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Antiretroviral therapy and lipid disorders among people living with HIV/AIDS in the West Region of Cameroon: a comparative cross-sectional study

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Abstract

Antiretroviral therapy (ART) has greatly reduced HIV morbidity and mortality, but long-term use increases metabolic complications, especially dyslipidemia. This study assessed the prevalence of lipid disorders and associated factors among people living with HIV (PLHIV) in the West Region of Cameroon. A comparative cross-sectional study was conducted in two major HIV/AIDS care units. A total of 450 participants were enrolled: PLHIV on ART for more than a year (n=150), PLHIV on ART for less than a year (n=150), and HIV-negative controls (n=150). Dyslipidemia was defined according to the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III criteria. Group comparisons used the Kruskal-Wallis test, and associations were assessed with multivariate logistic regression. Mean levels of total cholesterol (TC), triglycerides (TGs), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) differed significantly between groups ($p<0.001$, $p=0.002$, $p<0.001$, $p<0.001$, respectively). In multivariate logistic regression, treatment duration above 1 year (adjusted odds ratio [aOR]=10.7; 95% confidence interval [CI]: 5.3-21.7; $p<0.001$) significantly increased the odds of hypercholesterolemia. Syphilis (aOR=3.1; 95% CI: 1.2-7.5; $p=0.012$) significantly contributed to the occurrence of elevated TG levels. Long-term ART was linked to significantly more lipid disorders, confirming treatment duration as a major driver of dyslipidemia in this population.

Key words: antiretroviral therapy; HIV-negative controls; PLHIV; lipid disorders; Cameroon.

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Introduction

AIDS continues to pose a major global health challenge, affecting more than 40 million people worldwide.[1] According to the Joint United Nations Program on HIV/AIDS, 1.3 million new infections and 680,000 AIDS-related deaths were recorded in 2024, reflecting a 40% decline in new infections compared to 2010, largely due to expanded access to antiretroviral therapy (ART).[2] Despite this progress, Sub-Saharan Africa (SSA) carries the greatest share of the epidemic, with about 27 million people living with HIV (PLHIV) in 2024, nearly two-thirds of the global total, where new infections and AIDS-related deaths are most concentrated, particularly among adolescent girls and young women. In SSA, the scale-up of ART has significantly reduced HIV-related morbidity and mortality, transforming HIV from a fatal disease into a chronic condition and thereby extending patient survival.[3,4] In Cameroon, adult HIV prevalence remains around 3-4%, with 14,000 new infections reported in 2024. ART coverage has improved markedly, rising from 26% in 2013 to 55% in 2018.[5]

Although ART has achieved remarkable success in extending life expectancy,[6] its long-term use is associated with metabolic complications, including dyslipidemia. Dyslipidemia reflects a disruption of lipid metabolism, characterized by abnormal levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides (TGs), and high-density lipoprotein cholesterol (HDL-C).[7] These alterations substantially increase the risk of cardiovascular disease (CVD), with PLHIV experiencing higher CVD morbidity and mortality compared to uninfected individuals.[8,9]

The impact of ART on lipids differs by drug class. Protease inhibitors raise TC, LDL-C, and TGs, promoting atherogenic dyslipidemia.[10,11] Non-nucleoside reverse transcriptase inhibitors, such as efavirenz (EFV), increase TC and LDL-C, but less than first-generation protease inhibitors.[11-13] In contrast, nucleoside reverse transcriptase inhibitors (NRTIs), such as tenofovir (TDF), lower most lipid parameters.[14] Integrase strand transfer inhibitors (INSTIs), including dolutegravir and raltegravir, are linked to weight gain and modest lipid changes, though obesity may worsen effects.[14,15] Furthermore, chronic exposure to ART contributes to

persistent systemic inflammation, marked by elevated levels of interleukin (IL)-6 and C-reactive protein (CRP), which further aggravate dyslipidemia and heighten CVD risk.[6,16]

The metabolic complications of ART are well-documented in high-income countries.[17] However, data from SSA, and Cameroon in particular, remain limited and reveal some distinctive patterns. Studies examining lipid profiles and metabolic complications among PLHIV have shown heterogeneous findings, often influenced by treatment regimens and patient characteristics.

Dyslipidemia is highly prevalent under ART.[4,18] Integrase inhibitors, especially dolutegravir, have been associated with weight gain and an increased risk of diabetes and hypertension, particularly among women and individuals of African descent.[19] Among NRTIs, stavudine raises TGs and lowers HDL□C, whereas TDF has a more favorable lipid profile.[14]

Epidemiological data on ART and dyslipidemia in Cameroon's West Region are scarce. The decentralized health system and local socio−anthropological contexts may influence treatment outcomes in regimen−, time−, and region−specific ways. This study is among the first to assess the metabolic status of PLHIV in this region, as the lack of data hinders objective evaluation of HIV care units and evidence−based adjustments. We therefore aimed to evaluate the time−dependent impact of ART regimens on lipid profiles, a critical step to improve locally tailored HIV care and reduce metabolic and cardiovascular complications.

Materials and Methods

Study design

We conducted a 2-month comparative cross−sectional study (September-October 2024) in two major HIV/AIDS care units in Cameroon's West Region: Bafoussam Regional Hospital and Mbouo Protestant Hospital. These facilities were selected for their diverse urban and peri−urban patient populations.

Study population and sampling strategy

PLHIV attending the HIV/AIDS care units of Bafoussam Regional Hospital and Mbouo Protestant Hospital were consecutively invited to participate in the study. Participants were adults of both sexes (≥18 years), who provided free and informed consent. For the control group, HIV-negative patients of the same age range, willing to participate, were selected among individuals attending one of the hospitals for conditions other than HIV or metabolic disorders. There is evidence that ART induces a time-dependent metabolic disorder in patients.[20] To examine this hypothesis in the present study, HIV-positive participants were divided into two groups of 150 individuals each, including PLHIV on ART for less than one year or more than one year, respectively. As the comparative approach focuses on the effect of ART rather than on hospital differences, no specific randomization was performed regarding the number or profile of participants from the selected hospitals. Moreover, specific ART regimens were not considered a differentiating factor, and participants were consecutively enrolled to meet the target sample size within the study period. Due to constraints in the availability of consumables for biochemical assays, the sample size was adopted based on feasibility rather than statistical calculation. Also, no other restrictive considerations were applied to control participants besides being HIV-negative, having no metabolic disorders, and giving consent to participate. Participants with incomplete data, hemolyzed blood samples, or known conditions that could compromise laboratory analyses were excluded from the study.

Data collection

PLHIV aged 18 years and above and hospital−based controls were invited to participate. After obtaining informed consent, socio-demographic and clinical data were collected *via* questionnaire. Anthropometric measurements, including weight and height, were recorded to estimate body mass index (BMI). BMI categories followed standard definitions,[21] including underweight (BMI <18.5 kg/m^2), normal weight (18.5-24.9 kg/m^2), overweight (25.0-29.9 kg/m^2), and obesity (BMI ≥30 kg/m^2).[22,23] After a resting (seated) period of 10 to 20 mins, fasting blood samples were collected, and blood pressure was measured in each participant. Blood pressure was measured using a calibrated electronic sphygmomanometer. Two readings were done, and the average value was recorded for statistical analysis. Glycemia was estimated using Accu-Check® glucometer following standard procedures, with blood obtained by finger prick under aseptic conditions. Clinical records of PLHIV were reviewed to retrieve the most recent viral load.

Blood sample collection and laboratory procedures

Fasting venous blood samples (approximately 4 mL) were collected from each participant by trained personnel using standard phlebotomy procedures. The serum was obtained after a 10-minute centrifugation at 3,000 rpm in dry tubes. Two mL of serum were transferred into labelled 2 mL Eppendorf tubes using sterile, disposable Pasteur pipettes. All aliquots were transported in insulated safety boxes with ice packs to the laboratory of the Evangelical University Institute of Cameroon and stored at −20 °C until analysis. Serum concentrations of TC, TGs, and HDL-C were measured using a commercial Dutch Diagnostics kit, following the manufacturer's instructions. Briefly, TC and TGs were determined by spectrophotometric reading against the blank. Standard and sample tubes were prepared according to the assay protocol provided by the kit. All the mixtures were prepared, mixed thoroughly, and incubated for 5 mins at 37 °C to allow optimal color development. The absorbance was read afterward against the blank using a SmartSpec 3000 spectrophotometer. HDL-C determination required prior protein precipitation and centrifugation; the resulting supernatant was then processed using the same analytical sequence and incubation conditions for TC and TGs. LDL-C was calculated using the Friedewald formula: LDL-C = TC−HDL-C−(TGs/5).[22] An abnormal lipid profile was defined as TC ≥200 mg/dL, HDL-C <40 mg/dL, LDL-C ≥160 mg/dL, or TGs ≥200 mg/dL, according to NCEP ATP III guidelines.[24]

Statistical analysis

Descriptive statistics (frequencies, percentages) were reported. Normality was assessed using the Shapiro−Wilk test and quantile plots. Due to non−normal distribution, group differences in lipid parameters were compared using the Kruskal−Wallis test. Multivariate logistic regression examined associations between abnormal lipid profiles (TC, TGs, HDL−C, LDL−C) and predictors. A $p<0.05$ was considered significant. All analyses were performed using SPSS version 23.

Ethical considerations

The study received ethical approval (No. 882/28/08/2024/CE/CRERSH-OU/VP). All participants gave written informed consent after the study information was explained. Participation was voluntary.

Results

Sociodemographic characteristics

A total of 450 participants were enrolled in the study, equally distributed across the three groups: PLHIV on ART for more than one year (n=150), PLHIV on ART for less than one year (n=150), and HIV-negative controls (n=150) (Table 1).

Among patients, 405 (90%) were recently at the Bafoussam Regional Hospital. The age distribution showed that most participants were between 38 and 57 years (55.4%). PLHIV, especially those on ART for more than one year, were more frequently represented in the older age categories (48-67 years), whereas younger age groups (18-37 years) were more common among HIV-negative individuals. Women represented 77.8% of participants, equally across groups; only 0.8% were pregnant. Married participants accounted for 42.4%, widowed for 23.6% (more common among PLHIV on ART), and single for 24.9% (more frequent among HIV−negative). Most had a high−school (56.0%) or primary/lower (32.7%) education; university education was higher in HIV−negative (16.7%). Unemployment was 65.8%, especially among PLHIV on ART for >1 year (70.7%). Urban residency was 68.0%, and 95.6% were non−smokers.

Anthropometric characteristics

The mean BMI of the total population was 28.3±4.5 kg/m^2, with similar values in PLHIV on ART for more than one year (28.8±4.9 kg/m^2) and less than one year (28.6±4.5 kg/m^2), and a slightly lower mean among HIV-negative individuals (27.6±3.9 kg/m^2). Only one participant (0.2%) was underweight, while 23.1% had a normal BMI. Overweight was the most common category (44.9%), and 31.8% of participants were obese. Obesity was

Table 1. Socio-demographic and clinical profiles of all participants.

Variables	Total, n=450 n (%)	PLHIV+ART >1 year, n=150 n (%)	PLHIV+ART <1 year, n=150 n (%)	HIV-negative, n=150 n (%)
Age (years)				
18-27	32 (7.1)	7 (4.7)	8 (5.3)	17 (11.3)
28-37	42 (9.3)	3 (2.0)	14 (9.3)	25 (16.7)
38-47	111 (24.7)	30 (20.0)	54 (36.0)	27 (18.0)
48-57	138 (30.7)	61 (40.7)	41 (27.3)	36 (24.0)
58-67	95 (21.1)	35 (23.3)	25 (16.7)	35 (23.3)
68-77	30 (6.7)	13 (8.7)	8 (5.3)	9 (6.0)
>77	2 (0.4)	1 (0.7)	0 (0.0)	1 (0.7)
Gender				
Female	350 (77.8)	123 (35.1)	110 (31.4)	117 (33.4)
Male	100 (22.2)	27 (27.0)	40 (40.0)	33 (33.0)
Pregnancy				
Yes	5 (0.8)	0 (0.0)	1 (0.9)	4 (3.4)
No	445 (99.2)	123 (96.9)	109 (99.1)	113 (96.6)
Marital status				
Single	112 (24.9)	30 (20.0)	35 (23.3)	47 (31.3)
Co-habitant	37 (8.2)	4 (2.7)	11 (7.3)	22 (14.7)
Divorced	4 (0.9)	0 (0.0)	0 (0.0)	4 (2.7)
Married	191 (42.4)	73 (48.7)	66 (44.0)	52 (34.7)
Widower	106 (23.6)	43 (28.7)	38 (25.3)	25 (16.7)
Education				
Primary and below	147 (32.7)	53 (35.3)	57 (38.0)	37 (24.7)
High school	252 (56.0)	82 (54.7)	84 (56.0)	86 (57.3)
University	42 (9.3)	14 (9.3)	3 (2.0)	25 (16.7)
Not determined	9 (2.0)	1 (0.7)	6 (4.0)	2 (1.3)
Employment				
Employee	53 (11.8)	14 (9.3)	16 (10.7)	23 (15.3)
Self-employed	101 (22.4)	30 (20.0)	41 (27.3)	30 (20.0)
Unemployed	296 (65.8)	106 (70.7)	93 (62.0)	97 (64.7)
Residency				
Rural	144 (32.0)	50 (33.3)	42 (28.0)	52 (34.7)
Urban	306 (68.0)	100 (66.7)	108 (72.0)	98 (65.3)
Smoking status				
Smoker	20 (4.4)	6 (4.0)	6 (4.0)	8 (5.3)
Non-smoker	430 (95.6)	144 (96.0)	144 (96.0)	142 (94.7)
Body mass index				
Underweight	1 (0.2)	1 (0.7)	0 (0.0)	0 (0.0)
Normal	104 (23.1)	29 (19.3)	31 (20.7)	44 (29.3)
Overweight	202 (44.9)	66 (44.0)	69 (46.0)	67 (44.7)
Obesity	143 (31.8)	54 (36.0)	50 (33.3)	39 (26.0)

PLHIV, people living with HIV; ART, antiretroviral therapy.

somewhat more frequent among PLHIV on ART for more than one year (36.0%).

Antiretroviral treatment characteristics and clinical profile

The most common ART regimen was TDF+3TC+TLD (86.3%), followed by TDF+3TC+EFV (4.6%). Among patients on ART <1 year, 53.7% received TDF+3TC+TLD, while 71.4% of those on TDF+3TC+EFV were in the >1 year group. Only 1.0% of PLHIV were non−adherent to treatment (Table 2), of whom 2 (66.7%) belonged to the PLHIV+ART >1 year group. Regarding viral suppression, 19 (6.34%) HIV-positive participants had a viral load above 40 copies/mm^3, and 89.5% of them were in the PLHIV+ART >1 year group. A total of 78 participants (26.0%) had been on ART for 10 years or more.

Lipid profile

As shown by the Kruskal−Wallis test, Table 3 reveals a significant difference among the four groups for TC (p<0.001), TGs (p=0.002), HDL-C (p<0.001), and LDL-C (p<0.001). Chi−square analyses confirmed an association between ART duration and lipid

Table 2. Distribution of ART regimens, adherence levels, viral load, duration of treatment, diabetes, and hypertension status among all participants.

Variables	Total, n=450 n (%)	PLHIV+ART >1 year, n=150 n (%)	PLHIV+ART <1 year, n=150 n (%)	HIV-negative, n=150 n (%)
ART regimen				
TDF+3TC+EFV	14 (4.6)	10 (71.4)	4 (28.6)	n.a.
TDF+3TC+TLD (INSTI-based)	259 (86.3)	120 (46.3)	139 (53.7)	n.a.
3TC+AZT+ATV	4 (1.3)	1 (25.0)	3 (75.0)	n.a.
TDF+3TC+ATV	5 (1.6)	2 (40.0)	3 (60.0)	n.a.
ABC+3TC+ATV	11 (3.6)	10 (90.9)	1 (9.1)	n.a.
ATV/ABC	2 (0.6)	2 (100.0)	0 0.0)	n.a.
TDF+3TC+LPVr	5 (1.6)	5 (100.0)	0 (0.0)	n.a.
ART adherence				
Yes	297 (99.0)	148 (49.8)	149 (50.1)	n.a.
No	3 (1.0)	2 (66.7)	1 (33.3)	n.a.
Viral load				
<40 copies/mm^3	281 (93.6)	133 (47.3)	148 (52.7)	n.a.
>40 copies/mm^3	19 (6.34)	17 (89.5)	2 (10.5)	n.a.
Duration of ART treatment				
3 months	24 (8.0)	n.a.	24 (16.0)	n.a.
4 months	46 (15.3)	n.a.	46 (30.7)	n.a.
5 months	58 (19.3)	n.a.	58 (38.7)	n.a.
6 months	22 (7.3)	n.a.	22 (14.7)	n.a.
1 to 2 years	4 (1.3)	4 (2.7)	n.a.	n.a.
3 to 4 years	22 (7.3)	22 (14.7)	n.a.	n.a.
5 to 6 years	16 (5.3)	16 (10.7)	n.a.	n.a.
7 to 8 years	30 (10.0)	30 (20.0)	n.a.	n.a.
≥10 years	78 (26.0)	78 (52.0)	n.a.	n.a.

PLHIV, people living with HIV; ART, antiretroviral therapy; TDF, tenofovir; 3TC, lamivudine; TLD, dolutegravir; EFV, efavirenz; INSTI, integrase strand transfer inhibitors; AZT, zidovudine; ATV, atazanavir; ABC, abacavir; LPVr, lopinavir; n.a., not applicable.

Table 3. Descriptive statistics of lipid profile parameters among the three study groups (PLHIV on ART >1 year, PLHIV on ART <1 year, HIV-negative controls). Mean values were compared using the Kruskal-Wallis test and categorical variables using the chi-square test.

Variables	PLHIV+ART >1 year n=150	PLHIV+ART <1 year n=150	HIV-negative n=150	p
TC				
Mean ± SD (CI 95%)	192.6±64.9^a	160.1±59.8^b	148.5±56.0^b	<0.001
≥200 mg/dL, n (%)	67 (44.7)	11 (7.3)	24 (16.0)	0.001
TGs				
Mean ± SD (CI 95%)	111.0±62.1^{ab}	105.5±56.5^b	124.5±50.2^a	0.002
≥200 mg/dL, n (%)	31 (20.7)	36 (24.0)	7 (4.7)	0.001
HDL-C				
Mean ± SD (CI 95%)	55.1±25.2^a	56.9±23.5^a	26.1±19.0^b	<0.001
<40 mg/dL, n (%)	49 (32.7)	39 (26.0)	126 (84.0)	0.001
LDL-C				
Mean ± SD (CI 95%)	115.3±70.0^a	78.8±38.4^b	65.8±39.4^b	<0.001
≥160 mg/dL, n (%)	35 (23.3)	15 (10.0)	0 (0.0)	0.001

PLHIV, people living with HIV; ART, antiretroviral therapy; TC, total cholesterol; TGs, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SD, standard deviation; CI, confidence interval; ^{a,b,ab}averages followed by the same letter in the same row are not significantly different according to the Kruskal-Wallis test at the 5% significance level.

abnormalities: hypercholesterolemia (44.7% in ART >1 year, 7.3% in ART <1 year, 16.0% in controls, p=0.001) and elevated LDL−C (23.3%, 10.0%, 0.0%, p=0.001).

In multivariate logistic regression, treatment duration <1 year (adjusted odds ratio [aOR]=30.7; 95% confidence interval [CI]: 14.7-64.0; p<0.001), and treatment duration >1 year (aOR=10.7; 95% CI: 5.3-21.7; p<0.001), significantly increased the odds of hypercholesterolemia (Table 4). Normal weight (aOR=1.7; 95% CI: 1.1-2.7; p=0.010), independent occupation (aOR=1.5; 95% CI: 1.0-2.2; p=0.043), and syphilis (aOR=3.1; 95% CI: 1.2-7.5; p=0.012) significantly contributed to the occurrence of elevated TG levels (Table 4). The use of INSTIs (dolutegravir) (aOR=0.3; 95% CI: 0.1-2.2; p<0.001) significantly contributed to the appearance of reduced TG levels. Secondary education level (aOR=2.5; 95% CI: 1.2-5.2; p=0.008) significantly promoted the onset of low HDL−C (Table 4).

Table 4. Multivariate logistic regression analysis of factors associated with lipid abnormalities among all participants (n=450).

Variables	TC ≥200 mg/dL		TGs ≥200 mg/dL		HDL-C <40 mg/dL	
	aOR (95% CI)	p	aOR (95% CI)	p	aOR (95% CI)	p
Marital status						
Single	1.6 (0.9-2.7)	0.084	n.a.		n.a.	
Co-habitant	2.1 (1.1-5.4)	0.048	n.a.		n.a.	
Divorced	1273646081.9 (n.a.)	0.999	n.a.		n.a.	
Married	0.9 (0.5-1.4)	0.585	n.a.		n.a.	
Widower	1.0		n.a.		n.a.	
Married						
Yes	0.6 (0.4-2.9)	0.130	n.a.		n.a.	
No	1.0					
Pregnancy						
Yes	1382706684.5 (n.a.)	0.999	n.a.		n.a.	
No	1					
Normal weight						
Yes	n.a.		1.7 (1.1-2.7)	0.010	n.a.	
No	n.a.		1			
Independent activity						
Yes	n.a.		1.5 (1.0-2.2)	0.043	n.a.	
No	n.a.		1			
Education						
Primary and below	n.a.		n.a.		1155100885.8 (n.a.)	0.999
High school	n.a.		n.a.		2.5 (1.2-5.2)	0.008
University	n.a.		n.a.		1	
Not formally recorded	n.a.		n.a.		4.1 (1.2-5.4)	0.111
Duration of treatment						
<1 year	30.7 (14.7-64.0)	<0.001	n.a.		n.a.	
≥1 year	10.7 (5.3-21.7)	<0.001	n.a.		n.a.	
HIV-negative	1		n.a.		n.a.	
ART switch						
Yes	1		n.a.		n.a.	
No	1.7 (0.9-3.2)	0.091	n.a.		n.a.	
HIV-negative	1		n.a.		n.a.	
Duration of taking medication						
3 months	1.7 (0.9-3.2)	0.091	n.a.		n.a.	
4 months	0.9 (0.2-2.4)	0.877	n.a.		n.a.	
5 months	1.1 (0.3-3.3)	0.847	n.a.		n.a.	
6 months	0.5 (0.1-2.4)	0.421	n.a.		n.a.	
1 to 2 years	2E9 (n.a.)	n.a.	n.a.		n.a.	
3 to 4 years	2.7 (0.8-8.7)	0.094	n.a.		n.a.	
5 to 6 years	2.0 (0.5-7.4)	0.257	n.a.		n.a.	
7 to 8 years	1		n.a.		n.a.	
≥10 years	1.7 (0.7-4.3)	0.224	n.a.		n.a.	
Syphilis						
Yes	n.a.		3.1 (1.2-7.5)	0.012	n.a.	
No	n.a.		1		n.a.	
INSTI (dolutegravir)						
Yes	n.a.		0.3 (0.1-2.2)	<0.001	n.a.	
No	n.a.		1		n.a.	

ART, antiretroviral therapy; TC, total cholesterol; TGs, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; aOR, adjusted odds ratio; CI, confidence interval; INSTI, integrase strand transfer inhibitors; n.a., not applicable.

Discussion

Before the introduction of ART, HIV infection itself was known to cause characteristic lipid abnormalities, characterized by a reduction in TC, LDL-C, and HDL-C and an increase in TGs. This pattern is largely attributed to chronic immune activation and elevated pro-inflammatory cytokines, such as interferon-α.[3]

The objective of this study was to determine the prevalence of lipid disorders and the factors associated with them in patients receiving ART for HIV/AIDS. Longer ART exposure was linked to more lipid abnormalities, consistent with known metabolic effects of sustained treatment.

Our results showed that PLHIV on ART for more than one year had significantly higher rates of hypercholesterolemia and elevated LDL-C compared to those on ART for less than one year and to HIV-negative individuals. These findings confirm that ART durably disrupts lipid metabolism. This is consistent with the pathophysiological understanding that metabolic complications are often cumulative over time. Studies in Cameroon report that hypercholesterolemia is associated with treatment duration >42 months, with lipid abnormalities peaking at 2−3 years.[25,26] This mechanism can be explained by the fact that HIV infection itself induces chronic inflammation and persistent immune activation, characterized by elevated pro-inflammatory cytokines such as IL-6, tumor necrosis factor-α, and biomarkers like CRP.[6,15] This inflammation promotes lipid dysregulation through lipoprotein lipase inhibition and increased lipolysis.[25] Prolonged duration amplifies ART toxicity, exacerbating these lipid disorders by inducing mitochondrial dysfunction and visceral fat accumulation.[10]

The first years of ART treatment are associated with a major increase in TC. This result is consistent with studies conducted in Cameroon, which report that the duration of treatment is an independent factor in dyslipidemia,[27] and shows that TC is higher in patients taking ART for more than 2 years.[4] This is explained by the fact that suppression of viral replication leads to weight gain and an increase in body fat, which contributes to hypercholesterolemia.[4,27]

Normal-weight PLHIV have an increased risk of high TGs. This result is consistent with a study that notes that TGs are elevated independently of the BMI in patients on ART.[4] This can be explained by chronic inflammation because even with normal weight, HIV infection, and some ART, maintains an inflammatory state that stimulates the hepatic production of TGs.[28,29]

Syphilis co-infection strongly increases the risk of high TGs. This result is explained by the fact that bacterial co-infections increase oxidative stress, which contributes to dyslipidemia.[30]

Dolutegravir significantly reduces the risk of hypertriglyceridemia. This statistic is consistent with other studies showing that INSTIs are associated with modest and often favorable metabolic changes.[31] While INSTIs can lead to weight gain, their impact on TGs remains neutral or even favorable.[19] This is explained by the fact that INSTIs do not induce mitochondrial dysfunction observed with first-generation NRTIs (stavudine, didanosine), which is a major factor in hypertriglyceridemia.[13]

Limitations

This study faced several limitations. Firstly, CD4 counts were unavailable, though viral load provided reliable virological control data. Secondly, the sample contained a high proportion of women (77.8%). While this reflects a well-documented pattern across African health surveys, where women are more likely than men to engage with healthcare services and participate in research, the resulting sex imbalance may have influenced gender-related associations, particularly those involving metabolic outcomes such as dyslipidemia. Finally, interpretation of differences between ART regimens was limited by the small sample sizes and high variability in non-dolutegravir-based groups, especially atazanavir-based protease inhibitor regimens.

Conclusions

This study confirms that prolonged antiretroviral therapy (>1 year) is a major driver of dyslipidemia among PLHIV in western Cameroon, heightening their cardiovascular risk. These findings underscore the urgent need for routine lipid screening and integrated management of both metabolic and infectious comorbidities in this region.

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