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The impact of HIV and malaria co-infection on apolipoprotein profile and CD4$^+$ T cell counts in adult HIV seropositives in Nauth Nnewi, South Eastern, Nigeria.

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Abstract

Aim: To determine the impact of HIV and malaria co-infections on Apolipoproteins and CD4$^+$ T cell counts in adult HIV seropositives in NAUTH Nnewi, South Eastern, Nigeria.

Methods: A total of 400 subjects aged between 17 and 58 years were recruited from patients that attended HIV Clinic, NAUTH, Nnewi for this study. The study design was a case-control study. Ethical approval was obtained from the Ethics Review Committee from NAUTH, Nnewi. The subjects were grouped based on WHO criteria for staging HIV infection and the presence or absence of malaria. Turbidimetric procedure was used forA-I, A-II, B, C-II, C-III and Apo E and Cyflow SL-Green for CD4 counts. Student t test was used for data analysis.

Results: The mean serum levels of Apo C-III and Apo E were significantly higher in symptomatic HIV on ART with malaria parasitaemia compared with those without malaria parasitaemia at ($P< 0.05$ respectively). But the mean blood CD4 count was significantly lower in symptomatic HIV on ART with malaria infection compared to those without malaria infection($p<0.05$). Also, the mean serum levels of Apo C-II and Apo E were significantly higher in symptomatic HIV not on ART with malaria parasitaemia compared with those without malaria parasitaemia at ($P< 0.05$ respectively).Also, the serum Apo E level was significantly higher in asymptomatic HIV subjects with malaria parasitaemia compared with those without malaria parasitaemia($p<0.05$).

Conclusion: Malaria endemicity poses a threat to the improved apolipoprotein status in HIV malaria co-infection.

Keywords: HIV, malaria, Apolipoproteins, CD4$^+$ T cell.

1. Introduction

Malaria is a debilitating and life threatening protozoan disease in Human commonly caused by any of the four species of the genus Plasmodium namely *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale and Plasmodium malariae*.- a mosquito-borne infectious disease. It is naturally transmitted by the bite of a female anopheles mosquito [1].

The signs and symptoms of malaria begin about 7 – 25 days after infection[2].The classical symptoms of malaria is febrile paroxysm which is a cyclical occurrence of sudden coldness followed by rigor, then fever and sweating which last for six hours, occurring every two days (tertian fever) for *P. vivax* and *P ovale* infection and every three days (quartan fever) in *P. malarae.* Severe malaria is mainly caused by *P. falciparum* infection and it usually arises between 6-14 days after infection [3,4].*P. falciparum* infection can cause recurrent fever every 36- 48 hours or a less pronounced and almost continuous fever[5].

These parasites spent about two weeks and several months in the liver and multiply within red blood cells causing symptoms like fever and headache. In severe cases, the disease worsens leading to hallucinations, coma and even death [6]. Other signs and symptoms of malaria include shivering, arthralgia, vomiting, anaemia, jaundice, anaemia, vomiting, shilvering, joint pain, headache, pyrexia, haemoglobinaemia, renal damage and convulsion [5,7,8].

The $CD4^+$ T cell counts are the primary target of HIV infection because of the affinity of the virus to the CD4 surface marker. Infection with HIV leads to a progressive impairment of cellular functions, which is characterized by a gradual decline in peripheral blood $CD4^+$ T cell counts levels and a wide variety of opportunistic, viral, bacterial, protozoal and fungal infections and to certain malignancies [9].

Apolipoproteins are proteins that bind to lipids to form lipoproteins, whose main function is to transport lipids. Apolipoproteins are important in maintaining the structural integrity and solubility of proteins (5). Hence, this study is intended to evaluate the impact of HIV and malaria on Apolipoprotein profiles and CD4 counts in adult HIV seropositive individuals in NAUTH, Nnewi.

2. Materials and Methods

2.1 Subjects

The study was conducted in Nnamdi Azikiwe University (NAUTH), Nnewi in Anambra state, South East Nigeria.Based on 3.1% prevalence rate of HIV in Nigeria [10], and using the formular of Naing *et al.*[11]. for sample size calculation, a total of 400 adult subjects were recruited by random sampling technique from subjects that attended HIV Clinic and ART unit of NAUTH, Nnewi, South Eastern Nigeria. All the subjectsparticipated voluntarily, with a written informed consent. Ethical approval was obtained from the Ethics committee of NAUTH, Nnewi. The subjects were screened for both HIV and malaria infection routinely and the subjects were classified, using WHO method [12].HIV staging as guide, into: (1) Symptomatic HIV on Antiretroviral therapy (ART) (n = 100) of these, 50 were symptomatic HIV on ART with malaria co-infection. (2) Symptomatic HIV not on ART (n = 100) of these, 50 were symptomatic HIV not on ART with malaria co-infection. (3) Asymptomatic HIV subjects (n = 100) of these, 50 were asymptomatic HIV with malaria co-infection. (4) HIV seronegative control subjects (n = 100) of these, 50 were malaria positive. Questionnaires were used to obtain background information of subjects such as age, sex, time of HIV infection, time of intake of ART, any or absence of disease.

2.2 Sample collection

Six milliliter (6 ml) of fasting blood samples were collected from all the participants in this study. 2ml of blood samples were collected into EDTA sample tubes for HIV and malaria screening and detections, and CD4+ T cell count. The remaining 4 ml of blood sample were collected into plain tube and allowed to clot, centrifuged, the serum separated and analyzed for serum Apo A-I, Apo A-II, Apo B, Apo C-II, Apo C-III and Apo E levels.

2.3 Quality control measures

Quality control sera were run along test in each batch of analysis these were compared with the reference values of the control sera. Standard deviation and coefficient of variation were calculated on them.

2.4 Methods of assaying

2.4.1 Determination of Antibodies to HIV-1 and HIV-2 in Human plasma.

Procedure

Two different methods were used, namely, Abbott determine TM HIV -1 and HIV-2 kit, which is an in-vitro visually read immunoassay (Abbott Japan Co.Ltd.Tokyo, Japan) and HIV-1 and 2 STAT-PAK Assay kit, which is an Immunochromatographic test for the quantitative detection of antibodies to HIV-1 and HIV-2 in Human plasma (CHEMBIO Diagnostic system, Inc, New York, USA). For the Abbott determine TM HIV -1 and HIV-2 kit, the procedure described by the manufacturer was used for the analysis. Briefly, 50 µl of participant serum samples separated from the corresponding whole blood samples in EDTA were applied to the appropriately labeled sample pad. After 15 minutes but not more than 60 minutes of sample application, the result was read. This method has inherent quality control that validates the results. For the Immunochromatographic method for HIV -1 and HIV-2, the procedure described by the manufacturer was used for the analysis. In brief, 5 ml of participant's plasma was dispensed into the sample well in the appropriately labeled sample pad. Three drops of the buffer supplied by the manufacturer was added into the appropriately labeled sample pad. The results of the test were read at 10 minutes after the addition of the running buffer. This method has inherent quality control and validates the results.

2.4.2 Determination of $CD4^+$T cells counts by CyFlows SL-Green

Procedure

200 ml EDTA whole blood was collected into PARTEC test tubes (Rohren tube). Then 20 µl of

$CD4^+$ T antibody was added into the tube. The contents was mixed and incubated in the dark for15 minutes at room temperature. 800 ml of CD4 buffer was added into the mixture and mixed gently. Then the Partec tube was plugged on the Cyflow counter and the $CD4^+$ T cells were displayed as peaks and interpreted as figures.

2.4.3 Quantitative determination of Apolipoprotein A-I in human sera

Principle

Turbidimetric test was used for the measurement of apolipoprotein A-I. Anti- Apo A-I antibodies were mixed with samples containing Apo A-I, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo A-I concentration of the patient's sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).

The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 ^{O}C. 800 µ/l of Reagent R1 (Tris buffer, 20 mmol/L. PEG, pH 8.3, sodium azide 0.95 g/L) was dispensed into a cuvette and 7 µ/l of calibrator was added respectively, mixed and the

absorbance (A_1) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as A_1 sample. Immediately, 200 µ/l of Reagent R2 (Anti-human apolipoprotein A-1 goat- polyclonal antibody, tris buffer, 50 mmol/L, pH 7.5, sodium azide 0.95 g/L) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 2 minutes as A_2 for calibrator and sample respectively.

Calculations

$\frac{(A_2-A_1) \text{ sample}}{(A_2-A_1) \text{ calibrator}}$ x Calibrator concentration = mg/dl Apo A-I

2.4.4 Quantitative determination of Apolipoprotein A-11 in human sera

Principle

Turbidimetric test was used for the measurement of apolipoprotein A-II. Anti- Apo A-II antibodies were mixed with samples containing Apo A-II, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo A-II concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain). The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 ^{O}C. 300 µ/l of Reagent R1 (2-amino-2-hydroxymethyl-1, 3-propanediol buffer, 100 mmol/L, pH 8.5, macrogol) was dispensed into a cuvette and 5 µ/l of calibrator was added respectively, mixed and the absorbance (A_1) of calibrator was read at 600 nm. The sample was treated the same way as the calibrator and its absorbance reading as A_1 sample. Immediately, 100 µ/l of Reagent R2 (Anti-human apolipoprotein A-11 goat- polyclonal antibody) was dispensed into each of the same cuvette, mixed and was read again at 600 nm after 5 minutes as A_2 for calibrator and sample respectively.

Calculations

$\frac{(A_2-A_1) \text{ sample}}{(A_2-A_1) \text{ calibrator}}$ x Calibrator concentration = mg/dl Apo A-II

2.4.5 Quantitative determination of Apolipoprotein B in human sera

Principle

Turbidimetric test was used for the measurement of Apolipoprotein B. Anti- Apo B antibodies were mixed with samples containing Apo B, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo B concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).

The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 ^{O}C. 800 µ/l of Reagent R1 (Tris buffer, 20 mmol/L, PEG, pH 8.3, sodium azide, 0.95 g/L) was dispensed into a cuvette and 7 µ/l of calibrator was added respectively, mixed and the absorbance (A_1) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as A_1 sample. Immediately, 200 µ/l of Reagent R2 (Anti-human apolipoprotein B goat- polyclonal antibody, Tris buffer, 50 mmol/L, pH 7.5, sodium azide, 0.95 g/L) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 2 minutes as A_2 for calibrator and sample respectively.

Calculations

$\frac{(A_2-A_1)\ sample}{(A_2-A_1)\ calibrator}$ x Calibrator concentration = mg/dl Apo B

2.4.6 Quantitative determination of Apolipoprotein C-II in human sera

Principle

Turbidimetric test was used for the measurement of apolipoprotein C-II. Anti- Apo C-II antibodies were mixed with samples containing Apo C-II, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo C-II concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).

The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 ^{O}C. 750 µ/l of Reagent R1 (Tris buffer, 100 mmol/L, PEG 4000, pH 8.5, sodium azide, 0.95 g/L) was dispensed into a cuvette and 25 µ/l of calibrator was added respectively, mixed and the absorbance (A_1) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as A_1 sample. Immediately, 250 µ/l of Reagent R2 (Anti-human apolipoprotein C-II goat- polyclonal antibody, Tris buffer, 100 mmol/L, pH 7.2, sodium azide, 0.95 g/L) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 5 minutes as A_2 for calibrator and sample respectively.

Calculations

$\frac{(A_2-A_1)\ sample}{(A_2-A_1)\ calibrator}$ x Calibrator concentration = mg/dl Apo C-II

2.4.7 Quantitative determination of Apolipoprotein C-III in human sera

Principle

Turbidimetric test was used for the measurement of apolipoprotein C-III. Anti- Apo C-III antibodies were mixed with samples containing Apo C-III, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo C-III concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).

The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 ^{O}C. 750 µ/l of Reagent R1 (Tris buffer, 100 mmol/L, PEG 4000, pH 8.5, sodium azide, 0.95 g/L) was dispensed into a cuvette and 20 µ/l of calibrator was added respectively, mixed and the absorbance (A_1) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as A_1 sample. Immediately, 250 µ/l of Reagent R2 (Anti-human apolipoprotein C-III goat- polyclonal antibody, Tris buffer, 100 mmol/L, pH 7.2, sodium azide, 0.95 g/L) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 5 minutes as A_2 for calibrator and sample respectively.

Calculations

$\frac{(A_2-A_1)\ sample}{(A_2-A_1)\ calibrator}$ x Calibrator concentration = mg/dl Apo C-III

2.4.8 Quantitative determination of Apolipoprotein E in human sera

Principle

Turbidimetric test was used for the measurement of apolipoprotein E. Anti- Apo E antibodies were mixed with samples containing Apo E, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo E concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).

The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 ^{O}C. 750 µ/l of Reagent R1 (Tris buffer, 100 mmol/L, PEG 4000, pH 8.5, sodium azide, 0.95 g/L) as dispensed into a cuvette and 25 µ/l of calibrator was added respectively, mixed and the absorbance (A_1) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as A_1 sample. Immediately, 250 µ/l of Reagent R2 (Anti-human apolipoprotein E goat- polyclonal antibody, Tris buffer, 100 mmol/L, pH 7.2, sodium azide, 0.95 g/L) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 5 minutes as A_2 for calibrator and sample respectively.

Calculations

$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrator}}$ x Calibrator concentration = mg/dl Apo E

2.4.9 Detection of Plasmodium falciparum parasite

(a) Using Plasmodium falciparum malaria antigen rapid test device.

Principle

The principle of Plasmodium falciparum antigen detection was based on a rapid chromatographic immunoassay for the qualitative detection of circulating *Plasmodium falciparum* antigen in the whole blood. This method utilizes gold conjugate to selectively detect plasmodium antigen.

Procedure

The procedure was as described by the manufacturer (Access Bio, Incorporated, New Jersey, USA). 5 µl of whole blood was added into sample well of the test device and 60 µl of assay buffer was added also into the sample well. The analysis was read within 20 minutes. The presence of two colour bands indicate a positive result but the presence of one colour band indicates a negative result.

(b) Determination of malaria parasitaemia by Thick and thin film as described by WHO [13.]

Principle

The principle of malaria parasite density was based on the examination of stained film using the x 100 oil immersion objective. 200 and 500 leucocytes were counted in a field and at the same time, the number of malaria parasite and density present in the field were counted.

Procedure

Thick and thin films will be prepared for each participant's blood sample. The thin films were fixed with methanol and both thick and thin films were stained with Giemsa (1 in10 dilution) for 10 minutes, after which they were examined microscopically with oil immersion (x 100) objective. The malaria parasite counting was done

using the thick blood films while the thin blood films were used for species identification. Malaria parasites were counted according to the method of World Health Organisation (1995). 200 leukocytes were counted and if 10 or more parasites were identified, then the number of parasites per 200 leucocytes was recorded; but if after counting 200 leukocytes and 9 or less parasites identified then, 500 leukocytes was recorded. In each case the parasite count in relation to the leukocyte count was converted to parasite per microlitre of blood using this mathematical formular: Malaria parasite density /µl= number of parasites x 8000 Number of leukocytes Where 8000, is the average number of leukocyte per microlitre of blood, which is taken as the standard (WHO, 1995).

2.5 Data analysis

The results of the analysis were statistically analyzed using Students't-test. The analyses were performed with the use of Statistical *Package for Social Sciences* (SPSS) statistical software package, version 16.0. P <0.05 is considered statistically significant.

3. Results

The mean serum levels of Apo C-III and Apo E were significantly higher in symptomatic HIV on ART with malaria parasitaemia compared with symptomatic HIV on ART without malaria parasitaemia at p<0.05, in each case. But the mean blood CD4 count was significantly lower in symptomatic HIV on ART with malaria infection compared to symptomatic HIV on ART without malaria infection at p<0.05. The values of Apo A-I, Apo A-II, Apo B and Apo C-IIdid not differ significantly in symptomatic HIV on ART with malaria infection compared to symptomatic HIV on ART without malaria infection at P>0.05 respectively.

Also, the mean serum levels of Apo C-11 and Apo E were significantly higher in symptomatic HIV not on ART with malaria parasitaemia compared with symptomatic HIV not on ART without malaria parasitaemia at P< 0.05, in each case. But the values of Apo A-I, Apo A-II, Apo B, Apo C-III and blood CD4 count were the same in symptomatic HIV not on ART with malaria infection compared to symptomatic HIV not on ART without malaria infection at P< 0.05 respectively.

Also, the serum Apo E level was significantly higher in asymptomatic HIV subjects with malaria parasitaemia compared with asymptomatic HIV subjects without malaria parasitaemia at p<0.05. But the values of Apo A-I, Apo A-II, Apo B, Apo C-II, Apo C-III and blood CD4 count were the same in asymptomatic HIV subjects with malaria infection compared to asymptomatic HIV subjects without malaria infection at P< 0.05 respectively.

Similarly, there were no statistical significant difference in the mean values of Apo A-I, Apo A-II, Apo B, Apo C-II, Apo C-III, Apo E and blood CD4 count were the same in control subjects with malaria infection compared to control subjects without malaria infection at P< 0.05 respectively (see table 1).

Table 1: Comparison of serum levels of Apolipoprotein and Lipid profiles in symptomatic HIV infected subjects on ART, without ART, in Asymptomatic HIV infected subjects and Control subjects each with and without malaria parasitaemia (mean ± SD).

Groups	Apo A-1 (g/L)	Apo A-II (g/L)	Apo B (g/L)	Apo C- II (g/L)	Apo C- III (g/L)	Apo E (g/L)	CD4 (cells/µL)
Symptomatic HIV on ART mp^+ (n=50)	1.08±0.23	0.28 ± 0.01	1.44 ± 0.72	0.07 ± 0.05	0.04 ± 0.02	0.09±0.06	535.58 ± 368.91
Symptomatic HIV on ART mp- (n=50)	1.09 ± 0.25	0.30 ± 0.12	1.50 ± 0.65	0.06 ± 0.05	0.02 ± 0.01	0.12±0.07	706.58 ± 400.11
t-value	0.820	0.562	0.664	0.289	0.000	0.021	0.026
p-value	p>0.05	p>0.05	p>0.05	p>0.05	P<0.05	P<0.05	P<0.05
Symptomatic HIV without ART mp^+ (n=50)	0.30 ± 0.19	0.85 ± 0.20	2.50 ± 1.00	0.40 ± 0.20	0.01 ± 0.01	0.27±0.12	390.06 ± 130.75
Symptomatic HIV without ART mp^+ (n=50)	0.40 ± 0.20	0.83 ± 0.21	2.32 ± 0.72	0.60 ± 0.20	0.01 ± 0.01	0.21±0.09	359.50 ± 110.90
t-value	0.016	0.000	0.301	0.001	0.362	0.004	0.211
p-value	P<0.05	P<0.05	p>0.05	P<0.05	p>0.05	P<0.05	p>0.05
Asymptomatic HIV mp^+ (n=50)	0.61 ± 0.24	0.45 ± 0.14	1.44 ± 0.52	0.04 ± 0.01	0.02 ± 0.02	0.14±0.08	416.18 ± 130.47
Asymptomatic HIV mp- (n=50)	0.58 ± 0.14	0.43 ± 0.12	1.55 ± 0.38	0.05 ± 0.01	0.03 ± 0.02	0.08±0.03	457.92 ± 126.94
t-value	0.016	0.000	0.301	0.001	0.362	0.004	0.111
p-value	P<0.05	P<0.05	p>0.05	P<0.05	p>0.05	P<0.05	p>0.05
Control subjects mp^+ (n=50)	1.27 ± 0.06	0.25 ± 0.11	0.62 ± 0.26	0.05 ± 0.02	0.03 ± 0.02	0.05 ± 0.01	935.02 ± 151.15
Control subjects mp- (n=50)	1.28 ± 0.53	0.23 ± 0.04	0.73 ± 0.31	0.05 ± 0.02	0.03 ± 0.02	0.04 ± 0.01	946.26 ± 147.83
t-value	0.196	0.200	0.055	0.919	0.916	0.208	0.708
p-value	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05	p>0.05

4. Discussion

This study revealed that the serum levels of Apo C-III and Apo E were significantly higher in symptomatic HIV subjects on ART with malaria parasitaemia more than in those without malaria parasitaemia. Onyenekwe *et al*,[14].have reported a tripled prevalence of malaria infection in symptomatic HIV infected subjects. The CD4 count was significantly lower in symptomatic HIV subjects on ART with malaria parasitaemia more than in those without malaria parasitaemia. Ezeugwunne *et al*, [15].observed that HIV and malaria co-infections were capable of lowering the value of CD4 counts in HIV individuals. CD4 count has been depleted in HIV disease progression [16].

Also, these elevations in the levels of Apo C-III and Apo E may be due to the adverse effect of malaria infection on body organs [17].Also, reports have it that Apo C-III inhibits lipolysis of TG-rich lipoproteins [18,19].

The present study showed that the serum Apo C-II and Apo E were significantly higher in symptomatic HIV infected subjects not on ART with malaria infection compared to those without malaria infection. Ezeugwunne *et al*,[20]reported a significant higher levels of serum Apo C-II and Apo E levels in symptomatic HIV infected subjects not on ART compared with asymptomatic HIV infected subjects. Apo E is an essential apolipoprotein for the normal catabolism of TG-rich lipoprotein constituents. The increased level of Apo E may explained the wasting disease found in HIV individuals and may be due to the increased activity of TG-rich lipoprotein catabolism in their body which may be as a result of the effect of the HIV infection on them [21].

Again, the serum level of Apo E was significantly higher in asymptomatic HIV infected subjects with malaria infection compared to those without malaria infection. Ezeugwunne *et al*, [20].reported a significant higher level of serum Apo Ein asymptomatic HIV infected subjects compared to HIV seronegative control subjects. Studies suggested that an increased Apo E level was found to increase hepatic synthesis of VLDL and decrease clearance of triglycerides with lipid abnormality [22].

Also, Apo E has been reported to influence psoriasis risk, which is an autoimmune disease with chronic skin reddish patches with scales. Psoriasis is one of the risk factors for hyperlipidemia [23].Again, Apo E level has been reported to linked with neurodegenerative conditions such as multiple sclerosis and Alzheimer's disease [24].

5. Conclusion

HIV and malaria co-infections were found to cause hyper-apolipoproteinemia with respect to Apo C-II, Apo C-III,Apo E and low CD4 count in symptomatic HIV subjects with malaria parasitaemia.

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Conflict of interest

There is no conflict of interest whatever with anyone or group of persons. The studied was sponsored by Tertiary Education trust Fund (TETFUND), Nigeria.

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