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CONCURRENT EXISTENCE OF MALARIA AND IRON DEFICIENCY AMONG UNDER-6 CHILDREN IN ABEOKUTA, NIGERIA

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Abstract: **Background: Iron deficiency and malaria are common scourges of children around the world and both conditions may coexist with the other in some individuals, especially in the tropics where malaria is endemic. This study set out to determine the concurrent existence of iron deficiency and malaria among children aged 0 - 5years in Abeokuta, Nigeria.**

Method: One hundred and eighteen children clinically assessed and queried for malaria were purposively recruited as subjects. Forty apparently healthy age-matched children were enrolled as the control group. The parameters assessed were packed cell volume (PCV), examination of red blood cell morphology by the thin blood film, malaria parasite by the Giemsa thick blood film and serum ferritin by enzymometric assay. Data were analysed using the chi-squared test to determine the effect of one variable on the other while comparison between two groups was carried out by the Student's T-test.

Result: The mean PCV of malaria parasite positive subjects (23.90 ± 0.91) was significantly lower than the mean PCV of malaria parasite negative subjects (33.32 ± 1.01) (t =5.72, *P*<0.05 (0.001)). Both low serum iron level and malaria positivity had significant relationship with the hematocrit of the subjects. Serum ferritin of the patients also showed a significant association with PCV (*P*<0.05 (0.001)). Children in the age range 1 to 3 manifested the greatest frequency for both low iron levels and malaria positivity.

Conclusion: The co-existence of iron deficiency and malaria among children in the study population is pronounced. Children between age 1 and 3 are at greatest risk of severe iron deficiency and malaria infection in malaria-endemic areas, unless preventive measures are taken. Low serum ferritin levels among children predisposes to malaria infection such that the lower the serum ferritin level is, the more the susceptibility to malaria.

Keywords: **anemia, children, iron deficiency, malaria.**

1. INTRODUCTION

Iron-deficiency anemia is a common anemia caused by inadequate dietary intake or poor absorption of, or even iron loss due possibly to bleeding, or a combination of these factors. Iron deficiency is the most common cause of the nutritional deficiency anemias, being found in every country of the world **1, 2**. Untreated iron deficiency may lead to iron deficiency anemia.

Among several vital functions of iron, the following are prominent: as a carrier of oxygen to the tissue from the lungs in the form of hemoglobin; as a transport medium for electrons within the cells in the form of cytochromes, and as an integral part of enzyme reaction in various tissues. Because of its high utility in the body, long-term imbalances in iron absorption against that which is utilized for body metabolism and the amount lost; could result in iron deficiency. The amount of iron required each day to compensate for loss from the body and for growth varies with age and sex; although normally, a positive iron balance exists in healthy individuals. Iron demand is highest in pregnancy, among children in general and toddlers in particular, as well as in adolescent and menstruating females **3**. Expectedly, persons belonging to any of these categories are particularly likely to develop iron deficiency if there is additional iron loss or a prolonged reduced intake **4**

It is estimated that one-quarter of the world's children suffer from iron deficiency anemia since iron deficiency is found in both developing and developed countries, although it is more prevalent in the former **2**. In developing countries, iron deficiency anemia may range between 30% and 80% in pre-school age children **3**. Worldwide, the prevalence of anemia among this group was found to be 47.4% **5**. Iron-deficient children are prone to developmental defects, affecting not only neural development but with effects that persist into middle childhood and adolescence. In the absence of sufficient iron for the infant child, iron utilization for hematopoiesis in the bone marrow takes precedence over that of other tissues. The brain may thus develop iron deficiency that can lead to permanent brain damage **6**. What makes iron deficiency rampant in many countries is the inability to meet the dietary iron needs of rapidly growing young children, consequent on the type of poorly-nourishing diets that are commonly fed to children in many parts of the world **7, 8.**

Although the iron requirement of babies is usually adequate from breast-milk in infancy, since the iron stores at birth is high; the demand after the first six months requires supplementary dietary intake in order to prevent iron-deficiency **9**. Studies suggest that although hepcidin, the regulatory protein for iron transport across tissues is available at this stage, its co-regulators in the intestine, the divalent metal-ion transporter 1 (DMT1) and ferroportin are insensitive to iron status in infancy and thus may not play any significant role in iron homeostasis until the baby has been weaned **10**. Consequently, low-dose oral iron supplementation has been advocated for normal infants and young children beyond the age of 6 months; and for low-birth weight infants beginning at 2 months of age **11**. This is in spite of the finding that iron supplementation has the potential of increasing the risk of severe malaria morbidity in children, since the malaria parasite thrives better when there are more reticulocytes in circulation **12**.

Iron deficiency is the most important cause of microcytic hypochromic anemia with blood films showing microcytic and hypochromic red cells and in which two other parameters of the red cell indices - mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), become reduced **13**. The abnormal blood morphological appearance seen in this condition is caused by a defect in hemoglobin synthesis as a result of inadequate iron supply. When inadequate iron supply is present, microcytosis occurs, since erythropoietic activity is enhanced in a bid to compensate for the anaemia; and the proportion of protoporphrin moieties in which zinc replaces iron is increased **4**. Serum ferritin measurement may be carried out to distinguish iron deficiency anemia from other conditions with similar RBC morphology. Since ferritin levels have direct correlation to the amount of iron stored in the body, its measurement is often undertaken in the laboratory in order to determine the iron status among individuals suspected of iron deficiency. In iron deficiency anemia, serum ferritin levels are found to be low relative to these other conditions. There are situations however, in which iron deficiency is present but has not manifested as iron deficiency anemia. In such non-severe types, the tissues are already functionally iron-deficient, but anemia is not yet reflected in the peripheral blood. When iron-deficiency becomes marked as to significantly affect erythropoiesis, anemia may then result **14**.

Iron deficiency caused by poor nutrition among children may be compounded by the presence of parasites. Malaria and helminth parasites, particularly hookworm infection are among those parasites capable of accelerating the onset of iron deficiency in children **15, 16**. Malaria is more rampant in many parts of the world than hookworm infections. More importantly, years of regular deworming campaigns in developing countries have nearly eradicated most pathogenic helminths **17, 16**. In Nigeria, regular deworming campaigns are carried out among school children by non-governmental organisations. This has drastically reduced the incidence of hookworm infestation among the populace.

On the contrary, malaria seems to have defied several control efforts to date. The global burden of malaria, instead of abating, seems to be on the rise **18**. WHO claims that children under 5 years are the group more particularly susceptible to the disease **18**. The risk of anemia is increased by malaria infection and if preventive measures are taken against malaria infection, the prevalence of anemia would decrease in many populations. Both clinical and asymptomatic malaria are capable of resulting in iron deficiency anemia. Severe malaria is usually caused by *P.falciparum*, although other species

may also have effects resulting in anemia. Malaria parasites cause the lysing of both infected and uninfected red cell, leading to hemoglobin breakdown, although with minimal iron loss **20** as iron from hemoglobin breakdown is often recycled. A great number of anemia cases in developing countries may be traced to malaria **8** while iron deficiency is the most common cause of anemia globally **11**. Because children are vulnerable to the two diseases, it becomes pertinent to investigate the concurrent existence of both diseases among children in the study population.

2. MATERIALS AND METHODS

Sample collection

Study subjects were 118 pediatric patients presenting at the Oba Ademola State Hospital, Ijaaye, Abeokuta. They consisted of both male and female children clinically assessed and queried for malaria within the age range of 0 to 5years. Subjects were purposively recruited and were unhealthy at the time of specimen collection. Forty age-matched, apparently healthy children were recruited as control subjects. Procedures adopted in this study for sample collection were in conformity with the Helsinki Declaration Ethical Standards for human subjects. The Ethical Review Committee of State Hospital, Ijaaye, Abeokuta gave approval for the commencement of the study. Only children whose mothers gave assent were included in the study. Children without assent from their mothers and those with known complications e.g HIV, sickle cell disease among others, were excluded from the study. Demographic data of mothers as well as diet pattern of subjects were obtained via questioning of the subject's mother or guardian using a semi-structured questionnaire; and the sample collection lasted for 3 months. Four *mls* of blood was collected from each child with the assent of the mother. Two *mls* of the blood sample was dispensed into a plain bottle for ferritin assay. Serum for ferritin assay was obtained after sample had clotted and the supernatant aspirated. Serum was kept at $4^{O}C$ until analyzed. The remaining two *mls* was anticoagulated in an EDTA bottle for the following investigations and the tests were performed within 6 hours of sample collection:

Thin blood film for red blood cell morphology

A thin blood film was made on a clean slide and was allowed to air-dry. It was stained using Leishman staining technique. Staining was carried out using the standard method prescribed by Dacie & Lewis **21**.

Hematocrit (PCV) determination

A micro-capillary tube was filled up to three-quarter level and spun in a micro-hematocrit (Hawksley) centrifuge at 5000rpm for five minutes. The percentage level of sedimented red blood cells was determined using the micro-hematocrit reader **21**.

Blood smear (thick film) examination for malaria parasite

A thick blood film was made on a clean, grease-free slide. It was allowed to air-dry; then stained using the Giemsa staining technique. Briefly, the stock Giemsa stain was appropriately diluted and the film was flooded with stain for 20 minutes. The stain was rinsed off with clean water, the back of the slide blotted dry and the film left to air-dry.

The film was then examined under the microscope using 100X oil immersion objective lens for the presence of malaria parasite **21**.

Results are as presented.

Estimation of serum ferritin by immuno-enzymometric sequential assay

The essential reagents required for an immune-enzymometric assay include high affinity and high specificity antibodies, with different and distinct epitope recognition, in excess; and native antigen. In this procedure, the immobilization takes place during the assay at the surface of a micro-plate well, through the interaction of streptavidin coated on the well and exogenously added biotinylated monoclonal anti-ferritin antibody. Upon mixing monoclonal biotinylated antibody and serum containing the native antigen, a reaction develops resulting in an antigen-antibody complex. Simultaneously, the biotin attached to the antibody binds to the streptavidin coated on the micro-well resulting in immobilization of the complex.

Procedure

The desired number of micro-plate wells was secured in the holder of a Rayto microplate reader RT-2100C (Rayto Company Ltd, Shenzen, China). A 25µl each of standard, control and the specimen was dispensed with new disposable

tips into appropriate wells according to the manufacturer's instruction. 100µl ferritin biotin reagent was dispensed into each well. The plate was thoroughly mixed for 30seconds and covered. The wells were incubated for 30minutes at room temperature.

A 100µl of ferritin enzyme conjugate was added to each well; not shaking the plate after enzyme addition). It was incubated for 30minutes at room temperature. The content of the well was discarded by decantation. The wells were again washed three times with wash buffer (300µl per well).

A 100µl of working substrate solution was added to all wells (no shaking after substrate addition), and incubated for 15minutes at room temperature. The absorbance in each well of micro plate reader was read at 450nm wavelength within 30minutes of adding the stop solution.

Manufacturer recommended reference ranges:

New born: 22-220ug/L

Between 1 and 2months: 190-610 ug/L

2-5months: 50-220 ug/L

6months -16years: 10-160 ug/L

3. RESULT

One hundred and eighteen samples were analyzed for hematocrit, serum ferritin, malaria parasite and red blood cell morphology. Results obtained were further analysed with the statistical package for social science (SPSS 20) software. Forty (40) apparently healthy children that were age-matched with the test subjects served as the control. A summary of the results has been presented here in tabular form. Reference values recommended by the manufacturer were applied to each age group. A *P-value* of less than 0.05 is considered statistically significant.

Table 1 shows the summary of the demographic data of the subjects' mothers and the children's feeding pattern as obtained by semi-structured questionnaire.

In table 2, the relationship between malaria parasite positivity and the age of pediatric patients attending State Hospital, Ijaaye, Abeokuta out-patient clinic was assessed using chi-squared. The result shows that there was no association existing between malaria positivity and the age of the subjects (X^2 =1.54, P>0.05 (0.46)). Also, the result shows that no significant association exists between malaria positivity and gender of the subjects [X^2=0.25, P >0.05 (0.62)].

Table 3 shows the relationship between malaria parasite positivity and serum ferritin as investigated using chi-squared. The result displayed a significant association in which the frequency of low ferritin was found to be significantly higher in malaria parasite positive subjects (50.6%) than in malaria parasite negative subjects (19.4%) [X^2 = 9.12, P< 0.05 (0.03)]. In all, 44 of the 118 (37.3%) subjects had concurrent iron deficiency and malaria infection at the same time.

In table 4, the relationship between the serum ferritin level and the age of pediatric patients in the study population as determined by chi-square is displayed. Out of the 118 children examined for serum ferritin 50 (42.4%) had low serum ferritin, 68 (57.6%) were within the normal range. The reference range of serum ferritin for normal persons varies with the age, even among children. Consequently, low and normal serum ferritin values were determined according to the reference range given by the reagent manufacturer. Ten (41.7%) children below age one were with low serum ferritin values while 14 (58.3%) were within the normal range. For children within the age range of 1-3years, 28 (45.2%) were with low ferritin values while 34 (54.8%) were within the normal range. Also within the age range 4-5yrs, 12 (37.5%) subjects had low serum ferritin values and 20 (62.5%) were within the normal range for serum ferritin. Result shows an insignificant association between serum ferritin level and the age of the pediatric subjects (X^2 =0.51, P >0.05 (0.77)).

In table 5 the effect of malaria parasite positivity on the PCV of test subjects was determined. Comparison shows a significant difference in which the mean PCV of malaria parasite negative subjects (33.32 ± 1.01) was significantly higher than that of malaria parasite positive subjects [(23.90 ± 0.91) (t =5.72, P <0.05 (0.001)]. The same table shows the relationship between serum ferritin and PCV of the patients in which a significant association [t = 5.13, P <0.05 (0.001)] was found.

Table 6 shows the data obtained for PCV and red cell morphology of the test subjects. In accordance with World Health Organisation (WHO) guidelines for assessing anemia, a hematocrit value less than 0.3(30%) is considered anemic and is

therefore recorded as low hematocrit in this study. Children with anemia (PCV< 0.3) and who had microcytic cells had a frequency of (92.7%), followed by anemic children with hypochromic cells (80%); the lowest frequency was recorded in subjects who had anemia with both morphological features (41.8%).

4. DISCUSSION

This study was carried out to investigate any association between malaria and iron deficiency among pediatric patients presenting at the Oba Ademola State Hospital, Ijaaye, Abeokuta. The parameters determined were packed cell volume (PCV), examination of red blood cell morphology (thin blood film), malaria parasite (thick blood film) and serum ferritin, while the results were compared to see if there is any effect or correlative relationship between these factors. The subjects with malaria parasites were 87 (73.7%) and those without malaria were 31 (26.3%) showing that most subjects were generally unhealthy at the time of sample collection. This high prevalence shows that malaria is endemic in the study population.

Out of the one hundred and eighteen (118) subjects enrolled in the study, there were 54 males and 64 females. The relationship between gender and malaria infection however had no association. This is in agreement with the findings of Anamudu *et al*., **22** who also found no relationship between gender and malaria infection among children in their study. However, a study in Kenya **23** has shown that women are 50% more likely to have malaria than men. This finding has been attributed to the immune down-regulating effect of both pregnancy and HIV among mostly adult women in the studied population. In our study, only children were involved and physiological differences between the male and female child would not yet have become pronounced in the age-groups studied, nor did the subjects include HIV-infected children; hence no significant difference in malaria and gender association was observed between the two sexes. Moreover, malaria infection among children would more likely be a consequence of exposure to *Plasmodium* parasite-infested mosquito bites than to gender-related physiological gap.

The study population was categorized into three distinct age groups based on physiological iron supply, regulation and metabolism among children. The relationship between the occurrence of malaria and age displayed an insignificant association [(p > 0.05, (0.463)]. It appears that this finding is in disagreement with previous works carried out by **24, 25, 26** probably because in this study, the age segmentation was among only the children-folk unlike in those other studies which investigated the association between age and malaria positivity across board in both the children and adults in the populations studied. Anamudu *et al*., **22**, who investigated iron deficiency in children with malaria, had similar results as ours. It was however observed that in our own study, children within the age group (>1 to 3) years showed a higher frequency of malaria parasite positivity than the other age groups. This should be expected, since immunity to malaria may not have been fully developed among children of this age bracket; whereas the extra care and protection usually given to the younger babies against exposure to malaria is now gradually withdrawn. It is not unusual to see children in this age range exposed to mosquito bites for the first time, since they are considered as coming of age and thus need less protection than the younger infants. It also appears that the severity of malaria infection partly affected the serum iron status of this group of subjects since the highest percentage of children with low serum ferritin level was found within this age bracket. Low iron levels not only lead to anemia but also predisposes to poor cellular immunity. Thus, the concurrent existence of iron deficiency and malaria in individuals is a vicious cycle.

Generally, this study shows that out of the 118 children examined for serum ferritin, (42.4%) had low serum ferritin while (57.6%) were within the normal range. The relationship between the serum ferritin level and age group of these pediatric patients was not significant (p > 0.05(0.77)). Age disparity had already been taken into consideration in the assessment of iron deficiency, based on the recommendation of the reagent manufacturer.

Iron deficiency has been known to be more prevalent among children of age 0 to 5 years because of growth demands for iron. Iron deficiency (ID) resulted in low serum ferritin levels in the children investigated as there was a significant difference between the mean serum ferritin concentration of the anemic and non-anemic subjects (p < 0.05 (0.001)), a finding which is in agreement with the work of Stroltzfus *et al.*, **27**. They suggested that the hemolysis that occurs during malaria attack and the consequent suppression of erythropoiesis results in severe anemia. Lakshmi and Bamji, **28** categorized stages of iron deficiency into three. Stage 1 manifests as loss of bone marrow iron stores although hemoglobin and serum iron levels remain normal. In both stages 1 and 2, latent iron deficiency occurs before the onset of observable anemia in the latter stage 3. Latent iron deficiency is characterized by depletion of iron stores and reduction of effective iron transport in the first two stages. This study had subjects with these different stages of iron deficiency as observed from the different levels of serum iron among the study population, a factor responsible for the findings. The

highest frequency of low serum ferritin was found in the age range >1 - 3 years. This age bracket is that in which growth of children is very rapid and iron demand is at its peak. In addition, most children in the age range >1 to 3 years would have been weaned and the regulatory role of the trio of hepcidin, DMT1 and ferroportin would already be in effect. Even many of those that are not weaned in this age bracket derive low-quality nutrient value from the breastmilk. Consequently, low iron levels should be expected where dietary iron is inadequate among families; and this is reflected in the high prevalence of low ferritin status among children under consideration. Incidentally, a great number of children within this age group are still learning to eat solid food and many of them usually crave for sweet but non-nutritious meals that may lack essential minerals and vitamins, thus paving the way for nutritional deficiencies. In addition, ignorance of nutritious diets for kids on the part of the mothers and the relative poor economic status of some of the parents in the population studied may also have accounted for the data obtained. The under-1 age group had the next high prevalence of low serum ferritin. By 6 months of birth, the baby's iron stores would have been depleted **2**. If such children are fed on cow milk formula or the mother's diet is deficient in iron even when baby is breast-fed, iron-deficiency may still be the outcome in the baby. These two factors are rampant in the study community. Our findings however differ from that of Anamudu *et al*., **22** who did not observe significant differences in the serum ferritin levels in the two groups.

Low hematocrit or packed cell volume (PCV) levels have been used as one of the parameters to indicate the occurrence of iron deficiency anemia. However, this test is low in specificity in consideration of iron deficiency **29** because only the third or the last stage of iron deficiency (IDA) may be reliably assessed by carrying out the hematocrit or hemoglobin estimation **28**. Besides, the cause of anemia could be multifactorial; as is the case in many of the subjects having both malaria infection and iron deficiency. While PCV alone may not be a reliable variable in the determination of iron deficiency status; in this study, there was a significant correlative relationship between PCV and serum ferritin concentration which is an indicator of iron status [$p < 0.05$ (0.001)]. Thus, a very low level of hematocrit in malaria infection of children should raise the suspicion of iron deficiency, especially in those settings where facilities to carry out iron studies are not available.

Additionally, there was a high level of abnormal red blood cell morphology features that are indicators of iron deficiency anemia (hypochromia and microcytosis) among those children with a hematocrit of less than 0.3(30%). Although Agarwal, **30** posited that plasma may be trapped within spaces in the centrifuged tube when the red blood cells have abnormal shapes leading to false hematocrit results, our observation from this study is that the hematocrit still reflects the abnormal red cell morphology seen in iron deficiency anaemia. Consequently, one is tempted to believe that the combination of these two blood parameters can help in determining iron deficiency anemia among children with or without any additional assessment factor. That the morphological features of microcytosis and hypochromia were caused by iron deficiency in this study is evidenced by the low serum ferritin recorded in many of the subjects.

Also, there was a significant relationship between malaria infection and serum ferritin: the serum ferritin levels in children decreased with increasing malaria parasite density, implying that children with heavy malaria burden are most likely to suffer iron deficiency. Moreover, the frequency of low serum ferritin was found to be significantly higher in malaria parasite positive subjects than in malaria parasite negative subjects [$P < 0.05$; (0.03)**]**. The precise mechanism for this result pattern is not known; however, several studies reported an increased serum transferrin receptor concentration and decreased serum ferritin in persons with symptomatic and mildly symptomatic *Plasmodium falciparum* **31, 32**. Our findings here are in disagreement with Gwamaka *et al*., **33** who concluded that iron deficiency is associated with reduced levels of clinical malaria. The reason adduced for reduced malaria in iron deficiency as cited in their study is the increase in the number of reticulocytes. However, the question is whether the level of increase of reticulocytes in ID would be significant in these situations; when iron which is the building block for hemoglobin synthesis is lacking. The evidences from **31** and **32** above also negate this line of thought. Because of its importance in children, the precise mechanism by which malaria affects iron status in children needs to be further investigated.

5. CONCLUSION

This study found a high prevalence of both malaria and low serum ferritin among the subjects studied. Children within the age >1 to 3 years had the highest frequency of the two pathological conditions. The hemoglobin level as evaluated by packed cell volume (PCV) or hematocrit is reflected in the red cell morphology and we suggest that the latter may be used to validate hematocrit results, when examination of blood film is handled by skilled laboratory personnel. PCV alone cannot be used as an indicator for iron deficiency or for iron deficiency anemia. However, in low resource settings, the finding of very low hematocrit in malaria infection should call for the investigation of a concurrent iron deficiency. Going

by the results in this study, it would be safe to conclude that the lower the serum ferritin level, the more the susceptibility to malaria among children and consequently also; that the lower the hematocrit value, the more the susceptibility to malaria infection.

6. RECOMMENDATIONS

The relationship between malaria and iron deficiency can be controlled by preventing the occurrence of malaria using the WHO recommended family protective measures and encouraging iron-rich diets for children. This becomes necessary in order to reduce the risk of iron deficiency, since malaria is not the only cause of iron deficiency in children. Prompt treatment when malaria is detected in children is advocated. Finally, when malaria is found to be positive in children, the PCV and blood morphology should be carried out, as the combination of these two tests can help in assessing the relative degree of iron deficiency where ferritin studies are not readily accessible. Similarly, when the PCV is low in children, the malaria smear and the thin blood film examination for morphology should be part of the work up.

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APPENDICES - A

TABLES AND FIGURES

Table 1: Demographic data of participating children and those of their mothers

Nature of mother's employment	Frequency	Percentage	Aver. H/hold income/month (N)
Teachers	27	22.9%	24,000
Civil Servants	6	5.1%	59,000
Traders	60	52.0%	35,000
Artisans	21	18.8%	17,000
Unemployed (full time housewife)	4	5.0%	-
Feeding Pattern of children			
Age 0 to 1			
Exclusive breast feeding	37	30%	
Infant milk formula	19	15%	
Infant formula + breastfeeding	39	42%	
Cereal formula	23	13%	
Age >1 to 3			
Exclusive breastfeeding	1	0.7%	
Infant milk formula only	4	3.0%	
Infant formula + breastfeeding	2	1.8%	
Cereal formula only	6	4.7%	
Cereal formula + breastfeeding	13	9.3%	
Solid food only	62	56%	
Solid food + fluid pap	21	17%	
Fluid pap alone	9	7.5%	
Age >3 to 5			
Solid food + fluid pap	37	33%	
Solid food alone	49	43.3%	
Fluid pap alone	1	0.9%	
Cereal formula	31	28%	

TABLE 2: Relationship between malaria parasite positivity and the age and gender of pediatric patients attending State hospital, Ijaaye, Abeokuta outpatient clinic.

Status of children	AGE (years)						Total	*P*
	<1		1-3		>3-5			
	N	(%)	N	(%)	N	(%)		
Malaria positive	18	(75.0)	48	(77.4)	21	(65.6)	87	
Malaria negative	6	(25.0)	14	(22.6)	11	(34.4)	31	0.46
Total	24	(100.0)	62	(100.0)	32	(100.0)	118	

Status of children	GENDER				Total	*P*
	Male	(%)	Female	(%)		
Malaria positive	41	(75.9)	46	(71.9)	87	
Malaria negative	13	(24.1)	18	(28.1)	31	0.62
Total	54	(100)	64	(100)	118	

TABLE 3: Relationship between malaria parasite positivity and patients' serum ferritin level

MALARIA STATUS

Ferritin Status	Negative		Positive		Total	*P*
	N	(%)	N	(%)		
Low	6	(19.4)	44	(50.6)	50	
Normal	25	(80.6)	43	(49.4)	68	0.03
Total	31	(100.0)	87	(100.0)	118	

TABLE 4: Relationship between the serum ferritin level and the age of participating subjects.

Serum ferritin	AGE (YEARS)						Total	*P*
	<1		1-3		>3-5			
	N	(%)	N	(%)	N	(%)		
Low	10	(41.7)	28	(45.2)	12	(37.5)	50	
Normal	14	(58.3)	34	(54.8)	20	(62.5)	68	0.77
Total	24	(100.0)	62	(100.0)	32	(100.0)	118	

TABLE 5: Effect of malaria parasite positivity and serum ferritin status on the hematocrit of subjects

Malaria parasite	N	Packed cell volume	*P*
		Mean ± SEM	
Positive	87	23.90 ± 0.91	
Negative	31	33.32 ± 1.01	0.001
Total	118		

Serum ferritin	N	Packed cell volume	p
		Mean ± SEM	
Low	50	21.94 ±1.24	
Normal	68	29.63 ± 0.091	0.001
Total	118		

TABLE 6: Record showing the hematocrit and red cell morphology of pediatric subjects

PCV of subjects	RBC Morphology						Total
	Microcytic cells		Hypochromic cells		Microcyctic +hypochromic		
	N	(%)	N	(%)	N	(%)	
Low/anaemic (PCV< 0.3)	51	(92.7)	8	(80)	22	(41.5)	81
Normal	4	(7.3)	2	(20)	31	(58.5)	37
Total	**55**	**(100.0)**	**10**	**(100.0)**	**53**	**(100.0)**	**118**