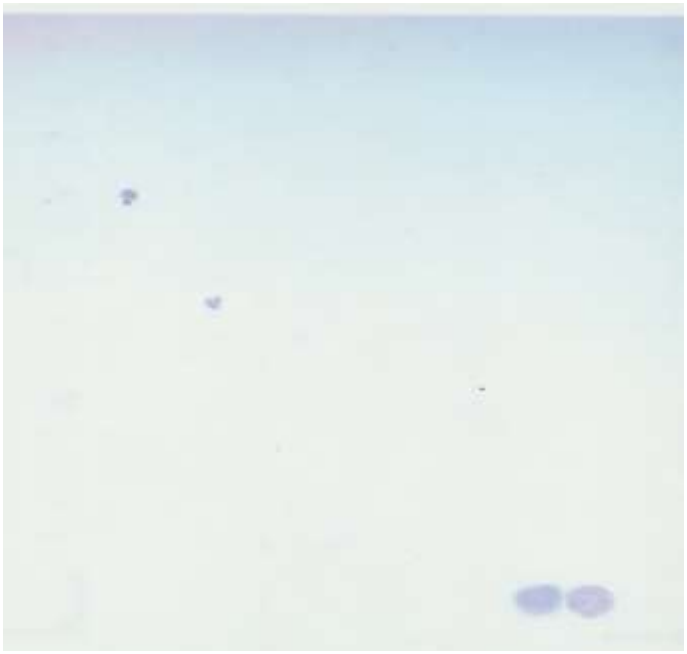

**Figure 6: Thick Film of Plasmodium *Vivax* with Scanty Parasite Density**

## 4. Conclusions

In conclusion, the results presented in this study compared to what is available in the literature are in support of the notion that malaria infections have effect on some haematological and biochemical parameters in children with malaria parasite infection when compared with non-infected children (control). Children of age two and five years are more vulnerable to malaria attack. Hypoglycaemia was found. Total protein level, albumin and globulin were reduced in children with malaria infection. Anaemia, thrombocytopenia, and moderate leucocytosis were found in children with malaria parasites infection. These features can be found more in children with heavy and severe parasites density of malaria attack. Efforts should be geared towards malaria parasite density in laboratories analysis for proper and good diagnosis. This will enable quick management of the patients by the clinicians.

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