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Variation of Some Hematological Parameters from the Normal Blood Counts as a result of *Plasmodium falciparum (*parasitaemia) Infection in children (6-59 Months). A Case Study of Bulumkutu Comprehensive Health Centre Maiduguri, Borno State – Nigeria

Y. Inuwa,[*] M. Kokori,[**] M. Dahiru.[***] R. E. Momoh.[****]
[*]*Department of Biological Sciences, Faculty of Science, University of Maiduguri, Maiduguri Nigeria.*
Department of Biological Sciences, Faculty of Science, Federal University of Kashere, Gombe State, Nigeria.
[**]*Department of Biological Sciences, Faculty of Science, University of Maiduguri, Maiduguri Nigeria.*
[***]*Department of Biological Sciences, Federal University of Kashere, Gombe State, Nigeria.*
[****]*Department of Chemical Sciences, Faculty of Science, Federal University of Kashere, P.M.B. 0182 Gombe State, Nigeria.*
Corresponding Author: Yahaya Inuwa

Abstract
This study was conducted to assess the influence of Plasmodium falciparum parasitaemia on some selected hemalogical parameters in children (6-59months), at Bulumkutu Health Centre, Maiduguri, Bono State, between Augusts to December 2018. A total of 210 children were enrolled in the study which consists of 88 (41.90%) patients with positive P. falciparum malaria and 122 (58.10%) negative malaria. Hematological parameters were analyzed using sysmex haematology auto-analyser (2011), while the Giemsa stained slides thick and thin blood films were prepared from the stock solution, and tested for Plasmodium falciparum malaria and count of malaria parasite density. This study also showed the variation of some hematological parameters from the normal blood counts. In which, the children within the age group 51-60 months (27.27%) had a highest percentage of low PCV counts, whereas the children within the age group 31-40 months (2.27%) had the lowest percentage of PCV counts. In addition, the results also showed that the children within the age group 51-60 months (10.23%) had the highest percentage of normal PCV counts (33%), whereas the children within the age group 6-10 months (1.14%) (>33%) had the lowest percentage of normal PCV. Furthermore, it was found that the malarial infected subjects within the age bracket 51-60 months recorded a high percentage of children with thromobocytopenia (5.68%) and leucocytopenia (2.27%) and also a high percentage of thrombocytosis (10.23%) and leucocytosis (13.63%) respectively. However, the result obtained on neutrophil counts showed that the children within the age group 11-20 months (5.68%) recorded the highest percentage of neutropenia, while the children within the age group 51-60 months (13.63%) recorded the highest percentage of neutrophilia. Moreover, the result also showed that the malarial infected subjects within the age bracket 51-60 months (2.72%) had the highest percentage of Eosinopenia, while the malarial infected subject within the age group 21-30 months (5.68%) had the highest percentage of eosinophilia. The result also showed that the children within the age group 51-60 months (13.63%) recorded highest percentage of lymphocytopenia, whereas the children within the age group 11-20 months (6.82%) recorded the highest percentage of lymphocytosis. Both monocytopenia and monocytosis was found among the malarial infected subjects within the age group 51-60 months had the percentage of occurrence of 9.09% for monocytopenia and 7.95% of monocytosis. From the result presented on table it was found that, about 78.41% of the subject had a low PCV, while 21.59% of the children tested positive had a fairly normal PCV as it was also reported that 14.77% of the subject had low thrombocytes, while 48.55% of the subjects had a normal thrombocytes and then 30.68% had a higher thrombocytes counts. Similarly, the results also revealed that 2.27% of the children tested positive had a white blood cell below normal counts, while 63.64% of the subjects are within the normal range, and 34.09% of the children had higher white blood cells counts. However 15.91% of the subjects had higher neutrophil and eosinophil counts. Whereas 15.91%, and 44.31% showed up with low neutrophil and eosinophil counts. The lymphocyte and monocyte counts were reported to have equal percentage of occurrence with normal counts. Whereas, 30.68% and 25.41% recorded a higher counts, while more than 15% where observed to be higher than the normal counts. By using normal blood counts of WHO, (1996).
Normal values: PCV → (33%), Platelet → (150,000-400,000 x 10^{-9}), WBC → (4.5 – 13x-9), Neutrophil → (30-65%), Eosinophil → (1-4%), Lymphocyte → (30-60%), Monocyte → (1-9%), N = Number (group) WHO, (1996).

Key words: *Malaria Infection, Plasmodium falciparum, Aneamia, parasitaemia Children*

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I. Introduction

Malaria is one of the leading communicable and deadly disease in the developing countries of the globe. It mostly occurs in the tropical and subtropical regions and accounts for considerable morbidity and death. It causes the death of approximately one million in Africa every year, and is responsible for fifteen percent (15%) of clinical illnesses in the tropical regions of the continent (WHO, 1997). Ten percent (10%) of death recorded in children aged below three years are cause by malaria in some parts of the tropical regions. Out of the projected annual 300-500 million clinical malaria cases, 1.5 to 2.7 million deaths are directly credited to malaria and the great majority occurs in immune compromise children especially in remote rural areas of the sub-Sahara Africa (Snow *et al*.,1999) Malaria is transmitted into human during the bite of anopheles mosquitoes and the injection of sporozoites, the invasive forms of *Plasmodium*.

Malaria parasites belong to the genus *Plasmodium* which approximately includes about 125 species infecting reptiles, birds, and mammals (Cater and Mendis, 2002). *Plasmodium* is the only genus in the family *Plasmodia*, the class *Haematozoa* and the phylum *Apicomplexa*. The genus *Plasmodium* is subdivided into 10 sub-genera. Human and primate malaria parasites are all included in the sub-genera. P. *Plasmodium* and P. *laverania*, whereas species infecting other mammals are members of the heterogenus sub-genus P. *Vinckeia*. The four species of malaria parasites known to infects humans are: *Plasmodiumfalciparum, Plasmodium malariae, Plasmodium vivax* and *Plasmodiumovale* (Collier *et al.*, 1998; WHO 2003).

Hematological changes that are associated with malaria infection includes anemia (PCV<33%), thrombocytopenia (platelet < $150{,}000 \times 10^{-9}$), *lymphocytosis (*lymphocyte>60%), *monocytosis* (monocyte>9%), and disseminated intravascular coagulation (Facer, 1994; Perrin *et al.,* 1982, Maina *et al.,* 2010, Chandra and Chandra, 2013). Therefore, well informed changes in blood parameters in malaria infection enable the clinicians to establish reliable diagnosis and therapeutic interventions.

Literature review

Heamatological Changes due to Malaria Infections

In tropical countries like Nigeria, Malaria remains an important health problem. Hematological changes, which are the most common complications, play a major role in these fatal complications. They include *lysis* of the red blood cells leading to *anaemia, cytoadherence* of infected red blood cells leukocyte changes followed by the induction of cytokine and thrombocytopenia, and (Pavithran, 2007) Severe cerebral malaria is characterized by clogging of the post capillary venules by *Plasmodium falciparum* infected red cells. As *P. falciparum* matures, Knobs appear on the surface of the parasitized red cells to endothelia cells in capillaries and post capillary *venules* of the brain, kidneys and other affected organs. This cytoadherence accounts for the observation that mature asexual *P. falciparum* parasite are not seen in the peripheral blood smeared because they are sequestered in the peripheral microcirculation. The *P. falciparum* erythrocytic membrane protein, present on the surface of the parasitized red cells mediate cytoadherence of infected red cells to vascular endothelium through adhesion molecules, E-selection and vascular cell adhesion molecule. The malaria parasite uses blood group antigen A present on the surface of uninfected red cells for rosette (Pile) formation resulting in cerebral Vaso-occlusion by sequestered parasitized erythrocytes (Chen *et al*, 2000).

Anaemia (PCV < 33%) in Malaria

Anaemia is the most common complication in malaria. The incidence of anaemia in malaria was reported to be as high as 74%. Severe anaemia was observed predominantly observed in *P. falciparum* infection which characterized by hyper *parasitaemia* and systematic complication such as disseminated intravascular coagulation (Pavithran, 2007; reported that *Plasmodium falciparum* malaria infection is a contributory factor to the etiology of the anaemia in children in malaria endemic areas of the world. Abro *et al*., (2008) reported incidence of anaemia of up to 64% and are usually normocytic normachromic type in majority of cases. (Menendez et. al., 2007) also reported anaemia as one of the most common complications in malaria especially in younger children and pregnant women in high transmission areas. The mechanism of anaemia in *Plasmodium falciparum* parasitized patients is either due to haemolysis of parasitized red cells, exacerbated removal of parasitized red blood cells, bone marrow suppression, decreased erythropoietin level or due to ineffective erythropoiesis (Pavithran, 2007). *Plasmodium falciparum* malaria is one of the commonest causes of anaemia and correlates with its infection (Erhabor *et. al.*, 2006).*Plasmodium falciparum* was found to be the causes of malaria among parasitized subjects 210 (100%). This finding is consistent with a previous report that found *Plasmodium falciparum* as the predominant cause of malaria in Nigeria (Erhabor *et. al.,* 2006).

The pathogenesis of anaemia in malaria is extremely complex and factorial. It is thought to result from a combination of *haemolysis* of parasite red blood cells and accelerated removal of both parasitized and innocently *non parasitized* red blood cells, depressed as well as ineffective *erythropoiesis* with

dyserythropoietic changes and anaemia of chronic disease. Other factors contributing to anaemia in malaria include decreased red blood cells deformability, *splenic* pooling and/or phagocytosis resulting in increased rate of clearance from circulation (Abro *et al.,* 2008).

Changes in Platelets/Thromobocytes in Malaria Infection

Thrombocytopenia or platelet dysfunction are the two most important changes in malarial infection. Thrombocytopenia is seen in 40%-90% of patients infected with *P. falciparum.* Maximum thrombocytopenia occurs on the fifth or sixth day of infection, and gradually return to normal within 5-7 days after *parasitaemia* ceased. (Ovuakporaye, (2011), Kumar, (2006) found thrombocytopeniaa common occurance in children infected with *Plasmodium falciparum* Parasitaemia. (Adedapo *et. al.,* 2007) also find thrombocytopenia as one of the haemolytic challenges associated with malaria infection among children. The mechanism of thrombocytopenia in malaria is due to decreased thromobopoiesis despite normal or increased megakaryocytes in bone marrow (Abro *et. al.,* 2008), peripheral destruction induced by *Plasmodium falciparum* in which immune complexes generated by malarial antigens lead to sequestration of the injured platelets by macrophages in the spleen and consumption by disseminated intravascular coagulation (Pavithran, 2007).

Two types of platelets dysfunction are usually seen in malarial initially there is a platelet hyperactivity which is followed by platelet hypoactivity. Platelet hyperactivity result from various aggregating agents like immune complexes, surface contracts of platelet membrane to malaria red blood cells and damage to endothelial cells. The injured platelets undergo lysis intravascularity. The release of platelets contents can activate coagulation cascade and contribute to disseminated intravascular coagulation. Transient platelets hypoactivity is seen following this phase and return to normal in 1 - 2 weeks (Pavithran, 2007).

The incidence of Disseminated intravascular coagulation (DIC) is reported to be 4-13% (Murthy *et al.,* 2000). It usually occur in patients with *P. falciparum* infection and *hyperration* of coagulation cascade by the release of various sources such as lysis of platelets and red blood cells, *Cytokins micro-birculatorystasis*. *P. falciparum* infection was associated with increased plasma levels of *plasmogen* activator inhibitor, factor VIII R: Ag and reduced levels of protein C, protein S, and *antithrombin* III.

Leucocytes Changes in *Plasmodium* Infections

Haemalologic changes which are the most common complications in Malaria play a major role in the fatal complications. They include anaemia, cytoadherence of infected red blood cells, leucocyte changes, thrombocytopenia and coagulopathy (Pavithran, 2007). Changes in leucocyte proliferation and function are seen with severe *Plasmodium* infection. Leucocyte proliferation is associated with release of cytokines which are involved in cytoadherence, thrombocytopenia, disseminated intravascular coagulation hypoglacemia and lactic acidosis (Pavithran, 2007).

The white blood cells play a pivotal role in defense against *Plasmodium* infection (Staedke *et.al.,* 2004). Changes in white blood cells counts are response to parasite densities during follow up periods in treated children in *Plasmodium falciparum* infection but varies (Abro *et.al.,* 2008) and as a result of either levels of acuteness of infection, parasitaemia or host immunity (Ladhani *et.al.,* 2002). The total white blood cell counts within acute *Plasmodium falciparum* infection in children are usually within normal ranges in healthy individual, but there may be a slight decrease or upsurge (Leucocytosis) from the normal reference range.

In innate immunity, monocyte macrophages are the main immune effectors for controlling malaria blood stage infection via phagocytiv activity (Seghides *et.al.,* 2003). Activation through pattern recognition receptors (PRRs) present on monocytes, dendritic cells and neutrophils indece release pro inflammatory cytokines and chemokines the development of acquired immunity (Kolli *et.al.,* 2013). During acute infection monocytes produce high level of IL-IB,OL-112 and TNF-α, where as malaria pigment, hemozoin (Hz), production by monocytes contributing to dendritic cell maturation (Jaramillo *et.al.,* 2004). In humans, an impaired function on the maturation of monocytes and DCs due to malaria has been indicated by the reduced numbers of blood DCs inducing pregnan women (Diallo *et.al.,* 2008).

Eosinophils may play a role in the protection against malaria (*Plasmodium falciparum*) by induction of parasite killing Walter, *et al, 1987*) but they may also contribute to pathology by release of granule proteins such as eosinoiphil catonic protein (EC) as eosinophil protein x leasinophil-derived neurotoxin (EPX) (Durack *et al.,* 1981). Acute malaria in children is associated with decreased numbers of easinophils in peripheral blood, it also induces eosinophil production. Eosinophils might be stimulate either directly by the parasites or other mediators produced during the malaria attack (Walters et al., 1987)

There is ample evidence showing the potential of malaria infection to affect the counts of lymphocyte subpopulations in the peripheral blood. This is because the pathogenesis as well as the disease outcome of malaria is highly dependent on host genetics (Allen, *et al*., (1997), (Hill *et al.,* (1991) and parasite factors. (Chotivanich *et al.,* (2000).

The mechanism of neutropenia in malaria has been postulated to involve increased margination and sequestration of neutrophils (Dole and Wolff , 1973) as a result of the increased express of cell adhesion molecules (ICAM-I and VCAM-I) that occurs in malaria (Clark, *et. al.,* 2006). Other changes include *easinophil*

concentration on *neutrophils* and lymphocyte, thrombocytes and neutrophilic leukocytosis (Pukrittayakarne *et al.,* 1989). *falciparum* malaria also causes accelerated turnover of coagulation cascade. In severe disease, there is increased fibrinogen consumption, but in most cases plasma concentrations are either normal or elevated (Pukrittayakarnee *et al.,* 1989).

METHODOLOGY

STUDY AREA

Maiduguri Lies on latitude 11^0 40'N and longitude 13^0 5'E. The state occupies the greater part of the Chad basin and is in the North eastern part of Nigeria, the state share borders with the republic of Niger to the North, Chad to the North east and Cameroon to the East. Within Nigeria, the state shares boundaries with Adamawa state to the south, Gombe state to the west and Yobe state to the North West.

Maiduguri is the Capital of Borno State. It is located in the Sahel Savannah region of northeast Nigeria. The climate of Maiduguri is favorable, with a mean annual rainfall and temperature of about 650 mm and 32^0C respectively. The month of March and April are the hottest periods of the year with temperatures ranging between 30^0C and 40^0C. It is usually cold and dry during the harmattan, November to January being the coldest months. (Borno State Ministry of Information, 2015).

Ethical Clearance

Ethical permission was obtained from the Ethical Committee of the University of Maiduguri Teaching Hospital, to carry out the blood analysis using sysmex *haemotology* auto-analyzer of Immunology laboratory and it was also obtained from Primary health Care Department, Maiduguri Metropolitan Council to collect the blood sample from children attending the general out patient department of Bulumkutu Health Center Maiduguri, Borno State. Subject and head of Bulumkutu Health Center, Maiduguri, Borno State were educated on the collection of the blood samples and significance of the study.

Inclusion Criteria

All consecutively recruited children aged between 6-59 months visiting the pediatric outpatient department of the Bulumkutu Comprehensive Health Centre, Maiduguri, Borno State with history of febrile illness and whose parents and guidance consented to their inclusion in this study will be eligible to participate as subjects for this study.

Exclusion Criteria

All children less than 6 months and greater than 59 months and whose parent did not give inform consent were excluded from participating in this study.

Collection of Venous Blood by venipuncture

(I) Label collection tubes and pre-cleaned slides preferably (frosted end) with the patient's name, date and time of collection
(II) Clean the slide with alcohol and allow it to dry
(III) Collect the venous blood in a vacuum tube containing anti-coagulant (preferably EDTA)
(IV) Prepare at least two thick smears and two thin smears as soon as possible after collection (CDC, 2004).

Preparation and Examination of Blood Films

The examination was conducted according to Cheesbrough (1999) in the following procedures: thick film is completely dry a drop of immersion oil was applied onto the film. Oil was spread to cover an area of 10mm in diameter. The preliminary scanning of the blood film was done with x10 and x40 objective to select area of good staining and correct thickness, the slides was viewed using x100 objective for presence of parasitized cells. (up to 100 high power microscope field was examined). Blood samples were obtained from patients by trained laboratory staff on duty. Thick and thin blood films were made by spreading a drop of blood on a clean, grease-free, labeled slide and then allowed to dry. The dried blood films were then stained with 10% Giemsa stain solution and washed after 10 min using clean water. The stained films were allowed to dry and on addition of a drop of immersion oil, each slide was examined under ×100 objective lens for malaria parasites. The examination was conducted according to Cheesbrough, 1999 while the densities of positive slides were estimated by the methods described by (WHO, 2008).`

Thick Blood Film

The drop of well mixed whole blood was placed on a clean grease – free slide. Using a glass spreader, it was spread to the size of a small coin. The thickness was made in such a way that the hands of a wrist watch can be seen through the film. It was allowed to air dry free from dust and flies and labeled with patient identity. (Cheesbrough, 1999).

Thin Blood Film

A drop of blood was placed at the Centre near one end of a clean grease free slide. A glass spreader was placed on the slide and drawn back to touch the drop of the blood. When the blood spreads to the edges of the spreader, the spreader was moved forward at an angle of 45^0 without interruption to obtain the thin blood film. It was allowed to air dry to free from dust and flies and labeled with patient identify.

Determination of parasite density

The thick film slide was stained for 30 to 45 minutes with 3% Giemsa for the assessment of parasite density. The samples were examined using objectives of a research microscope (x100) asexual parasites were counted alongside with 200 leukocytes. In an even that parasite count was <10 parasites/200 leukocytes; count was continued per 500 leucocytes. The parasite density was expressed as the number of asexual parasites per ml of blood by assuming a mean normal leukocyte count of 8000/µl of blood Gilles and Warrell, 1993 and modified by (WHO, 2008). Parasitaemia (per µl) = number of parasites x 8000 / number of leucocytes (200/500).

Blood Analysis

The collected samples were transferred to the laboratory for the estimation of blood parameters such as, packed cell volume, platelate, white blood cell, neutrophil, eosinophil, monocytes and lymphocytes by using sysmex hematology Autoanalyser, (2011).The Standard Operating Procedure for Sysmex Hematology Autanalyser (2011).

(I) Switch on the machine and wait until, the visual Display unit (VDU) is in the "ready" mode, this takes about five (5) minutes (SOP, 2011).
(II) Place the sample on a mixer to mix for about five (5) minutes.
(III) Open the well–mixed sample of blood in EDTA and prime it with probe.
(IV) Using the sysmexautoanalyser aspirate the sample to analyze automatically.
(V) Remove from the auto-analyser as bit sound is made.
(VI) The result of blood parameters such as White Blood Cells, Red Blood Cells, Platelets, Packed Cell Volume, lymphocytes counts, neutrophlyles, Haemoglobin and percentage are recorded.
(VII) Any blood sample where haemoglobin concentration of less than 8.0d/dl considered prick value to be reported to the clinic immediately.viii.
(VIII) The result are recorded in result sheets after quality officer is certified. `

Statistical Analysis

Data collected were subjected to descriptive statistic using the statistical package for social science SPSS version 20.0 (Armand and Jon peck, 2011) and analysis software statistics version 8.0 (Microsoft, 2013) measure of central tendencies (standard deviation percentages) were determined. Differences were considered significant when $P<0.01$ or 0.05.

II. Results

Results presented in table 1 showed the characteristics of the base line of enrollment in the study population. A total number of 210 children were enrolled for the study52 (24.76%) were male tested negative, 64(30.48%) tested positive and 36 (17.14%) were female tested negative and 58(27.62%) were female tested positive. Mean S.D to estimate variability in the data set was observed, consequently the age of the subject were highly disperse between 6-59 months from the mean SD of 42.0± 55.55 tested positive and 31.0±18.96 tested negative.

Table 1: Characteristics Baseline of Enrolment of the participant in Bulumkutu Comprehensive Health Centre, Maiduguri.

Variables	Tested positive	Tested negative	Total
No enroll age (month)	88	122	210
Mean	42.00	31.00	73.00

S.D	55.55	18.96	74.51
Range (in month)	6-59	6-59	6-59
Gender			
Male	52.0(24.76%)	64.0(30.48%)	116
Female	36.0(17.14%)	58.0(27.62%)	94

Table 1: Generally, more patients were reported to have relatively low Packed Cell Volume (PCV) counts in age group 6-10, 11-20, 21-30, and 51-60 months as shown in fig. i, ii, iii and vi respectively. In addition, the age group 31-40 and 51-50 months recorded equal number of patients with low and normal Packed Cell Volume (PCV) as indicated in fig iv and v respectively.

It was observed that the age group 51-60 month had highest platelet counts, and white blood cell counts, while the age group 31-40 had the lowest platelets and while blood count as reported in fig vi and iv respectively.

The result of table 8 also revealed that the children with the age group 51-60 months had highest neutrophil and monocytes counts, while the children within the age group 41-50 recorded lowest neutrophil and monocytes counts as presented on fig v.

Moreover, the children within the age group 6-10 months recorded low Eosinophil and lymphocyte counts as showed in fig I where as other age groups had fairly normal counts.

Table 2: Variation of blood cells counts from the normal counts based on age.

	Age(6-59 months)																							
Blood	6 – 10				11 – 20				21 – 30				31 – 40				41 – 50				51 – 60			
Cell	N	Low	Nor	High	N	Low	Nor	High	N	Low	Nor	High	N	Low	Nor	High	N	Low	Nor	High	N	Low	Nor	High
Indices																								
PCV	08	07	01		14	14	00		19	17	02		04	02	02		10	05	05		33	24	09	
Platelet	08	01	05	02	14	03	06	05	19	02	12	05	04	00	01	03	10	02	05	03	33	05	19	09
WBC	08	01	03	04	14	00	09	05	19	01	11	07	04	00	04	00	10	00	08	02	33	00	24	09
Neutrophil	08	02	05	01	14	05	08	01	19	04	08	07	04	01	03	00	10	00	07	03	33	02	19	12
Eosinophil	08	01	04	03	14	04	09	01	19	08	06	05	04	02	02	00	10	04	05	01	33	20	09	04
Lymphocyte	08	01	06	02	14	02	06	06	19	07	08	04	04	01	01	01	10	04	06	00	33	12	19	02
Monocyte	08	03	05	00	14	04	07	03	19	07	09	03	04	02	02	00	10	01	06	03	33	08	18	07

Normal values: PCV → (33%), Platelet → (150,000-400,000 x 10^{-9}), WBC → (4.5 – 13x-9), Neutrophil → (30-65%), Eosinophil → (1-4%), Lymphocyte → (30-60%), Monocyte → (1-9%), N = Number (group). WHO, (1996)

The result presented in table 3 showed the variation of some hematological parameters from the normal blood counts. In which, the children within the age group 51-60 months (27.27%) had a highest percentage of low PCV counts, whereas the children within the age group 31-40 months (2.27%) had the lowest percentage of PCV counts as shown in figure 6 and 4 respectively. In addition, the results also showed that the children within the age group 51-60 months (10.23%) had the highest percentage of normal PCV counts, whereas the children within the age group 6-10 months (1.14%) had the lowest percentage of normal PCV counts as shown in figure 6 and 2 respectively.

Furthermore, it was found that the malaria infected subjects within the age bracket 51-60 months recorded a high percentage of children with thromobocytopenia (low platelets counts) and leucocytopenia (low WBC counts) with an occurrence of 5.68% and 2.27% and also a high percentage of thrombocytosis (high platelets counts) and leucocytosis (high WBC counts) with an occurrence of 10.23% and 13.63% respectively as indicated in figure 6.

However, the result obtained on neutrophil counts showed that the children within the age group 11-20 months (5.68%) recorded the highest percentage of neutropenia (low neutrophil counts), while the children within the age group 51-60 months (13.63%) recorded the highest percentage of neutrophilia (high neutrophil counts) as shown in figure 3 and 6 respectively.

Moreover, the result presented in table 3 showed that the malarial infected subjects within the age bracket 51-60 months (2.72%) had the highest percentage of Eosinopenia (low eosinophil counts) as shown in figure 6, while the malarial infected subject within the age group 21-30 months (5.68%) had the highest percentage of eosinophilia (high eosinophil counts) as indicated in figure 3

The result presented in table 3 showed that the children within the age group 51-60 months (13.63%) recorded highest percentage of lymphocytopenia (low lymphocyte counts), whereas the children within the age group 11-20 months (6.82%) recorded the highest percentage of lymphocytosis (High lymphocyte counts) as indicated in figure 6 and 2 respectively.

Both monocytopenia (low monocyte counts) and monocytosis (high monocyte counts) was found among the malarial infected subjects within the age group 51-60 months had the percentage of occurrence of 9.09% for monocytopenia and 7.95% of monocytosis as indicated in figure 6

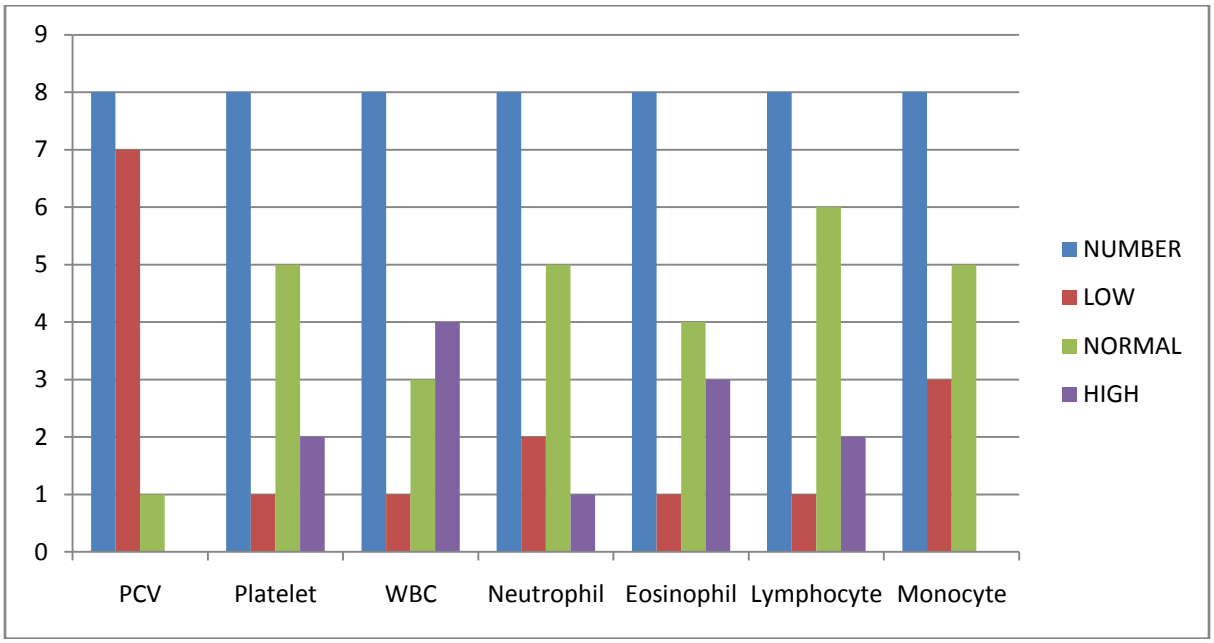

Fig 1: Variation of Blood Cell Counts from the Normal Count with Age 6-11 month

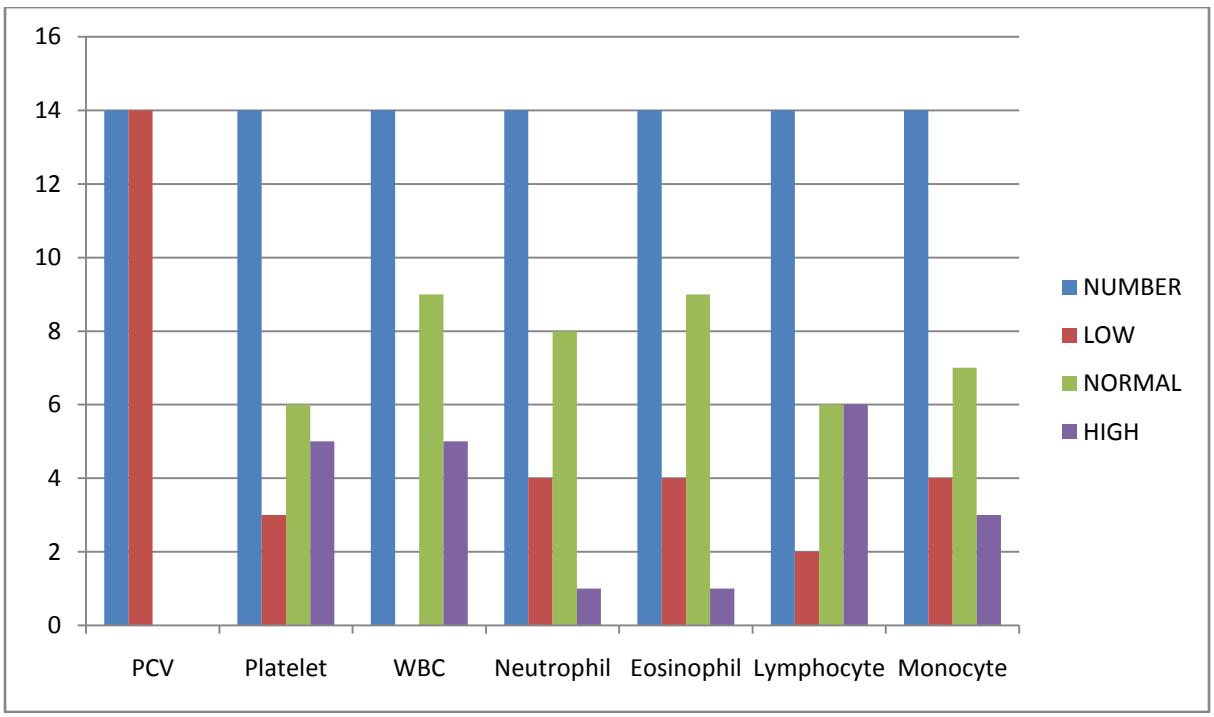

Fig 2: Variation of Blood Cell Counts from the Normal Count with Age 11-20 month

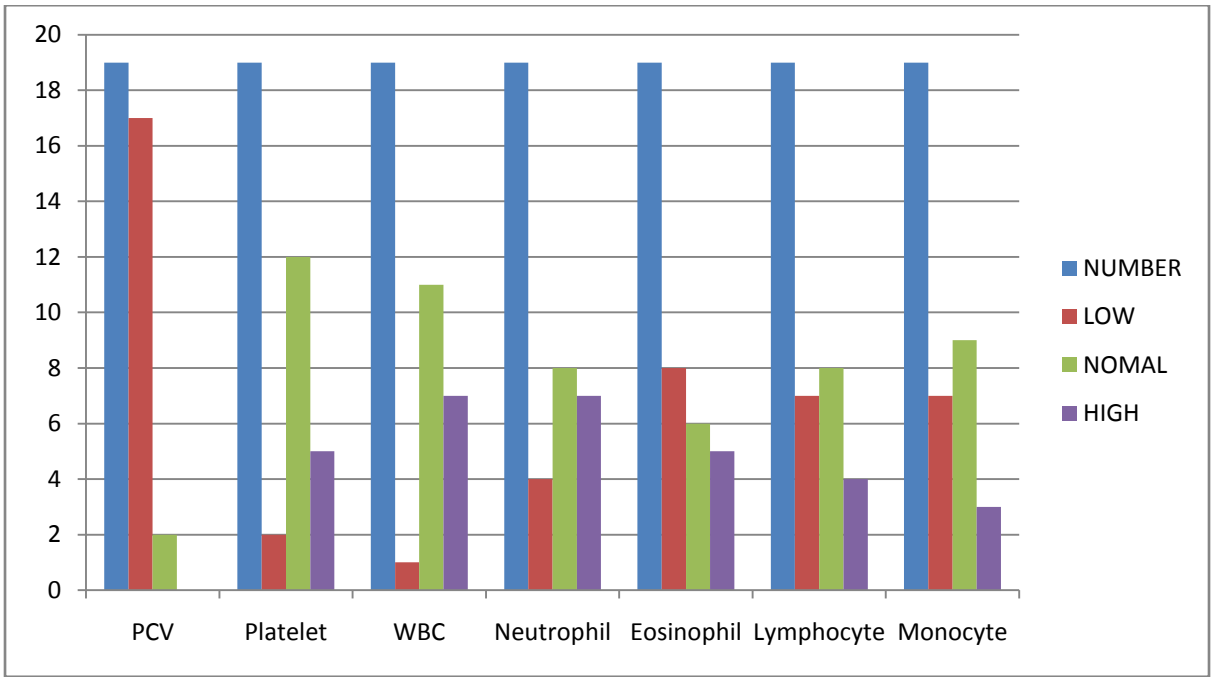

Fig 3: Variation of Blood Cell Counts from the Normal Count with Age 21-30 month

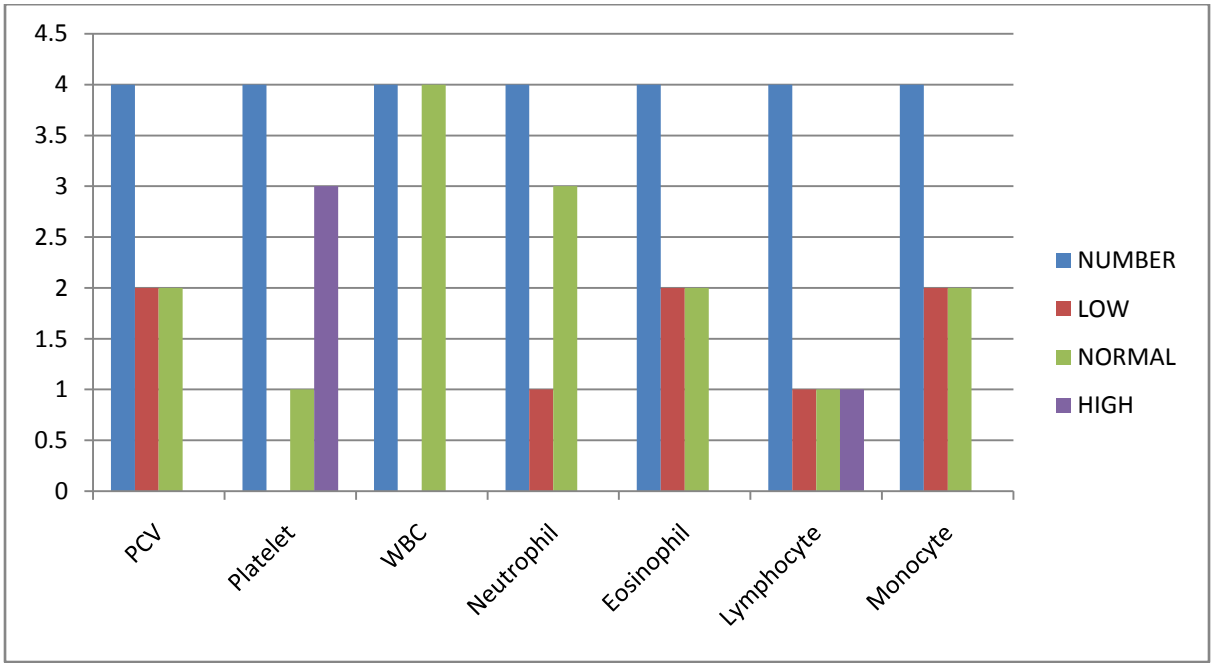

Fig 4: Variation of Blood Cell Counts from the Normal Count with Age 31-40 month

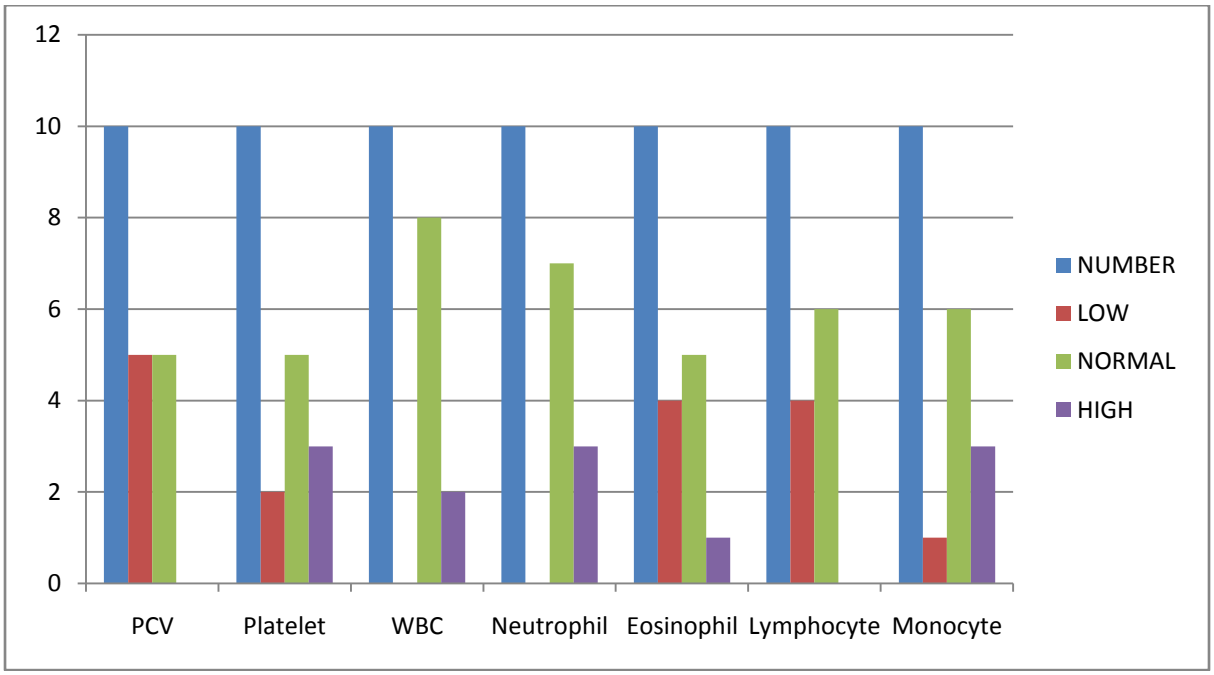

Fig 5: Variation of Blood Cell Counts from the Normal Count with Age 41-50 month

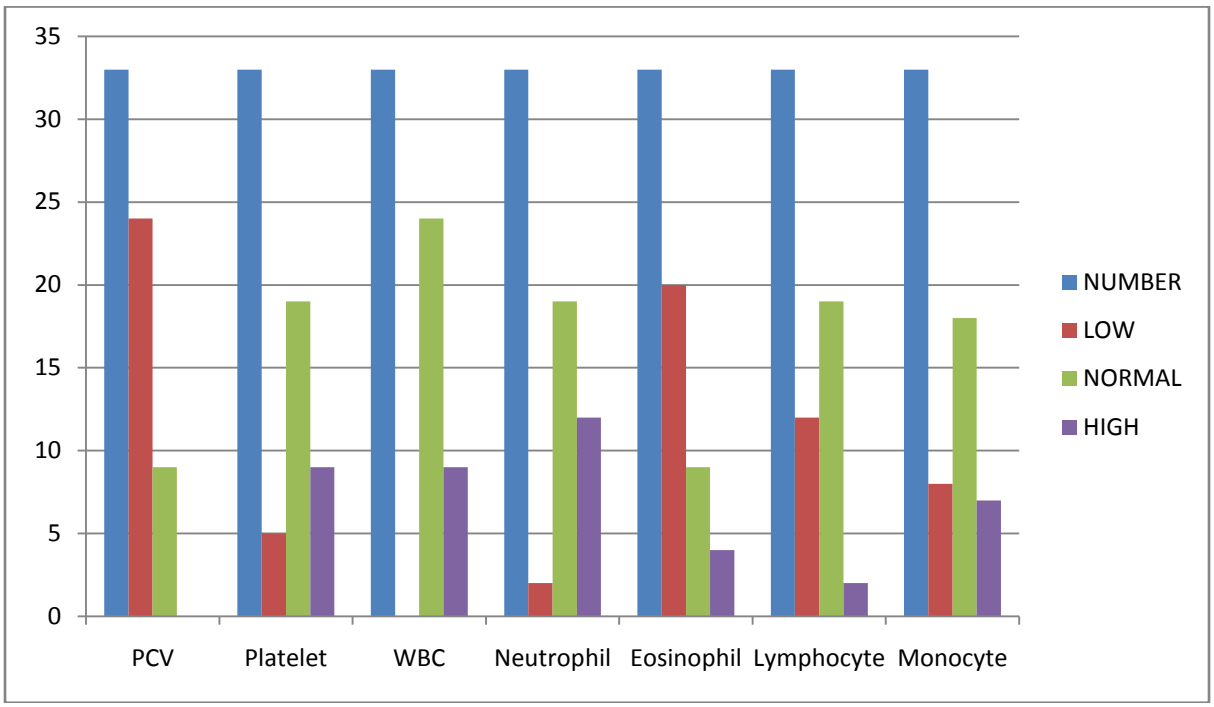

Fig 6: Variation of Blood Cell Counts from the Normal Count with Age 51-59 month

Table 3: Variation of the PCV, Platelets, WBC and different leucocyte blood counts from normal counts

	AGE(in month)											
Blood		**6**	**-**	**10**		**11**	**-**	**20**		**21**	**-**	**30**
parameters	**N**	**Low**	**Normal**	**High**	**N**	**Low**	**Normal**	**High**	**N**	**Low**	**Normal**	**High**
PCV	88	7.95%	1.14%	-	88	15.91%	0.00%	-	88	19.32%	2.27%	-
Platelets	88	1.14%	5.68%	2.27%	88	3.41%	6.82%	5.68%	88	2.27%	13.64%	5.68%
WBC	88	1.14%	3.41%	4.55%	88	0.00%	10.23%	5.68%	88	1.14%	12.5%	7.95%
Neu	88	2.27%	5.68%	1.14%	88	5.68%	9.09%	1.14%	88	4.55%	9.09%	7.95%
Eosi	88	1.14%	4.55%	3.41%	88	4.55%	10.23%	1.14%	88	9.09%	6.82%	5.68%
Lym	88	1.14%	6.82%	1.14%	88	2.27%	6.82%	6.82%	88	7.95%	9.09%	4.55%
Mon	88	3.41%	5.68%	0.00%	88	4.55%	7.9%	3.41%	88	7.95%	10.23%	3.41%
		31	**-**	**40**		**41**	**-**	**50**		**51**	**-**	**60**

	N	Low	Normal	High	N	Low	Normal	High	N	Low	Normal	High
PCV	88	2.27%	2.27%	-	88	5.68%	5.68%	-	88	27.27%	10.23%	-
Platelets	88	0.00%	1.14%	3.41%	88	2.27%	5.68%	3.41%	88	5.68%	21.59%	10.23%
WBC	88	0.00%	4.55%	0.00%	88	0.00%	9.09%	3.41%	88	2.27%	21.59%	13.64%
Neu	88	1.14%	3.41%	0.00%	88	0.00%	9.09%	3.41%	88	2.27%	21.59%	13.64%
Eosi	88	2.27%	2.27%	0.00%	88	4.55%	5.68%	1.14%	88	2.27%	10.23%	4.55%
Lym	88	1.14%	2.27%	1.14%	88	4.55%	6.82%	0.00%	88	13.63%	21.59%	2.27%
Mon	88	2.27%	2.27%	0.00%	88	1.14%	6.82%	3.41%	88	9.09%	20.45%	7.95%

Normal values: PCV → (33%), Platelet → (150,000-400,000 x 10^{-9}), WBC → (4.5 – 13x-9), Neutrophil (30-65%), Eosinophil → (1-4%), Lymphocyte → (30-60%), Monocyte → (1-9%), N = Number (group) WHO, (1996).

From the result presented on table 4 it was found that, about 78.41% of the subject had a low PCV, while 21.59% of the children tested positive had a fairly normal PCV as it was also reported that 14.77% of the subject had low thrombocytes, while 48.55% of the subjects had a normal thrombocytes and then 30.68% had a higher thrombocytes counts. Similarly, the results also revealed that 2.27% of the children tested positive had a white blood cell below normal counts, while 63.64% of the subjects are within the normal range, and 34.09% of the children had higher white blood cells counts. However 15.91% of the subjects had higher neutrophil and eosinophil counts. Whereas 15.91% and 44.31% showed up with low neutrophil and eosinophil counts. The lymphocyte and monocyte counts were reported to have equal percentage of occurrence with normal counts. Whereas, 30.68% and 25.41% recorded a higher counts, while more than 15% where observed to be higher than the normal counts.

Table 4: Variations of the overall percentage of PCV, Platelets, WBC and different leucocyte counts from normal counts

Blood Cell Indices	N	Low	Normal	High	Total
PCV	88	69 (78.41%)	19 (21.59%)	-	100%
Platelets	88	13 (14.77%)	48 (54.55%	27 (30.68%)	100%
WBC	88	02 (2.27%)	56 (63.64%)	30 (34.09%)	100%
Neutrophil	88	14 (15.91%)	50 (56.82%)	24 (27.27%)	100%
Eosinophil	88	39 (44.31)	35 (39.77%)	14 (15.91%)	100%
Lymphocytes	88	27 (30.68%)	47 (53.41%)	14 (15.91%)	100%
Monocytes	88	25 (28.41%)	47 (51.41%)	16 (18.18%)	100%

Normal values: PCV → (33%), Platelet → (150,000-400,000 x 10^{-9}), WBC → (4.5 – 13x-9), Neutrophil → (30-65%), Eosinophil → (1-4%), Lymphocyte → (30-60%), Monocyte → (1-9%), N = Number (group) WHO, (1996).

III. Discussion

The white blood cell plays a pivotal role in defence against *Plasmodium falciparum* infection Staedke *et al.,* (2004). The total white blood cell and differential leucocytes counts within acute *Plasmodium falciprum* infection in children are usually with normal ranges in healthy individual, but there may be a slight decrease from the normal reference range Rwagacondo, *et al,* (2004).

Hematological changes are the most common complications that play a major role in those fatal complications. They include lysis of the red blood cell, leading to anaemia cytoadherence of infected red cells, leucocytes changes coagulopathy, particularly intravascular coagulated (Pavithran, 2007), others are lymphocytosis, leucopenia leucocytosis, neutrophilia and monocytosis have all been reported Abro *et al.,* (2008).

The study showed parasite densities influenced some hematological parameters in positive malaria in children (6-59months). A case study of bulumkutu health centre Maiduguri Borno State. During this study it was observed that 210 (41.90%) children aged between 6-59 months visited the pediatric outpatient department were positive for *Plasmodium falciparum* malaria. This finding is concurrent with previous reports from Nigeria by (FMOH, 2005) that obtained 40% annual prevalence rate found in Nigeria. This finding is also concurrent with previous report by Ojukwu, (2002) 50% in North East, North Central, North West and South South regions of Nigeria respectively. But, this study contradicted other finding by Ojukwu, (2002) who in a similar research, in South Eastern part of Nigeria report 17% prevalence rate.

There was a relatively higher prevalence of infection 52 (59.09%) among males than females 36 (40.91%) of female subject (p>0.05%). However reports indicated higher prevalence in males than females (WHO, 2005; WHO, 2006) with no evidence on higher prevalence to gender susceptibility to malaria infection is not influenced by gender Giles and warell, (1993). The higher prevalence rate among male could just be by chance.

This study linked *hematological* abnormalities as a hallmark for assessing malaria infection. The abnormalities previously reported include changes in packed cell volume (anemia/pcv<33%), platelets, leucocytes, differential leucocytes counts and disseminated intravascular coagulation (DIC) Reyburn *et al.,* (2007).

The results presented in table 3 showed the variation of some hematological parameters from the normal blood counts Pcv → (33%), platelate → (150,000 – 400,000x10^3), White blood cell → (4.5 – 13x10^3), Neutrophil → (30 – 65%), Eosinophil → (1- 4%), Lymphocyte → (30 – 60%), Monocyte → (1 – 9%) (WHO 1996). During this study, it was observed that the children within the age group 51 – 60 months (27.27%) had a highest percentage of low Pcv counts, whereas the children within the age group 31 – 40 months (2.27%) had the lowest percentage of Pcv counts as shown in figure 6 and 4 respectively. In addition, the results also showed that the children within the age group 51 – 60 months (10.23%) had the highest percentage of normal Pcv counts whereas the children with the age group 6 – 10months (1.4%) had the lowest percentage of normal Pcv counts (33%) as shown in figure 6 and 2 respectively. The result obtained in this study tallies with Evans *et.al,* 2006 that anemia is relative to *Plasmodium falciparum* malaria especially in severe cases of younger (<5years) children. It was also reported that the malarial infected children within the age group 51 – 60 months (5.68%) recorded a highest percentage of children with low Platelate count (thrombocytopenia), similarly, children within the age group 51-60 (10.23%) months also recorded a highest percentage of subjects with high Platelate counts (throobocytosis) as indicated in figure 6 which is concordance with previous report by Kumar (2006); Ovuakporaye (2011) that thrombocytopenia is a common occurrence in children infected with *Plasmodium falciparum* parasitaemia. Furthermore, it was also found out that the malarial infected subjects within the age group 51-60 months (2.27%) recorded a high percentage of children with low white blood cell count (Leucocytopenia), similarly, children with the age group51-60 months (13.63%) recorded a high percentage of children with high white blood cell counts (Leucocytosis). This finding is in line with Ladhani *et al.,* 2002 that the total white blood cells count within acute *Plasmodium falciparum* infection in children are usually within normal ranges, but there may be a slight decrease or upsurge. However, the result obtained on neutrophil counts showed that the children within the 11-20 months (5.68%) recorded the highest percentage of neutropenia (low neutrophil counts), while the children within the age group 51-60 moths (13.63%) recorded the highest percentage of neutrophilia (high neutrophil counts as shown in figure 3 and 6 respectively. This finding is in agreement with Pukrittayakarne *et.al* 1989 that neutrophilia has also been found to be associated with malaria Parasitaemia. Moreover, the result presented in table 3 showed that the malaria infected subjects within the age group 51-60 months (2.27%) had the highest percentage of Eosinophenia (low eosinophil counts) as shown in figure 27, while the malaria infected subjects within the age group 21-30 months (5.68%) had the highest percentage of eosinophilia (high eosinophil counts) as indicated in figure 3. This finding is consistent with Davis *et al*., 2003 who reported that the acute malaria with or without limited previous exposure to *Plasmodium* infection is usually associated with high eosinophil counts, followed by persistent eosinophila. Furthermore, the result presented in table 3 showed that the children within the age group 51-60months (21.59%) of children with normal lymphocyte count. Similarly, children within the age group 51-60 months 13.63% also recorded highest percentage of lymphocytopenia (low lymphocyte count) whereas the children within the age group 11-20 months (6.28%) recorded the highest percentage of lymphocytosis (high lymphocyte counts) as indicated in figure 6 and 2 respectively. This finding agrees with a previous study of Abdalla and Pasvol, 2004 that the lymphocyte count remains normal during an acute malaria infection. But disagrees with maina *et al*., 2010. Who reported lymphocytopenia as a common finding in acute malaria in children found in malaria endemic Areas. Similarly, the result also revealed that both monctopenia (low monocyte counts) and monocytosis (high monocyte counts) was found among the malaria infected subjects within the age group 51-60 months with these percentage of occurances 9.09% for monocytopenia and 7.95% for monocytosis as indicated in figure 6. This finding agrees with Abdalla and Pasvol, 2004 reported monoctosis as one of the most consistent observations reported from prior studies done on hematological studies that characterized malaria.

The incidence of anaemia in malaria infected subject in this group is (78.41%) as presented in table 4. The result is in agreement with Abro *et al.,* (2008) with the incidence of anaemia to 64% and are usually normochromic type in majority of cases.

The thromboccyte counts in children malaria showed a downward trend thrombocyte concentration (thrombocytopenia) with an increase in parasiteamia. The incidence of thrombocytetopenia in this study was (30.68%) in children infected with *Plasmodium falciparum*. This finding is in line with report from Kumar, (2006) who reported thrombocyletopenia in 40-90% of patients infected with *Plasmodium falciparum*.

There is a marginal difference of lencocytes concentration (34. 09%) in the present study. This finding is concordance with a previous report by Pavithran, (2007) that changes in leucocyte proliferation (leucocytosis) and its functions are seen with severe *Plasmodium falciparum* Infection. The differential lencocyte count showed a normal neutrophil count in majority of *Plasmodium falciparum* infected children (56.82%). This finding is similar to the mechanism of neutropenia in malaria has been postulated to involve increased

margination and sequestration of neutrophils (Dole and Wolff, 1973). This study also revealed a marginal difference of eosinophic concentration (27. 27%) in malaria positive children. This tallies with Akhtar *et al.,* (2012) and disagrees with Maina *et al*., (2010). In this study the incidence of lymphocytopenia was higher in malaria infected subjects (30. 68%). This is in agreement with Allen *et al*., (1997), Hill *et al*., (1991) and Chotivanich *et al*., (2000) respectively who stated that there is ample evidence showing the potential of malaria infection on to affects the counts of lymphocyte sub- population in the peripheral blood. This is because the pattogenesis as well as the disease outcome of malaria is highly dependent on host genetics. There is also a marginal difference of monocytosis concentration (18.18%) in the present study. This finding is in line with a previous study of (Abadalla *et al.,* 1988) who reported high monocyte count in patients with uncomplicated malaria.

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AUTHORSPROFILE

Prof. Mustapha Kokori, received B. Sc. degree. Zoology and General Biology, University of Maiduguri in 1990, M.Sc. in Applied Medical Parasitology of University of Ibadan, Nigeria in 1994. He did Ph.D in Applied Medical Parasitology - with reference to "Efficacies of Combination Therapies of Antimalarial Drug Safety in Children of Tropical Africa in 2004: A case study on Rural settlements of Lake alau, Borno State, Nigeria. Currently he is a Professor in the department of Biological Sciences, faculty of science, University of Maiduguri, Nigeria.

Dr. Musa Dahiru did B.Sc in Microbiology, Bayero University, Kano, M.Sc and PhD in Environmental and Public Health Microbiology in University of Benin. He is currently an Associate Professor in the Department of Biological Sciences, Faculty of Science, Federal University of Kashere Gombe State, Nigeria.

Mr. Yahaya Inuwa did B.Sc in Zoology and General Biology, M.Sc Applied Entomology and Parasitology University of Maiduguriin 2007 and 2019 respectively with reference to " Influence of Plasmomium falciparum Parasitaemia on Some Heamatological Parameters Among Children (6-59 Months). A Case Study of Bulumkutu Comprehensive Health Centre, Maiduguri, Borno State, Nigeria.

Mrs Rashidat Ejura Momoh received B.Sc in Biochemistry, from University of Jos, M.Sc in Pharmaceutical Chemistry from University of Lagos. She is currently an Assistant Lecturer in the Department of Chemical Sciences, Facutly of Science, Federal University of Kashere, Gombe State, Nigeria.

Yahaya Inuwa, et. al. " Variation of Some Hematological Parameters from the Normal Blood Counts as a result of Plasmodium falciparum (parasitaemia) Infection in children (6-59 Months). A Case Study of Bulumkutu Comprehensive Health Centre Maiduguri, Borno State – Nigeria." *IOSR Journal of Pharmacy and Biological Sciences (IOSR-JPBS)*, 16(1), (2021): pp. 01-11.