# Seasonal Prevalence of Malaria Parasites Infection and Hematological Alteration Among Children (6-59 months) at Pediatric Out-Patient Department of Bulumkutu Comprehensive Health Centre, Maiduguri, Borno State. Nigeria

Y. Inuwa, U Babagana, I. Linus

Department of Biological Sciences, Faculty of Science, University of Maiduguri, Maiduguri Nigeria

Email: ibninuways@vahoo.com

Department of Basic Sciences, Yobe State College of Agriculture Gujba, PMB 1104, Damaturu, Nigeria.

Email: <u>bgumar2005@gmail.com</u>

# Department of Biological Sciences, Faculty of Science, University of Maiduguri, Maiduguri Nigeria

#### **ABSTRACT**

This study investigated the influence of P. falciparum Parasitaemia on some selected haematological parameters in children (6-59 months), at Bulumkutu Health Centre, Maiduguri Borno State, Nigeria. Between November 2015 to February 2016. The aim was to determine the influence of parasites densities on some selected haemotological parameters, to assess the influence of sex distribution of children on the influence of parasteamia in relation to some haemotological parameters in children malaria. A total of 210 children were enrolled in this study consisting of 8 (41.90%) patients infected with P. falciparum malaria a total of 52 (.24.76%) were male tested positive and 36 (17.14%) were females tested positive. While 122 (58.10%) were negative malaria children, out of which 64 (30.48%) were male tested negative and 58 (27.62%) were female tested negative. Haemotological parameters were analyzed using sysmexhaemaology auto-analyser (2011), while the Giemsa stained was prepared from the stock solution, and the testing for p. falciparum malaria and malaria parasite density count was carried out using the Giemsa stained thick and thin blood film. The mean PCV, Platelet Counts and lymphocyte of subjects was significantly lower compared to malaria negative individual. (27.31%, 342.5 x  $10^{-9}/\mu$ / and 41.80%) versus (34.52%, 382.5 x  $10^{-9}/\mu$ / and 43.89% respectively p = 0.05). While the mean WBC, Neutrophil, Eosinophil, Lymphocyte and Monocyte of malarial infected subjects was significantly higher among malarial infected subjects compared to the malaria negative children. (11.61 x 10<sup>-9</sup>/µ/, 52.53%, 2.28%, and 4.121%) versus (10.25 x  $10^{-9}/\mu$ /, 48.77%, 2.14%, and 4.057%) respectively (p = 0.05). The result further showed parasite density count also significantly influences the haematological parameters of male and female subjects as mean of PCV, WBC, lymphocytes and monocytes of male subject was significantly lower compared to their female counterparts. (26.85%,  $11.398X10^{-9}$ / µl, 43,782% and 4.424% respectively P=0.05) Hence, the need for prompt and treatment of clinical malaria episode with anti-malaria drugs for children infected with malaria in Nigeria.

Key words: Prevalence, Malaria, Plasmodium, Children

#### INTROUCTION

Malaria is an acute and chronic parasitic disease caused by an obligate intracellular protozoan of the genus Plasmodium. (Eneanya, 1996). It is caused by the protozoan parasites of Plasmodium species and transmitted through the bites of infected female Anopheles mosquitos. The miseries these parasites inflict on humans remain a major health challenge and a problem worldwide (Oluwafemi, 2003). The genus Plasmodium has four species, causes malaria in man, namely Plasmodium vivax, Plasmodium malariae, Plasmodium ovale and Plasmodium falciparum (Ukagaet al., WHO, 2003 2000). Plasmodium falciparum is the most virulent species and accounts for over 90% of human malaria infection (Ekanamet al., 1990). Malaria is an endemic parasitic infection in more than 99 countries and it is one of the world major causes of death worldwide (Fortin et al., 2002). Haemalologic changes are the most common complications Malaria which plays a major role in the fatal complications. They include anaemia, cytoadherence of infected red blood cells, leucocyte changes, thrombocytopenia and coagulopathy (Parithran, 2007). Changes in leucocyte proliferation and function are seen with severe Plasmodium infection. Leucocyte proliferation associated with release of cytokines which are involve in cytoadherence, thrombocytopenia, disseminated intravascular coagulation hypoglacemia and lactic acidosis (Parithran, 2007).

The previous studies confirmed hematological abnormalities are considered a hallmark of malaria infection are common and are pronounced in Plasmodium falciparum malaria infection, probably due to higher levels of parasiteamia found in these children. The abnormalities previously reported include changes in parked cell volume, platelets, leucocyte, differential leucocyte counts and disseminated intravascular coagulation (DIC) (Reyburn et.al., 2007), (Wickramasignleet.al., 2000), (Richards et.al., (1998). (Bushawriet.al., (2002) (Chiwakataet.al., (2000), reported that there was no significance difference in white blood cells count between malaria infected and non-infected groups. Atypical Lymphocitosis, leucopenia and leukocytosis neutrophilia, eosinophilia and monocytosis have all been reported (Abro et al., 2008) leucopenia appears as a common finding in a patient with Plasmodium falciparum malaria when white blood cell counts may be as low as 1-2X10<sup>9</sup>/<sup>1</sup> (Erhabor et al., 2006). Changes in leucocyte proliferationsuch as leucopenia have been reported in severe Plasmodium falciparum (Abro et al., 2008).

Eosinophil are white blood cells of the immune system that are responsible for combatting infection in vertebrates and they also control mechanisms associated with allergic reactions. Eosinophil (Acid loving cells) make up about 1-4% of the white blood cells, and are about 12-17 micrometers in size (WHO, 1996), (Richards et.al., 1998). The decrease in lymphocyte counts associated with malaria parasitaemia may be due to reflecting redistribution of lumphocytes sequestration in the spleen (Wickramasinghe et al., 2000). The high monocyte count had been reported in patient with an uncomplicated malaria (Abdalla et.al., 1988).

# **Study Area**

Maiduguri Lies on latitude 11<sup>0</sup> 40'N and longitude 13<sup>0</sup> 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°C respectively. The month of March and April are the hottest periods of the year with temperatures ranging between 30°C and 40°C. 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 carried out the blood analysis using sysmexhaemotology auto-analyzer of Immunology laboratory and it was also be obtained from Primary health Care Department, Maiduguri Metropolitan Council. Subject and head of Bulumtuku, comprehensive health centre 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.

# **Preparation and Examination of Blood Films**

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, labelled 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  $\times 100$  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 Kolhatkar, (2007).

#### 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. (Cheesbough, 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<sup>0</sup> without interruption to obtain the thin blood film. It was allowed air dry to free from dust and flies and labeled with patient identify.

# **Determination of parasite density**

The technique followed on both thick and thin films as described by John and Kolhatkar, (2007). The white cell remains intact even in thick films. It's necessary that the films should be evenly spread so that the white cell and parasite are evenly distributed.

Using the microscope in oil emersion (objective x 100) count 100 white blood cell and the number of parasite in the area covered. Repeat the same procedure twice and take average of the three count. Count the number of white blood cells per cubic mm and calculate the number of parasite as

# **Blood Analysis**

The collected samples was transferred to the laboratory for the estimation of blood parameters such as white blood cells packed cell volume, lymphocytes, monocytes, neutrophil, eosinophil, platelets and by using sysmex hematology Autoanalyser, (2011). The result will be recorded alongside findings of each subject's data.

# **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.

## **RESULTS**

Results presented in table 1 showed the characteristics 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  $42.0\pm55.55$  tested positive and  $31.0\pm18.96$  tested negative.

Table 2 shows comparison of heamatological parameters between positive and negative Malaria in Children. The mean standard deviation of park cell volume (PCV) count for positive was lower PCV 25.4±1.2 compared to the negative Malaria Children with a mean 30.6±0.9. Also, the mean standard deviation of platelet count 38.5184.9 among negative children was significant (p = 0.05) compared to 342.5±204.6 positive subjects. The results further showed that parasite density count significantly influences white blood cells as mean SD 11.61±5.36 recorded in positive subject was significant compared to children tested negative. 10.25±8.94 Similar finding was also obtained in neutrophil with significant (P=0.05) mean SD 52.53±19.07 in positive subject compared to 48.77±18.92 in negative children. Similar, result was recorded in eosinophil with a significant (P=0.05) means SD 2.29±3.17 in positive subject compared to 2.14±2.82 in negative individuals. The mean SD of relative lymphocyte count 41.08±19.21 was significantly lower in subject tested positive compared to 43.89±18.40 in negative Malaria Negative Children. Similar result was also seen in monocytes count with mean SD 4.05±4.184 in negative Malaria Negative Children were significant (P=0.05) in positive subjects respectively.

Results presented on table 4, 5, and 6 shows to influence of Plasmodium falciparum parasitaemia on haematological parameters in relation to sex.

During the course of this study mean SD Parked cell volume (PCV) values were found to be higher in males (25.20±1.1) than in females 22.17±0.9, where mean SD platelet counts were higher in male subjects (371.12±2.30) compared to females (307.32±186.86).

Mean SD WBC total count lower in male.  $(11.398\pm5.489)$  than in the females  $(11.557\pm5.256)$ . The relative neutrophil counts shows males  $(54.490\pm19.888)$  was higher than females  $(51.866\pm28.261)$ . Means relative eosinophil count was higher in males  $(2.710\pm3.608)$  than females  $(1.469\pm2.944)$ . The means relative lymphocyte and monocytes counts were found to be lower in males  $(39.840\pm39.840)$ ,  $(4.024\pm4.562)$  than their female's counterparts  $(43.782\pm19.862)$ ,  $(4.421\pm3.945)$  respectively.

Table 1: Characteristics Baseline of Enrolment of the participant in Bulunkutu 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	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 2: Mean Effect of Plasmodium falicparum Parasitemia on Hematological Parameters on Positive and Negative Children 6-59 Months at Bulunkutu, Health Centre Maiduguri

H. parameter	Participant group	Mean + S.D	St. mean	Df	p-value
Malaria parasitamia	Positive	8.38±2.46	0.263	87	**
(µl)					
	Negative	0.00		121	
PCV (%)	Positive	27.31±1.2	10.61	87	*
	Negative	33.52±0.9	01.54	121	

Platelet (x10 <sup>9</sup> /µl)	Positive	342.5±204.6	21.93	87	**
	Negative	382.5±184.9	16.74	121	
White blood cell	Positive	11.61±5.36	0.574	87	**
$(x10^{9}/\mu l)$					
	Negative	$10.25\pm8.94$	0.809	121	
Neutrophil (%)	Positive	52.53±19.07	2.045	87	*
	Negative	48.77±19.92	1.713	121	
Eosinophil (%)	Positive	2.28±3.17	0.339	87	*
	Negative	$2.14\pm2.82$	0.254	121	
Lymphocytes (%)	Positive	43.89±18.40	2.059	87	*
	Negative	41.08±19.21	1.665	121	
Monocytes (%)	Positive	4.057±4.18	0.448	87	*
	Negative	$4.12\pm4.042$	0.366	121	
-					<del></del>

**Key;** \*\* highly significant \* Significant

Table 3: Mean Effect of Plasmodium falicparum Parasitemia on Hematological Parameters on Positive Children 6-59 Months at Bulunkutu, Health Centre Maiduguri

Parameters	Mean + SD	T. value	P. value
PVC (%)	27.31±1.2	8.25	*
Platelet (x10 <sup>9</sup> /µl)	$342.5 \pm 203$	15.6	**
White blood cell	$11.61 \pm 5.39$	20.2	**
$(x10^{9}/\mu l)$			
Neutrophil (%)	$52.53 \pm 18.97$	25.6	*
Eosinophil (%)	$2.278 \pm 3.16$	6.71	*
Lymphocyte (%)	43.89±18.40	19.9	*
Monocytes (%)	$4.057 \pm 4.16$	9.04	*

Table 4: Mean Effect of Plasmodium falciparum Parasitema on Hematological Parameters on Male and Female Subjects

Parameters	Participant	Mean + SD	T-value	P. value
Malaria	Male	8.420±2.643	22.39	
parasitamia (µl)				
	Female	$8.474\pm2.458$	21.25	**
PVC (%)	Male	26.85±7.616	4.728	*
	Female	27.91±6.035	4.828	
Platelet	Male	371.12±230.260	11.39	*
$(x10^9/\mu l)$				
	Female	11.398±5.256	21.25	**
	Female	11.557±5.256	13.55	*
Neutrophil (%)	Male	$54.490 \pm 19.888$	19.35	
	Female	51.866±28.261	11.31	*
Eosinophil (%)	Male	$2.710\pm3.608$	5.213	
	Female	1.469±2.944	3.075	*
Lymphocytes	Male	39.840±39.840	14.10	
(%)				
	Female	$43.782 \pm 19.862$	13.58	*
Monocytes (%)	Male	$4.024\pm4.562$	6.160	NS
	Female	4.421±3.945	6.908	

**Key;** \*\* highly significant \* Significant "NS" non-significant

Table 5: Mean Effect of Plasmodium falciparum Parasitema on Hematological Parameters on Male Subjects

Parameters	Mean + SD	T. value	P. value	
Malaria parasitamia	8.420±2.643	22.39	**	
(µl)				

PCV (%)	26.85±7.616	4.728	*
Platelet (x10 <sup>9</sup> /µl)	371.12±230.260	11.39	*
White blood cell	11.40±5.487	14.62	**
$(x10^{9}/\mu l)$			
Neutrophil (%)	54.490±19.888	19.35	*
Eosinophil (%)	2.710±3.608	5.213	*
Lymphocyte (%)	39.840±39.840	14.10	*
Monocytes (%)	4.024±4.562	6.160	NS
<del></del>			

Key; \*\* highly significant \* Significant "NS" non-significant

Table 6: Mean Effect of Plasmodium Falciparum Parasitema on Hematological Parameters on Female Subjects

Parameters	Mean + SD	T. value	P. value	
Malaria parasitamia	8.474±2.458	21.25	**	
(µl)				
PCV (%)	27.91±6.035	4.828	*	
Platelet (x10 <sup>9</sup> /µl)	307.315±186.86	21.25	**	
White blood cell	11.557±5.256	13.55	**	
$(x10^{9}/\mu l)$				
Neutrophil (%)	51.866±28.261	11.31	*	
Eosinophil (%)	1.469±19.944	3.075	**	
Lymphocyte (%)	43.782±19.862	13.58	*	
Monocytes (%)	4.421±3.945	6.908	*	

**Key;** \*\* highly significant \* Significant

Table 7: The result that Packed Cell Volume of malarial infected subject was observed with the following values: PCV% M; 6-10 months =  $28.05 \pm 0.70$ , PCV% M; 11-20 months =  $25.42 \pm 0.76$ ; PCV% M; 21-30 months =  $25.43 \pm 0.76$ ; PCV% M; 31-40 months =  $32.08 \pm 0.69$ ; PCV% M; 41-50 months =  $31.24 \pm 0.72$ ; PCV% M; 51-60 months =  $27.23 \pm 0.$ ; P>0.05, with values below the reference interval : PCV% = 33%.

Furthermore, the platelets counts of malarial infected subject with age brackets 6-10, 11-20, 21-30, 41-50, and 51-60 months were within the reference interval; PLT x  $10^{-9}/\mu$ /; 6-10 months =  $384.30\pm198.23$ ; PLT x  $10^{-9}/\mu$ /; 11-20 months =  $381.43\pm103.43$ ; PLT x  $10^{-9}/\mu$ /; 21-30 months =  $276.05\pm177.44$ ; PLT x  $10^{-9}/\mu$ /; 41-50 months =  $329.95\pm268.92$ ; PLT x  $10^{-9}/\mu$ /; 51-.60 months =  $350.99\pm219.04$ ; P>0.05, were found to be within the reference interval: platelets x  $10^{-9}/\mu$ / = 150,000-400,000 x  $10^{-9}/\mu$ /. While the platelets counts of malarial infected subjects between the ages bracket 31-40 were above the reference interval PLT x  $10^{-9}/\mu$ /; 31-40 months =  $441.75\pm168.34$ .

WBC counts of malarial infected subjects within the age group 6-10, 11-20, 21-30, the reference interval (WBC x  $10^{-9}/\mu$ / = 4.5 - 13 x  $10^{-9}/\mu$ /. Similarly, the relative neutrophil counts of malarial infected subjects between the following age brackets; 6-10, 11-20, 21-30, 31-40, 41-50, 51-60 months were found to be within the normal reference interval (neutrophil% = 30-65%).

The result also revealed that relative eosinophil counts of malarial infected subjects within the age groups 6-10, 11-20, 21-30, 31-40 months were within reference interval of (eosinophil 1-40%) counts of malarial infected subject within the age group 21-30, 41-50 and 51-60 months were below the reference interval of (1-4%.) Similarly the relative lymphocyte counts of malarial infected subjects between the age group 6-10, 11-20, 21-30, were found to be within the normal reference interval. (Lymphocytes = 30-60%) while the relative lymphocytes counts of malarial infected subject within the age group 31-40, 41-50 and 51-60 months were below the reference interval, (lymphocyte = 30-60%).

Monocytes of malarial infected subjects within the age group 6-10, 11-20, 21-30, 31-40, 41-50, 51-60 months were within the reference range (monocytes = 1-9%).

Table 7: Age Distribution on the Influence of Parasitaemia on some Hematological Parameters in children (6-39 months)

Age	Mean	Mean	Mean	Mean	Mean	Mean	Mean	Mean
Group	PDC	PVC/HCT	PLT	WBC	Neu	EOS	Lym.	MON
6-10	32.00 <u>+</u> 94.28	28.05 <u>+</u> 6.2	384.30 <u>+</u> 198.23	12.59 <u>+</u> 3.81	41.90 <u>+</u> 11.14	1.40 <u>+</u> 2.59	2.00 <u>+</u> 1.89	55.90 <u>+</u> 15.32
11-20	313.17 <u>+</u> 113.64	25.42 <u>+</u> 2.56	381.43 <u>+</u> 103.43	11.44 <u>+</u> 4.90	35.98 <u>+</u> 14.47	3.85 <u>+</u> 2.91	3.63 <u>+</u> 3.05	55.80 <u>+</u> 24.23
21-30	325.33 <u>+</u> 97.59	25.43 <u>+</u> 2.66	276.05 <u>+</u> 177.44	11.45 <u>+</u> 5.76	40.50 <u>+</u> 22.59	4.13 <u>+</u> 3.54	5.39 <u>+</u> 3.02	48.33 <u>+</u> 21.84
31-40	260.00 <u>+</u> 69.28	32.08 <u>+</u> 3.44	441.75 <u>+</u> 168.34	9.43 <u>+</u> 2.81	44.95 <u>+</u> 20.97	2.07 <u>+</u> 2.06	3.56 <u>+</u> 3.56	13.96 <u>+</u> 13.96
41-50	340.00 <u>+</u> 105.41	31.24 <u>+</u> 3.89	329.95 <u>+</u> 268.92	10.75 <u>+</u> 3.39	51.13 <u>+</u> 17.95	4.80 <u>+</u> 4.68	4.67 <u>+</u> 4.69	18.66 <u>+</u> 18.67
51-60	347.11 <u>+</u> 102.13	27.23 <u>+</u> 8.2	350.99 <u>+</u> 219.04	11.45 <u>+</u> 6.18	57.50 <u>+</u> 16.73	22.78 <u>+</u> 22.78	4.39 <u>+</u> 4.39	16.35 <u>+</u> 16.35

Normal values: PCV  $\rightarrow$  (33%), Platelet  $\rightarrow$  (150,000-400,000 x 10<sup>-9</sup>), WBC  $\rightarrow$  (4.5 – 13x-9), Neutrophil  $\rightarrow$  (30-65%), Eosinophil  $\rightarrow$  (1-4%), Lymphocyte  $\rightarrow$  (30-60%), Monocyte  $\rightarrow$  (1-9%), N = Number group. (WHO, 1996).

Packed Cell Volume was found to be negatively correlated with parasite densities among malaria infected subject age 6-59 months ( $r^2 = 0.508$ , P = 0.005) and as well as among males infected subjects ( $r^2 = 0.680$ , P = 0.005) and females infected subjects ( $r^2 = 0.537$ , P = 0.005) as shown in figure 1, 8 and 15 respectively. Platelate was also negatively correlated with parasite densities among malaria infected subjects age between 5-59 months, as well among males and females subjects with ( $r^2 = 0.760$ , P = 0.001) ( $r^2 = 0.680$ , P = 0.005), and ( $r^2 = 0.810$ , P = 0.001) as shown in figures 2, 9 and 15 respectively.

The white blood cells were also found to be positively correlated with mean parasite densities among the malaria infected children 6-59 months ( $r^2$  0.831, P = 0.001) and also among males subjects ( $r^2 = 0.735$ , P = 0.001), and female subjects ( $r^2 = 0.710$ , P = 0.001) as indicated in figure 3,10 and 17 respectively.

This study also indicated that there was a positive association between parasite densities and granulocytes (Neutrophil and Eosinophil) among malarial infected subjects and as well as males and females, infected subjects ( $r^2$  0.635, P = 0.005), ( $r^2$  = 0.510, P = 0.005), ( $r^2$  = 0.602, P = 0.005), ( $r^2$  = 0.504, P = 0.005 and ( $r^2$  = 0.890, P = 0.001), ( $r^2$  0.623, P = 0.005) as indicated in figure 4,5,11,12 and 18,19 respectively. Hence, the agranulocyte (Lymphocytes, Monocytes) were also found to be positively correlated with mean parasite densities among the malaria positive children 6-59 months ( $r^2$  = 0521, P = 0.005), ( $r^2$  = 0.520, P = 0.005) and as well as among lymphocyte of male positive children ( $r^2$  = 0.6231 P = 0.005) and females infected subjects ( $r^2$  = 0539, P = 0.005), ( $r^2$  = 0.607, P = 0.005) as indicated in figure 6, 7, 13, and 20, 21 respectively. However, a non-significant and negative correlation was observed between parasite density and monocyte of male positive children ( $r^2$  = 0.410) as shown in figure 14.

## **Discussion**

In this present study influence of plasmodium falciparum parasitaemia on some haematological parameters in children (6-59 months). During the study it was observed that 210 (41.90%) children aged between 6-59months visited the pediatric out-patient department were positive for plasmodium falciparum malaria. This finding is consistent with finding from previous reports from various parts of Nigeria by (FMH, 2005a) who obtained 40% annual prevalence rate found in Nigeria. The finding is also consistent with previous reports by (Ojukwu, 2002) who obtained malaria prevalence of 50% in North East, North Central, North West and South South regions of Nigeria respectively. This study contradicted other findings by (Ojukwu, 2002) who in a similar research, in South Eastern part of Nigeria reported 17% prevalence rate.

The males had a relatively higher prevalence rate of 52 (59.09%) compared with their female counter parts that had prevalence rate of 36 (40.91%) that was statistically significant (p <0.05%). Similar reports had indicated higher prevalence in males than females (WHO, 2005b; WHO, 2006) but there is no scientific evidence to higher prevalence being related to gender as susceptibility to malaria infection is not influence by gender. (Giles and Warell, 1993). The higher prevalence rate among male could just be by chance.

The high prevalence 41.90% plasmodium falciparum infection obtained in this study may be due to the facts that there are ecological alterations favouring the breeding of the mosquito vector which facilitate the spread of the malaria infection. Other incriminating factors include rapid rate of urbanization of Maiduguri, and its attendant sanitation and public health problems. These problems have arisen as a result of inadequate waste disposal facilities, poor drainage system and poor water supply among others.

This study confirms that haematological abnormalities considered hallmark of malaria infection are common and more pronounced in plasmodium falciparum malaria infection, probably due to higher levels of parasitaemia found in these children. The abnormalities previously reported include changes in parked cell volume (anaemia), platelets, leucocytes, differential leucocytes counts and disseminated intravascular coagulation (DIC) (Reyburnet. al.,(2007), (Wickramasingheet. al.,) (2000), (Richards et. al., (1998).

The mechanism of anaemia in plasmodialparasitized patients is either due to haemolysis of parasitized red cells, exacerbated removal of parasitized red blood cells, bone marrow suppression, and decreased erythropoietin level or due to ineffective erythropoiesis (Pavithran, 2007). Plasmodium falciparum malaria is one of the commonest cause of anaemia and correlates with the severity of the infection (Erhaboret. al., 2006).Plasmodium falciparum was found to be the cause of malaria among parasitized subjects 88 (49.10 %).

The result obtained from Parked Cell Volume (PCV) shows that subject tested positive had lower PCV with a mean SD 26.72±29.41 compared to negative subject with means SD 34.83±32.91, this was consistent with report from (Kwadwoet. al., 2000). The mechanism of thrombocytopenia in malaria is due to decreased thromobopoiesis despite normal or increased megakaryocytes in bone marrow (Abroet. al., 2008). This study revealed that the mean standard deviation of absolute platelet count 382.5±184.9 in negative subject was significantly (P = 0.05%) higher compared to 342.5±204.6 in positive subject, which shows that malaria parasite exerted a significant reduction in platelet count in parasitized subject. This is concordant with an earlier report by (Pavithran, 2007). The above finding is consistent with previous report by (FMH and NSP for RBC in Nigeria, (2001).

The result of this study further shows that parasite density significantly influence, white blood cell as means SD  $11.61\pm5.36$  recorded in positive subject was significantly (p = 0.05%) higher than mean SD  $10.25\pm5.22$  obtained in negative Malaria Negative Children . The result obtained in this study is consistent with a report from (Abro et. al., 2008) The means SD neutrophil count was normal for both parasitemic subject  $52.53\pm19.07$  and non parasitemic Malaria Negative Children  $48.77\pm18.92$  in this study. These findings are similar to those from two studies. One involving 400 cases in a malaria endemic region of India, in which about 85% of the patients and had normal neutrophil counts (Akhtar,et. al., 2012). In contrast though, some earlier studies had reported neutropenia (Dole and Wolff, 1973) or neutrophilia (Abdalla, 1988) among malaria cases, especially in the pediatric patient (Maina,et. al., 2010). The result of this study further revealed that plasmodium falciparum parasitaemi a significantly influences eosinophil as mean SD  $2.278\pm3.166$  recorded in positive subjects was significantly (p = 0.050%) higher than the mean SD  $2.139\pm2.816$  (obtained in negative Malaria Negative Children . The result obtained in this study is consistent with a report from (Walters, 1987) who stated that eosinophil might be stimulate either directly by the parasites or other mediators produced during the malaria attack.

The results from lymphocytes shows assessment positive test had lower lymphocytes with a mean SD 41.08±19.21 compared to negative Malaria Negative Children with mean SD 43.89±18.40. This was consistent with a report from (Allen, et al, (1997), (Hill et al, (1991) and (Chotivanichet al, (2000) respectively who stated that there is an sample evidence showing the potential of malaria infection to affect to counts of lymphocyte sub population in the peripheral blood. This is because the pathogenesis as well as the disease outcome of malaria is highly dependent on host genetics. The study further revealed that the means standard deviation of relative monocyte count 4.121±4.042 in positive subject was higher compared to 4.057±4.184 in negative Malaria Negative Children. This was concordant with a report from (Murthy et al, (2000) and Pavithran, 2007) who in a similar study reported that the monocyte procogprocagulant activity was also found to be high in plasmodium falciparum infection.

## References

- Abdalla S.H (1988) Peripheral blood and bone marrow lencocytes in Gambia children with malaria: numerical changes and evaluation of phagocytosis. Ann trop predator.1988; 8: 250- 258.
- Abro, A.H., Ustadi, A.M., Younis, N.J., Abdou, A.S., Hamed, D.A., &Saleh, A.A., (2008). Malaria and haematological changes Pakistani Journal of Medical Sciences 24 (2): 27-287-291.
- Akhtar S, Gumashta R, Mahore S, and maimoon S. (2012) "Haematological change in malaria a comparative study change of pharmacy and biological sciences, Vol.2, no.4, pp. 15-19, 2012.
- Allen, S. J., A. O'Donnell, N. D., Alexander, M. P., Alpers, T. E., Peto, J. B., Clegg, & Weatherall D. J., (1997). a<sup>+</sup>-Thalassemia protects children against disease caused by other infections as well as malaria. Proc Natl. Acad. Sci USA 94:14736-14741.
- Centre for Disease Control and Prevention (2004). Biology of Malaria, accessed online at www. cdc. gov/malaria! Biology / index.htm.
- Cheesbrough, M, (1999). District Laboratory practice in tropical countries. Cambridge University Press. Volume 1:244-251.
- Chotivanich, K., Udomsangpetch, R. Simpson, J. A., Newton, P. Pukrittayakamee, S. Looareesuwan, S.& White N. J., (2000) Parasite multiplication potential and Forney J.P., Wongsirchanalai C, Magill A.J., Craig L.G., Sirichalaisinthop J, Bautista C.T., Miller R.S., Ockenhouse C.F., Kester K.E., Aronson N.E., Anderson E.M., CluinoAscura H.A., Vidal C, Moran K.A., Murray C.A., De Witt C.C., Heppner D.G., Kain K.C., Ballou W.R., and Jr Gasser R.A (2003). "Devices for rapid diagnosis of malaria: evaluation of prototype assays that detect Plasmodiumfalciparumhistidinrich protein 2 and Plasmodiumvivax specific antigen". Journal of clinical microbiology. 41:2358-2366, 2003.
- Ekanam, G.N., (1990). Pathology of malaria in West Africa. BlitMedical Journal, 1, 7 15-718.
- Erhabour O., Babatude S. and Uko K.E (2006) some hematological parameters in plasmodia parasitized individual in Nigeria. Nigerian journal of medicine bold 52-55.
- FMH (2005a) national treatment guideline federal ministry of health publication of the FMH Nigeria P. 44.
- Fortin, A, Stevenson, M.M., Gros, p. (2002) Susceptibility to malaria as a complex trait: big pressure from a tinycreature. Human Molecular Genetics, 11(20). 2469-2478.
- Gilles H.M, warrell D.A (1993). In Bruce- chwatt's essential malariology, 3<sup>rd</sup> Ed. Edward Arnold pp. 19-24.
- Gilles H.M, warrell D.A (1993). In Bruce- chwatt's essential malariology, 3<sup>rd</sup> Ed. Edward Arnold pp. 19-24.
- Kumar A.S (2006), thrombocytopenia an indicial for of acute malaria. Indian pattol microbial 2006; 49(4): 505-508
- Kwadwo A, Koramt Seth Owusn Agyci, DavidJ, fryauf T, Abraham Hogson and Francis K, Nkrumah (2003). Seasonal profile of malaria infection anaemia and
- Maina, R.N., Walsh, D., Gaddy, C., Hongo, G., Waitumbi, J., Otieno, L., Jones, D. &Ogutu, B.R., (2010).Impact of Plasmodium falciparum infection on haematological parameters in children living in Western Kenya. M. J. 9, S4,

- Medical laboratory science, theory and practice by Dr. John O. Ochei, Ph.D, FMLS &DrArundhati A. Kolhatakar, Ph.D, AIMLS, D, HA. Sixth reprint 2007 RXCARRYDCRLY.
- Murthy, G.L., Sahey, R.K., &Srinivasan, V.R., (2000) clinical profile of falciparum malaria in a tertiary care hospital. Journal of Indian Medical Association 98: 160-169.
- Ojukwu, J.U., (2002). Patter and outcome of Paediatric malaria admissions in Abakaliki, Nigeria EbonyiMedical Journal j (1), 1720.
- Okiro E.A, Al-Tiar A, reyburn H, Idro R, Berkley J.A, Snow R.W (2009) Age patterns of severe pediatric malaria and their relationship to plasmodium falciparum transmission intensify. Malar J. 2009; 8:4.
- Oluwafemi, 0, Oguntibeju (2003). Parasitic Infestation and Anaemia: The Prevalence in a Rural Hospital Setting Journal, Indian Academy of Clinical Medicine, 4(3).
- Pavithran, K, (2007). Haematological changes in Malaria. Clinical pharmacology 1:1-3.
- Reyburn H, Mbakilwa H, Mwangi R, Mwerinde O, Olomi R, Drakeley C, Whitty C.J (2007). Rapid diagnostic tests compared with malaria microslopy for guiding outpatient treatment of febrile illness in Tanzania randomized trial BMJ. 2007; 334: 403.Doi 10.1136/bmj. 39073. 496829. AE.
- Richard M.W, Behrens R.H, Doherty J.F (1998) Hematological change in acute, imported plasmodium falciparum malaria.AM.J. Trop med Hyg.1998; 59:859.
- Ukaga, C.N., Nwoke, B.E.B., Onyeka P.L.K., (2003). Integrating Woman in disease Management: Case of Malarial. The Nigeria journal of parasitology, 24, 53-58.
- Water N.C., Edstein M.D., (2012). "8 aminquinolines: primaquine and tafenoquine" (<a href="https://books.google.com/">https://books.google.com/</a>Books? id=cNuY6tryyrU C&pg=PA69). In staines HM, Krishna S. Chemistry, action and use, Springer, pp. 69-93 ISBN 978-303446-0447-6
- WHO (2005a). Making every matter and child count world health organization, Geneva. The world health report.
- WHO (2006) World health statistics, NHS, Nigeria fact sheet, No .3, p.7.
- WHO, 2005. Susceptibility of plasmodium falciparum to antimalarial drugs: report on global monitoring, 1996-2004 WHO/HTM/MAL/2005.1103.
- Wickramasinghe SN, Abdalla SH. Bailliere's ClinHematol. Vol. 13. Harcourt Pub Ltd; 2000. Blood and bone marrow changes in malaria; pp. 277–299. [PubMed]
- World Health organization (2005). World malaria report 2005. A-5 minute briefing 1:1-5.