Assessment of Some Nutritional and Immunlogical Parameters of HIV Subjects Co-Infected with Plasmodium Species in Uromi, Edo State, Nigeria

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Abstract
Background: Human Immunodeficiency virus (HIV) is known to compromise the immune system, Malaria a disease caused by plasmodium specie which has its own disease burden can compound the already depressed immune status and nutritional status of HIV patients.
Aim: This cross sectional study was undertaken using patients attending HIV clinic at General hospital and St. Camillus hospital both in Uromi, Edo State Nigeria, to determine the disease burden of HIV sero-positive patients due to malaria parasite co-infection.
Methods: A total of 360 subjects were randomly recruited; 116 HIV and malaria positive (test group), 152 HIV positive and malaria negative (control group I) subjects, and 92 apparently healthy (control group II) subjects aged between 7 and 63 years. Eight milliliter (8ml) of venous blood samples was drawn from each subject. Some immunological parameters and biochemical parameters were assayed. Malaria parasite density was equally determined.
Results: Result from this study showed that the prevalence of malaria and HIV sero-positive subjects (co-infection) within the study area was 43.4%. Mean granulocytes count for co-infected group (58.01±23.36%) was significantly higher (p<0.05) than the control groups. Co-infected subjects had lower mean values for lymphocytes (36.50±22.16 %) (p<0.05), monocytes (5.49±4.04 %) (p<0.05) and total white blood cells (9.26±7.37 x10^9/l) when compared with control groups. Nutritional indices revealed that co-infected subjects had significantly lower (p<0.05) glucose concentration of 65.38±16.59mg/dl when compared to control group I (76.26±47.44mg/dl). Albumin concentration was significantly higher (p<0.05) in co-infected group (4.07±0.66g/dl) than control group I (3.88±0.66g/dl).
Conclusion: HIV co-infection with malaria parasites further increase the disease burden of HIV.

Key words: HIV, Malaria, Co-infection, Disease

Introduction
Malaria and HIV are among the two most important global health problems of our time.

Together, they cause more than 4 million deaths each year. Malaria accounts for more than a million deaths each year, of which over 80% occur in tropical Africa, where malaria is the leading cause of mortalityin children under five years of age. Aside from young children, pregnant women are among the most affected by the disease (W.H.O 2004). Malnutrition is also a major public health problem in tropical areas where malaria prevails, with estimated 38 % stunted, 28 % underweight, and 9 % wasted in Africa (UNICEF, 2007).

Nutritional status impacts on mortality among children less than 5 years due to diarrhoea, respiratory diseases, malaria and measles (Rice *et al.,* 2000). In relation to morbidity, more studies have shown that children and adolescents with chronic malnutrition (stunting) and low weight for age (underweight) besides thin adult have protection against prevalent cerebral malaria (Nacher *et al.,* 2001; Nacher *et al.,* 2002), stunted and underweight children and adolescents have less prevalence and incidence of hyperparasitaemia (Thuma *et al.,* 2011; Mitangala *et al.,* 2013) and, to a lower extent, children and adolescents with wasting or stunting were protected against new episodes of clinical malaria (Fillol *et al.,* 2009). Although limited by the small number of studies, malnutrition may contribute to deaths from malaria, even though the significance was not high compared with other diseases (Faye *et al.,* 1998; Man*et al.,* 1998). However, some studies found no association between nutrition and subsequent mortality from malaria (Van *et al.,* 1993; Genton *et al.,* 1997).

HIV and malaria infections often coexist in patients in many parts of the world due to geographic overlap of these two diseases. This is particularly true in sub-Saharan Africa, where an estimated 40 million people are living with HIV and more than 350 million episodes of malaria occur yearly. There is also evidence of a negative interaction between these two infections. HIV increases the risk of malaria infection and the development of clinical malaria. Conversely, malaria increases HIV replication (Hewitt *et al.*, 2006).

HIV infection compromises the nutritional status of infected persons and in turn poor nutritional status can affect the progression of HIV infection (Friis and Michaelsen, 1998; Piwoz and Preble, 2000; Fawzi, 2003).It has been shown that deficiencies of nutrients may affect the immune function in ways that may influence viral expression and replication, which further affect progression of HIV disease and mortality of the patient (Semba and Tang, 1999). Hormones such as glucagon, insulin, epinephrine and cortisol which are involved in the metabolism of protein, carbohydrate and fat, have been reported to be affected by HIV infection (Macallan, 1999). Increased levels of these hormones are believed to contribute to weight loss and the wasting syndrome seen in HIV/AIDS patients (Macallan, 1999; Piwoz and Preble, 2000). Research studies have confirmed that nutrient deficiencies are associated with immune dysfunction and accelerated progression to AIDS (Macallan, 1999). Furthermore, deficiencies of protein and essential fatty acids interfere with immune function.

Increased availability of highly active antiretroviral therapy (HAART) in LMICs has led to some improvement of the nutritional status of patients [Gupta *et al.,* 2011; Johannessen *et al.,* 2011; Padmapriyadarsini *et al.,* 2010]. However, for certain individuals, undernutrition and weight loss persist despite therapy (Mangili*et al.,* 2006; Hadgu *et al.,* 2013). Just like with HIV, HAART and malnutrition contribute to a deadly cycle. Highly active antiretroviral therapy (HAART) leads to increased requirements for macro- and micronutrients, high metabolic demands (Shevitz *et al.,* 1999) and low appetite (Wakeham *et al.,* 2010) which perpetuate undernutrition (Ivers *et al.,* 2009)

Materials and Method
Study Location
The study area was carried out in Uromi, the administrative headquarter of Esan North-East L.G.A. in Edo State, Nigeria.

Research Design
This was a cross sectional study. Human subjects positive to HIV were used for the study. The recruitment of malaria co-infected participants was by simple random sampling method. The control subjects II were apparently healthy subjects within the study community.

Sample Size
A total number of 360 subjects were recruited; 116 HIV-infected malarial parasitaemia subjects, 152 HIV-infected malarial aparasitaemia subjects and 92 apparently healthy subjects between 7 to 63 age group visiting HIV clinic at General Hospital, Uromi and St. Camillus Hospital, Uromi, Edo State.

The sample size was calculated using the formula:

$$N = Z^2 pq / D^2$$

- Where N = sample size, Z = standard deviation (1.96), p = prevalence, q = 1-p and D = degree of freedom (0.05), and a 4.6% HIV prevalence for Edo state. (EDOSACA, 2014).

Extrapolation from method

$$N = \frac{(1.96)^2 \times 0.046 \times (1 - 0.046)}{(0.05)^2}$$

$$\frac{3.8416 \times 0.046 \times 0.954}{0.0025}$$

$$\frac{0.1686}{0.0025}$$

67.4 or 67

To increase reliability of result, 116 test samples were collected.

Selection Criteria
Inclusion Criteria:
Only HIV subjects positive to malaria infection were recruited as test subjects for the study. HIV patients negative to malaria were recruited as Control I, while apparently healthy subjects were recruited as Control II

Exclusion Criteria:
With the aid of verbal questionnaire, patients with clinical illness (condition) other than malaria and HIV were excluded from the study

Ethical Approval
Ethical approval was obtained from the ethics and research committee of Ambrose Alli University, Ekpoma and informed consent of the patients was sought before sample collection.

Anthropometric Measurement

Weight and height of all subjects were measured to evaluate the Body Mass Index (BMI) which indicates the usual health and nutritional status of an individual (Razak, 2007).

Sample Collection

About 8mls of venous blood was collected from each patient. Three milliliter (3mls) was dispensed into EDTA anticoagulant bottle for evaluation of haematological and immunological parameters as well as thick film preparation for malaria. Five milliliter (5mls) was dispensed into lithium heparin bottle for biochemical assay. These samples were immediately analyzed in the Laboratory Department of General Hospital, Uromi.

Laboratory Analytical Methods
Malaria Parasitaemia by Blood Film Examination

A thick blood film was prepared according to Cheesbrough, (2004) using giemsa stain. The thick blood film was examined; 200 or 500 white blood cells were counted and the number of malaria parasites was also taken. The percentage of parasitaemia was calculated as recommended by *Denis et al.*, (2012).

That is: number of malaria parasites   x   Assumed WBC count (10000/µl)
            number of WBC counted

Full Blood Count and Biochemical Assay

Full Blood Count was carried out using automation – Sysmex Kx-21N automated Haematology Analyzer. Biochemical analysis was carried out using spectrophotometric methods; Serum total protein was processed by Biuret Method as described by Johnson, (2006); Serum Albumin was determined by Bromocresol Green method as described by Daumas *et al.*, (1971); Total cholesterol and Triglyceride were estimated by the Enzymatic method of Nader and Warnick, (2006), while Glucose oxidase method was used to estimate serum glucose as described by David, (2006).

Data Analysis

Data generated was presented in a table form. Data obtained from assayed parameter was statistically analysed using ANOVA (SPSS 20.0). Values were expressed as Mean ± SD. A p-value of ≤ 0.05 was considered as significant in all statistical analysis.

Results

This study was carried out to determine the nutritional status and cellular immunity profile of HIV subjects co-infected with malaria in Uromi, Edo State between September 2016 and March 2017.

Demographic Attributes of the Population of Study

Table 1 shows the demographic characteristics of the population of study. From the table, the total number of the study population was 360. The number of HIV-malaria co-infected subjects (Test Group) studied was 116 (32.2%), the total number of HIV non-malaria infected subjects (Control I) was 152 (42.2%), while 92 (25.6%) apparently healthy subjects were used as Control II. Ninety two (92) females and twenty four (24) males made up test group. There were 112 females and 40 males in control I. The control II group was made up of 32 females and 60 males. In test group, there were 84(31.3%) ART (anti-retroviral therapy) and 32(11.9%) non-ART subjects. While in control group I, the distribution was 132(49.2%) and 20(7.5%) for

ART and non-ART respectively. The age group with predominant co-infected subjects (45) was 30-39 while 18years and below had the least distribution (4).

Table 1: Demographic Distribution of Study Subjects According to Age and Sex

Age (Years)	CONTROL			MP&HIV /ART			MP&HIV/ NOART			HIV/ART			HIV/NOART			TOTAL INFECTED (%)		
	M	F	T	M	F	T	M	F	T	M	F	T	M	F	T	Coinfec.	HIV Pos.	Study Pop.
≤18	0	4	4	0	0	0	4	0	4	0	4	4	0	0	0	4(3.4%)	8(3.0%)	12(3.3%)
19-29	40	8	48	1	4	5	2	3	5	0	12	12	0	4	4	10(8.6%)	26(9.7%)	74(20.6%)
30-39	16	8	24	0	31	31	1	13	14	12	43	55	4	3	7	45(38.8%)	107(39.9%)	131(36.4%)
40-49	3	12	15	2	12	14	1	4	5	8	17	25	3	5	8	19(16.4%)	52(19.4%)	67(18.6%)
50-59	1	0	1	5	12	17	4	0	4	12	20	32	1	0	1	21(18.1)	54(20.1%)	55(15.3%)
60-69	0	0	0	4	13	17	0	0	0	0	4	4	0	0	0	17(14.7%)	21(7.8%)	21(5.8%)
Total	60	32	92	12	72	84	12	20	32	32	100	132	8	12	20	116(100%)	268(100%)	360(100%)

KEY: MP&HIV/ART=HIV-Malaria co-infected subjects on Anti-retroviral drug, **MP&HIV/NOART**= HIV-Malaria co-infected subjects not on Anti-retroviral drug, **HIV/ART=** HIV non-Malaria infected subjects on Anti-retroviral drug, **HIV/NOART**= HIV non-Malaria infected subjects not on Anti-retroviral drug. **Coinfec.=** Coinfected group, **HIV Pos.=** HIV sero-positive group, **Study Pop.=** Study population, M = Male, F = Female, T = Total

The sex differences and similarities of the mean weight (kg) and body mass index (BMI) of the study population were shown in table 2. In control II group, the mean weight of the female (63.88±16.47kg) was higher than that of the male (60.60±9.37kg) but the difference was not statistically significant (p>0.05). Conversely, the average weight of control I subjects showed that the female group (61.86±14.87kg) was lower than their male counterpart (62.30±13.12kg) (p>0.05). Data from test subjects reveals statistically significant (p<0.05) difference between the male (66.20±11.15kg) and the female (58.38±14.83kg). Similarly, there was significance (p<0.05) difference between the mean weight of male subjects in test (66.20±11.15kg) and control I (60.60±9.37kg) groups. Also, the mean weight of control II female subjects was statistically significantly (p<0.05) higher than their female counterpart among the test subjects. Other differences were not significant (p>0.05). The control II male showed significantly (p<0.05) lower BMI (21.23±3.20kg/m^2) when compared to female control (25.11±5.40kg/m^2). Comparing BMI of the same sex across the three groups, the statistical differences in the average BMI followed the same pattern as seen with the weight.

Table 2: Mean Weight and BMI Values of the Study Subjects with Respect to Sex.

Parameters	Control (Male) n=60	HIV (Male) n=40	HIV& MP (Male) n=20	Control (Female) n=32	HIV (Female) n=112	HIV& MP (Female) n=96	F-value	P-value
Weight (Kg)	60.60±9.37[ab]	62.30±13.12[ab]	66.20±11.15[b]	63.88±16.47[ab]	61.86±14.87[ab]	58.38±14.83[a]	1.69	0.14
BMI(kg/ m²)	21.23±3.20[ad]	21.99±4.78[ad]	23.52±3.93[bcd]	25.11±5.41[b]	24.23±5.28[bc]	23.07±4.81[c]	6.26	0.00

P>0<0.05
KEY: Values in a row with different superscript are statistically significantly different at p<0.05. **HIV&MP** = HIV-Malaria co-infected subjects, **HIV**= HIV non-Malaria infected subjects.

Cellular Immunity Profile of the Study Groups

Table 3 demonstrates the mean distribution of cellular immunity of the study population and the inter-group comparison. The control II group revealed the highest mean CD4 value of 928.39±179.89cells/mm^{3}which was significantly (p<0.05) higher than the other two groups. The mean CD4 for control I subjects (436.87±263.31cells/mm^3)was significantly lower than that of test subjects (559.38±398.60 cells/mm^3). Similarly, the mean Lymphocytes showed significant differences (p<0.05) across the groups. The mean monocytes distribution showed that control group II (5.36±3.13%) and test subjects (5.49±4.04%) were significantly (p<0.05) lower than the mean monocytes value of control I subjects (7.65±6.09%). There was no significant difference in the mean WBC count across the three groups.

The mean CD4 value of the male control group II was observed to be significantly (p<0.05) higher than the other groups except the female control II (p>0.05). The mean CD4 value of male control I group was significantly (p<0.05) lower than the mean values of the other groups.

Table 3: Cellular Immunity Profile of the Study Groups

Parameters	Control n=92	HIV&MP n=116	HIV n=152	F-value	P-value
CD4 (cells/mm^3)	928.39±179.89[a]	559.38±398.60[b]	436.87±263.31[c]	79.75	0.00
LYMP (%)	29.93±14.65[a]	36.50±22.16[b]	42.56±21.78[c]	11.22	0.00
MON (%)	5.36±3.13[a]	5.49±4.04[a]	7.65±6.09[b]	9.25	0.00
GRAN (%)	64.71±16.47[a]	58.01±23.36[b]	49.82±25.19[c]	12.90	0.00
TWBC(x10^{9}l)	6.47±2.42[a]	9.26±7.37[b]	9.96±9.12[b]	6.72	0.01

P>0<0.05

KEY: Values in a row with different superscript are statistically significantly different (P<0.05). **CD4**= Cluster of differentiation 4, **LYMP**= Lymphocyte, **MON**= Monocytes, **GRAN**= Granulocyte, **TWBC**=Total white blood cell count, **HIV&MP**= HIV-Malaria co-infected subjects, **HIV**= HIV non-Malaria infected subjects.

The least mean lymphocyte value (28.94±14.52%) was seen in male control group II while the highest (44.56±21.56%) was seen in control group I. The difference was statistically significant (p<0.05). The mean monocyte differences between male and female within the same group were significant (p<0.05) except for test group (p>0.05). The highest mean monocyte was observed among male control group I (10.11±6.40%) and the smallest mean value was observed in male control group II (4.61±2.75%). The difference was statistically significant (p<0.05). The mean monocyte value of male test group (7.24±5.41%) was significantly (p<0.05) higher than male control group II and lower than male control group I. Control group I male had significantly lower mean granulocyte (45.33±25.96%) when compared with male control II subjects (66.45±16.03%). The mean granulocyte value for female test group (58.45±25.02%) was significantly (p<0.05) higher than the mean granulocyte value for female control I subjects (51.42±24.84%). The mean WBC count for male control group I (10.37±11.96x10^9/l) was higher (p<0.05) than female control group II (5.41±1.28x10^9/l). (See table 4)

Table 4: Cellular Immunity Status of Study Groups According to Sex.

PARAMETERS	Control (Male) n=60	MP&HIV (Male) n=20	HIV (Male) n=40	Control (Female) n=32	MP&HIV (Female) n=96	HIV (female) n=112	F-value	P-value
CD4 (cells/mm^3)	954.33±171.87^a	464.80±274.72^{bd}	262.40±217.26 ^{c}	879.75±187.17 ^{a}	579.08±418.28 ^{d}	499.18±250.69 ^{b}	38.48	0.00
LYMP(%)	28.94±14.52^a	36.84±14.21^{abcd}	44.56±21.56^c	31.79±14.94^{ab}	36.43±23.54^{bd}	41.85±21.92^{cd}	4.65	0.00
MON (%)	4.61±2.75^a	7.24±5.41^{bd}	10.11±6.40^c	6.76±3.35^{bd}	5.12±3.62^{ab}	6.77±5.75^d	8.41	0.00
GRAN(%)	66.45±16.03 ^{a}	55.92±12.87^{abcd}	45.33±25.96^{cd}	61.45±17.05^{ab}	58.45±25.02^b	51.42±24.84^d	5.84	0.00
WBC(x10^{9}l)	7.03±2.70^a	7.44±3.62^{abcd}	10.37±11.96^{cd}	5.41±1.28^a	9.63±7.90^{bcd}	9.82±7.93^d	3.22	0.01

P>0<0.05
KEY: Values in the same row with different superscript are statistically significantly different (P<0.05). **CD4**= Cluster of differentiation 4, **LYMP**= Lymphocyte, **MON**= Monocytes, **GRAN**= Granulocyte, **TWBC**=Total white blood cell count, **HIV&MP**= HIV-Malaria co-infected subjects, **HIV**= HIV non-Malaria infected subjects.

Nutritional Indices Profile of the Study Subjects
The nutritional indices pattern was demonstrated in table 5. The control group II gave a significantly (p<0.05) higher mean Packed Cell Volume (PCV) value (38.83+2.61%) than the two HIV sero-positive groups. Glucose evaluation reveals that the test group had significantly (p<0.05) lower mean values than the control groups. The test group gave a significantly (p<0.05) higher mean albumin concentration than the control groups. There was significant difference between the mean globulin of test group (3.22+0.81g/dl) and control group I (3.57+1.17%). Control group II with mean total cholesterol concentration of 139.65+37.98g/dl was significantly (p<0.05) lower than the mean values obtained from the HIV sero-positive groups. The HIV non-malaria infected group showed a mean triglyceride (Tg) concentration (78.29+34.41g/dl) that was not significantly different from the control group and HIV-malaria co-infected group. The test group had a significantly higher (p<0.05) mean Tg concentration (85.48+30.77g/dl) than that of control group II (75.04+25.29g/dl).

Table 5: Trend of Some Nutritional Indices of the Study Subjects

PARAMETERS	Control n=92	HIV&MP n=116	HIV n=152	F-value	P-value
PCV (%)	38.83±2.61[a]	29.49±6.49[b]	29.39±6.34[b]	93.54	0.00
GLUCOSE (mg/d)	72.17±21.98[ab]	65.38±16.59[a]	76.26±47.44[b]	3.36	0.036
TOTAL PROTEIN (g/dl)	7.44±0.70[a]	7.29±0.75 [a]	7.46±0.84 [a]	1.64	0.20
ALBUMIN (g/dl)	4.04±0.50 [ab]	4.07±0.66 [a]	3.88±0.66[b]	3.45	0.03
GLOBULIN (g/dl)	3.40±0.86 [ab]	3.22±0.81 [a]	3.57±1.17[b]	4.16	0.02
TOTAL CHOLESTEROL (g/dl)	139.65±37.98[a]	150.31±30.70[b]	155.50±33.64[b]	6.28	0.00
TRIGLYCERIDE (g/dl)	75.04±25.29[a]	85.48±30.77[b]	78.29±34.41[ab]	3.19	0.04

P>0<0.05

KEY: Values in the same row with different superscript are statistically significantly different (P<0.05). **PCV**= Packed cell volume, **HIV&MP**= HIV-Malaria co-infected subjects, **HIV**= HIV non-Malaria infected subjects.

Table 6 demonstrates some nutritional indices with reference to sex and apt comparison outcome. The male control group II produced crest mean PCV of 40.07+1.78% which showed to be significantly (p<0.05) higher than the mean value obtained from all other groups. The male subjects from the test group had significantly (p<0.05) higher mean PCV (34.80+7.23%) than its male counterpart in control I group (28.24+6.52%). There was no significant (p>0.05) difference in the mean PCV values of female subjects between the HIV sero-positive groups. Male subjects in control group I gave the maximum mean glucose concentration (83.80+26.89mg/dl) which was statistically significantly (p<0.05) higher than the least mean

glucose value obtained from male subjects of the test group (56.60 ± 8.27mg/dl). The differences in the mean glucose concentration of the female subjects across the three groups were not significant ($p > 0.05$). The mean total protein value obtained with the male test group was significantly ($p < 0.05$) lower than values from the male control group II. The crest mean albumin concentration (4.44 ± 0.47g/dl) was obtained amid male subjects of test group which was significantly ($p < 0.05$) higher than the mean value obtained from male subjects of control group I (3.56 ± 0.41g/dl). The female subjects across the groups revealed no significant ($p > 0.05$) difference in their mean albumin values. Conversely, globulin evaluation reveals a maximum mean value with male subjects of control group I which was significantly ($p < 0.05$) higher than the mean values of both control II and test groups.

Female subjects of control group I had a mean total cholesterol level that was statistically significantly ($p < 0.05$) higher than the female control II counterpart but showed no significant ($p > 0.05$) difference with the mean value obtained from the female test subjects. Triglyceride assay showed peak mean concentration (89.00 ± 30.94g/dl) with the male subjects among the test group which was significantly higher ($p < 0.05$) than the mean Tg value for male subjects of control group I (69.50 ± 23.69g/dl). The mean Tg concentration obtained from male subjects of HIV sero-positive groups were significantly higher ($p < 0.05$) than the mean value from the female control II subjects.

Table 6: Nutritional Indices of Study Subjects with Reference to Sex

PARAMETERS	Control (Male) n=60	HIV&MP (Male) n=20	HIV (male) n=40	Control (female) n=32	HIV&MP (female) n=96	HIV (female) n=112	F-value	P-value
PCV	40.07±1.78^a	34.80±7.23 ^{b}	28.24±6.52 ^{c}	36.50±2.32^b	28.38±5.78 ^{c}	29.79±6.24^c	47.55	0.00
GLUCOSE (mg/dl)	77.40±23.92^a	56.60±8.27^{bc}	83.80±26.89^a	62.37±13.32^{bc}	67.21±17.32^{ac}	73.57±52.72^{ab}	3.05	0.01
TOTAL PROTEIN (g/dl)	7.64±0.68 ^{a}	7.24±1.10^{bc}	7.35±1.11^{ab}	7.08±0.60 ^{b}	7.30±0.66^{bd}	7.49±0.74^{acd}	3.14	0.00
ALBUMIN (g/dl)	4.15±0.51 ^{ac}	4.44±0.47 ^{a}	3.56±0.41 ^{e}	3.84±0.41^{bde}	4.00±0.67^{cd}	4.00±0.69^{cbd}	7.50	0.00
GLOBULIN (g/dl)	3.27±0.78^{ac}	2.80±1.09^c	3.79±1.17^{bd}	3.65±0.95^{ab}	3.31±0.72^a	3.49±1.17^{ad}	3.71	0.00
TOTAL CHOLESTEROL (g/dl)	141.87±32.66^{ac}	147.40±20.63^{ad}	155.00±45.61^{cd}	135.50±46.67^a	150.92±32.46^{cd}	155.68±28.44^d	2.68	0.02
TRIGLYCERIDE (g/dl)	78.27±25.15^{ab}	89.00±30.94^b	69.50±23.69^a	69.00±24.83^a	84.75±30.85^b	81.43±37.40^b	2.59	0.03

P>0<0.05

KEY: Values in the same row with different superscript are statistically significantly different (P<0.05). **PCV**= Packed cell volume, **HIV&MP**= HIV-Malaria co-infected subjects, **HIV**= HIV non-Malaria infected subject

Discussion

Body Mass Index (BMI) statistics from this study revealed that the co-infected female had significantly ($p<0.05$) lower values ($23.07\pm4.81kg/m^2$) than the control female subjects ($25.11\pm5.41kg/m^2$). This may be due to loss of appetite associated with malaria.

Cellular immunity profile of the study population showed that infected groups had significantly ($p<0.05$) higher lymphocytes ($36.50\pm22.16\%$ and $42.56\pm21.78\%$) and total White Blood Cell (WBC) count (9.26 ± 7.37 x10^9l and 9.96 ± 9.12 x10^9l) than the apparently healthy group with $29.93\pm14.65\%$ and 6.47 ± 2.42 x10^9l for lymphocytes and total WBC count respectively. This could be as a result of increased mobilization of leucocytes associated with infection. There was higher ($p<0.05$) mean granulocytes count in co-infected group ($58.01\pm23.36\%$) than their non-co-infected counterpart ($49.82\pm25.19\%$). This deviation may be by reason of the association of granulocytes in immune response against parasitic infection.

Total WBC count was slightly lower in co-infected group (9.26 ± 7.37x10^9l) than the non-co-infected complement (9.96 ± 9.12 x10^9l). The difference was not statistically significant ($p>0.05$). The reduction in total WBC count may be because of increased destruction of leucocytes allied with multiple infections.

Generally, in this study, the presence of malaria parasitaemia was not associated with anaemia. This remark could be ascribed to the fact that all the HIV individuals with malaria in this study had very low parasite density. This was in agreement with the study of Ebomwonyi, *et al.,* (2013) who reported reduction in the proportion of severe malaria in Esan Northeast and Owan West. These low density parasites may not have had significantly negative impact on the red blood cells (PCV) of co-infected subjects in this study. Clinical malaria is well studied and reported to be linked with a reduction in haemoglobin levels frequently leading to anaemia (Halliday *et al.,* 2012; Mohandas and An, 2012). The mean PCV values for malaria co-infected and non-co-infected HIV positive subjects were $29.49\pm6.49\%$ and $29.39\pm6.34\%$ respectively. There was no significant difference ($p>0.05$). This finding agreed with the study reported in Osogbo, Osun state, Nigeria where the mean haemoglobin level of malaria positive HIV individuals (10.86 g/dL SD $\pm$ 1.87) in comparison to their malaria negative counterpart (10.80 g/dL SD $\pm$ 1.71) was not statistically significant (Olusola *et al.,* 2014).

Albumin obtained with the co-infected group ($4.07\pm0.66g/dl$) reveals significantly higher value than their non-co-infected compliment ($3.88\pm0.66g/dl$). This produced corresponding significant ($p<0.05$) decrease globulin values of co-infected group ($3.22\pm0.81g/dl$) when compared with the non-co-infected group ($3.57\pm1.17g/dl$). The reduced globulin among co-infected subjects could be caused by depletion in immunoglobulin occasioned by immune response to parasitic infection (Stewart, 2012). Lower total cholesterol was obtained with co-infected group ($150.31\pm30.70g/dl$) than non-co-infected group ($155.50\pm33.64g/dl$). The difference was not significant ($p>0.05$). The reduction obtained in co-infected group may be because of loss of appetite occasioned by malaria. The result obtained in glucose evaluation revealed a significant ($p<0.05$) drop within co-infected group ($65.38\pm16.59mg/dl$) when put side by side with the glucose values from the non-co-infected group ($76.26\pm47.44mg/dl$). This report was in line with the findings of Annalisa *et al.,* (2012) who recorded a significantly lower level of glucose ($4.8\pm1.5mmol/L$) among HIV/malaria co-infected subjects in Mozambique. This reduction in glucose could be attributed to its utilization by the parasite (Monika *et al.,* 2006) as well as loss of appetite due to malaria.

Co-infected female group had lower ($p>0.05$) PCV ($28.38\pm5.78\%$)than the non-co-infected counterpart ($29.79\pm6.24\%$). The disparity may be due to haemolytic activity of malaria parasites (CDC, 2015). Contrarily, co-infected male subjects had significantly ($p<0.05$) higher

mean PCV value (34.80±7.23%) than the non co-infected complement (28.24±6.52%). This variation may be due to haemo-concentration, or as a result of medical intervention such as blood transfusion (Carson *et al.,* 2012).

Recommendation

Considering the findings from this research endeavor, we recommend the following;

- **I.** Routine malaria parasite screening should be carried out for sero-positive patients during HIV clinics. Also mosquito nets should be given to HIV patients
- **II.** Patients of malaria co-infection with HIV should be managed with food supplements.
- **III.** Estimation of fasting blood sugar is advised for malaria co-infected HIV clients.

Conclusion

We concluded from this study that malaria infection compounds HIV disease burden and the health indices of malaria-HIV co-infection adversely affected include lymphocytes, total WBC, PCV, glucose, total protein, globulin, total cholesterol and triglycerides. Of the affected indices, total WBC count, lymphocytes, PCV, glucose, and globulin are diagnostic in monitoring the prognosis and management of HIV disease burden.

Acknowledgement

We appreciate the Management and staff of the General Hospital, Uromi and St. Camillus Hospital, Uromi, Edo State, Nigeria for their support.

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AUTHOR'S CONTRIBUTIONS

Okparaku, S. O. was responsible for Manuscript preparation and execution of the research.

Okogun, G.R.A was involved in development of the research design.

Dic-Ijiewere, O.E was responsible for data collation and statistical analysis while

Airhomwanbor, K.O. was involved in resource and quality control

Idehen, I.C. was involved in resource and quality control