

# Lipid Profile of Anti-Retroviral Treatment-Naïve HIV-Infected Patients in Jos, Nigeria

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## Abstract

**Background:** Human immunodeficiency virus (HIV) infection and its treatment are associated with lipid abnormalities. Data on lipid profile of treatment-naïve HIV-infected patients in Nigeria are limited, and available studies did not exclude the role of major host-related risk factors for dyslipidemia. **Aim:** We assessed the lipid profile of normotensive, non-diabetic, and non-obese treatment-naïve HIV-infected patients to identify their abnormalities in comparison with age- and sex-matched HIV-negative control. **Subjects and Methods:** One hundred and six normotensive, non-diabetic, and non-obese HIV positive patients and 98 age- and sex-matched HIV-negative controls had lipid profile estimation in the fasting state. The CD4+ cell count of the HIV-infected patients was also quantified. **Results:** The median (IQR) triglyceride was significantly higher in HIV-positive patients than in the controls [1.75 (1.30-2.40) mmol/L vs. 1.55 (1.30-1.90) mmol/L,  $P = 0.01$ ]. HIV-positive patients also had significantly lower mean total cholesterol, TC [4.18 (1.04) mmol/L vs. 4.64 (1.01) mmol/L,  $P = 0.001$ ] and HDL-C [1.17 (0.35) mmol/L vs. 1.29 (0.43) mmol/L,  $P = 0.03$ ]. The mean LDL-C [2.20 (0.87) mmol/L vs. 2.19 (0.75) mmol/L,  $P = 0.97$ ] and TC/HDL-C ratio [3.95 (1.42) vs. 3.84 (1.14) mmol/L,  $P = 0.52$ ] were similar between the HIV-positive patients and controls. The HIV-infected patients had a significantly higher proportion of subjects with low HDL-C [36.8% (39/106) vs. 23.5% (23/98),  $P = 0.04$ ] and hypertriglyceridemia [31.1% (33/106) vs. 11.2% (11/98),  $P = 0.001$ ] while the controls had significantly higher proportion of subjects with hypercholesterolemia [22.4% (22/98) vs. 10.4% (11/106),  $P = 0.02$ ]. Lower HDL-C was associated with CD4+ cell count < 200 cells/ $\mu$ L ( $P = 0.02$ ). **Conclusion:** Lipid abnormalities are common in treatment-naïve HIV-infected patients even in the absence of major host-related risk factors for dyslipidemia. HIV-infected patients should, therefore, be routinely screened for lipid disorders before commencement of anti-retroviral therapy.

**Keywords:** Africa, Dyslipidemia, Human immunodeficiency virus, Lipid profile

## Introduction

The human immunodeficiency virus (HIV) pandemic has led to unprecedented consequences in global health statistics in the past three decades. By far, the impact of the disease has been greatest in sub-Saharan Africa, which is home to 70% of the people living with HIV (PLHIV) globally.<sup>[1]</sup> With the introduction of anti-retroviral therapy (ART) and improved care for a number of ART-naïve populations, an increase in the life

expectancy of PLHIV has been documented in both developed and developing countries.<sup>[2,3]</sup> As HIV-infected patients live longer and suffer less infective complications, non-infective conditions have emerged as important complications of the disease and its treatment.<sup>[4]</sup>

Before the introduction of highly active anti-retroviral therapy (HAART), lipid abnormalities were reported in HIV-infected patients.<sup>[5-7]</sup> Although there are fewer studies in developing countries, various forms of dyslipidemia have been found to occur more frequently in ART-naïve HIV-infected patients compared to HIV-negative individuals.<sup>[8-10]</sup> While the introduction of HAART has undeniably revolutionized the management of PLHIV, some forms of dyslipidemia have been found to be commoner and more severe in ART-experienced patients.<sup>[11,12]</sup> This justifies routine assessment of the lipid profile of PLHIV before commencement of HAART.

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Data on lipid profile of HIV-infected patients in Nigeria are limited.<sup>[13-16]</sup> By far, majority of the studies are from Southern Nigeria.<sup>[13-15]</sup> The role of major host-related risk factors for dyslipidemia was not accounted for in nearly all the studies. In this survey, we assessed the lipid profile of normotensive, non-diabetic, and non-obese treatment-naïve HIV-infected patients in a tertiary hospital in Northern Nigeria to identify their abnormalities in comparison with age- and sex-matched HIV-negative control.

## Subjects and Methods

### Study setting and design

This was a cross-sectional study conducted at the adult HIV and the medical out-patient clinics of the Jos university teaching hospital, North-central Nigeria between July and November 2010. The HIV clinic offers ambulatory HIV care and treatment on every week day to over 15,000 patients. About 100-150 treatment-naïve HIV-infected patients are enrolled in the clinic on monthly basis. The region where the hospital is located has the highest burden of HIV/AIDS in the country.<sup>[17]</sup> The medical out-patient department (MOPD) from where the control subjects were recruited is run from Monday to Thursday with an average of two physician clinics per day. At the MOPD, over 800 patients are seen per month including over 100 new cases.

A total of 120 HIV-positive patients were selected using a simple random sampling technique from a sampling frame comprising 365 treatment-naïve patients while 120 HIV-negative controls were selected from medical patients attending the MOPD using simple random sampling from a frame of 411 patients stratified on age and sex to allow frequency matching with HIV-positive subjects. Individuals with hypertension, diabetes mellitus, or obesity as well as those on lipid-lowering agents were excluded from the study. Obesity was defined as body mass index (BMI)  $>30 \text{ kg/m}^2$ .<sup>[18]</sup>

Based on the exclusion criteria and exclusion of subjects with insufficient data, 14 HIV-positive patients and 22 HIV-negative controls were further excluded from the 120 patients already selected from each group, giving a final sample of 106 ART-naïve HIV-infected patients and 98 HIV-negative controls.

### Data collection

Information obtained from each participant included socio-demographic characteristics, history of alcohol consumption and cigarette smoking as well as drug history. Anthropometric measurements (weight, height, and BMI) and cardiovascular system examination were carried out for each subject.

The HIV status of the patients was confirmed by western blot assay, and CD4+ T-lymphocyte count was quantified

using flow cytometry (Partec, Germany). HIV-infected patients were divided into three categories using the Centers for Disease Control and Prevention (CDC) classification; category 1:  $\geq 500 \text{ cells}/\mu\text{L}$ , category 2:  $200\text{-}499 \text{ cells}/\mu\text{L}$ , and category 3:  $<200 \text{ cells}/\mu\text{L}$ .<sup>[19]</sup>

Fasting blood glucose was done on the spot using a glucometer (ACCU-CHEK® Aviva, United States) for each participant to exclude those with undiagnosed diabetes mellitus. After a 12-hour overnight fast, five milliliters of blood samples were collected from each participant by venepuncture into plain sample tubes. The blood samples were allowed to coagulate and spun at 3,000 rpm for 10 minutes. The serum samples were collected and stored at  $-4^\circ\text{C}$  and subsequently assayed for total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), and triglyceride (TG) within 48 hours of sample collection using a dry reagent chemistry technique (Reflotron® System, Roche Diagnostics, Germany). Low-density lipoprotein cholesterol (LDL-C) was estimated using the Friedewald formula.<sup>[20]</sup> Presence of a derangement in any of the lipid profile parameters was defined as dyslipidemia according to the World Health Organization (WHO) criteria thus hypercholesterolemia ( $\text{TC} > 5.2 \text{ mmol/L}$ ), hypertriglyceridemia ( $\text{TG} > 1.7 \text{ mmol/L}$ ), low HDL-C ( $<0.9 \text{ mmol/L}$  for men and  $<1.0 \text{ mmol/L}$  for women), high LDL-C ( $>3.5 \text{ mmol/L}$ ), and high TC/HDL-C ratio ( $>5$ ).<sup>[18]</sup>

### Ethical considerations

The study was approved by the ethics committee of the hospital. Participation was voluntary, and an informed consent was obtained from the participants. Confidentiality was ensured.

### Statistical analysis

The Epi Info version 3.5.1 statistical software (CDC, Atlanta, Georgia, USA) was used for data analysis. Means (standard deviation, SD) and counts (proportions) were used to describe continuous and categorical variables, respectively. The Student “*t*” test was used to compare the mean values of normally distributed variables while non-normally distributed continuous data were compared using the Kruskal-Wallis test. Where more than two group means were compared, analysis of variance (ANOVA) test was used. The Chi-square test was used to compare observed differences in proportions where appropriate. Multivariate analysis (logistic regression) was used to determine factors associated with dyslipidemia using variables that had a *P* value  $< 0.5$  on univariate analysis. The independent variables included in the logistic model were age, alcohol consumption, BMI, HIV status, sex, and smoking. *P* values of  $< 0.05$  were considered significant.

## Results

### Characteristics of the study participants

The HIV-infected patients comprised 66 females (62.3%) and 40 males (37.7%), and there was no difference in sex distribution

between them and the controls,  $P = 0.88$  [Table 1]. The mean (SD) age of the HIV-infected patients and controls were 33 (8) years and 34 (8) years, respectively,  $P = 0.79$ . The mean BMI in both groups was within normal, but the HIV-infected patients had a significantly lower BMI ( $P < 0.0001$ ). There was no statistically significant difference between the HIV-positive patients and controls in terms of proportion of subjects with a positive history of alcohol consumption ( $P = 0.41$ ) or cigarette smoking ( $P = 0.31$ ). The median CD4+ cell count of the patients was 215 (Interquartile range, IQR 101-314) cells/ $\mu\text{L}$ , and 47.2% (50/106) had severe immunosuppression reflected by CD4+ cell count below 200 cells/ $\mu\text{L}$  [Table 1].

### Lipid profile and lipid abnormalities

The lipid profile values are shown in Table 2. The median (IQR) TG was significantly higher in the HIV-infected patients than in the controls, 1.75 (1.30-2.40) mmol/L vs. 1.55 (1.30-1.90) mmol/L,  $P = 0.01$ . The HIV-infected patients also had significantly lower mean TC: 4.18 (1.04) mmol/L vs. 4.64 (1.01) mmol/L,  $P = 0.001$  and HDL-C 1.17 (0.35) mmol/L vs. 1.29 (0.43) mmol/L,  $P = 0.03$ . The mean LDL-C ( $P = 0.97$ ) and TC/HDL-C ratio ( $P = 0.52$ ) were similar in the two groups.

Among the HIV-infected patients, the mean HDL-C was significantly lower in those with CD4+ cell count  $<200$  cells/ $\mu\text{L}$  compared to those with CD4+ cell count of 200-499 and  $\geq 500$  cells/ $\mu\text{L}$ ,  $P = 0.02$  [Table 3]. The difference across the three CD4+ cell categories was not significant for the TC ( $P = 0.38$ ), LDL-C ( $P = 0.79$ ), TG ( $P = 0.57$ ), and TC/HDL-C ( $P = 0.51$ ).

Table 2 also shows the proportion of participants with dyslipidemia. Overall (i.e., irrespective of the specific lipid abnormality), the HIV-infected group had a significantly higher proportion of patients with dyslipidemia than the controls, 62.3% (66/106) vs. 33.7% (33/98),  $P < 0.0001$ . Given the significant difference in BMI between HIV-positive patients and the controls and that BMI may affect lipid levels, the subjects were further compared independent of BMI and other variables in a multivariate analysis, and HIV seropositivity remained strongly associated with dyslipidemia: Odds ratio (OR) = 3.10, 95% confidence interval (C.I) = 1.70-5.65,  $P = 0.0002$ . Age ( $P = 0.73$ ), alcohol consumption ( $P = 0.87$ ), BMI ( $P = 0.69$ ), sex ( $P = 0.60$ ), and smoking ( $P = 0.34$ ) had no significant association with dyslipidemia on multivariate analysis.

In terms of specific lipid abnormalities, compared to the controls, the HIV-infected patients had a significantly higher proportion of subjects with low HDL-C, 36.8% (39/106) vs. 23.5% (23/98),  $P = 0.04$  and hypertriglyceridemia 31.1% (33/106) vs. 11.2% (23/98),  $P = 0.001$ . The control group had significantly higher proportion of subjects with hypercholesterolemia, 22.4% (22/98) vs. 10.4% (11/106),  $P = 0.02$ . There was no difference between the patients

and controls in the proportion of subjects with high LDL-C ( $P = 0.37$ ) or high TC/HDL-C ratio ( $P = 0.91$ ).

## Discussion

This study assessed the lipid profile of treatment-naïve HIV-infected Nigerians who have none of the major host-related risk factors for dyslipidemia namely hypertension, diabetes

**Table 1: Characteristics of HIV-infected patients and controls**

Variable	HIV positive N=106	Controls N=98	P value
Age (years), mean (SD)	33 (8)	34 (8)	0.79
Sex (M/F)	66/40	60/38	0.88
BMI (Kg/m <sup>2</sup> ), mean (SD)	22.33 (3.62)	24.49 (2.85)	<0.0001
Alcohol consumption, yes, n (%)	29 (27.4)	32 (32.7%)	0.41
Cigarette smoking, yes, n (%)	12 (11.3)	7 (7.1)	0.30
CD4+ cell count (cells/ $\mu\text{L}$ ), n (%)			
$\geq 500$	11 (10.4)	-	-
200-499	45 (42.4)	-	-
<200	50 (47.2)	-	-

BMI: Body mass index, SD: Standard deviation

**Table 2: Lipid profile and lipid abnormalities of: Human immunodeficiency virus-infected patients and controls**

Variable	HIV positive N=106	Controls N=98	P value
TC, mean (SD), (mmol/L)	4.18 (1.04)	4.64 (1.01)	0.001
HDL-C, mean (SD), (mmol/L)	1.17 (0.35)	1.29 (0.43)	0.03
LDL-C, mean (SD), (mmol/L)	2.20 (0.87)	2.19 (0.75)	0.97
TG, median (IQR), (mmol/L)	1.75 (1.30-2.40)	1.55 (1.30-1.90)	0.01
TC/HDL-C, mean (SD), (mmol/L)	3.95 (1.42)	3.84 (1.14)	0.52
Dyslipidemia, n (%)	66 (62.3)	33 (33.7)	<0.0001
Hypercholesterolemia, n (%)	11 (10.4)	22 (22.4)	0.02
Low HDL, n (%)	39 (36.8)	23 (23.5)	0.04
High LDL, n (%)	6 (5.7)	3 (3.1)	0.37
Hypertriglyceridemia, n (%)	33 (31.1)	11 (11.2)	0.001
High TC/HDL-C ratio, n (%)	19 (17.9)	17 (17.3)	0.91

TC: Total cholesterol, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, TG: Triglyceride, IQR: Interquartile range, SD: Standard deviation

**Table 3: Lipid profile of human immunodeficiency virus-infected patients according to CD4+ cell category**

Variable mean (SD) (mmol/L)	CD4+ $\geq 500$ N=11	Cell category 200-499 N=45	(cells/ $\mu\text{L}$ ) N=50	P value
TC	4.11 (0.90)	4.34 (0.95)	4.04 (1.15)	0.38
HDL-C	1.17 (0.41)	1.27 (0.35)	1.07 (0.31)	0.02
LDL-C	2.15 (0.81)	2.27 (0.81)	2.15 (0.93)	0.79
TG <sup>†</sup>	1.60 (1.30-2.30)	1.90 (1.30-2.40)	1.60 (1.30-2.60)	0.78
TC/HDL-C	3.85 (1.09)	3.79 (1.12)	4.12 (1.70)	0.51

TC: Total cholesterol, HDL-C: High density lipoprotein cholesterol, LDL-C: Low density lipoprotein cholesterol, TG: Triglyceride, <sup>†</sup>TG values are expressed as median (Interquartile range, IQR)

mellitus, and obesity. Our findings show that treatment-naïve HIV-infected patients have significantly lower levels of TC and HDL-C as well as significantly higher levels of TG compared to age- and sex-matched apparently healthy individuals. The LDL-C and TC/HDL-C ratio were, however, similar in both groups.

Our observations are in agreement with previous studies in United States and other parts of sub-Saharan Africa that documented significantly lower levels of TC and HDL-C as well as higher levels of TG in treatment-naïve HIV-infected individuals.<sup>[6,8,9]</sup> However, these studies also found lower levels of LDL-C in their HIV-positive patients unlike what we found. Iffen and colleagues in Calabar, South-south Nigeria also made comparable observations in their HIV-infected patients who had significantly lower TC, HDL-C but significantly higher levels of LDL-C and TG.<sup>[15]</sup> Of all the lipid parameters in treatment-naïve HIV-infected patients, LDL-C appears to have a rather inconsistent report. While studies in the United States,<sup>[7]</sup> Ghana,<sup>[8]</sup> and Cameroon<sup>[9]</sup> documented lower LDL-C levels in ART-naïve HIV-infected patients compared to apparently healthy populations; other studies in Belgium<sup>[21]</sup> and Nigeria<sup>[15,16]</sup> found higher levels of LDL-C. Our findings regarding LDL-C differed from all of them.

A possible explanation for the LDL-C difference between our study and previous reports may be the variable stages of immunosuppression of the study participants. For instance, the median CD4+ cell count of the HIV-infected subjects in Ghana<sup>[8]</sup> was twice that of our participants. It has been shown that HIV infection induces an early decrease of cholesterol typically affecting TC first, followed by HDL-C then LDL-C, and a late increase of TG and these changes have been shown to correlate with the degree of immunosuppression in a number of studies.<sup>[8,21,22]</sup>

In terms of the relationship between lipid profile changes and immune status of the patients, our study showed significantly lower HDL-C in HIV-infected patients with severe immunosuppression compared to those with better immune status but the changes in levels of TC, LDL-C, TG, and TC/HDL-C in relation to immune status of the patients were, however, not statistically significant. Other reports from Nigeria have been conflicting. Whereas Iffen and co-workers<sup>[15]</sup> in Southern Nigeria found that lower HDL-C and higher TG levels significantly correlated with severity of immunosuppression, Adewole, *et al.*<sup>[16]</sup> in Northern Nigeria found no significant association between any of the lipid parameters and immune status.

The mechanism of lipid disorders in ART-naïve HIV-infected patients is thought to be cytokine-mediated. An association between plasma TG levels and circulating interferon (IFN)- $\alpha$  levels has been observed in persons with AIDS.<sup>[23]</sup> IFN- $\alpha$  is believed to bring about increased TG levels through a decrease in TG clearance as well as an increase in *de novo* hepatic lipogenesis and VLDL synthesis.<sup>[23]</sup> Although the mechanism

of hypocholesterolemia is poorly understood, low levels of TC, HDL-C, and LDL-C have been associated with elevated levels of  $\beta$ -2 microglobulin.<sup>[22]</sup>

Although dyslipidemia in HIV-infected patients is believed to carry a similar risk as in HIV-negative populations,<sup>[24]</sup> the consistent findings of low HDL-C in combination with hypertriglyceridemia can easily increase the burden of cardiovascular diseases in HIV-infected patients by unwanted proportions. This is because hypertriglyceridemia and low HDL-C are recognized independent risk factors for coronary artery disease.<sup>[25,26]</sup> The scenario is even more complex considering the fact that ART, especially PI-based therapy, is associated with lipid changes, which may initially result in normalization of some lipid disorders but may lead to hypertriglyceridemia and hypercholesterolemia on a long-term basis.<sup>[11,12]</sup>

Our study was not without limitations. Familial causes of dyslipidemia were unaccounted for due to lack of facilities for such screening. Considering that the study design was cross-sectional, it is not possible to determine the temporal relationship between acquisition of HIV infection and development of dyslipidemia. There is need for prospective studies to investigate the evolution of lipid abnormalities in HIV-infected patients.

In conclusion, lipid abnormalities are common in treatment-naïve HIV-infected patients, even in the absence of major host-related risk factors for dyslipidemia. The combination of hypertriglyceridemia and low HDL-C is the most consistent abnormality. HIV-infected patients should, therefore, be routinely screened for lipid disorders before commencement of ART, and those found to have dyslipidemia, should be appropriately treated. Population-based prospective studies are needed to further explore the relationship between lipid profile changes and immunological status of HIV-infected patients in the Nigerian setting.

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