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Evaluation of Some Biochemical Parameters of *Plasmodium falciparum* Infected Inhabitants of Ekpoma Metropolis, Nigeria

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Abstract

This study was carried out to determine the impact of malaria parasite infection caused by *Plasmodium falciparu*m on some biochemical parameters. One hundred and five (105) malaria parasite positive male and female subjects and fifty (50) apparently healthy subjects were recruited for this study. Malaria Parasite diagnosis was done by Microscopic Examination of Thick and Thin Stained Blood Films. Age range 15-25 had the highest prevalent rate of 17.14% for males and 14.29% for females while the age range 55-60 had the least prevalent rate of 0.95% for both male and female. when control values were compared with the mean values obtained from the biochemical analysis of *Plasmodium falciparum* parasitaemia subject samples, it was significantly lower for glucose (p=0.04), ALT (p=0.00), AST (p=0.00), ALP (p=0.00), total protein (p=0.01), Globulin (p=0.02) and Creatinine (p=0.04). Total cholesterol (p=0.12), Triglyceride (p=0.06), Albumin (p=0.49) and Urea (p=0.15) were statistically not significant for malaria parasitaemia subject samples. Glucose, Total cholesterol, Liver enzymes, creatinine changes were seen involved in malaria infection caused by *Plasmodium falciparum.* The prevention of malarial infection is still the most valid method of preventing these conditions and early diagnosis and treatment are the measures likely to decrease malarial complications commonly determined by changes in these biochemical parameters.

Keywords

Malaria, Parasitaemia, Biochemical Profile, Infected, *P. falciparum*

1. Introduction

Malaria parasites are members of the genus plasmodium (Phylum Apicomplex). In humans malaria is caused by *P. falciparum, P. malarie, P. ovale, P. vivax and P. knowlesi* [1-2]. Malaria is a mosquito-borne infectious disease of human caused by eukaryotic prostis of the genus plasmodium. It is widespread in tropical and subtropical regions, including much of sub-saharan Africa, Asia and the America. Malaria is prevalent in these regions because of the significant amounts of rainfall and consistent high humidity along with stagnant water in which their larvae mature and provide mosquitoes in the environment [3]. Malaria was once common in most of Europe and North America [4] where it is no longer endemic [5] though imported cases do occur. A total number of 216 million estimated malaria cases occurred in 2008 as reported by World Health Organization. A total of 106 countries of the world were highly affected of which Nigeria was included [6] 81% of malaria cases occurred in the Africa region and 43% of the reported cases occurred in Nigeria out of which 655,000 deaths was reported worldwide. Malaria develops via two phases: an exoerythrocytic and an exoerythrocytic phase. The exoeryrthrocytic phase involves infection of the hepatic system or liver, whereas the erythrocyte or red blood cell when an infected female anopheles mosquito pierces a person's skin to take a blood meal, sporozoites in the mosquito saliva enters the blood stream and migrate to the liver. Within minutes of being introduced into the human host, the sporozoites infect hepatocytes, multiplying

asexually and asymptomatically for a period of 8-30 days [7]. Once in the liver, these organisms differentiate to yield thousands of merozoites, which following rupture of their host cells, escape into the blood and infect red blood cells, thus beginning the erythrocytic stage of the life cycle [7]. The parasite escapes from the liver undetected by wrapping itself in the cell membrane of the infected host liver cell [8] Within the red blood cells, the parasite multiply further, again asexually, periodically breaking out of their hosts to invade fresh blood cells. Several such amplification cycles occur [8]. Thus classical description of waves of fever arises from simultaneous waves of merozoites escaping and infecting red blood cells. Some P. vivax and P. ovalesporozoites do not immediately develop into exoerythrocytic phase merozoites but instead produce hypozoites that remain dormant for period ranging from several months (6-12 months is typical) to as long as three years. After a period of dormancy, they reactivate and produce merozoites. Hypozoites are responsible for long incubation and late relapses in these two species of malaria [9]. It was hypothesized that the malaria parasite uses cholesterol and phospholipids from its host, resulting in a decrease of serum HDL. Prior to this report, Angus *et al.* [10]. utilized lipoprotein electrophoresis in rhesus monkeys infected with *Plasmodium knowlesi* to study serum lipids in malaria. Their results were not conclusive because lipoprotein bands could barely be detected in the serum of controls. Subsequently, several clinical studies showed lipid profile changes in the setting of both uncomplicated and complicated malaria [11-12].

2. Materials and Methods

2.1. Research Design

A total of one hundred and five (105) malaria parasite positive male and female subjects between the ages of 15-60 years visiting the various health centres and general hospital laboratories in Ekpoma town and adjourning rural settlements were recruited for this study. A total of fifty (50) (25 males and 25 females) apparently healthy male and female subjects between the ages of 15-60 years who tested negative for malaria parasite were recruited as control subjects for this study. Ethical approval was obtained and the study was conducted with informed consent of the patients.

2.2. Geographical Description of the Study Area

This study was carried out in Ekpoma a town in Edo state, Nigeria. It is the headquarters of the Esan West Local Government Area. It is a semi urban area with an estimated population of 125,842 people at the 2006 census [13]. The town is home to the Ambrose Alli University. Ekpoma has the following coordinates; 6°45'N 6°08'E [14].

2.3. Sample Collection

Ten mililitre (10ml) of venous blood samples were collected by vene-puncture from the subjects into accurately labeled plain containers for both subjects and control. For this study, the test subjects were those that presented at the various health centres and General hospitals with symptoms associated with malaria infection. The signs and symptoms of malaria infection in humans are caused by the asexual blood stage of the parasite which includes: headache, joint pains, nausea, vomiting, abdominal upset, fever, and digestive disorders [15]. Thick and thin stained blood films were made, and the remaining blood samples were centrifuged at 3500 rpm for ten minutes at room temperature within one hour of sample collection and the serum separated into clean plain containers which were kept frozen until required for analysis.

2.4. Malaria Parasite Diagnosis by Microscopic Examination of Thick and Thin Stained Blood Films

The Giemsa's staining technique was used for the staining of the thick blood films for malaria parasite detection and malaria parasite count, while the thin blood films were stained with Leishman staining technique for plasmodium species identification as described by Monica Cheesbrough [16]. The changes in parasitized red cells helped to identity plasmodium species and to detect mixed infection of malaria parasite. The number of asexual *Plasmodium falciparium* and other species per 200 leukocytes were counted and when ten or more parasites were identified, the number was recorded, a blood sample was regarded as negative when the examination of thick films failed to show the presence of asexual parasites. The parasite count in relation to the leukocyte count was converted to parasite per micro litre of blood using this mathematical formula;

$$\frac{\text{Number of parasites}}{\text{Number of leukocytes}} \times \text{Total WBC count} = \text{Parasite per micro litre of blood}$$

2.5. Procedure for Staining Thick Blood Film

This involved making thick blood films on clean grease free glass slides, allowed to air dry and stained with prepared Giemsa stain for 30 mintues. The Giemsa stain was prepared by diluting stock Giemsa stain (commercially obtained) in buffered water immediately before use. Stained slides were rinsed in clean water and allowed to air dry before examination under a microscope using X100 objective lens. Chromatin of malaria parasite was stained dark red and cytoplasm stained blue with Giemsa's stain. The presence of malaria parasite, identification of the species of human parasites and relative malaria parasite count in each blood sample was determined from the Giemsa stained thick films and Leishman stained thin blood films. Malaria Parasitaemia was confirmed by microscopic examination using X100 objective lens (oil immersion lens). A slide was scored as negative when 100 high power fields had been examined for about 30 minutes without seeing any parasites. The amount of relative parasite count (Occurrence) in positive smears was done using a simple

code from one to four crosses (+ - ++++) [15], although none of the subjects had ++++. Malaria ParasitaemiaOccurrence was graded as; + = 1 – 10 parasites per 100 thick film field; ++ = 11 – 100 parasites per 100 thick film field; +++ = 1 – 10 parasites per single thick film field; ++++ = more than 10 parasites per single thick film field after staining for 30 minutes as described by Monica Cheesbrough [16].

2.6. Procedure for Staining Thin Blood Film

Thin blood films were made on clean grease free glass slide and stained using Leishman staining technique as described by Monica Cheesbrough [16]. The films were allowed to air dry and covered with Leishman stain for four minutes. The stain on the slides were diluted with buffered distilled water and allowed to stain for ten minutes. Slides were rinsed with water, allowed to air dry and examined under microscope using X100 objective lens.

2.7. Malaria Parasite Count

Quantitative parasitaemia count (Parasite density) was determined by counting the number of asexual parasites (trophozoites, schizonts) present in as many microscopic fields (100x) necessary to count 200 leukocytes in each thick blood film and multiplies by the total white blood cells count of each blood sample. Parasitaemia was graded as low (parasite 1000-9,999 μL^{-1}) and high (>10,000 μL^{-1}) [17].

$$\text{parasites / ul of blood} = \frac{\text{Number of observed asexual parasites}}{200\ (\text{Number of leucocytes counted})} \times \text{total WBC count / ul}$$

2.8. Biochemical Analysis

Alanine Amino Transferase (ALT) and Aspartate Amino Transferase (AST) activity was determined using the method described by Rietman and Frankel [18], while the activities of Alkaline Phosphatase (ALP) determined using the method described by Rec. Gscc (DGKC) [19].

The Bromo-cresol Green (BCG) binding method was used for the determination of albumin [20].

3% Sulphosalicylic acid (SSA) colorimetric method was used for the determination of Total protein.

The Jaffe-Slot modified alkaline picrate colorimetric method was used for Creatinine level estimation [21].

Urea levels were determined using the diactetylmonozine methods [22].

Glucose oxidase method was used for the estimation of plasma glucose [23].

Plasma total Cholesterol was determined colorimetrically using cholesterol oxidase method as described by Allain *et al.* [24].

2.9. Statistical Analysis

The data generated from the study was subjected to basic statistical measurement for mean and standard deviation, and comparisons were carried out to test for significant differences, using parametric analysis of variance (ANOVA) with the aid of the computer SPSS 20.0 windows application. Statistical significance was accepted at $p < 0.05$.

3. Result

The statistical data obtained from the analyzed subjects and control samples are shown in Tables 1-5 below. Values from Control samples were compared with values from the test subjects, and multiple comparisons were carried out with the one-way ANOVA statistics between groups and within groups and the control and test subject values are presented as mean ± Standard Deviation (SD).

The Demographic characteristic of the study population showed that a total of 105 test subjects was used for this study, 59 (56.18%) were males of which 18 (17.14%) of them were within the ages of 15-25, those within the ages of; 26-35 were 16 (15.23%), 36-45 were 14 (13.33%), 46-55 were 11 (10.48%). 28 (26.60%) males had '+' occurrence of malaria parasitaemia, 24 (22.85%) had '++' occurrence of malaria parasitaemia and 7 (06.60%) males had '+++' occurrence of malaria parasitaemia. A total of 46 (43.81%) female test subjects were used for this study, within the ages of 15-55. Those within the ages of 15-25 were 14 (13.33%), within the ages of 26-35 were 15 (14.29%), within the ages of 36-45 were 08 (07.62%), and those within the ages of 46-55 were 09 (08.57%). 30 (28.51%) females had '+' occurrence of malaria parasitaemia, 14 (13.33%) had '++' occurrence of malaria parasitaemia and 03 (02.81%) females had '+++' occurrence of malaria parasitaemia.

Table 1. *Demographic characteristic of the study population.*

Age (Years)	Male Subjects	Female Subjects
15-25	18 (17.14%)	15 (14.29%)
26-35	16 (15.23%)	14 (13.33%)
36-45	14 (13.33%)	08 (07.62%)
46-55	10 (09.52%)	08 (07.62%)
>55	01 (0.95%)	01 (0.95%)
Total (100%)	59 (56.17%)	46 (43.81%)
Severity of Malaria Parasitaemia		
+	28 (26.60%)	30 (28.51%)
++	24 (22.85%)	14 (13.33%)
+++	07 (06.60%)	03 (02.81%)
Total (100%)	56%	44%

As shown in table 2; when control values were compared withthe mean values obtained from the biochemical analysis of*Plasmodium falciparum*parasitaemia subject samples, it was significantly lower for glucose (p=0.04), ALT (p=0.00), AST (p=0.00), ALP (p=0.00), total protein (p=0.01), Globulin (p=0.02) and Creatinine (p=0.04). Total cholesterol (p=0.12), Triglyceride (p=0.06), Albumin (p=0.49) and Urea (p=0.15) were statistically not significant for malaria parasitaemia subject samples.

Table 2. *Mean±SD of some biochemical parameters of Plasmodium falciparum malaria Infected inhabitants of Ekpoma metropolis compared with healthy controls.*

Parameter	Control	Malaria Infected Subjects	f-ratio	p-values
Glucose (mg/dl)	79.21±11.24	64.34±8.47	4.34	0.04*
Total Cholesterol (mg/dl)	154.65±27.98	149.34±20.77	6.28	0.12
Triglyceride (mg/dl)	85.04±25.39	81.48±24.77	3.19	0.06
ALT (IU/L)	10.66±5.34	24.34±3.15	4.34	0.00*
AST (IU/L)	13.48±3.43	28.50±2.38	2.75	0.00*
ALP (mg/dl)	44.13±13.65	64.23±5.24	5.25	0.00*
Albumin (g/dl)	3.76±0.73	3.85±0.32	3.02	0.49
Total protein (g/dl)	7.85±0.31	6.11±0.35	4.50	0.01*
Globulin (g/dl)	4.09±0.12	2.26±0.23	2.34	0.02*
Creatinine (mg/dl)	0.68±0.16	0.89±0.44	3.45	0.04*
Urea (mg/dl)	15.44±1.12	17.68±1.23	4.21	0.15

KEY: *Statistically significant (p<0.05), ALT; Alanine Amino Transferase, AST; Aspartate Amino transferase, ALP= Alkaline phosphatase, g/dl; gram per decilitre, IU/L; international unit per litre, mg/dl; milligram per decilitre, SD; standard deviation.

As shown in table 3 below; when male control subject values were compared with mean values obtained from the biochemical analysis of male *Plasmodium falciparum* parasitaemia subject samples, it was significantly lower for glucose (p=0.01), ALT (p=0.02), AST (p=0.01), ALP (p=0.00) and Creatinine (p=0.04). Total cholesterol (p=0.06), Triglyceride (p=0.08), total protein (p=0.21), Globulin (p=0.78), Albumin (p=0.13) and Urea (p=0.22) were statistically not significant for malaria parasitaemia subject samples.

Table 3. *Mean±SD of some biochemical parameters of Plasmodium falciparum Malaria infected male inhabitants of Ekpoma metropolis compared with healthy male controls.*

Parameter	Male Control	Male Infected Subjects	f-ratio	p-values
Glucose (mg/dl)	80.12±9.20	69.14±8.47	3.36	0.01*
Total Cholesterol (mg/dl)	148.47±20.28	145.34±30.00	7.11	0.06
Triglyceride (mg/dl)	89.04±15.21	83.18±11.01	4.11	0.08
ALT (IU/L)	11.54±4.30	25.74±2.18	6.14	0.02*
AST (IU/L)	12.23±3.13	27.43±3.12	4.15	0.01*
ALP (mg/dl)	45.13±8.55	69.13±8.09	4.15	0.00*
Albumin (g/dl)	4.16±1.00	4.89±0.42	2.09	0.13
Total protein (g/dl)	7.98±1.01	8.16±1.05	3.71	0.21
Globulin (g/dl)	3.82±0.76	3.22 ±0.32	4.32	0.78
Creatinine (mg/dl)	0.79±0.11	0.95±0.22	5.33	0.03*
Urea (mg/dl)	16.17±1.00	18.08±0.91	7.20	0.22

KEY: * Statistically significant (p<0.05), ALT; Alanine Amino Transferase, AST; Aspartate Amino transferase, ALP= Alkaline phosphatase, g/dl; gram per decilitre, IU/L; international unit per litre, mg/dl; milligram per decilitre, SD; standard deviation.

As shown in table 4 below; when female control subject values compared with mean values obtained from the biochemical analysis of female *Plasmodium falciparum* malaria parasitaemia subject samples, it was significantly lower for glucose (p=0.01), ALT (p=0.00), AST (p=0.01), ALP (p=0.04) and Creatinine (p=0.00). Total cholesterol (p=0.09), Triglyceride (p=0.11), total protein (p=0.08), Globulin (p=0.13), Albumin (p=0.06) and Urea (p=0.27) were statistically not significant for malaria parasitaemia subject samples.

Table 4. *Mean±SD of some biochemical parameters of Plasmodium falciparum Malaria infected female inhabitants of Ekpoma metropolis compared with healthy female controls.*

Parameter	Female Control	Female Infected Subjects	f-ratio	p-values
Glucose (mg/dl)	72.22±5.30	62.23±5.14	4.12	0.01*
Total Cholesterol (mg/dl)	154.11±23.00	150.34±30.00	5.01	0.09
Triglyceride (mg/dl)	86.01±8.20	85.48±14.00	2.05	0.11
ALT (IU/L)	10.13±3.21	23.63±1.29	7.06	0.00*
AST (IU/L)	11.13±2.22	25.21±4.00	9.04	0.01*
ALP (mg/dl)	36.10±3.15	65.25±6.11	5.26	0.04*
Albumin (g/dl)	3.70±0.90	3.51±0.72	4.43	0.06
Total protein (g/dl)	7.67±2.13	7.91±2.00	6.22	0.08
Globulin (g/dl)	3.97±0.18	3.68±0.67	3.15	0.13
Creatinine (mg/dl)	0.59±0.06	0.80±0.22	9.08	0.00*
Urea (mg/dl)	12.38±1.08	14.09±1.01	3.99	0.27

KEY: * Statistically significant (p<0.05), ALT; Alanine Amino Transferase, AST; Aspartate Amino transferase, ALP= Alkaline phosphatase, g/dl; gram per decilitre, IU/L; international unit per litre, mg/dl; milligram per decilitre, SD; standard deviation.

As shown in table 5 below; When the various levels of severity of *Plasmodium falciparum* malaria parasite in the test subjects were compared and multiple comparisons carried out with the one-way ANOVA statistics between groups and within groups, Mean Glucose value for + (1 plus) malaria parasitaemia (67.25±6.34) was significantly higher when compared with ++ (2 plus) Malaria parasitaemia (62.65±4.33) and +++ (3 plus) Malaria parasitaemia (59.17±5.21), p=0.04. Mean Total cholesterol value for + (1 plus) malaria parasitaemia (151.14±1.77) was not significant in comparison with ++ (2 plus) Malaria parasitaemia, but was significantly higher in comparison with +++ (3 plus) Malaria parasitaemia (144.10±24.23), p=0.03. Mean ALT levels for + (1 plus) malaria parasitaemia (18.34±3.15) was not significant in comparison with ++ (2 plus) Malaria parasitaemia, but was significantly lower in comparison with +++ (3 plus) Malaria parasitaemia (26.01±1.19), p=0.01. Mean AST levels for + (1 plus) malaria parasitaemia (16.43±2.08) was significantly lower in comparison with ++ (2 plus) (23.20±1.12) and +++ (3 plus) (29.15±2.02) malaria parasitaemia,++ (2 plus) was significantly lower in comparison with +++ (3 plus) Malaria parasitaemia (p=0.02). Mean Creatinine levels for + (1 plus) malaria parasitaemia (0.68±0.32) was significantly lower in comparison with ++ (2 plus) (0.85±0.15) and +++ (3 plus) (1.3±0.62) malaria parasitaemia,++ (2 plus) was significantly lower in comparison with creatinine levels for +++ (3 plus) Malaria parasitaemia (p=0.00). Triglyceride, ALP, Albumin, Total protein, globulin and Urea were not statistically significant.

Table 5. *Effect of different levels severity of Plasmodium falciparum parasitaemia on Some Biochemical Parameters.*

Parameter	+ (1 plus) Malaria parasitaemia	++ (2 plus) Malaria parasitaemia	+++ (3 plus) Malaria parasitaemia	f-ratio	p-values
Glucose (mg/dl)	67.25±6.34 [a]	62.65±4.33 [b]	59.17±5.21[b]	2.69	0.04*
Total Cholesterol (mg/dl)	151.14±1.77 [a]	148.21±31.41[ab]	144.10±24.23[b]	8.08	0.03*
Triglyceride (mg/dl)	87.68±24.77[a]	87.08±15.70[a]	80.19±30.11[a]	1.94	0.07
ALT (IU/L)	18.34±3.15[a]	21.21±1.05[ab]	26.01±1.19[b]	5.00	0.01*
AST (IU/L)	16.43±2.08[a]	23.20±1.12[b]	29.15±2.02[c]	6.25	0.02*
ALP (mg/dl)	59.21±1.14[a]	64.00±6.03[a]	69.41±3.44[a]	3.12	0.10
Albumin (g/dl)	3.75±0.02[a]	3.55±0.32[a]	3.80±0.14[a]	3.11	0.21
Total protein (g/dl)	7.10±1.05[a]	7.41±1.12[a]	7.81±1.63[a]	4.67	0.32
Globulin (g/dl)	3.35±0.34[a]	3.86±0.56[ab]	4.01±1.00[b]	4.04	0.05
Creatinine (mg/dl)	0.68±0.32[a]	0.85±0.15[b]	1.3±0.62[c]	8.03	0.00*
Urea (mg/dl)	14.23±4.03[a]	13.44±2.05[a]	16.08±3.03[a]	1.21	0.18

KEY: * Statistically significant (p<0.05), ALT; Alanine Amino Transferase, AST; Aspartate Amino transferase, ALP= Alkaline phosphatase, g/dl; gram per decilitre, IU/L; international unit per litre, mg/dl; milligram per decilitre, SD; standard deviation.

As shown in table 6 below, when the various age groups of malaria parasitaemia test subjects were compared and multiple comparisons carried out with the one-way ANOVA statistics between groups and within groups it was not significant for any of the biochemical parameters tested (p>0.05).

Table 6. *Some Biochemical Profile of Plasmodium falciparum Infected inhabitants of Ekpoma metropolis based on Age.*

Parameter	15-25years	26-35 years	36-45 years	46-55 years	55-60 years	f-ratio	p-values
Glucose (mg/dl)	68.12±5.23	66.24±5.66	70.07±6.23	66.43±8.91	67.43±6.87	6.67	1.23
Total Cholesterol (mg/dl)	146.56±30.12	149.72±24.26	150.23±20.34	147.35±27.18	145.58±23.11	4.93	0.68
Triglyceride (mg/dl)	80.37±19.24	69.26±12.56	62.34±23.43	78.48±18.34	79.79±13.67	5.07	0.09
ALT (IU/L)	23.21±2.28	20.14±1.79	21.66±0.79	19.29±2.11	24.16±34	7.12	0.26
AST (IU/L)	26.34±1.79	25.56±2.52	27.34±3.01	26.67±2.10	22.78±1.65	4.19	1.08
ALP (mg/dl)	74.17±4.81	69.18±3.78	67.18±8.12	65.84±6.43	70.21±9.21	7.89	0.06
Albumin (g/dl)	3.66±0.51	3.45±0.11	3.54±0.20	3.19±0.18	3.65±0.67	3.02	0.16
Total protein (g/dl)	6.52±0.54	6.25±1.23	6.41±0.97	6.37±0.24	6.74±1.23	8.06	0.08
Globulin (g/dl)	2.86±0.21	2.80±0.43	2.87±0.65	3.18±0.29	3.09±0.48	3.97	0.15
Creatinine (mg/dl)	0.86±0.52	1.03±0.32	1.11±0.37	0.98±0.28	1.02±0.55	6.23	0.06
Urea (mg/dl)	15.08±2.14	18.11±1.45	16.58±2.01	14.98±1.22	16.18±2.31	3.78	1.35

KEY: * Statistically significant (p<0.05), ALT; Alanine Amino Transferase, AST; Aspartate Amino transferase, ALP= Alkaline phosphatase, g/dl; gram per decilitre, IU/L; international unit per litre, mg/dl; milligram per decilitre, SD; standard deviation.

4. Discussion

The result of demographic characteristic of the study population showed that males had higher prevalence of Malaria parasitaemia than females, which agrees with studies done by Mbanugo and Ejim, [25], Uzoegwu, and Onwurah [26], Nwaorgu and Orajiaka [27], Okafor and Oko-Ose [28]. These researchers reported higher prevalences in males than females.

From this study, the age range 15-25 had the highest prevalent rate of 17.14% for males and 14.29% for females, followed by 26-35 with prevalent rate of 15.23% for males and 13.33%, while the age range 55-60 had the least prevalent rate of 0.95% for both male and female and this

was in conformity with findings of Aribodor *et al.* [29], Nwaorgu and Orajiaka [27] which all reported decline in prevalence by age.

The mean Glucose values were significantly lower for malaria parasitaemia subjects for both male and female. A significant decline in glucose value with increase in severity was observed for glucose. It was observed from this study that glucose reduction in malaria parasitaemia was not affected by age. Some studies have reported that Plasmodium falciparum parasites fully depend on glucose as an energy source, hence hypoglycemia occurs during the management of patients with malaria [30-31], Binh *et al*. [32] reported hypoglycemia in malaria infected patients, stated host glucose production becomes insufficient for both host and parasite demand as infection progressed. Contrary to this study, Esan [33] reported that Glucose level in control subjects were observed lower compared to value obtained in pretreatment and post-treatment during malaria parasite infection.

This study showed decreased Total cholesterol and triglyceride levels when compared with control values, though it was not statistically significant, this was in conformity with a systematic review on Serum lipids and lipoproteins in malaria by Benjamin Visser *et al.* [34]. The increased oxidative stress in malaria, which accounts for the degradation of the lipoproteins, may originate from several sources including intracellular of parasitized erythrocytes and extracellular of haemolysed erythrocytes or host immune responses [35]. These events may be related to the decrease, as significant decrease in total cholesterol levels with increase in severity was observed.

This study established the fact that *P. falciparum* infection has significant effect on the liver, as results of this study showed some significant increases in enzymes activities of aspartate amino transferase (AST), alanine amino transferase (ALT), and alkaline phosphatase (ALP) among patients with P. falciparum malaria parasitaemia, which increased with increase in severity of the malaria parasitaemia. The increased serum levels of hepatic enzymes AST and ALT, and ALP are the biomarkers of liver disorders. This results was in conformity with studies by Ignatius *et al.* [36]; Onyesom and Onyemakonor [37]; Mohamed Al-Salahy *et al.* [38].

The study revealed that total protein, urea, albumin and globulin values of malaria parasitaemia subjects were not statistically significant in comparison with the healthy control, this was at variance with the study done by Melita *et al.* [39]; Prakash *et al.* [40] who recorded significant levels in malaria infected patients. Significantly higher values were recorded for Creatinine in both males and females, and these values increased significantly with increase in severity. It is well known that creatinine concentration is used for the assessment of renal efficiency, hence when their values are higher than normal, deficiency in renal function is suspected. Elevation of plasma creatinine concentration could be suggestive of ineffective filtering ability of the kidneys which could lead to renal function impairment. This was consistent with the studies by Parkash et al. [40]; Ogbadoyi and Tsado [41].

5. Conclusion

Glucose, Total cholesterol, Liver enzymes, creatinine changes were seen involved in malaria infection caused by *Plasmodium falciparum.* The prevention of malarial infection is still the most valid method of preventing these conditions, early diagnosis and treatment are the measures likely to decrease malarial complications commonly determined by changes in these biochemical parameters in developing countries. Prompt visits to centers equipped to provide necessary anti malarial therapy and support could further reduce mortality and enhance recovery.

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