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ORIGINAL RESEARCH

Trends in Serum Lipid Profile in HIV, Tuberculosis and Tuberculosis/HIV Co-Infected Patients at Three-Day Care Centres in Fako Division, Southwest Region of Cameroon

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Abstract

Background: Despite the reasonable goals and achievements with Antiretroviral (ARV) Therapy and Anti-Tuberculosis Drugs (ATD), several metabolic disturbances, such as abnormal serum lipid profile, have been observed during treatment with ARVs and ATD.
Objectives: To evaluate serum lipid profile in HIV and TB patients (before and after initiation of treatment) and their association with some possible risk factors.
Methods: One hundred and twenty-five patients were consecutively recruited at initiation to treatment and followed up for 12 weeks. Serum Total-Cholesterol (TC), High-Density-Lipoprotein-Cholesterol (HDLc), Low-Density Lipoprotein-Cholesterol (LDLc) and Triglycerides (TG) were measured spectrophotometrically to determine the serum lipid profile of participants.
Results: A significant association was recorded comparing the cumulative incidence of abnormal serum lipid profile (HDLc and LDLc) of the study participants before the initiation of treatment and after initiation of therapy, with 2.26 and 3.57 times likelihood of those on treatment to developing abnormal HDL and LDLc respectively ($\chi 2$ =7.4, p = 0.007; O.R; 2.26, 95% C.I; 1.25-4.10; $\chi 2$ =14.79, p = 0.00012; O.R; 3.57, 95% C.I; 1.82-6.98, respectively). There was a significant association between the level of TC and the type of drug regimen, with those on ATD/HAART having a higher frequency (8/17(47.1%)) of abnormal TC compared to their counterpart. There is a significant association between the level of TG and body mass index BMI ($\chi 2$ = 13.95, p = 0.003).
Conclusion: This study shows TB and HIV/AIDS alter lipid levels, and their respective treatments can exacerbate these effects, with TC levels being affected significantly by combination ATD/HAART therapy. Therefore, understanding these changes is crucial to patient management.

Keywords: Antiretroviral Therapy, Anti-Tuberculosis Drugs, Cholesterol, HIV/AIDS, Lipid Profile, Tuberculosis.

Introduction

Despite the reasonable goals and achievements with Antiretroviral (ARV)/Highly Active Antiretroviral Therapy (HAART) and Anti-Tuberculosis Drugs (ATD), several metabolic disturbances have been observed in the course of treatment with these drugs. One of the major metabolic disturbances observed is altered lipid metabolism in patients with Human immunodeficiency virus/Acquired immune deficiency syndrome (HIV/AIDS) and Tuberculosis (TB). [1, 2] These disturbances have been highly reported to be a hallmark risk of cardiovascular diseases. [1-3]

Cellular levels of cholesterol are known to be regulated by basic cellular metabolism and hormones.[4] Cholesterol also participates in enzyme activity, phagocytosis, cell growth, and fluidic cell membrane functions.[4] The transportation of cholesterol back and forth between the liver and tissues is by low-density lipoprotein cholesterol (LDL) and high-density lipoprotein cholesterol (HDL).[4] Both high levels of total cholesterol (TC) and LDL are traditional risk factors associated with coronary heart disease, among other metabolic disorders. High levels of LDL are known to cause plaque build-up along blood vessels that reduces blood flow to vital organs, depriving them of oxygenated blood. [1-3] Also, low levels of HDL are widespread in naïve/untreated HIV patients.[5]

Hypertriglyceridaemia is the most prevalent dyslipidaemia in HIV patients, and the second most common is hypercholesterolaemia.[2] Hypocholesterolaemia has been reported to promote the activation of latent TB to TB since it weakens immune defences by impairing macrophage function. In contrast, hypercholesterolaemia protects against TB, boosts immune responses, enhances macrophage activity, and restricts TB growth. [4, 6] Pulmonary TB has been reported to have a negative effect on the level and function of lipid profile.[4, 6,7] However, it is not clear whether a low lipid profile (LDLc, TG, TC, HDLc) leads to an advancement of the disease or active TB leads to a low lipid profile. Recent studies have suggested that genetic factors with substantial individual variation (diet, environmental factors and lifestyles) may also contribute to hyperlipidaemia in these patients. [1, 2, 8] It has also been reported that lower HDL and central obesity have been associated among HIV/AIDS patients with individual risk behaviours such as alcohol consumption and smoking habits.[8]

This study was aimed at determining lipid profiles in HIV/AIDS and TB patients before and during the first 12 weeks of initiation of treatment. This study helped outline some critical interactions of lipid metabolism, HIV/AIDS, and TB, particularly regarding treatment. Monitoring the lipid profiles within the first 12 weeks of treatment helps provide insight into metabolic changes that may affect patient outcomes in patients with cardiovascular risks and complications related to treatments. It enlightens findings that inform not only specific patient care but also overall public health strategy for the care of HIV/TB co-infected patients.

Methods

Study area and design

This prospective study was conducted at the HIV/AIDS and TB Treatment Centres in three hospitals, including Limbe Regional Hospital (LRH), Buea Regional Hospital Annex (BRHA), and Mutengene Baptist Hospital (MBH), in Fako Division, Southwest Region of Cameroon, between September 2018 and November 2019. This study area covers one of the biggest HIV/TB treatment centres in Cameroon, which is among the 223 diagnostic and treatment centres for tuberculosis patients coordinated by the Cameroon National Tuberculosis Control

Program. It included naïve HIV/AIDS, TB and HIV/TB co-infected patients who accepted to be part of the study and initiated treatment.

Study population

The study population included naïve HIV/AIDS, TB and HIV/TB co-infected patients who accepted to be part of the study and initiated treatment. The participants were on the first-line regimen of HAART [Tenofovir Disoproxil Fumarate (TDF), Lamivudine (3TC), and Efavirenz (EFZ)] or ATD [Isoniazid (INH), Pyrazinamide (PZA), Rifampicin (RIF), and Ethambutol (EMB)], and, all the participants on HAART were on Cotrimoxazole and TB prophylaxis (INH).

Inclusion criteria: Participants who did not have a previous history of ARV or ATD therapy, were 18 years and above, and did not have other complications or disease processes related to lipid abnormalities such as diabetes and high blood pressure.

Exclusion Criteria: Patients on other hepatotoxic, nephrotoxic drugs or diagnosed with other co-morbidities that markedly affect the blood lipid profile, glucose, and blood pressure (patients with renal failure on dialysis, thyroid disease, and liver disease have deranged lipid profile). Acutely ill patients who require medical/surgical treatment or admission. Those with documented hypertension, diabetes, and dyslipidaemia before beginning HAART, as well as pregnant and lactating women were also excluded.

Sample size

The expected sample size was determined using a 13.8% prevalence of abnormal TC/HDL-c ratio among HIV-infected patients on ATD taken from a similar setting, [9] 95% confidence interval, and a margin of error 0.05. We enrolled 183 participants consecutively; 125 of these were finally involved in the study. The consecutive sampling technique was used to collect the data from patients who met the sampling criteria.

Data collection and patient monitoring

Treatment naïve HIV/AIDS and TB patients reporting for scheduled and unscheduled check-ups were consecutively enrolled into the study. Hospital records of potential participants were reviewed to evaluate their medical history, and their blood samples were screened for hepatitis B and C to check whether they fulfilled the study inclusion criteria. Participants who fulfilled the inclusion criteria signed the informed consent and were interviewed using a standardised, validated structured questionnaire. The questions were designed to capture information on socio-demographic characteristics and clinical and epidemiological data, including gender, age, alcohol abuse status, smoking, ethnicity, consumption of antioxidant food, other diseases, and concomitant use of different medication(s). Alcoholism was estimated following international guidelines based on the participant's alcohol consumption status, average volume of consumption, frequency and volume of heavy episodic drinking. [10, 11]

After enrolment, participants were followed up, and blood samples were collected during the 12-week follow-up period: at initiation, week one, week four, week eight, and week 12 after initiation of treatment. A morbidity questionnaire was administered four weeks after treatment initiation and before the week 12 sample collection. The morbidity questionnaire was used to monitor some abnormal serum lipid profile clinical signs such as fever, nausea, vomiting, tiredness, episodes of malaria, other medications (anti-malaria, paracetamol, tramadol and the presence of other opportunistic infections (herpes, fungi/candidiasis, toxoplasmosis, bacterial infections, syphilis).

Specimen collection and analyses

Following eight to 12 hours overnight fast, 2mls of venous blood specimens were obtained by venipunctures under sterile conditions from the antecubital vein of the patients into dry test

tubes. The specimens were allowed to clot and then centrifuged to obtain sera into an Eppendorf tube and stored at -20°C, was used to assay for total serum cholesterol, triglyceride, LDL and HDL levels. Serum TC, TG, LDL, and HDL were determined using spectrophotometers (using kits from Cypress Diagnostic, Belgium). This was done before the initiation of treatment (Baseline) and at 1, 4, 8 and 12 weeks after commencement of therapy.

At treatment initiation, a rapid HIV-1 antibody test was done for all those who did not know their HIV status but were TB positive, using the Alere Determine (Belgium) kit. A confirmatory sputum smear microscopy test using the Ziehl-Nielsen staining method for TB was done for all those who did not know their TB status but were HIV positive. A rapid glucose test (fasting blood sugar test) was also done using Cypress Diagnostic kit (Belgium) to exclude participants with diabetes mellitus. Moreover, a sphygmomanometer was used to measure the participants' blood pressure (BP) before enrolment, and all those at risk of high BP were excluded.

Body weight was measured to the nearest 0.5kg with the participant upright on a weight measuring scale (Seca, Hamburg, Deutschland). The height was measured without shoes to the nearest 0.1cm with a Shahe stature meter (Shanghai, China). The Body Mass Index was calculated by dividing weight in kilograms by the square of standing height in metres (kg/m2). The World Health Organization (WHO) diagnostic criteria were used to determine underweight (<18.50), normal BMI (18.50–24.99), overweight (25.00–29.00), and obese (≥30.00). Blood Pressure (BP) was measured with an electronic sphygmomanometer (Omron M5-1 from Omron Health Care of Kyoto, Japan) on both arms, using the "Pan-African Society of Cardiology" guidelines. [12] Each participant was seated with their arms resting on an armrest, feet on the floor and back resting on the chair. The BP measurement was taken after being seated for at least 10 minutes. High BP was defined as values above 140 mmHg for systolic and/or above 90 mmHg for diastolic.

Laboratory analyses were done according to the same clinical schedule in all studied participants at the Infectious Disease Laboratory-FHS and at the Routine Laboratory of Saint Albert the Great Clinic. The normal reference ranges for lipid profile were set based on the Cypress Diagnostic Kit (Belgium) used for analysis and were as follows: HDL Cholesterol ≥ 60 mg/dL, LDL Cholesterol < 100 mg/dL, Triglycerides (TG) < 150 mg/dL, Total Cholesterol (TC) < 200 mg/dL. Abnormal serum lipid profile was defined as the presence of abnormal levels of all lipid parameters in the blood, including elevated TC, LDLc ("bad" cholesterol), TG (Fatty Acid), and as well as reduced HDLc ("good" cholesterol). [13, 14]

Statistical analysis

Data were analysed with IBM-SPSS Statistics 21.0 software for Windows (IBM-SPSS Corp., Chicago, USA). The Chi-Square (χ^2) test was used to compare socio-demographic characteristics with the health districts and binary logistic regression to identify significant correlates of the main outcomes. p values ≤ 0.05 were considered statistically significant.

Ethical considerations

The study protocol was approved by the Institutional Review Boards of the Faculty of Health Sciences, University of Buea (Ref: 2018/153/UB/SG/IRB/FHS) and Cameroon Baptist Convention (Ref: IRB2018-48). Administrative clearances were obtained from the Southwest Regional Delegation for Public Health and the Directors of the three hospitals involved in the study. An oral presentation was made to the participants, educating them on the objectives, purpose, risks, and benefits of participating in this study according to the

Declaration of Helsinki principles. After the presentation, written informed consents were obtained from each study participant aged 18 years and above, with participation being voluntary, and they were free to withdraw from the study at any time without any justification. Participants were assigned unique identification numbers on the questionnaires, tubes of sample collection, and results form.

Results

The mean age and mean BMI of the 125 participants who completed the study over 8-12 weeks were 39.54±10.69 years and 24.32±4.23 Kg/m^2, respectively (Table I). Also, 31.2% (39/125), 45.6% (57/125) and 23.2% (29/125) had primary, secondary and tertiary levels of education, respectively (Table I).

Table I: Baseline characteristics of the study population, stratified by treatment groups

Variable	*Subclass*	*HIV (%)*	*TB (%)*	*TB/HIV (%)*	*Total (%)*	χ^2	*p-value*
Gender	Female	46 (60.5)	15 (46.9)	07 (41.2)	68 (54.4)	3.079	0.214
	Male	30 (39.5)	17 (53.1)	10 (58.8)	57 (45.6)		
Age (years)	< 40	34 (44.7)	22 (68.8)	6 (35.3)	62 (49.6)	6.805	0.33
	≥ 40	42 (55.3)	10 (31.2)	11 (64.7)	63 (50.4)		
	Mean age (±SD)	39.54±10.69	39.54±10.69	39.54±10.69	39.54±10.69		
BMI	Underweight	04 (5.3)	05 (15.6)	01 (5.9)	10 (8.0)	7.444	0.11
	Normal	40 (52.6)	20 (62.5)	12 (70.6)	72 (57.6)		
	Overweight	32 (42.1)	07 (21.9)	04 (23.5)	43 (34.4)		
	Mean BMI (±SD)	24.32±4.23	24.55±4.35	24.32±4.23	24.32±4.23		
Education	NFE + Primary	23 (30.3)	10 (31.2)	06 (35.3)	39 (31.2)	7.728	0.10
	Secondary	41 (53.9)	11 (34.4)	05 (29.4)	57 (45.6)		
	Tertiary	12 (15.8)	11 (34.4)	06 (35.3)	29 (23.2)		
Marital status	Married	28 (36.8)	11 (34.4)	06 (35.3)	45 (36.0)	0.064	0.96
	Unmarried	48 (63.2)	21 (65.6)	11 (64.7)	80 (64.0)		
Occupation	Skilled	13 (17.1)	02 (6.3)	03 (17.6)	18 (12.8)	11.656	0.07
	Unskilled	54 (71.1)	22 (68.8)	12 (70.6)	88 (72.0)		
	Student	03 (3.9)	07 (21.8)	2 (11.8)	12 (9.60)		
	Unemployed	06 (7.9)	01 (3.1)	00 (0.00)	07 (5.60)		
BCG scar	Yes	44 (57.9)	20 (62.5)	09 (52.9)	73 (58.4)	0.438	0.80
	No	32 (42.1)	12 (37.5)	08 (47.1)	52 (41.6)		
Malaria	Yes	17 (22.4)	12 (37.5)	07 (41.2)	36 (28.8)	3.984	0.1
	No	59 (77.6)	20 (62.5)	10 (58.8)	89 (71.2)		
Sample size	-	*n* = 76	*n* = 32	*n* = 17	*N* = 125		

BMI - Body Mass Index; NFE - No formal education; χ2 – Chi-Square

Serum Lipid Profile

There was a significant and gradual decrease of some serum lipid parameters, including HDL (χ^2 =15.60, p = 0.004), TG (χ^2 =16.33, p = 0.0026), and TC (χ^2 = 14.30, p = 0.0064) from baseline to week 12 of follow-up (Figure 1). Furthermore, a high point prevalence [32/125 (25.6%)] of abnormal HDL at the initiation of treatment and a gradual decrease during the follow-up period was observed. This was similar to other parameters

(LDC-C, TC, and TG) of the serum lipid profile, as shown in Figure 2.

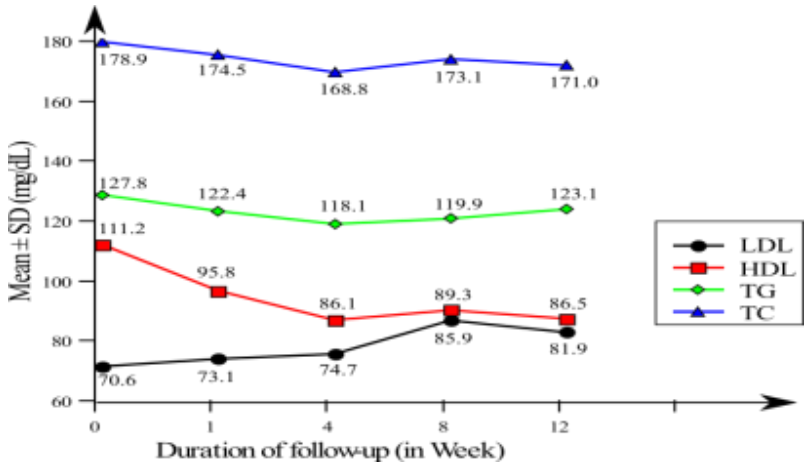

Figure 1: Variation of the Serum Lipid Profile in relation to follow-up care duration

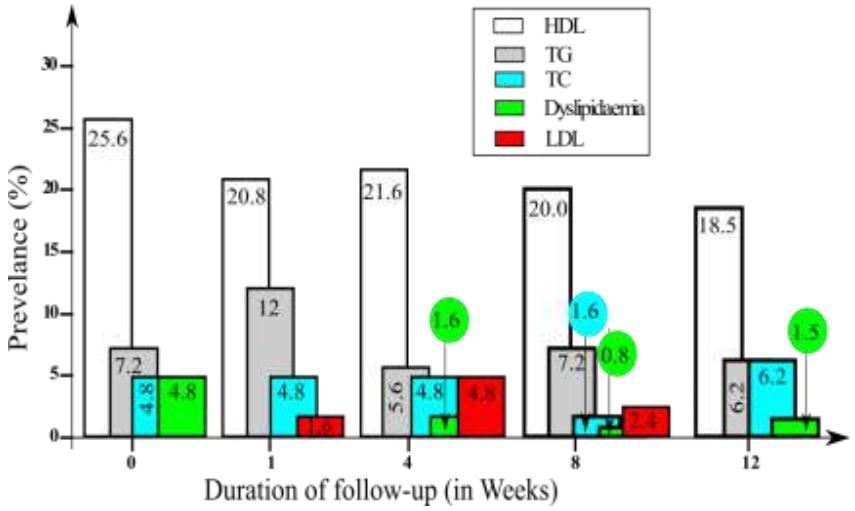

Figure 2: Point prevalence of abnormal serum lipid profile in relation to weeks of follow-up care

Comparison of the cumulative incidence of abnormal serum lipid profile before and after initiation of treatment

Comparing the cumulative incidence of the abnormal serum lipid profile before the initiation of treatment (control) and after treatment (case) showed a significant association observed in HDL and LDLc. This showed that there was a 2.26 and 3.57 times likelihood of those on treatment to develop abnormal HDL and LDLc, respectively ($\chi^2 = 7.4$, p = 0.007; O.R = 2.26, 95%C.I = 1.25-4.10; $\chi^2 = 14.79$, p = 0.000012; O.R = 3.57, 95%C.I = 1.82-6.98, respectively) (Table II).

At the end of the follow-up period, the following incidence rates: 45/72 (62.5%), 37/111(33.33%), 32/92(34.78%), and 31/95(32.63%) of abnormal HDLc, LDLc, TG, and TC respectively, were recorded. The incidence rates determined how quickly the participants developed abnormal serum lipid profiles. The incidence rate of

abnormal HDLc, LDLc, TG, and TC observed was; 45/519 (8.7 cases per 100 person-weeks), 37/1003 (3.7 cases per 100 person-weeks), 32/822 (3.0 cases per 100 person-weeks), and 31/857 (3.6 cases per 100 person-weeks) respectively.

Table II: Comparison of the incidence of abnormal lipid profile before and after initiation of treatment

	Before Initiation of Treatment		*After Initiation of Treatment*			
Parameters	**Normal (%)**	**Abnormal (%)**	**Normal (%)**	**Abnormal (%)**	**O.R (95 C.I)**	χ^2
HDL	72/125 (57.6)	53/125 (42.4)	27/72 (37.5)	45/72 (62.5)	2.26 (1.25-4.10)	7.40
LDL	111/125 (88.8)	14/125 (11.2)	74/110 (67.3)	37/111 (33.3)	3.57 (1.82-6.98)	14.79
TG	92/125 (73.6)	33/125 (26.4)	60/92 (65.2)	32/92 (34.8)	1.49 (0.83-2.67)	1.77
TC	95/125 (76.0)	30/125 (24.0)	64/95 (67.4)	31/95 (32.6)	1.53 (0.84-2.78)	2.00

O.R - Odds Ratio; χ2 - Chi Square

Relationship between some risk factors and abnormal lipid profile

Although no significant association was observed between any of the possible risk factors and the level of HDL, there was a high frequency [7/11 (63%)] of abnormal HDL observed among those at risk of obesity risk (Table III). In addition, a significant association was observed between opportunistic infection and level of HDLc, with those infected with some opportunistic infections being 0.39 times likely to develop abnormal HDLc as compared to their counterparts (χ^2 = 5.45, p = 0.021; O.R = 0.39, 95% C.I = 0.17-0.87) (Table III).

Table III: Relationship between some risk factors and abnormal HDLc levels in the study participants

Parameters		*HDL*		*Total (%)*	*Odd Ratio (CI)*	χ^2/*F*
		Abnormal (%)	**Normal (%)**			
Gender	Male	25 (51.02)	32 (42.11)	57 (45.6)	1.43 (0.70-2.95)	0.95
	Female	24 (48.98)	44 (57.89)	68 (54.4)	1.0	
Age Group	40<	26 (53.06)	36 (47.37)	62 (49.6)	1.26 (0.61-2.58)	0.38
(years)	≥40	23 (46.94)	40 (52.64)	63 (50.4)	1.0	
Drug Regimens	ATD	12 (24.49)	20 (26.3)	32 (25.6)		0.23
	HAART	31 (63.27)	45 (59.21)	76 (60.8)		
	ATD/HAART	06 (12.24)	11 (14.47)	17 (13.6)		
Other	Yes	10 (20.4)	27 (35.53)	37 (29.6)	0.47 (0.20-1.08)	3.27
Medication	No	39 (79.6)	49 (64.47)	88 (70.4)	1.0	
Alcoholism	Yes	29 (59.18)	50 (65.79)	79 (63.2)	1.33 (0.63-2.78)	0.75
	No	20 (40.82)	26 (34.21)	46 (36.8)	1.0	
OIs	Yes	19 (38.78)	15 (19.74)	34 (27.2)	0.39 (0.17-0.87)	5.45
	No	30 (61.22)	61 (80.26)	91 (72.8)	1.0	
Smoking	Yes	01 (2.04)	1 (1.32)	02 (1.6)	1.60 (0.10-26.25)	0.11
	No	48 (97.96)	75 (98.68	123 (98.4)	1.0	
BMI	Underweight	01 (2.04)	9 (11.84)	10 (8.0)		6.83
	Normal	30 (61.22)	42 (55.26)	72 (57.6)		
	Overweight	11 (22.44)	21 (27.63)	32 (25.6)		
	Obesity Risk	7 (14.29)	4 (5.26)	11 (8.8)		
Total		49 (39.2)	76 (60.8)	125 (100)		

Binary logistic regression; CI=Confidence Interval; OIs=Opportunistic Infection; BMI= Body Mass Index; ATD – Antituberculosis Drugs; HAART – Highly Active Antiretroviral Therapy

In addition, a significant association existed with LDLc levels in patients who took other medications (antimalarial, paracetamol) within the follow-up period, implying that those on other medications are 0.23 times likely to induce abnormal LDL levels (χ^2 = 3.31, p = 0.0009; OR = 0.23; 95% C.I = 0.09-0.55) (Table IV).

Table IV: Relationship between some risk factors and abnormal LDLc levels

Parameters		*LDLc*		*Total (%)*	*O.R (95%C.I)*	χ^2/*F*
		Abnormal (%)	Normal (%)			
Gender	Males	21 (53.85)	36 (41.86)	57 (45.6)	0.7 (0.35-1.67)	0.657
	Females	18 (46.15)	50 (58.14)	68 (54.4)	1.0	
Age Group	40<	18 (46.15)	44 (51.16)	62 (49.6)	0.9 (0.41-1.98)	0.239
	≥40	21 (53.85)	42 (48.84)	63 (50.4)	1.0	
Drug	ATD	8 (20.51)	24 (27.91)	32 (25.6)		2.596
Regimens	HAART	23 (58.98)	53 (72.09)	76 (60.8)		
	ATD/HAART	8 (20.51)	9 (10.47)	17 (13.6)		
Other	Yes	16 (41.02)	21 (24.42)	37 (29.6)	0.23 (0.09-0.55)	3.310
Medication	No	23 (58.98)	65 (75.58)	88 (70.4)	1.0	
Alcoholism	Yes	11 (28.21)	35 (40.70)	79 (63.2)	1.7 (0.76-3.96)	1.334
	No	28 (71.79)	51 (59.30)	46 (36.8)	1.0	
OIs	Yes	11 (28.21)	23 (26.74)	34 (27.2)		
	No	28 (71.79)	63 (73.26)	91 (72.8)		
Smoking	Yes	1 (2.56)	1 (1.16)	02 (1.6)	0.43 (0.02-7.17)	0.580
	No	38 (97.44)	85 (98.84)	123 (98.4)	1.0	
BMI	Underweight	3 (7.69)	7 (8.14)	10 (8.0)		0.174
	Normal	23 (58.98)	49 (56.97)	72 (57.6)		
	Overweight	10 (25.64)	22 (25.58)	32 (25.6)		
	Obesity Risk	3 (7.69)	8 (9.30)	11 (8.8)		
Total		39 (31.2)	86 (68.8)	125 (100)		

O.R - Odds Ratio; CI - Confidence Interval; OIs - Opportunistic Infection; BMI - Body Mass Index; ATD – Antituberculosis Drugs; HAART – Highly Active Antiretroviral Therapy

A high frequency of abnormal LDLc (8/17(47.05%) was observed in participants on HAART/ATD, followed by (23/76(30.26%) among patients on HAART, and lastly (8/32(25%) among those on ATD only.

Furthermore, a significant association between TG level and BMI (χ^2 =13.95, p = 0.003), with a high frequency [6/11(54.5%)] of abnormal TG, was observed among participants at risk of developing obesity (Table V). Based on the respective infection groups, a high frequency of abnormal TG was observed in those on HAART [23/76(30.3%)], followed by HAART/ATD [5/17 (29.4%)] and lastly, ATD [8/32(25.0%)].

There was a significant association between TC level and the type of drug regimen (χ^2 = 6.98, p = 0.03), with a higher frequency [8/17(47.1%)] of abnormal TC observed among ATD/HAART participants compared to their counterparts. Also, there was a 0.42 times likelihood for females to develop abnormal TC than males (χ^2 = 2.03, p = 0.043; OR = 0.42, 95% C.I = 0.18-0.97) (Table VI). There was no significant relationship between the TC and age group, but participants above ≥40 years had a high frequency [21/33(63.64%)] and 0.48 likelihood of developing abnormal TC (Table IV).

Discussion

Lipid abnormalities have been reported to occur in HIV infection because of the infection-induced acute phase response. Lipid abnormalities may also result from host response because of cytokines (TNF, interleukins and interferon) produced by the infection. [15] Because of the combined immune system deterioration and metabolic dysfunction caused by HIV and TB, the host's health has been known to deteriorate. [15] The study was aimed at assessing the variation of the levels of lipid parameters in HIV, TB and TB/HIV patients during a follow-up period of 12 weeks. The study compared the levels of serum lipid profiles before the initiation of treatment and after the initiation of therapy.

Table V: Relationship between some risk factors with abnormal triglycerides levels

Parameters		*TG*		*Total (%)*	*Odd Ratio (CI)*	χ^2/F
		Abnormal (%)	Normal (%)			
Gender	Male	13 (36.11)	44 (49.44)	57 (45.6)	0.57 (0.26-1.28)	1.35
	Female	23 (63.89)	45 (50.56)	68 (54.4)	1.0	
Age Group	40<	15 (41.67)	47 (52.81)	62 (49.6)	0.64 (0.29-1.40)	1.13
(years)	≥40	21 (58.33)	42 (47.19)	63 (50.4)	1.0	
Drug Regimens	ATD	08 (22.22)	24 (26.97)	32 (25.6)		0.70
	HAART	23 (63.89)	53 (59.55)	76 (60.8)		
	ATD/HAART	5 (13.89)	12 (13.48)	17 (13.6)		
Other	Yes	9 (25.0)	28 (31.46)	37 (29.6)	1.03 (0.42-2.51)	0.055
Medication	No	27 (75.0)	61 (68.54)	88 (70.4)	1.0	
Alcoholism	Yes	11 (30.56)	35 (39.33)	46 (36.8)	0.68 (0.30-1.55)	0.92
	No	25 (69.44)	54 (60.67)	79 (63.2)	1.0	
OIs	Yes	8 (22.22)	26 (29.21)	34 (27.2)	1.39 (0.56-3.46)	0.71
	No	28 (77.78)	63 (70.79)	91 (72.8)	1.0	
Smoking	Yes	0 (00.0)	2 (2.25)	02 (1.6)		
	No	36 (100)	87 (97.75)	123 (98.4)		
BMI	Underweight	4 (11.11)	6 (6.74)	10 (8.0)		13.95
	Normal	24 (66.67)	48 (53.93)	72 (57.6)		
	Overweight	2 (5.56)	30 (33.71)	32 (25.6)		
	Obesity Risk	6 (16.67)	5 (5.62)	11 (8.8)		
Total		36 (28.8)	89 (71.2)	125 (100)		

CI - Confidence Interval; OIs - Opportunistic Infection; BMI - Body Mass Index; ATD – Antituberculosis Drugs; HAART – Highly Active Antiretroviral Therapy

The high frequency of 42.4% (53/125) of abnormal HDLc in the treatment-naïve patients recorded in this study was in line with previous reports. [4, 5] It has been reported that low levels of high-density lipoprotein (HDL-C) are typical in naïve/untreated TB and HIV patients, which could be due to chronic inflammation and immune system activation.[5] Comparing the incidence of these lipid abnormalities during the follow-up period shows that the alteration is much more noticeable between 4-12 weeks, in line with the observations of Wilson *et al*. [16]. The steady decrease of the HDLc was in line with other studies, which reported an association of decreased plasma concentration of HDL with immune activation.[17, 18] This may be because of the increased activity of cholesterol ester transfer protein (CETP), leading to an increased cholesterol ester transfer from HDL to apo B-lipoproteins. In addition, there is a negative correlation between CETP activity and HDLc, with cholesterol being diverted to very low-

density lipoprotein and LDL-C by the high activity of CETP. [15, 17] There was a gradual and significant decrease in HDLc during the follow-up period. This could be related to some infections, such as malaria, within the follow-up period, which several studies have associated with the alteration of lipid profile. [19, 20]

Table VI: Relationship between some risk factors and abnormal triglycerides levels

Parameters		*TC*		*Total (%)*	*O.R(95%C.I)*	χ^2/F
		Abnormal (%)	*Normal (%)*			
Gender	Male	10 (30.3)	47 (51.09)	57 (45.6)	0.42 (0.18-0.97)	4.23
	Female	23 (69.7)	45 (48.91)	68 (54.4)	1.0	
Age Group	40<	12 (36.36)	50 (54.35)	62 (49.6)	0.48 (0.21-1.08)	1.76
(years)	≥40	21 (63.64)	42 (45.65)	63 (50.4)	1.0	
Drug Regimens	ATD	4 (12.12)	28 (30.43)	32 (25.6)		6.98
	HAART	21 (63.64)	55 (59.78)	76 (60.8)		
	ATD/HAART	08 (24.24)	9 (9.78)	17 (13.6)		
Other	Yes	14 (42.42)	23 (25.0)	37 (29.6)	2.21 (0.96-5.10)	3.53
medication	No	19 (57.58)	69 (75.0)	88 (70.4)	1.0	
Alcoholism	Yes	10 (30.3)	36 (39.13)	46 (36.8)	0.68 (0.28-1.59)	0.90
	No	23 (69.7)	56 (60.87)	79 (63.2)	1.0	
OIs	Yes	8 (24.24)	26 (28.26)	34 (27.2)	1.23 (0.49-3.08)	
	No	25 (75.76)	66 (71.74)	91 (72.8)	1.0	
Smoking	Yes	00 (00.0)	2 (2.17)	02 (1.6)		
	No	33 (100)	90 (97.83)	123 (98.4)		
BMI	Underweight	2 (6.06)	8 (8.70)	10 (8.0)		1.18
	Normal	20 (60.6)	52 (56.52)	72 (57.6)		
	Overweight	7 (21.21)	25 (27.17)	32 (25.6)		
	Obesity Risk	4 (12.12)	7 (7.61)	11 (8.8)		
Total	-	33 (26.4)	92 (73.6)	125 (100)		

O.R - Odds Ratio; CI - Confidence Interval; OIs - Opportunistic Infection; BMI - Body Mass Index; ATD – Antituberculosis Drugs; HAART – Highly Active Antiretroviral Therapy

A significant association was observed between the level of TG and BMI ($\chi^2 = 13.95$; $p = 0.003$), with those at risk of developing obesity having a high frequency of abnormal TG [6/11 (54.5%)]. The increase in TG among participants at risk of developing obesity could be due to decreased clearance of triglyceride-rich lipoproteins and increased hepatic production of very low-density lipoproteins (VLDL) particles. [21] As suggested by some previous studies, [9, 19] the main metabolic risk factor for the development of atherogenic dyslipidaemia is increased plasmatic lipoproteins rich in TG and atherogenic particles, among other factors, including alcoholism. [8, 16] Alcoholic consumption has been reported by Ali *et al*. [22] to be related to the increase in serum TG levels, just as observed in this study. The biosynthesis of lipids in the liver has been reported to be accelerated by excessive consumption of alcohol. [23, 24]

One of the leading causes or risk factors for cardiovascular diseases such as arteriosclerosis is hypercholesterolaemia. [22] Its high frequency observed in females may be because of hormonal and metabolic alterations. Total cholesterol has been reported to be affected by age and sex, among other factors. [13, 25-27] Ageing affects the liver's ability to metabolise LDL cholesterol by up to about 35% due to shortened telomeres on chromosomes, causing cells to lose the ability to reproduce and replace damaged cells.

Cholesterol levels generally increase with age.[13, 23-25] This was also observed in the current study, with participants aged ≥40 years showing a higher risk of developing abnormal TC. The high TC frequency in older people could be related to youths being more physically active than the ageing population. Also, the high frequency of 33.8% (23/68) of abnormal TC observed in women when compared to men could be hormonal-related, most especially in older women who have gone through menopause due to lower oestrogen levels. [25-27]

Rational and Strengths of the Study

This was a prospective study conducted over 12 weeks, which revealed the most liable risk factors, causes and effects of serum lipid abnormalities in HIV/AIDS, TB and HIV/AIDS/TB patients on treatment. This study contributed by explaining some ways to improve care for HIV, TB, and HIV/TB co-infected persons. Furthermore, it indicates that persons living with HIV/TB co-infections are more at risk of suffering from cardiovascular and metabolic abnormalities; thus, early screening and checking of serum lipid profiles are essential.

Limitations of the Study

A limitation of the study was that there was no deliberate monitoring of the medications consumed during the three months of follow-up, which may have consequences on the liver.

Conclusion

This study reinforces the fact that serum lipid profiles should be routinely checked in patients with HIV/AIDS and TB infections on treatment since there is a likelihood for patients on treatment to develop abnormal serum lipid profiles. Also, it shows that TB and HIV/AIDS alter serum lipid levels, and their respective treatments can exacerbate these effects, with TC levels being affected significantly by the combined ATD/HAART therapy. The study showed that the impact of ATD/HAART on serum lipids is more noticeable between 4-12 weeks after initiation of treatment. Therefore, understanding these changes is crucial to patient management.

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