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Research Article

Lipid profile of antiretroviral therapy-naive HIV-infected patients attending infectious diseases service of University Teaching Hospital of Kinshasa, Democratic Republic of the Congo (DRC)

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Abstract

Introduction: HIV infection leads to metabolic disorders. The objective of this work was to study the lipid profile of HIV + patients followed at the University Teaching Hospital of Kinshasa (UTHK).

Methods: This study analyzes the lipid profile of HIV + patients, aged at least 18 years, followed at the UTHK from January 1, 2008 to December 31, 2014. The medians of different types of lipids, the frequency of lipid disorders, the general clinical characteristics of patients and factors associated with dyslipidaemia were studied. Haemoglobin (Hb), White Blood Cells (WBC), Leukocyte Formula (LF), Blood Sugar, Urea, Creatinine, Transaminases, Uric Acid, CD4s+ count were analyzed.

Results: The lipid balance was performed in 38.8% of patients; 38.1% of them had dyslipidaemia. Total hypercholesterolaemia (28.6%), elevated LDL-C (19%), hypertriglyceridemia (23.8%) and HDL hypocholesterolaemia (42.9%) were observed. The medians of TG (128 mg / dL), HDL-C (51 mg/dL) and LDL-C (78 mg/dL) were high. Risk factors associated with dyslipidaemia were represented by WHO stage 4, tuberculosis (TB) and hyperglycaemia. The highest levels of LDL-C and TG and the lowest HDL-C were seen when CD4s+ were below 200 elements/µL.

Conclusion: The HIV/AIDS dyslipidaemia characterized in this study by HDL-C hypocholesterolaemia, hypertriglyceridemia and total and LDL hypercholesterolemia can be considered as an indicator of the progression of HIV infection.

More Information

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Keyword: HIV/AIDS; Lipid profile; DRC

Introduction

With the advent of antiretroviral therapy (ART), there has been a reduction in morbidity and mortality of HIV infection which has become a chronic affection. HIV infection leads to a decline in immunity, manifested biologically by a drop in CD4s$^+$ cells. It is also characterized by an increase in viral load [1,2]. In addition to the occurrence of opportunistic infections [OIs (bacterial, parasitic, fungal, and viral)], the HIV is responsible, in the long term, of metabolic disturbances including hyperglycaemia associated with insulin resistance and dyslipidaemia or both. Dyslipidaemia can be caused by the action of HIV *per se*, OIs, or ART, especially those of the first generation. In addition to these factors mentioned, other factors are incriminated in the occurrence of dyslipidaemia: weight, lipodystrophy, distribution of fats in the body, age, and diet [3]. The risk and severity of these metabolic alterations are usually increased by individual factors of susceptibility to diabetes mellitus and dyslipidaemia [3]. Restoration of health, changes in body composition and traditional risk factors increase the atherogenic risks responsible of cardiovascular diseases [3].

In general, acute infection induces changes in lipids, lipoproteins and apoproteins serum. These changes occur during the acute phase, convalescence and after complete recovery. HIV infection is a chronic infection in which changes in the lipid profile and markers of humoral and cellular immunity have also been observed [4-10]. These lipid abnormalities are related to changes in humoral and cellular immunity and are correlated with immune status and clinical course of HIV infection, which worsens when the immune deficiency widens. These changes are proportional to the drop in CD4s$^+$ cells. Lack of treatment with ARVs is responsible for the development of advanced HIV disease (CD4s$^+$ < 200 cells/mm^3 or WHO stages 3 or 4). HIV infection involves extensive immune activation and chronic inflammation leading to atherosclerosis. Dyslipidaemia occurring during HIV infection plays a role in the progression of atherosclerotic disease through the inflammation for which it is responsible. Immunosuppression plays a role in the decrease in VLDL clearance via the decrease in the synthesis of hepatic lipoprotein lipases exposing to hypertriglyceridemia. De novo lipogenesis, mediated by certain cytokines, major oxidative stress, and HDL decline explain this correlation between advanced immunosuppression and dyslipidemia. Thus, when the CD4s + count is low, as in advanced HIV disease, immune activation, inflammation, and dyslipidaemia are much greater. This explains why the lipid changes are proportional to the drop in CD4s$^+$ cells. [3,6,10,16]

The data for HIV-related dyslipidaemia are very variable. Dyslipidaemia in HIV/AIDS is characterized by total hypocholesterolaemia, decrease of HDL-C and LDL-C and increase of triglycerides and VLDL [10-19]. Hypocholesterolaemia is usually early and is associated with an immune dysfunction [14]. Nevertheless, the blood level of total cholesterol can, sometimes, be within normal limits [14] or high [15]. HDL-C and LDL-C levels decrease while immunosuppression increasing in the advanced stage of the disease [16]. In some cases, the LDL-C blood level is rather high [14].

Dyslipidaemia with or without ART during HIV infection have also been described in Africa [20-26].

Hypertriglyceridemia appears late in the course of the disease but was the first lipid abnormality described in HIV infection. It is due to:

- The decrease in the clearance of circulating lipoproteins resulting from the decrease in lipoprotein lipase (LPL) level.

- Stimulation of the synthesis of hepatic lipids through either the synthesis of hepatic fatty acids or a re-etherification of fatty acids derived from lipolysis.

Ideally, the follow-up of a person infected with HIV should include the blood sugar test and the lipid profile at the initial management and at least once a year according to the clinical context of the patient (normal or disturbed initial assessment, personal predispositions to diabetes mellitus or dyslipidaemia etc) [3].

Given the high costs of biochemical examinations, the PNLS (National Control Program) recommends exploring carbohydrate and lipid metabolisms at initial care but does not make it compulsory. In the ART follow-up, the PNLS, also, recommends evaluating these two metabolisms to detect hyperglycaemia and dyslipidaemia in time in the course of the disease under treatment. Little is known on the lipid profile of People living with HIV (PLWHIV) in the DRC. As in the management of HIV + patients in health centres and general hospitals, the lipid profile is not systematically carried out, this assessment is usually not available.

Considering disturbances in the lipid profile described in developed countries, the aim of the work is to describe the lipid profile to the patients followed at the UTHK, DRC.

Methods

This study carried out at the UTHK analyzes the data of a retrospective cohort of patients followed in consultation and hospitalization in Infectious Diseases Service from January 1, 2008 to December 31, 2014. It assesses the lipid profile of these HIV + patients at inclusion in the cohort before ART. It studies:

- sociodemographic characteristics: age, sex, marital status, profession, socioeconomic level, ethnicity.

- WHO clinical stages, history of the disease, clinical characteristics, and OIs.

- the median values of different types of lipids (Total Cholesterol = TC; High Density Lipoprotein Cholesterol = HDL-C; Low Density Lipoprotein Cholesterol = LDL-C; Triglyceride = TG).

- the general characteristics of the population in link to dyslipidaemia.

- the clinical characteristics of patients.

- risk factors associated with dyslipidaemia.

Besides the lipid profile, other haemato-biochemical parameters were evaluated:

- haemoglobin (Hb).

- white blood cells (WBC).

- leukocyte formula (LF).

- glycaemia.

- urea and creatinine.

- transaminases [Aspartate-Amino-Transferase (ASAT) and Alanine-Amino-Transferase (ALAT)].

- Uric acid.

The reference values used in this study are given in table 1.

Statistical analysis

Statistical analysis was performed using SPSS (Statistic Package for Social Sciences) software for Windows version 24.

The data are presented in the form of the absolute (*n*) and relative (%) frequencies for the categorical variables, as a mean (± standard deviation) for the quantitative variables with normal distribution and as a median (interquartile range = IQR) for the quantitative variables non-Gaussian distribution. The comparisons between PLWHIVs with a normal lipid profile and atherogenic dyslipidaemia were made by the Pearson Chi-square test or Fischer's exact test as appropriate for the categorical variables, the Student's *t* test for the continuous variables. The search for factors associated with atherogenic dyslipidaemia was carried out by the logistic regression test in exploratory univariate analysis.

Table 1: Reference values for haematological and biochemical assessment.

	Variables	Reference Values
1	Hb	12.5 to 15 g/dl for men and 10 to 15 g/dl for women
2	Ht	38 to 52% for men and 32 to 45% for women.
3	WBC	4,000 to less than 10,000 cells/mm^3
4	L F: Neutrophils	30% to 60%
	Lymphocytes	26% to 60%
	Eosinophils	0% to 12%
5	CD4s$^+$	From 410 to 1590 cells/mm^3
6	Urea	10 to 42 mg%.
7	Creatinine	0.5 to 1.2 mg%
8	ASAT	0 to 40 IU/L
9	ALAT	0 to 45 IU/L
10	High TC	if value > 200 mg/ dL
11	Hypertriglyceridemia	if TG > 150 mg/dL
12	High LDL-C	if value > 130 mg/dL
13	Low HDL-C	if value < 40 mg/dL for men and < 50 mg/dL for women

When differences were observed between atherogenic dyslipidaemia and the independent variables, the effect of potential confounders was studied by adjustment in conditional logistic regression in multivariate analysis. The ORs and their 95% CIs were calculated finally to determine the degree of association between atherogenic dyslipidaemia and the independent variables. The significance level retained was $p < 0.05$.

Ethical considerations

The study concerns a retrospective cohort. Data was collected from medical records. They were entered anonymously and in accordance with ethical rules. The study respected the rules of confidentiality, justice and charity of PLWHIV when collecting data anonymously. The service staff took care of the ethical aspects related to this study. Using deidentified data, no approval or consent from an ethics or institutional review board was required.

Results

Out of 270 PLWHIV followed in the UTHK and having been the subjects of this study, 105 (or 38.8%) of them had carried out the lipid assessment.

Median values and quartiles of different lipid types

The median values of the TC, its fractions and the TGs shown in table 2 are close to the third quartile; 25% of patients respectively have values higher than 92 mg/dl for LDL-C, 132.6 mg/dl for TG and 25% of patients had an HDL-C level lower than 51 mg/dl. The maximum values in our series are much higher than the reference values for TC, LDL-C and TG. For HDL-C, the minimum value is 6.3 mg/dl.

Frequency of lipid troubles in the study population

The frequency of atherogenic dyslipidaemia in general was 38.1% in PLWHIV followed in our service. Hypercholesterolemia, low HDL-C, high LDL-C and hypertriglyceridaemia were observed in this work as shown in table 3.

Table 2: Median values of classes of lipid.

Type of lipid, *n* = 105	Me (IQR)	Min-Max
TC, mg/dl	144 (125-148)	62,0-260
LDL-c, mg/dl	78,6 (35-92)	19-155,4
HDL-c, mg/dl	51,0 (42,3-67,5)	6,3-190,4
TG, mg/dl	128,0 (99-132,6)	66-286,5

Me = Median; IQR = Interquartile ratio; Min = minimum; Max: Maximum; TC = Total Cholesterol; LDL-C = Low Density Liprotein Cholesterol; HDL-C = High Density Lipoprotein Cholesterol; TG = Triglycerid

Table 3: Frequency of lipid troubles in the study.

Variable	*n* = 105	%	CI 95%
Dyslipidaemia in general	40	38,1	28,6-48,5
Hypercholesterolaemia	30	28,6	20,0-38,1
Low HDL-c	45	42,9	33,3-52,4
High LDL-c	20	19,0	11,4-26,7
Hypertriglyceridemia	25	23,8	16,2-32,4

General characteristics according to dyslipidaemia

The table on the general characteristics of PLWHIV according to dyslipidaemia indicates that women had a significantly high frequency of dyslipidaemia ($p = 0.024$), the widowed ($p = 0.023$), PLHIV with a low level of education ($p = 0.005$), those of the Revival Churches ($p = 0.006$), the Luba and Swahili ethnic groups ($p < 0.001$) (Table 4).

Clinical characteristics of patients

Table 5 shows that the frequency of dyslipidaemia was significantly higher in PLWHIHs with WHO's stage 4 ($p < 0.001$), in those with TB ($p < 0.001$) and anaemia ($p = 0.010$). Fever and elevated mean RR were significantly more encountered in PLHIV with dyslipidaemia. The frequency of vomiting is low in this group of PLWHIVs ($p = 0.016$).

Biological examinations

In PLHIV with dyslipidaemia, the average values of Hb were significantly lower while the average values of blood sugar ($p = 0,001$), urea and WBC were higher ($p < 0.05$) (Table 6).

Table 4: General characteristics according to dyslipidaemia.

Variable	All = 105	Normal $n = 65$	Dyslipidaemia $n = 40$	p
Age (year) ± SD	44,5 ± 11,5	44,9 ± 10,8	43,9 ± 11,5	0,651
Sex				0,024
Male	40(38,1)	30(46,2)	10(25,0)	
Female	65(61,9)	35(53,8)	30(75,0)	
Civil Status				0,023
Maried	45(47,4)	30(50,0)	15(42,9)	
Divorced	5(5,3)	5(8,3)	0(0,0)	
Single	30(31,6)	20(33,3)	10(28,6)	
Widower	15(15,8)	5(8,3)	10(28,6)	
Profession				0,443
Unemployed	40(47,1)	25(45,5)	15(50,0)	
State worker	15(17,6)	10(18,2)	5(16,7)	
Junior Company Agent	5(5,9)	5(9,1)	0(0,0)	
Liberal Profession	25(29,4)	15(27,3)	10(33,3)	
Level of studies				0,005
Primary	5(8,3)	0(0,0)	5(25,0)	
Secondary	35(58,3)	25(62,5)	10(50,0)	
University level	20(33,3)	15(37,5)	5(25,0)	
Religion				0,006
Catholic	30(42,9)	20(57,1)	10(28,6)	
Revival Church	35(50,0)	15(42,9)	20(57,1)	
Other	5(7,1)	0(0,0)	5(14,3)	
Socioeconomic level				0,056
Intermediary	35(87,5)	15(75,0)	20(100,0)	
Low	5(12,5)	5(25,0)	0(0,0)	
Ethnic group				< 0,001
Kongo	40(50,0)	30(60,0)	10(33,3)	
Ngala	15(18,8)	15(30,0)	0(0,0)	
Luba	15(18,8)	5(10,0)	10(33,3)	
Swahili	10(12,5)	0(0,0)	10(33,3)	

SD = Standard Deviation

Table 5: Patient's clinical characteristics.

Variables	All $n = 105$	Normal $n = 65$	Dyslipidaemia $n = 40$	p
WHO Stages				< 0,001
Stage 1	15(14,3)	10(15,4)	5(12,5)	
Stage 2	5(4,8)	5(7,7)	0(0,0)	
Stage 3	30(28,6)	30(46,2)	0(0,0)	
Stage 4	55(52,4)	20(30,8)	35(87,5)	
Antecedents				
TB	30(28,6)	5(7,7)	25(62,5)	< 0,001
Meningeal cryptococcosis	10(9,5)	5(7,7)	5(12,5)	0,313
Oral candidiasis	15(14,3)	10(15,4)	5(12,5)	0,458
Co-infection VHB-VHC	10(11,1)	5(9,1)	5(14,3)	0,332
HTA	15(14,3)	10(15,4)	5(12,5)	0,458
Smoking	10(9,5)	5(7,7)	5(12,5)	0,313
Alcohol intake	20(19,0)	15(23,1)	5(12,5)	0,139
Clinic				
Fever	80(76,2)	45(69,2)	35(87,5)	0,026
Weight loss	50(47,6)	30(46,2)	20(50,0)	0,428
Diarrhoea	95(90,5)	60(92,3)	35(87,5)	0,313
Cough	90(85,7)	55(84,6)	35(87,5)	0,458
Vomiting	90(85,7)	60(92,3)	30(75,0)	0,016
Headache	10(9,5)	5(7,7)	5(12,5)	313
Pallor	30(28,6)	25(38,5)	5(12,5)	0,003
Lymphadenopathy	15(14,3)	10(15,4)	5(12,5)	0,458
Lateralization signs	10(9,5)	5(7,7)	5(12,5)	0,313
SBP	124,2 ± 17,1	123,7 ± 12,6	125,0 ± 22,8	0,706
DBP	79,3 ± 14,5	79,4 ± 11,4	79,1 ± 18,6	0,930
Pulse	87,9 ± 12,2	89,4 ± 13,8	85,6 ± 8,9	0,127
RR	21,5 ± 4,9	20,8 ± 2,6	22,9 ± 7,1	0,041
BMI	24,8 ± 7,8	25,3 ± 8,6	22,8 ± 2,1	0,368
Comorbidities				
TB	30(28,6)	5(7,7)	25(62,5)	< 0,001
Oral candiasis	15(14,3)	10(15,4)	5(12,5)	0,458
Anaemia	25(23,8)	10(15,4)	15(37,5)	0,010
Meningeal cryptococcosis	10(9,5)	5(7,7)	5(12,5)	0,313

WHO = World Health Organization; TB = Tuberculosis; VHB = Viral Hepatitis B; VBC = Viral Hepatitis C; HTA = Hypertension; SBP = Systolic Blood Pressure; RR = Respiratory Rate.

Table 6: Biological examinations.

Variable	n	All	Normal	Dyslipidaemia	p
Hb (g/l)	90	9,4 ± 2,9	10,5 ± 2,9	8,8 ± 2,9	0,008
Ht (%)	70	30,3 ± 7,7	31,7 ± 6,4	27,8 ± 9,3	0,042
WBC (Cell/mm^3)	90	5847,8 ± 186,1	5254,1 ± 176,6	7035,0 ± 144,7	0,001
Neutrophile (%)	90	57,9 ± 12,9	57,5 ± 12,8	59,0 ± 13,2	0,602
Lymphocyte (%)	90	38,1 ± 12,1	37,7 ± 11,3	39,0 ± 13,6	0,636
Glycaemia (mg/dl)	60	96,8 ± 25,9	88,9 ± 16,9	120,3 ± 33,6	< 0,001
Urea (mg/dl)	95	31,1 ± 4,9	21,4 ± 6,5	52,0 ± 3,8	< 0,001
Creatinine (mg/dl)	95	1,4 ± 0,4	1,4 ± 0,6	1,2 ± 0,5	0,562
ASAT (UI/l)	95	31,1 ± 13,8	32,0 ± 14,8	28,9 ± 12,6	0,508
ALAT (UI/l)	95	30,4 ± 12,8	26,6 ± 9,3	38,7 ± 14,3	0,053
Uric Acid (mg/dl)	105	1,7 ± 0,4	1,9 ± 0,2	1,4 ± 0,5	0,460
CD4$^+$ cell/mm^3	105	229,5 ± 21,7	220,6 ± 21,5	243,9 ± 22,3	0,597

Hb = Haemoglobin; Ht = Haematocrit; WBC = White Blood Cell; g/l = gram per litter; Cell/mm^3 = Cell per cubic milliliter; mg/dl = milligram per deciliter; ASAT = Aspartate-Amino-Transferase; ALAT = Alanine-Amino-Transferase; CD4+ = Cluster of differentiation four.

Risk factors associated with atherogenic dyslipidaemia in the study population.

The risk factors associated with atherogenic dyslipidaemia in the univariate analysis were female, stage 4 of the WHO, presence of TB, anaemia, and hyperglycaemia.

After adjustment in multivariate analysis, female with risk multiplied by 3, WHO stage 4 (risk multiplied by 2), TB (risk multiplied by 9) and hyperglycaemia (risk multiplied by 8) were the factors patients associated with atherogenic dyslipidaemia (Table 7).

The LDL-C and TG levels were significantly higher and the HDL-C level low when the CD4 count was less than 200 elements per mm^3 (Table 8).

Discussion

This study analyzes the lipid profile of HIV + patients who have been followed up at UTHK. It highlights the lipid abnormalities observed in patients who have performed the lipid balance. Lipid assessment was only performed in 38.8% of patients. This score is low. The low number of patients who have performed the lipid assessment is because the examinations are expensive and are not accessible to all PLWHIV. Healthcare providers do not systematically ask for the lipid profile in the care of PLWHIV either. The PNLS (AIDS and STI Control Program) recommends the realization of the lipid profile when including patients in the care, but it does not have the possibility of subsidizing the biological assessment. Hence only those who have the financial resources do the exams.

Table 7: Factors associated with atherogenic dyslipidaemia in the study.

Variable	Univariate Analysis		Multivariate Analysis	
	p	OR (CI95%)	*p*	ORa (CI95%)
Sex				
Male		1		1
Female	0,033	2,57(1,08-6,11)	0,017	3,10(1,22-7,86)
WHO Stage 4				
No		1		1
Yes	0,002	2,27(1,34-3,85)	0,007	2,11(1,23-3,62)
TB				
No		1		1
Yes	0,000	6,00(2,56-9,60)	0,000	8,67(5,72-10,89)
Anaemia				
No		1		1
Yes	0,012	3,30(1,30-8,36)	0,749	1,22(0,36-4,18)
Hyperglycaemia				
No		1		1
Yes	0,000	6,00(3,87-12,21)	0,009	8,00(3,69-17,67)

OR = Odds Ratio; ORa = Odds Ratio a; CI = Confidence Interval

Table 8: Lipid profile according to $CD4^+$ count.

Lipid profile	< 200 *n* = 65	200-499 *n* = 30	≥ 500 *n* = 10	*p*
TC, mg/dL	162,6 ± 50,4	153,0 ± 14,3	146,0 ± 28,2	0,097
LDL-c, mg/dL	86,1 ± 15,2	74,2 ± 23,3	50,6 ± 16,2	0,067
HDL-c, mg/dL	44,0 ± 24,2	54,6 ± 24,3	66,6 ± 25,9	0,041
Triglycéride, mg/dL	158,9 ± 25,5	120,9 ± 27,2	94,6 ± 25,3	0,001

TC = Total Cholesterol; LDL-C = Low Density Lipoprotein Cholesterol; HDL-C = High Density Lipoprotein Cholesterol;

Median values and quartiles of different types of lipid

The median values of CT, LDL-C, HDL-C and TG indicate that there are lipid abnormalities. The median value of the CT is high and is above 50% of the value of the upper limit of the reference value. The same is true of the medians of LDL-C and TG. The minimum value of HDL-C is extremely low and is a good indication for atherogenic dyslipidaemia.

Frequency of lipid disorders in the study population

Table 9 shows the results of some studies on dyslipidaemia in HIV-infected patients. These results are compared to ours.

Regarding to the atherogenic risk, the 2 most important parameters in dyslipidaemia are HDL-C and TG compared to changes in TC and LDL-C [10]. Acute infection [4,5] and HIV infection are the basis of lipid abnormalities [6,8,13].

General characteristics according to dyslipidaemia

In this study, dyslipidaemia is predominant among women, members of revival churches, Luba and Swahili ethnicities, and low educational levels. The differences observed are statistically significant. However, we do not have a specific explanation based on scientific evidence to provide. Subject to the small sample size, it is difficult to draw a conclusion. A study with a larger sample considering the food composition of different subgroups, genetics, environment, and many other factors is needed to decide.

The advanced stage (stage 4) of the disease is a factor influencing the lipid profile. The occurrence of OIs may also explain the dyslipidaemia in HIV patients. TB is an OI disease with chronic inflammation and the supply of inflammatory cytokines may explain dyslipidaemia. Anaemia is common in HIV infection. It is a consequence of chronic inflammation with the possibility of iron sequestration in macrophages. In this study, PLHIV with dyslipidaemia have low mean Hb and higher level of blood sugar values. Low Hb is linked to chronic inflammation as mentioned above and probably to the frequent malnutrition in PLHIV with advanced HIV infection. During HIV infection, cardiometabolic complications may be observed. The elevated blood glucose values observed in this series in some patients may be placed in the context of the metabolic complications of HIV disease. On the other hand, the high mean values of urea can be linked to comorbidity (dehydration? Chronic kidney disease?). This study shows, in univariate analysis, that stage 4, TB and hyperglycaemia are risk factors associated with dyslipidaemia. Stage 4 is a pejorative factor leading to dyslipidaemia. The same is true of TB which is a chronic inflammatory condition and hyperglycaemia. In this series, after adjustment in multivariate analysis, the risk is multiplied by 3 in women, by 2 for stage 4 and by 9 for TB. High blood sugar is an 8-fold risk factor.

Advanced immunosuppression increases the risk of developing hypertriglyceridemia and LDL hypercholesterolemia, especially when the CD4 count is below 200 elements per mm^3.

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Table 9: Comparable literature data of some studies on dyslipidaemia in HIV patients.

Authors Years [Reference] Countries	Location	Cases (Ca)	Controls (Co)	Frequency of Dylipidae-mia	Age (Year; mean ± SD or median)	TC (Mean ± SD or median)	LDL-C (Mean ± SD or median)	HDL-C (Mean ± SD or median)	TG (Mean ± SD or median)	CD4s⁺ (Cells/µL) Mean ± SD or median
Singh, et al. 2014 [30] India	ART Center in Rohtak, Haryana	100	100	31 %	Co: 39 ± 4.5 Ca: 43 ± 9.62	Mean: 171.17 ± 52.24 mg/dL. TC>230 mg/dL in 8% of patients	Mean:108.62 ± 38.67 mg/dL. LDL-C >160 mg/dL: 7% of patients.	Mean: 26.86 ± 12.69 mg/dL. HDL-C<30 mg/dL: 10% of patients	Mean: 178.46 ± 58.41 mg/dL. TG>160 mg/dL: 29% of patients	CD4s⁺ 150.86 ± 67.32
Dave, et al. 2016 [28] South Africa	Depart-ment of Medicine, University of Cape Town	406 HIV+ ART ≥ 6 months	551 HIV+ No ART	HIV+ No ART: 90 %; HIV+ ART: 85 %	Median: 34 (Range: 19-68)	No hypercholes-terolemia in ART-naïve patients	Higher level (0,2 % in ART-naive patients)	Lower level (71 % of ART-naïve patients; HDL-C <1 mmol/L; 43 % in ART-patients)	no patient with high level in ART- naïve group	TG & HDL-C Signifi-cantly associated with CD4s⁺
Dayinam, et al 2013 [22] Nigeria	Jos University teaching Hospital	106	98	Co: 33.7 % Ca: 62.3 %	Co: 34 ± 8 Ca: 33 ± 8	Lower level for Ca (mmol/L) Co: 4.64 ± 1.01 Ca:4.18 ± 1.04	No significant difference (mmol/L) Co: 2.19 ± 0.75 Ca: 2.20 ± 0.87	Lower level for Ca (mmol/L) Co: 1.29 ± 0.75 Ca: 1.17 ± 0.35	Higher level for Ca (Median) Co:1.55(1.30-1.90) Ca:1.75(1.30-2.40)	Lower level HDL-C when CD4 < 200 cells/µL HDL-C: 1.07 ± 0.31
Kuti, et al. 2015 [29] Nigeria	HIV clinic of UCH Univer-sity of Ibanda	1316	No Controls	No Indicated globally. Indicated separately in TC and its fractons and in TG	35.2 ± 10.0	Higher level (mmol/L) TC > 35.2 ± 10.0 in 11.5 % of patients. Mean Value: 3.8 ± 1.2	Higher level (mmol/L) LDL-C>4.1 in 2.7 % of patients. Mean Value: 2.0 ± 1.0	Lower level (mmol/L) HDL-C <1 in 56.5 % of patients. Mean Value: 1.0 ± 0.5	Higher level in TG>1.7 in 27.6% of patients. Mean Value: 1.4 ± 0.7	CD4s⁺(Me-dian): 189.0 (70.0-366.3)
Anyabolu,et al. 2017 [30] Nigeria	HIV clinic in FMC, Oweri, South-East Nigeria	375	No Controls	No Indicated globally. Indicated separately in TC and its fractions and in TG	39 ± 11	Mean Value of TC: 4.26 ± 0.90	Elevated LDL-C in 17 % of Patients. Mean Value: 2.05 ± 0.5 mmol/L	Lower level HDL-C in 34 % of patients. Mean Value HDL-C: 1.18 ± 0.39 mmol/L	Elevated TG in 34 % of patients. Mean Value: 1.23 ± 0.37 mmol/L	Mean Value CD4s⁺ cells: 371 cells/µL. Significant association between LDL-C and CD4s⁺ cells.
Njoroge, et al. 2017 [31] Kenya	Volontary Counseling Centers Naïrobi	99	97	Co: 78% Ca: 83 %	Median Co: 32 (IQR: 24-40) Ca: 32 (IQR: 23-42)	Co: 95.30 Ca:96.56 Comparable for Co and Ca	Not mentioned	Co:31.56 Ca:30.93 Comparable for Co and Ca	Not mentioned	Median CD4s : 393 cells/µL (IQR: 57-729)
Nayyar, 2019 [27] India	Saraswati-Dhanwant-ri Dental College and Hospital Parbhani, Maharash-tra, India	HIV: 500 AIDS: 500	Co : 500	Not mentionned	Not mentioned	Co: 219.29 ± 37.46 HIV: 219.29 ± 43.01 AIDS: 200.18 ± 39.36 (mg/dL). Significant difference	Co: 114.09 ± 43.44 HIV: 138.47 ± 46.48 AIDS: 119.28 ± 27.89 (mg/dL) Significant difference	Co: 46.57 ± 22.54 HIV: 45.05 ± 17.84 AIDS: 45.69 ± 14.70non-significant difference	Co: 158.23 ± 49.20 HIV:140.88 ± 67.79 AIDS:167.43 ± 75.40 Significant difference	CD4s⁺ Co: 1125.38 ± 154.73 HIV: 501.35 ± 140.20 AIDS:256.41 ± 67.09
Mbula, et al. 2020 DRC	University teaching Hospital of Kinshasa	105	No Controls	38.1 % of patients	44.5 ± 11.5	Higher level in 28.6 % of patients. Median Value: 144.0 (125-148) mg/dL	Higher level in 19 % of patients. Median Value: 78.6 (35-92) mg/dL	Lower lever in 42.9 % of patients. Median Value: 51.0 (41.3-67.5)	Higher level in 23.8 % of patients. Median Value: 128 (99-132.6)	Highest levels of LDL-C and TG and lowest HDL-C when CD4s⁺< 200 Cells/

Conclusion

Lipid abnormalities were observed in patients who have performed the lipid balance in this study. Atherogenic dyslipidaemia was demonstrated. It was associated with certain independent factors (female gender, WHO stage 4, TB and hyperglycemia). Dyslipidaemia gives an indication of the progression of the disease in PLHIV.

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