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LIPID PROFILE AND WHITE BLOOD CELL COUNT AMONG HIV-POSITIVE MEN ON HIGHLY ACTIVE ANTIRETROVIRAL THERAPY: A COMPARATIVE STUDY IN OGBOMOSO, OYO STATE, NIGERIA

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ABSTRACT

Background: HIV infection and long-term antiretroviral therapy may influence lipid metabolism and hematological parameters, potentially contributing to increased cardiovascular and other chronic disease risks. However, evidence regarding these changes among men living with HIV in Nigeria remains limited.

Objective: This study evaluated lipid-profile parameters and white blood cell (WBC) count among HIV-positive men receiving highly active antiretroviral therapy (HAART) in Ogbomoso, Oyo State, Nigeria, compared with HAART-naïve HIV-positive men and HIV-negative controls.

Methods: A comparative cross-sectional study was conducted between March and October 2025 among 90 adult men comprising 50 HIV-positive participants receiving HAART, 10 HAART-naïve HIV-positive participants, and 30 HIV-negative controls. Fasting blood samples were collected for determination of WBC count, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Data were analysed using appropriate comparative statistical procedures, with statistical significance set at $p < 0.05$.

Results: WBC count did not differ significantly among controls (6,343.33 ± 427.20 cells/µL), HAART-naïve participants (7,210.00 ± 908.04 cells/µL), and HAART-treated participants (6,696.00 ± 363.00 cells/µL; $p = 0.625$). In contrast, TG concentrations differed significantly ($p < 0.001$), with the highest value among HAART-treated participants (161.58 ± 5.12 mg/dL). HDL-C also differed significantly ($p < 0.001$), while LDL-C showed a significant between-group difference ($p = 0.005$), with the highest LDL-C among HAART-naïve participants.

Conclusion: HIV and HAART status were associated with significant differences in individual lipid parameters but not total WBC count. Routine lipid monitoring should therefore form an integral component of long-term HIV care to facilitate early recognition and management of dyslipidemia and associated cardiovascular.

Keywords: Human immunodeficiency virus, Highly active antiretroviral therapy, Dyslipidemia, White blood cell, Oyo state.

1 INTRODUCTION

Human immunodeficiency virus (HIV) infection remains an important public health concern despite substantial advances in antiretroviral therapy (ART). The introduction and widespread use of highly active antiretroviral therapy (HAART) have markedly improved viral suppression, immune recovery and survival among people living with HIV. However, effective treatment has also changed the clinical profile of HIV infection from a predominantly fatal disease to a chronic condition in which metabolic and haematological complications may persist or emerge during long-term care. Consequently, attention has increasingly shifted from viral suppression alone to the broader metabolic and systemic consequences of HIV infection and its treatment [1-4]. Lipid abnormalities are among the important metabolic complications reported in people living with HIV. HIV infection itself may disturb lipid metabolism through persistent immune activation, inflammation and alterations in lipid handling, while antiretroviral drugs may further modify circulating lipid concentrations [4,5]. The resulting dyslipidaemia may include elevated triglycerides, total cholesterol and low-density lipoprotein cholesterol (LDL-C), together with alterations in high-density lipoprotein cholesterol (HDL-C). These abnormalities are clinically important because they may contribute to the increased burden of cardiovascular disease observed among people living with HIV [5-8]. The relationship between antiretroviral therapy and lipid metabolism is, however, complex. Different antiretroviral classes and individual drug combinations may have distinct effects on lipid concentrations. Contemporary studies have demonstrated differences in lipid responses according to treatment regimen, particularly following the introduction of integrase strand transfer inhibitors and newer nucleoside reverse transcriptase inhibitor formulations [9-13]. Tenofovir alafenamide-containing regimens, for example, have been associated with less favourable lipid changes in some populations, whereas other contemporary antiretroviral regimens may exert relatively neutral or even favourable effects on lipid parameters [12,13]. Thus, the interpretation of dyslipidaemia among treated patients requires consideration of both the underlying effects of HIV infection and the metabolic consequences of treatment. Evidence from sub-Saharan Africa indicates that dyslipidaemia remains common among people receiving ART. Studies from Uganda and Ethiopia have reported substantial burdens of dyslipidaemia among adults receiving contemporary antiretroviral regimens, while systematic evidence suggests that lipid abnormalities remain prevalent among ART-treated populations [14,15]. Nigerian studies have similarly documented alterations in lipid profiles among people living with HIV, including differences between treatment-naïve and treatment-experienced individuals [16-19]. These findings suggest that metabolic monitoring should form an important component of long-term HIV care, particularly in settings where patients are increasingly surviving for longer periods on ART. In addition to metabolic disturbances, HIV infection may influence haematological parameters. Abnormalities in white blood cell profiles may arise from direct and indirect effects of HIV infection, immune dysregulation, opportunistic infections, nutritional factors and drug-related effects. Leukopenia, neutropenia and other alterations in circulating leukocyte populations have been documented among people living with HIV, although the magnitude and direction of these changes may vary according to disease stage, treatment status and the specific antiretroviral regimen [20-23].

The restoration of immune function following ART may improve some haematological abnormalities, although persistent alterations may occur in some patients despite treatment [21-24]. Recent Nigerian studies have reported differences in haematological parameters among HIV-positive individuals receiving different antiretroviral regimens, including tenofovir-, zidovudine- and dolutegravir-based therapies [24-26]. Similarly, studies from other African settings have demonstrated changes in white blood cell and related haematological parameters following initiation of ART [27-29]. Nevertheless, findings across populations have not been entirely consistent, highlighting the importance of generating local evidence on haematological responses to long-term ART. Importantly, lipid metabolism and haematological status may be influenced by overlapping biological processes associated with chronic HIV infection, immune activation and antiretroviral exposure. Experimental evidence has linked altered immune-cell function with lipid abnormalities, suggesting a biological interface between immune dysregulation and metabolic disturbances in HIV infection [4]. However, much of the available literature has examined lipid abnormalities or haematological changes independently.

Furthermore, relatively limited evidence is available from smaller urban centres in Nigeria, particularly Ogbomoso in Oyo State, where HIV care is delivered through multiple public and teaching-hospital facilities. The present study therefore assessed the lipid profile and white blood cell count among HIV-positive men receiving HAART in Ogbomoso, Oyo State, Nigeria, using HIV-positive HAART-naïve men and apparently healthy HIV-negative men as comparison groups. The comparative design provides an opportunity to distinguish findings observed among individuals with HIV infection before treatment from those observed among patients receiving established antiretroviral therapy. Such evidence may contribute to improved understanding of metabolic and haematological patterns among men receiving long-term HIV care and may support the incorporation of appropriate laboratory monitoring into routine clinical management.

2 MATERIAL AND METHODS

2.1 Study design and setting

A comparative cross-sectional study was conducted to evaluate the lipid profile and white blood cell (WBC) count among HIV-positive men receiving highly active antiretroviral therapy (HAART), HIV-positive HAART-naïve men, and apparently healthy HIV-negative men in Ogbomoso, Oyo State, Nigeria. The study was conducted between March and October 2025 at selected healthcare facilities providing comprehensive HIV care services in Ogbomoso, including Oyo State General Hospital, Bowen University Teaching Hospital, and Ladoke Akintola University of Technology Teaching Hospital.

Ogbomoso is an important urban centre in Oyo State and serves as a healthcare hub for surrounding communities in Oyo and neighbouring states. The participating facilities provide HIV counselling and testing, antiretroviral treatment, clinical follow-up and laboratory monitoring services. The study setting therefore provided access to both treatment-experienced and newly diagnosed HIV-positive men, as well as apparently healthy HIV-negative individuals from the same geographical environment. A three-group comparative design was adopted to permit assessment of differences associated with HIV infection and antiretroviral treatment status. The inclusion of a HAART-naïve HIV-positive group was particularly important because it provided an untreated HIV reference group against which findings among individuals receiving established HAART could be compared. The HIV-negative control group provided a reference for comparison with individuals without documented HIV infection.

2.2 Study population and participant groups

The study population comprised adult men aged 18–65 years residing in Ogbomoso. Participants were categorized into three groups according to HIV status and antiretroviral treatment history. Group 1 comprised 40 HIV-positive men receiving HAART for at least six months. Group 2 comprised 10 HIV-positive men who were HAART-naïve at the time of recruitment and had not commenced antiretroviral treatment. Group 3 comprised 30 apparently healthy HIV-negative men who served as controls. The total sample size was therefore 80 participants.

HIV-positive participants were recruited from the participating HIV treatment centres, while HIV-negative controls were recruited from the same geographical setting. The use of participants from the same environment was intended to minimize substantial differences in background demographic and environmental characteristics that could influence lipid or haematological parameters.

2.3 Eligibility criteria

Participants were eligible if they were male, aged 18–65 years and had resided in Ogbomoso for at least 12 months. HIV-positive participants were required to have documented HIV infection. Participants assigned to the HAART group must have received antiretroviral therapy continuously for at least six months, whereas HAART-naïve participants had not commenced antiretroviral treatment at the time of enrolment. Apparently healthy HIV-negative men within the specified age range were eligible for inclusion in the control group. HIV-negative status was confirmed using the appropriate HIV testing algorithm before enrolment.

Participants were excluded if they had a documented history of chronic liver disease, chronic kidney disease, hepatitis B or hepatitis C infection, current hormonal or steroid therapy, current cigarette smoking, and heavy alcohol consumption, use of mineral or antioxidant supplements, acute illness, or active infection at the time of recruitment. These criteria were applied to reduce potential confounding from conditions or exposures that could independently influence lipid metabolism or white blood cell counts.

2.4 Sample size and sampling procedure

The sample size was determined using Cochran's formula and subsequently adjusted to obtain an adequate number of participants for the intended comparative analysis. The final study population consisted of 90 men distributed across the three study groups as follows: 50 HIV-positive men receiving HAART, 10 HAART-naïve HIV-positive men, and 30 HIV-negative controls.

A stratified sampling approach was used for recruitment across the participating healthcare facilities. Eligible HIV-positive participants were identified during routine clinic attendance and screened according to the predefined inclusion and exclusion criteria. Newly diagnosed HIV-positive men who had not commenced treatment were recruited into the HAART-naïve group. HIV-positive participants with documented treatment exposure of at least six months were recruited into the HAART group. Apparently healthy HIV-negative men from the same geographical area were recruited as controls.

The recruitment process was conducted throughout the study period, and all participants underwent the same pre-analytical procedures for blood collection and laboratory assessment.

2.5 Ethical considerations

Ethical approval was obtained from the Oyo State Ministry of Health Ethical Review Committee, Nigeria (Approval No. NHREC/OYOSHRIEC/10/11/22). The study was conducted in accordance with internationally accepted principles for research involving human participants.

Before participation, eligible individuals were informed about the objectives and procedures of the study, the type and volume of biological specimens required, potential risks and benefits, and their right to decline participation or withdraw at any stage without affecting their access to healthcare services. Written informed consent was obtained from each participant before questionnaire administration and blood collection. Confidentiality was maintained throughout the study. Participants were assigned unique identification codes, and names were not used on laboratory specimens or analytical datasets. Access to study information was restricted to authorized members of the research team.

2.6 Collection of socio-demographic and clinical information

Socio-demographic and relevant clinical information was collected using a structured interviewer-administered questionnaire. Information obtained included age, marital status, educational attainment, occupation, income and other relevant demographic characteristics.

For HIV-positive participants, clinical information included duration of HIV diagnosis, treatment status, duration of HAART and current antiretroviral regimen where available. Information was obtained from participant interviews and clinical records as appropriate.

Anthropometric measurements were also obtained using standardized procedures. Body weight was measured using a calibrated weighing scale, while height was measured using a portable stadiometer. Measurements were obtained by trained research personnel using consistent procedures.

2.7 Blood sample collection and processing

Participants were instructed to undergo an overnight fast of approximately 10–12 hours before venous blood collection. Fasting was used to minimize the acute influence of food intake on circulating lipid concentrations, particularly triglycerides. Approximately 10 mL of venous blood was collected from each participant under aseptic conditions. Blood intended for haematological analysis was dispensed into an ethylenediaminetetraacetic acid (EDTA) tube, while blood intended for lipid analysis was collected into a lithium-heparin tube. The specimens were labelled using unique participant identification codes and transported promptly to the designated laboratory for processing. EDTA-anticoagulated specimens were analysed within six hours of collection for haematological parameters. Specimens intended for lipid analysis were processed within 12 hours of collection.

2.8 Determination of white blood cell count

White blood cell count was determined as part of the full blood count performed on EDTA-anticoagulated whole blood. The analysis was performed using the routine automated haematology procedure established in the participating laboratory. The WBC count was reported in cells/μL.

All samples were inspected for appropriate identification, adequate volume and evidence of clotting before analysis. Samples that did not meet the laboratory's acceptance criteria were not included until an acceptable specimen was obtained.

Because WBC count may be influenced by acute infection and other inflammatory conditions, participants with known acute illness or active infection were excluded according to the study protocol. This approach was intended to improve the comparability of WBC measurements across the three study groups.

2.9 Determination of lipid profile

Fasting lipid profile was determined using lithium-heparin plasma obtained from the collected blood samples. The parameters assessed were TC, TG, HDL-C and LDL-C. Commercial Randox reagent kits were used according to the manufacturer's instructions and standard laboratory procedures. Total cholesterol was measured using the enzymatic cholesterol oxidase-peroxidase (CHOD-PAP) method. In this method, cholesterol undergoes enzymatic oxidation and subsequent reactions producing a coloured compound, the intensity of which is proportional to the cholesterol concentration and measured spectrophotometrically.

Triglycerides were determined using the enzymatic glycerol phosphate oxidase-peroxidase (GPO-PAP) method. The assay measures the products generated through enzymatic hydrolysis and oxidation of triglycerides, with the resulting colour intensity being proportional to the triglyceride concentration. HDL-C was determined following precipitation of non-HDL lipoproteins, after which cholesterol in the HDL-containing supernatant was measured spectrophotometrically.

LDL-C was calculated using the Friedewald equation where triglyceride concentrations were below 400 mg/dL:

LDL-C = TC − HDL-C − TG/5

All lipid concentrations were expressed in mg/dL. The same analytical procedures and reagent systems were used for samples from all study groups to minimize inter-assay variability.

2.10 Laboratory quality assurance

Quality assurance was maintained throughout the pre-analytical, analytical and post-analytical stages of the study. Blood collection, specimen identification, transportation and processing were standardized for all participants. Reagents were checked for appropriate storage conditions and expiration status before use. Laboratory equipment was calibrated according to established laboratory procedures, and internal quality-control materials were analysed alongside participant specimens during analytical runs. Appropriate control procedures were applied to monitor analytical performance and identify potential analytical errors. External quality assurance/proficiency testing was also undertaken through the relevant reference laboratory arrangements. Where applicable, samples were analysed in duplicate to assess analytical reproducibility. Laboratory results were reviewed for completeness and consistency before entry into the study database.

2.11 Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics. Continuous variables were summarized as mean ± standard error of the mean or mean ± standard deviation, as appropriate, while categorical variables were presented as frequencies and percentages.

The primary comparative analyses assessed differences in WBC count, TC, TG, HDL-C and LDL-C among the three study groups: HIV-negative controls, HAART-naïve HIV-positive men and HIV-positive men receiving HAART. Because the study involved three independent groups, one-way analysis of variance (ANOVA) was used to evaluate overall differences in normally distributed continuous variables. Prior to analysis, the distributional assumptions of the data were assessed. Where the assumptions of parametric analysis were not satisfied, the appropriate non-parametric alternative was considered. Categorical variables were compared using the appropriate chi-square or Fisher's exact test depending on expected cell frequencies. All statistical tests were two-tailed, and statistical significance was established at $p < 0.05$.

2.12 Data management

Questionnaires and laboratory records were checked for completeness before data entry. Each participant was assigned a unique study identification number, and data were entered into a computerized database for statistical analysis. Data cleaning procedures were undertaken to identify inconsistencies, missing values and potential data-entry errors. Only de-identified data were used for statistical analysis. Electronic records were stored securely, and access was restricted to members of the research team.

3 RESULTS AND DISCUSSION

3.1 Socio-demographic characteristics of the study participants

The study included 90 male participants comprising 30 HIV-negative controls, 10 HIV-positive HAART-naïve participants and 50 HIV-positive participants receiving HAART. The socio-demographic characteristics of the participants are presented in Table 1.

There was no statistically significant difference in age distribution among the three groups ($\chi^2 = 3.515$, $p = 0.742$). Similarly, marital status ($\chi^2 = 4.686$, $p = 0.585$), number of children ($\chi^2 = 11.157$, $p = 0.084$) and educational status ($\chi^2 = 5.102$, $p = 0.277$) did not differ significantly between the groups. However, occupation was significantly associated with study group ($\chi^2 = 6.072$, $p = 0.048$), while monthly income also differed significantly across the groups ($\chi^2 = 16.889$, $p < 0.001$).

Table 1: Socio-demographic characteristics of the study participants

Variable	Category	Control n (%)	HAART-naïve n (%)	HIV on HAART n (%)	χ^2	df	p-value
Age (years)	≤30	6 (6.7)	2 (2.2)	19 (21.1)	3.515	6	0.742
	31–40	7 (7.8)	2 (2.2)	9 (10.0)			
	41–50	11 (12.2)	4 (4.4)	14 (15.6)			
	≥51	6 (6.7)	2 (2.2)	8 (8.9)			
Marital status	Single	5 (5.6)	2 (2.2)	8 (8.9)	4.686	6	0.585
	Married	20 (22.2)	8 (8.9)	34 (37.8)			
	Divorced	5 (5.6)	0 (0.0)	5 (5.6)			
	Widowed	0 (0.0)	0 (0.0)	3 (3.3)			
Occupation	Employed	30 (33.3)	10 (11.1)	43 (47.8)	6.072	2	0.048
	Unemployed	0 (0.0)	0 (0.0)	7 (7.8)			
Educational status	No formal education	0 (0.0)	1 (1.1)	3 (3.3)	5.102	4	0.277
	Primary education	4 (4.4)	0 (0.0)	10 (11.1)			
	Post-primary education†	26 (28.9)	9 (10.0)	37 (41.1)			

3.2 Clinical, anthropometric characteristics and WBC count

The clinical and anthropometric characteristics of the participants are presented in Table 2. Mean age was 40.97 ± 2.01 years among controls, 42.40 ± 3.42 years among HAART-naïve participants and 37.80 ± 1.70 years among participants receiving HAART, with no significant difference among the groups (p = 0.340). Similarly, mean body weight, height and BMI did not differ significantly among the groups. Mean body weight was 66.46 ± 1.53 kg, 64.80 ± 4.91 kg and 66.50 ± 1.86 kg among controls, HAART-naïve participants and HAART-treated participants, respectively (p = 0.917). Mean BMI was 25.50 ± 2.55 kg/m^2 in controls, 30.15 ± 7.64 kg/m^2 in HAART-naïve participants and 27.37 ± 2.60 kg/m^2 among participants receiving HAART (p = 0.760).

The mean WBC count was highest among the HAART-naïve group (7,210.00 ± 908.04 cells/μL), followed by the HAART-treated group (6,696.00 ± 363.00 cells/μL) and controls (6,343.33 ± 427.20 cells/μL). However, the difference was not statistically significant (p = 0.625).

Table 2: Anthropometric Characteristics and *white blood cell count (WBC)* of Study Groups

Parameter	Control (n=30)	HAART-naïve (n=10)	HIV on HAART (n=50)	p-value
Age (years)	40.97 ± 2.01	42.40 ± 3.42	37.80 ± 1.70	0.340
Weight (kg)	66.46 ± 1.53	64.80 ± 4.91	66.50 ± 1.86	0.917
Height (m)	167.23 ± 3.23	160.20 ± 9.28	165.20 ± 3.27	0.687
BMI (kg/m^2)	25.50 ± 2.55	30.15 ± 7.64	27.37 ± 2.60	0.760
WBC (cells/μL)	6,343.33 ± 427.20	7,210.00 ± 908.04	6,696.00 ± 363.00	0.625

3.3 Haematological parameters among the study groups

The haematological findings are presented in Table 3. Most erythrocyte and platelet parameters were comparable across the three groups. Mean haemoglobin concentration was 12.83 ± 0.53 g/dL among controls, 12.40 ± 0.94 g/dL among HAART-naïve participants and 12.70 ± 0.38 g/dL among HAART-treated participants (p = 0.913). Similarly, RBC count, MCV, MCH, MCHC and platelet count showed no statistically significant differences among the groups.

In contrast, significant differences were observed in neutrophil and lymphocyte percentages. Neutrophils were 62.63 ± 0.95% among controls, 44.10 ± 2.30% among HAART-naïve participants and 61.54 ± 0.85% among HAART-treated participants ($p < 0.001$). Lymphocyte percentages were 32.23 ± 0.85%, 49.80 ± 2.11% and 33.42 ± 0.79%, respectively ($p < 0.001$). Monocytes, eosinophils and basophils did not differ significantly.

Table 3: Haematological parameters of the study participants

Parameter	Control	HAART-naïve	HIV on HAART	p-value
Haemoglobin (g/dL)	12.83 ± 0.53	12.40 ± 0.94	12.70 ± 0.38	0.913
RBC ($\times 10^{12}$/L)	4.29 ± 0.18	4.15 ± 0.31	4.24 ± 0.13	0.920
MCV (fL)	90.02 ± 0.16	89.82 ± 0.28	89.97 ± 0.11	0.809
MCH (pg)	30.04 ± 0.03	30.03 ± 0.04	30.04 ± 0.02	0.954
MCHC (g/dL)	33.30 ± 0.01	33.32 ± 0.02	33.31 ± 0.01	0.822
Neutrophils (%)	62.63 ± 0.95	44.10 ± 2.30	61.54 ± 0.85	<0.001
Lymphocytes (%)	32.23 ± 0.85	49.80 ± 2.11	33.42 ± 0.79	<0.001
Monocytes (%)	2.40 ± 0.10	2.40 ± 0.16	2.36 ± 0.07	0.935
Eosinophils (%)	1.77 ± 0.10	1.90 ± 0.18	1.82 ± 0.09	0.823
Basophils (%)	0.90 ± 0.06	0.90 ± 0.10	0.88 ± 0.05	0.957
Platelets ($\times 10^{9}$/L)	208.57 ± 5.24	203.10 ± 13.49	207.00 ± 5.09	0.910

3.4 Fasting lipid-profile parameters among the study groups

The fasting lipid-profile results are presented in Table 4. Mean total cholesterol (TC) concentration was 210.33 ± 5.28 mg/dL among controls, 219.60 ± 8.88 mg/dL among HAART-naïve participants and 213.24 ± 4.83 mg/dL among HIV-positive participants receiving HAART. The difference was not statistically significant ($p = 0.727$).

In contrast, significant differences were observed in HDL-C, TG and LDL-C. Mean HDL-C was lowest among HAART-naïve participants (42.20 ± 5.17 mg/dL), compared with 63.36 ± 2.84 mg/dL among controls and 65.38 ± 1.11 mg/dL among HAART-treated participants ($p < 0.001$). Mean TG concentration was highest among participants receiving HAART (161.58 ± 5.12 mg/dL), followed by HAART-naïve participants (129.73 ± 6.36 mg/dL) and controls (114.21 ± 3.72 mg/dL), with a statistically significant difference ($p < 0.001$).

LDL-C concentration was highest among HAART-naïve participants (151.45 ± 10.04 mg/dL), followed by controls (128.36 ± 4.78 mg/dL), and lowest among HAART-treated participants (115.54 ± 4.98 mg/dL), with a significant overall difference ($p = 0.005$).

Table 4: Fasting Lipid Profile of the study participants

Lipid parameter	Control (n=30)	HAART-naïve (n=10)	HIV on HAART (n=50)	P value
Total cholesterol (mg/dL)	210.33 ± 5.28	219.60 ± 8.88	213.24 ± 4.83	0.727
HDL-C (mg/dL)	63.36 ± 2.84	42.20 ± 5.17	65.38 ± 1.11	<0.001
Triglycerides (mg/dL)	114.21 ± 3.72	129.73 ± 6.36	161.58 ± 5.12	<0.001
LDL-C (mg/dL)	128.36 ± 4.78	151.45 ± 10.04	115.54 ± 4.98	0.005

3.5 Distribution of BMI categories among study groups

The distribution of participants according to BMI category is presented in Table 5. Normal BMI was the predominant category overall, particularly among HIV-positive participants receiving HAART, where 35 (38.9%) participants were classified as having normal BMI, compared with 22 (24.4%) controls and 7 (7.8%) HAART-naïve participants.

Overweight participants comprised 7 (7.8%) controls, 1 (1.1%) HAART-naïve participant and 8 (8.9%) participants receiving HAART. Obesity was observed in 1 (1.1%) control, 2 (2.2%) HAART-naïve participants and 7 (7.8%) HAART-

treated participants. The distribution of BMI categories did not differ significantly among the three groups ($\chi^2 = 3.701$, df = 4, p = 0.448).

Table 5: BMI classifications among the study Participants.

BMI category	Control n (%)	HAART-naïve n (%)	HIV on HAART n (%)	χ^2	df	p-value
Normal	22 (24.4)	7 (7.8)	35 (38.9)	3.701	4	0.448
Overweight	7 (7.8)	1 (1.1)	8 (8.9)			
Obese	1 (1.1)	2 (2.2)	7 (7.8)			

4 DISCUSSION

The present study examined white blood cell (WBC) count and lipid-profile parameters among HIV-positive men receiving highly active antiretroviral therapy (HAART) in Ogbomoso, Oyo State, Nigeria, using HAART-naïve HIV-positive men and HIV-negative men as comparison groups. The principal finding was a differential response of the haematological and metabolic parameters investigated. Total WBC count did not differ significantly among the three groups, whereas significant differences were observed in triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). This pattern suggests that, in this study population, lipid metabolism was more clearly differentiated by HIV and treatment status than total circulating leukocyte count. The finding is clinically relevant because the success of antiretroviral therapy has transformed HIV into a chronic condition in which long-term metabolic and cardiovascular health has become an increasingly important component of comprehensive care. Despite the numerically higher WBC count in the HAART-naïve group, the overall difference was not statistically significant (p = 0.625). This finding indicates that total circulating leukocyte counts were broadly comparable across the study groups. Although HIV infection is associated with immune activation, chronic inflammation and several haematological abnormalities, these effects do not necessarily translate into persistent changes in total WBC count in clinically stable individuals. Previous studies from sub-Saharan Africa have reported variable changes in WBC and other haematological indices according to HIV disease status, treatment exposure and clinical characteristics [20-29]. The absence of a significant difference in the present study may therefore reflect preservation of overall leukocyte numbers despite underlying alterations in immune-cell composition or function.

Importantly, total WBC count is a relatively nonspecific haematological measure. Changes in lymphocyte, neutrophil or monocyte populations may occur without producing a significant change in the aggregate WBC concentration. Consequently, the present finding should not be interpreted as evidence that HIV infection or HAART has no immunological effect. Rather, it suggests that total leukocyte concentration alone may have limited sensitivity for identifying treatment-related or HIV-associated immunohaematological alterations in a clinically stable outpatient population. The exclusion of participants with acute illness or active infection may also have contributed to the similarity observed between groups because such conditions can substantially influence leukocyte concentrations. Furthermore, effective ART-mediated suppression of viral replication and subsequent immune restoration may contribute to preservation of peripheral blood-cell counts among treatment-experienced individuals [20-26].

The relatively small number of HAART-naïve participants (n = 10) warrants particular consideration. Although their mean WBC count was numerically higher than those of both treated participants and controls, the considerable variability within this group may have reduced the ability to detect a statistically significant difference. Thus, the non-significant result should be interpreted as an absence of demonstrated statistical difference rather than proof that the groups are biologically equivalent. Larger studies incorporating differential leukocyte counts, CD4 cell counts and other markers of immune activation may provide a more comprehensive assessment of the haematological consequences of HIV and antiretroviral treatment.

In contrast to WBC count, marked differences were observed in lipid metabolism. Mean TG concentration increased from 114.21 ± 3.72 mg/dL in HIV-negative controls to 129.73 ± 6.36 mg/dL in HAART-naïve participants and 161.58 ± 5.12 mg/dL in HAART-treated participants, with a highly significant overall difference (p < 0.001). The substantially higher TG concentration among HAART-treated men is consistent with evidence that dyslipidaemia remains an important metabolic concern among people living with HIV receiving ART [14,15]. Similar alterations have been documented in Nigerian and other sub-Saharan African populations, although the magnitude and pattern of dyslipidaemia vary across studies [16-19].

The elevated TG concentration observed among treated participants may reflect the complex interaction between HIV infection, chronic inflammation and antiretroviral exposure. HIV-associated immune activation can alter hepatic lipid synthesis and peripheral lipid handling, while individual antiretroviral agents may further modify lipoprotein

metabolism [4,5,9-13]. Importantly, ART should not be regarded as having a uniform metabolic effect. Lipid responses vary according to drug class, specific regimen, duration of exposure, baseline metabolic status and patient characteristics [9-13]. Therefore, although the cross-sectional design prevents attribution of the observed triglyceride elevation directly to HAART, the marked difference between treated participants and both comparison groups identifies hypertriglyceridaemia as an important metabolic feature requiring clinical attention in this population.

The clinical relevance of this finding extends beyond the lipid measurement itself. Elevated TG may contribute to an adverse cardiovascular and metabolic profile, particularly when accompanied by other lipid abnormalities. As individuals receiving effective ART increasingly survive into older age, cardiovascular and metabolic diseases become increasingly relevant components of HIV care. Periodic lipid assessment may therefore facilitate early recognition of dyslipidaemia and provide an opportunity for appropriate dietary counselling, physical-activity interventions and pharmacological treatment when clinically indicated.

HDL-C demonstrated an equally important but different pattern. Mean HDL-C was 63.36 ± 2.84 mg/dL among controls, fell to 42.20 ± 5.17 mg/dL among HAART-naïve participants and increased to 65.38 ± 1.11 mg/dL among those receiving HAART, with a significant overall difference ($p < 0.001$). The markedly lower HDL-C among untreated HIV-positive participants is compatible with the adverse metabolic effects associated with active HIV infection. Persistent inflammation and immune activation may interfere with lipid transport and lipoprotein metabolism, while alterations in HDL may have implications beyond cholesterol transport because HDL is also involved in anti-inflammatory and antioxidant processes [4,5].

The comparatively high HDL-C concentration among HAART-treated participants is particularly noteworthy. It was higher than that observed among HAART-naïve participants and marginally higher than that of the HIV-negative controls. This finding illustrates the heterogeneous effects of ART on individual lipid fractions and cautions against characterizing HAART as uniformly detrimental to lipid metabolism. Contemporary antiretroviral regimens differ considerably in their metabolic effects, and some may exert relatively favourable effects on particular lipid fractions [9-13]. The observed HDL-C pattern may therefore reflect treatment-related effects, improved health following viral suppression, characteristics of the antiretroviral regimens used, duration of therapy, or differences in dietary and lifestyle factors. Because these factors were not examined longitudinally, the present finding should be interpreted as an observed association rather than evidence of a direct treatment effect.

LDL-C also differed significantly among the groups ($p = 0.005$). The highest mean concentration occurred among HAART-naïve participants (151.45 ± 10.04 mg/dL), compared with 128.36 ± 4.78 mg/dL among controls and 115.54 ± 4.98 mg/dL among HAART-treated participants. The higher LDL-C concentration among untreated HIV-positive men suggests that active HIV infection may be associated with adverse alterations in lipid metabolism, while effective treatment may modify this pattern. Previous evidence indicates that HIV infection and ART can exert different, and sometimes opposing, effects on individual lipid fractions [9-13]. Nigerian studies have likewise reported variation in lipid profiles according to HIV and treatment status [16-19].

The lower LDL-C observed among treated participants may reflect improved metabolic status following effective HIV treatment or the relatively favourable lipid effects of the regimens used in this population. Nevertheless, this interpretation should remain cautious. The cross-sectional design cannot establish that HAART caused the lower LDL-C concentration, and the groups may have differed in duration of HIV infection, nutritional status, body composition, physical activity and other cardiometabolic characteristics. The small HAART-naïve group also limits the precision of estimates involving untreated participants. Nevertheless, the significant difference in LDL-C highlights the value of assessing individual lipid fractions when evaluating cardiovascular risk among people living with HIV.

Taken together, the lipid findings demonstrate that HIV infection and treatment status were associated with distinct rather than uniformly adverse lipid patterns. HAART-treated participants had the highest TG concentration, whereas HAART-naïve participants had the lowest HDL-C and highest LDL-C concentrations. This heterogeneity is consistent with the multifactorial nature of HIV-associated dyslipidaemia, in which viral infection, persistent inflammation, immune activation, antiretroviral exposure, specific drug combinations, treatment duration and lifestyle factors may interact to produce different lipid phenotypes [4,9-15]. The findings also demonstrate why individual lipid fractions should be interpreted collectively rather than relying on a single parameter. A favourable HDL-C or LDL-C concentration does not necessarily exclude an adverse metabolic profile when TG or another lipid fraction is elevated.

The findings have important implications for HIV care in Nigeria. The local evidence generated from Ogbomoso contributes to the relatively limited literature describing the interaction between HIV treatment status and lipid metabolism among Nigerian men. Differences between this study and reports from other populations may reflect variations in antiretroviral regimens, duration of treatment, dietary patterns, physical activity, body composition, genetic background and socioeconomic circumstances. These differences reinforce the importance of generating

population-specific evidence rather than assuming that metabolic responses to HIV and ART are uniform across settings.

From a clinical perspective, the findings support the integration of metabolic monitoring into routine HIV care. While HAART remains essential for viral suppression, immune preservation and improved survival, long-term management should also address potentially modifiable cardiovascular risk factors. The significantly higher TG concentration among treated participants warrants particular attention to periodic lipid assessment and appropriate risk-reduction strategies. Conversely, the absence of a significant difference in total WBC count suggests that WBC measurement alone may have limited value for distinguishing stable treated HIV-positive men from untreated or HIV-negative individuals. Differential leukocyte counts and more specific immunological markers may provide greater insight into treatment-related haematological changes.

5 CONCLUSION

This study demonstrates significant differences in selected lipid-profile parameters according to HIV and HAART status among men in Ogbomoso, Nigeria. HAART-treated participants had significantly higher triglyceride concentrations, whereas HAART-naïve participants exhibited higher LDL-C and lower HDL-C concentrations. In contrast, WBC count did not differ significantly among the three groups. These findings highlight the heterogeneous metabolic effects associated with HIV infection and antiretroviral treatment and emphasize the importance of routine lipid-profile monitoring as part of comprehensive long-term HIV care. Early identification and management of dyslipidaemia may contribute to reducing the growing cardiovascular risk among people living with HIV.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Ethical approval was obtained from the Oyo State Ministry of Health Ethical Review Committee, (Approval No. NHREC/OYOSHRIEC/10/11/22). Furthermore, study participants were appropriately educated on the significance of the research to get their optimal support.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

REFERENCES

[1] Buru, A. S. (2020). Assessment of the effect of anti-retroviral therapy on haematological parameters in HIV positive individuals in Zaria. Journal of AIDS and HIV Research, 12(2), 17–23. https://doi.org/10.5897/JAHR2018.0467

[2] Jegede, F. E., Oyeyi, T. I., Abdulrahman, S. A., & Mbah, H. A. (2020). Malaria parasite density as a predictor of hematological parameter changes among HIV infected adults attending two antiretroviral treatment clinics in Kano, Northwest Nigeria. Journal of Tropical Medicine, 2020, 3210585. https://doi.org/10.1155/2020/3210585

[3] Seedat, F., Raal, F., & Martinson, N. (2020). Lipid and lipoprotein levels in HIV-infected adults with sepsis compared to healthy HIV-infected controls. African Journal of Infectious Diseases, 14(2), 1–9. https://doi.org/10.21010/ajid.v14i2.1

[4] Bowman, E. R., Cameron, C. M., Richardson, B., et al. (2020). Macrophage maturation from blood monocytes is altered in people with HIV, and is linked to serum lipid profiles and activation indices: A model for studying atherogenic mechanisms. PLOS Pathogens, 16(10), e1008869. https://doi.org/10.1371/journal.ppat.1008869

[5] Olaleye, M. T., Ademuyiwa, A. I., et al. (2022). Impact of highly active antiretroviral drug therapy (HAART) on biochemical, hematologic, atherogenic and anthropometric profiles of human immunodeficiency virus patients at a tertiary hospital in Owo, Nigeria. Bulletin of the National Research Centre, 46, 216. https://doi.org/10.1186/s42269-022-00953-3

[6] Aghimien, E. C., & Okhimamhe, F. A. (2021). Differences in lipid profile findings in HAART naive vs. HAART treated HIV positive patients. West Journal of Medicine and Biomedical Sciences, 2(2). https://doi.org/10.46912/wjmbs.47

[7] Nnamdi, D. B., Meludu, S. C., Ogbodo, E. C., et al. (2022). Pattern of lipid profile levels in HIV serodiscordant couples in Jos Plateau State, Nigeria. Asian Journal of Medicine and Health, 20(10), 126–135. https://doi.org/10.9734/ajmah/2022/v20i1030513

[8] Okunorobo, M. N., Nnamah, N. K., Ude, U. A., & Ude, E. A. (2023). Lipids and apolipoproteins C-III and E among treatment-naïve and treatment-experienced persons with HIV in Nigeria. African Journal of Laboratory Medicine, 12(1). https://doi.org/10.4102/ajlm.v12i1.2018

[9] Ukodei, F. U., Nnamah, N. K., Onuegbu, A. J., Onah, C. E., Ezenwa, O. B., et al. (2023). Evaluation of thyroid and lipid profile of HIV patients seen in a faith-based health facility in Anambra State, Nigeria. Annals of Clinical Endocrinology and Metabolism, 7, 014–020. https://doi.org/10.29328/journal.acem.1001028

[10] Echefu, S. N., Udosen, J. E., Akwiwu, E. C., & Akpotuzor, J. O. (2023). Effect of dolutegravir regimen against other regimens on some hematological parameters, CD4 count and viral load of people living with HIV infection in South Eastern Nigeria. Medicine, 102(47), e35910. https://doi.org/10.1097/MD.0000000000035910

[11] Umeozulu, C., Ibeh, N., & Obafemi, I. (2024). Comparison of viral load, CD4 and hematological parameters amongst HIV patients on tenofovir and zidovudine-based ARV therapy in Nasarawa State, Nigeria. International Journal of HIV/AIDS Prevention, Education and Behavioural Science, 10(1), 1–17. https://doi.org/10.11648/j.ijhpebs.20241001.11

[12] Ngwu, A. M., & Eneh, J. C. (2024). Haematological changes in HIV subjects receiving antiretroviral treatment in Enugu State University of Science and Technology Teaching Hospital, Enugu, Nigeria. African Journal of Health Sciences, 35(4), 417–425.

[13] Damtie, S., Workineh, L., Kiros, T., Eyayu, T., & Tiruneh, T. (2021). Hematological abnormalities of adult HIV-infected patients before and after initiation of highly active antiretroviral treatment at Debre Tabor Comprehensive Specialized Hospital, Northcentral Ethiopia: A cross-sectional study. HIV/AIDS – Research and Palliative Care, 13, 477–484. https://doi.org/10.2147/HIV.S308422

[14] Prevalence of leucopenia and associated factors before and after initiation of ART among HIV-infected patients, North East Ethiopia: Cross-sectional study. (2021).

[15] Immuno-haematological abnormalities of HIV-infected patients before and after initiation of highly active antiretroviral therapy in Dessie Town, Ethiopia. (2022). Relevance: Reported significant changes in WBC and absolute neutrophil count following HAART.

[16] Gudina, A., Wordofa, M., & Urgessa, F. (2024). Immuno-hematological parameters among adult HIV patients before and after initiation of dolutegravir-based antiretroviral therapy, Addis Ababa, Ethiopia. PLOS ONE, 19(10), e0310239. https://doi.org/10.1371/journal.pone.0310239

[17] Demango, F., Tadasa, E., & Kiya, G. T. (2025). Hematological profiles of adults coinfected with HIV and malaria receiving highly active antiretroviral therapy at Bonga Gebretsadik Shawo General Hospital, Southwest Ethiopia: A comparative cross-sectional study. Advances in Hematology, 2025, 3894305. https://doi.org/10.1155/ah/3894305

[18] Opie, J., Verburgh, E., Bailly, J., Mayne, E., & Louw, V. (2024). Hematological complications of human immunodeficiency virus (HIV) infection: An update from an HIV-endemic setting. Open Forum Infectious Diseases, 11(4), ofae162. https://doi.org/10.1093/ofid/ofae162

[19] Kigongo, V. J., Nankabirwa, J. I., Kitutu, F. E., et al. (2024). Dyslipidemia among adult people living with HIV on dolutegravir-based antiretroviral therapy at a private tertiary hospital in Kampala, Uganda: Burden and determinants. BMC Infectious Diseases, 24, 53. https://doi.org/10.1186/s12879-023-08892-8

[20] Mohammed, O., Alemayehu, E., Debash, H., et al. (2024). Dyslipidemia among HIV-infected patients in Ethiopia: A systematic review and meta-analysis. BMC Infectious Diseases, 24, 27. https://doi.org/10.1186/s12879-023-08910-9

[21] Woyesa, S., Mamo, A., Mekonnen, Z., Abebe, G., Gudina, E. K., & Milkesa, T. (2021). Lipid and lipoprotein profile in HIV-infected and non-infected diabetic patients: A comparative cross-sectional study design, Southwest Ethiopia. HIV/AIDS – Research and Palliative Care, 13, 1119–1126. https://doi.org/10.2147/HIV.S339539

[22] Seedat, F., Raal, F., & Martinson, N. (2020). Lipid and lipoprotein levels in HIV-infected adults with sepsis compared to healthy HIV-infected controls. African Journal of Infectious Diseases, 14(2), 1–9. https://doi.org/10.21010/ajid.v14i2.1

[23] Relationship between endothelial function, antiretroviral treatment and cardiovascular risk factors in HIV patients of African descent in South Africa: A cross-sectional study. (2021).

[24] Lipid profile of people living with HIV on antiretroviral therapy monitored in a hospital setting in Kinshasa, Democratic Republic of the Congo. (2025). Orapuh Journal, 6(6). https://doi.org/10.4314/orapj.v6i6.55

[25] Ayanful-Torgby, R., Shabanova, V., Essuman, A. A., Boafo, E., Amoah, L. E., & Paintsil, E. (2025). Alterations in lipid profiles in children with perinatally acquired HIV infection living in Ghana: A cross-sectional study. PLOS ONE, 20(7), e0318314. https://doi.org/10.1371/journal.pone.0318314

[26] Wala, S. I., Elechi, H. A., Imam, R. A., Rabasa, A. I., & Bakari, H. B. (2025). Fasting lipid profile of HIV-infected children on antiretroviral therapy living in an area of prolonged armed conflict. South African Journal of Child Health, 19(4). https://doi.org/10.7196/SAJCH.2025.v19i4.2703 C. International contemporary lipid/ART studies

[27] Bianconi, V., Schiaroli, E., Pirro, M., et al. (2020). Effects of antiretroviral therapy on proprotein convertase subtilisin/kexin 9: Focus on lipids, inflammation and immunovirological parameters. HIV Medicine, 21(8), 512–522. https://doi.org/10.1111/hiv.12884

[28] Gazzola, L., Tagliaferri, G., De Bona, A., et al. (2021). Dyslipidaemia after switch to tenofovir alafenamide (TAF)-based cART regimens in a cohort of HIV-positive patients: What clinical relevance? HIV Medicine, 22(2), 140–145. https://doi.org/10.1111/hiv.12984

[29] Chaudhary, N. S., Kind, T., Willig, A. L., et al. (2021). Changes in lipidomic profile by antiretroviral treatment regimen: An ACTG 5257 ancillary study. Medicine, 100(30), e26588. https://doi.org/10.1097/MD.0000000000026588

[30] Giacomelli, A., et al. (2021). Impact of switching to TAF/FTC/RPV, TAF/FTC/EVG/cobi and ABC/3TC/DTG on cardiovascular risk and lipid profile in people living with HIV: A retrospective cohort study. BMC Infectious Diseases, 21, 595. https://doi.org/10.1186/s12879-021-06304-3

[31] Drechsler, H., Ayers, C., Cutrell, J., Arasaratnam, R., & Bedimo, R. (2021). Consistent use of lipid lowering therapy in HIV infection is associated with low mortality. BMC Infectious Diseases, 21, 150. https://doi.org/10.1186/s12879-021-05787-4

[32] Do all integrase strand transfer inhibitors have the same lipid profile? Review of randomised controlled trials in naïve and switch scenarios in HIV-infected patients. (2021).

[33] Oliveira, D. P. R., Macêdo, A. P. A., Gonçalves, M. S., & Couto, R. D. (2022). Dyslipidemia secondary to the use of HAART in adults: A systematic review. Current Drug Research Reviews, 14(3), 239–246. https://doi.org/10.2174/2589977514666220519165549

[34] Lee, D. (2022). HIV: How to manage dyslipidaemia in HIV. Drugs in Context, 11, 2021-8-7. https://doi.org/10.7573/dic.2021-8-7

[35] Boccara, F., Caramelli, B., Calmy, A., et al. (2022). Long-term effects of evolocumab in participants with HIV and dyslipidemia: Results from the open-label extension period. AIDS, 36(5), 675–682. https://doi.org/10.1097/QAD.0000000000003175

[36] Sun, L., et al. (2022). Higher risk of dyslipidemia with coformulated elvitegravir, cobicistat, emtricitabine, and tenofovir alafenamide than efavirenz, lamivudine, and tenofovir disoproxil fumarate among antiretroviral-naive people living with HIV in China. Journal of Acquired Immune Deficiency Syndromes.

[37] Liu, S., Wei, B., Liang, W., Chen, T., Deng, L., Zhao, M., & Wan, J. (2023). The effects of ART on the dynamics of lipid profiles in Chinese Han HIV-infected patients: Comparison between NRTI/NNRTI and NRTI/INSTI. Frontiers in Public Health, 11, 1161503. https://doi.org/10.3389/fpubh.2023.1161503

[38] Zhang, J., Jin, H.-L., Jian, F.-B., et al. (2022). Evaluation of lipid metabolism imbalance in HIV-infected patients with metabolic disorders using high-performance liquid chromatography-tandem mass spectrometry. Clinica Chimica Acta, 526, 30–42. https://doi.org/10.1016/j.cca.2021.12.010

[39] Yoo, et al. (2024). Risk of dyslipidaemia in people living with HIV who are taking tenofovir alafenamide: A systematic review and meta-analysis. Journal of the International AIDS Society. https://doi.org/10.1002/jia2.26358

[40] Savinelli, S., Heeney, A., et al. (2026). People living with HIV on modern antiretrovirals do not display a pro-atherogenic lipid profile and have similar body composition compared to healthy controls. HIV Medicine. https://doi.org/10.1111/hiv.70118