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Pattern of Lipid Profile and Fasting Blood Glucose Among Human Immunodeficiency Virus Positive Patients on First Line Drugs Over 24 Months Period in Metabolic Syndrome Study in Sokoto Nigeria

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ABSTRACT

Background: The introductions of highly active antiretroviral therapy (HAART) has improved the live expectancy of patients with HIV/AIDS, but is associated with increase cardiovascular related morbidity among the recipient of the drugs.
Objectives: The aim of this study was to determine the prevalence of dyslipaedemias and the factors associated with lipid profile derangement among HIV positive patients on first line drugs.
Materials and Methods: This prospective study was conducted between July 2019 and June 2021. Eighty-six (86) HIV seropositive HAART naïve subject were recruited for the study at institute of human virology centres (IHVN) of Usmanu Danfodiyo University Teaching Hospital Sokoto. Lipid profile and fasting blood glucose were estimated after overnight fast and National cholesterol Education program adult treatment panel was used to determine dyslipaedemias and cut off for fasting blood glucose level. Baseline sample were taking before commencing the participants on first line ART regiment and flow up for twenty-four months (24 months). Repeated sample collection was done at six months intervals. The data were collected using interviewer administer questionnaire and analyses using SPSS IBM Statistical package version 23v.
Results: Of the eighty-six-participant recruited for the study, 47(54.7%) were female and 39(45.3%) are male. The baseline data was collected before placing the patients on first line antiretroviral drugs containing Lamivudine Tenofovir and Dolutegravil combination for all the patient. The mean age was 34.98 years, mean body mass index 19.22kg/M^2 and mean WHR was 0.84. Mean SBP and DBP was 110.6mmHg and 75.3mmHg respectively. The prevalence of hyperglycaemia, hypercholesterolemia, hypertriglyceridemia's, LDL-C hypercholesterolaemia and decrease HDL-C at baseline were 5.9%, 0%, 52.3%, 1.2%, and 55.9% respectively. Nonetheless the prevalence of dyslipidaemias and hyperglycaemia was statistically increase after commencing ART over the period of 24 months.
Conclusion: In this study of newly diagnosed HIV positive patients' lipids were irregularly elevated at baseline through 24 months, HDL-C is the most derange lipid component seen among HIV positive patients HAART naïve and HAART exposed patients.

Key word: Pattern, Lipid profile, Blood glucose, metabolic syndrome, Study.

INTRODUCTION

Abnormalities in serum lipid and lipoprotein levels (dyslipidaemia) are recognized as major modifiable cardiovascular disease (CVD) risk factors[1] and have been identified as independent risk factors for essential

hypertension. Although some study reported that dyslipidaemia is not risk for CVD [2], dyslipidaemia remain a major risk factor for coronary heart disease (CHD) and it refers to lipid abnormalities such as high total cholesterol (TC), elevated low density lipoprotein cholesterol (LDL-C), hypertriglyceridemia, low high density lipoprotein cholesterol (HDL -C) and high levels of lipoprotein (a). The World Health Organization estimated dyslipidaemia to be associated with more than 50% of cases of ischemic heart disease world -wide and more than 4 million deaths annually [3].

Dyslipidaemia could be familial or secondary to several conditions which include type 2 diabetes mellitus, hypothyroidism, excessive alcohol consumption, cholestatic liver disease, kidney disease, smoking and obesity[4]. Since the early days of the human immunodeficiency virus (HIV) epidemic, the effects of HIV disease on serum lipids have been described. With the use of potent antiretroviral therapy in patients with HIV disease, changes in lipid parameters and glucose homeostasis have been noted. However, these effects have been difficult to interpret because of the varied demographic and treatment characteristics of the cohorts and the complexity of differentiating the effect of HIV disease from that of the drugs used in its treatment[5]. Both HIV disease and demographic characteristics were found to influence lipid values and glucose homeostasis in the absence of antiretroviral treatment. More advanced HIV disease was associated with less favourable lipid and glucose homeostatic profiles [6]. It has been postulated that HIV infected individuals experience low level of LDL-C and HDL-C and elevated serum concentration of triglyceride years prior to developed AIDS [7]. Treatment with PIs and NNRTIs is associated with many metabolic disorders such as dyslipidaemia that may increase risk of coronary heart disease. Furthermore, HIV infected persons are subject to dyslipidaemia and other complication due to HAART which are called HIV MetS[8]. Studies among HIV infected individual revealed a range of lipids disorders, even before ART are readily available. Report have indicated low concentration of various lipid components among HIV infected persons [9]. Lipid values and glucose homeostasis were influence by the disease and demographic characteristic even in the absence of ART, with less favourable lipid and glucose homeostasis in more advance disease. Lipids and HIV RNA level have independent association that suggest viral replication had effect on lipid concentrations [6]. Elevated total cholesterol and decrease HDL cholesterol increase the risk of cardiovascular disease independent of hs-CRP [10]. The independent association between HIV RNA levels and various lipid parameters suggests that viral replication had a direct effect on lipid levels. Interpretation of the effects of various HIV treatment regimen and drugs on metabolic parameters must take into account the stage of HIV disease and the demographic characteristics of the population studied. Shafran *et al.*, (2005) [11] reported that, Ritonavir dosed at 100mg bid significantly increased the concentration of total cholesterol, LDL cholesterol, total/HDL cholesterol ratio and triglycerides and reduced HDL cholesterol concentration. The addition of LPV 400mg bid to RTV 100mg bid further increased both total and HDL cholesterol levels without affecting the total/HDL ratio. Reported by Buchacz *et al.*, in rural Ugandans [12] that with advanced HIV disease, initiating Nevirapine- or Efavirenz-based HAART experienced in-frequent elevations in TC, LDL-c, and TG at baseline and after 24 months of therapy. Increases in HDL-c levels were substantial and proportionally greater than increases in TC or LDL-c levels. The risk of CVD and how it is affected by lipid changes in this rural African population are unknown[12]. However, the changes observed after 24 months of highly active antiretroviral therapy (HAART) seem unlikely to increase the risk of CVD. However, HAART is linked to increase in total cholesterol [9] and

triglycerides [13]. Although, total cholesterol increase in therapy could signify a revival of pre infection level to some extent [9], but increased risk of MI is linked to use of HAART. However, exposure to some class of drugs in HAART combination such as protease inhibitors (PI) is widely linked to dyslipidaemia [14,15].while some studies suggest that lipid profile improves when PI is replaced with non-nucleoside reverse transcriptase inhibitors (NNRTI) in the therapy [16,17]. Furthermore, treatment of HAART naïve patient with Nevirapine or Efavirenz as first line drugs may lead to increase in HDL-C levels [18], but most of these studies lack adequate flow up period(< 1 year) or have in adequate sample size(<100).

MATERIALS AND METHODS

This is a Prospective study which was conducted at the Department of Chemical Pathology and immunology, Faculty of Basic Clinical sciences, College of Health Science, Usmanu Danfodiyo University Sokoto and Institute for Human virology of Nigeria (IHVN) in Sokoto metropolis for the period of two year. Eighty-six (86) HIV positive HAART naïve patients aged 18 years and above were recruited for the study. Baseline sample were taking before commencing the participants on first line ART regiment and flow up for twenty-four months (24 months). Repeated sample collection was done at six months intervals. We lost two patients within first six after commencing ART, but they are within the 20% attrition factor considered during sample size estimation.

The patients were instructed to fast over night for ten hours. Seven millilitres (7mls) of venous blood were taken 2mls was transfer in to lithium oxalate bottle for glucose analysis while five millilitres (5mls) were allowed to clot and serum harvested and stored at -20^{o}C for lipid analysis later. The patients were initiated on regiment containing dolutegravir, lamivudine and Tenofovir (DTG) base HAART regiment. Repeated sample collection and anthropometric measurement were done at six months intervals.

The serum triglyceride (TG) concentrations were estimated using the method of Trinder [19] using Kits procure from Randox laboratories England. The concentration of serum cholesterol was estimated by the enzymatic method of Allain, Poon, Chan, Richmond and Fu [20] Using reagent Kits which was procure, from Randox laboratories England. The serum high density lipoprotein cholesterol (HDL-C) Concentration were estimated by method of Burstein [21] using reagent Kits procure, from Randox laboratories England. Precipitation of LDL-C is done by addition of heparin to obtained HDL-C and very low-density lipoprotein (VLDL in supernatant after centrifugation and are measured enzymatically by the CHOD-PAP method. The LDL-C concentration is calculated as the difference of total cholesterol and that of supernatant. Serum glucose was measured by the glucose oxidase using the method of Trinder [22] using the laboratory kit from Randox laboratory England. Chemistry semi auto analyser was used for estimation of the analytes. The ethical clearance was obtained from Usmanu Danfodiyo University Teaching Hospital Sokoto (UDUTH) research and ethical committees. This was part of the study titled: "Assessment of metabolic syndrome and its Components among human immunodeficiency syndrome (HIV) Seropositive patients in Sokoto" with reference number (UDUTH/HREC/2019/ NO. 827). Likewise, written informed consent was also obtained from the individual subjects. All the participants that fulfil inclusion criteria were eighteen years and above.

STATISTICAL ANALYSIS

The cut-off values for lipid concentration were determine using national cholesterol education programme adult treatment panel III (NCEP-ATP III) as TC≥200mg/dl LDL ≥130mg/dl, HDL < 40mg/dl and TG ≥150mg/dl define as abnormally elevated. Baseline lipids concentration was compared

with measurement done at six months intervals post ART using one way ANOVA. Spearman's correlation was used to determine the association of viral load with lipid parameter among study subjects. The data were collected using interviewer administer questionnaire and analyses using SPSS IBM Statistical package version 23v.

RESULTS

The results of the current study were presented in table 1 to 4

Table 1: Shows the prevalence of lipid profile and blood glucose levels at baseline through 24 months post ART exposure. At baseline reduced HDL-C is the most prevalent lipid component seen followed by increase triglycerides (55.8% and 52.3%) respectively. The percentage reduces gradually and raise high again at twenty-four-month post ART. These indicated that reduced HDL-C is the most lipid abnormality in this study. Likewise, elevated fasting blood glucose level is seen in 5.8% of study subjects at baseline but rose to 52.4% at 24 months post ART exposure. However, none of the subjects was seen with elevated total cholesterol at baseline but its raises to 29.8% at 24 months post exposure.

Table 1: Prevalence of lipid profile component and blood glucose levels among subjects at baseline and at six months interval before and after ART initiation using NCEP ATP III Criteria.

Parameters	Baseline n (%)	Six months n (%)	Twelve months n (%)	Eighteen months n (%)	Twenty-four months n (%)
TC (mg/dl)	0(0)	12(14.3)	7(8.3)	19(22.6)	25(29.8)
TG (mg/dl)	45(52.3)	34(40.5)	18(21.4)	24(28.6)	30(35.7)
HDL (mg/dl)	48(55.8)	34(40.5)	36(42.9)	27(32.1)	41(48.8)
LDL (mg/dl)	1(1.2)	12(14.3)	4(4.8)	9(10.7)	14(16.7)
FBG (mmol/L)	5(5.8)	52(61.9)	51(60.7)	32(38.1)	44(52.4)

TC, Total cholesterol; TG, Triglycerides; HDL, High density lipoprotein cholesterol; LDL, Low density lipoprotein cholesterol; FBG, Fasting blood glucose. Statistic by Descriptive statistics Data are frequency

Table 2: Is comparison of mean hip circumference, waist to hip ratio, systolic blood and diastolic blood pressure, lipid profile parameters and fasting blood glucose over twenty-four months. The anthropometric measurements were statistically higher after commencing HAART than at base line and increases with increasing duration of HAART. Likewise, SBP and DBP were significantly higher after commencing HAART. However, the mean concentration of lipid profile parameter was significantly higher at various levels of measurement. TC, TG and LDL-C were significantly higher post HAART exposure and the mean concentration increases with increasing duration of HAART. Furthermore, decrease in HDL-C concentration is statistically higher after HAART exposure than at baseline.

Table 2: Comparison of mean hip circumference, waist to hip ratio, systolic blood and diastolic blood pressure, lipid profile parameters and fasting blood glucose over twenty-four months (Mean± SE)

Parameters	Baseline	Six months	Twelve months	Eighteen months	Twenty four months	P value
HC (cm)	85.42±1.20	92.06±1.05	91.08±0.94	92.06±1.05	93.27±1.14	0.000
WHR (cm)	0.84±0.01	0.86±0.01	0.86±0.01	0.86±0.01	0.85±0.01	0.000
BMI (kg/m^2)	19.2±0.49	21.5±0.40	23.5±0.41	21.5±0.41	21.5±0.41	0.012
SBP (mmHg)	110.6±2.04	128.3±1.87	126.6±1.53	126.9±1.49	125.1±2.36	0.000
DBP (mmHg)	75.3±1.25	82.9±1.24	82.0±0.93	81.8±0.94	84.6±1.48	0.000
TC (mmol/l)	3.02±0.07	3.43±0.15	3.62±0.12	4.11±0.12	4.12±0.16	0.000
TG (mmol/l)	1.88±0.10	1.32±0.08	1.24±0.05	1.36±0.07	1.72±0.10	0.000
HDL (mmol/)	0.98±0.04	0.95±0.06	1.24±0.06	1.50±0.06	1.23±0.06	0.000
LDL (mmol/l)	1.26±0.08	2.12±0.10	1.92±0.08	2.14±0.14	2.11±0.14	0.000
FBG (mmol/l)	4.62±0.12	6.22±0.14	6.34±0.21	5.96±0.16	6.35±0.13	0.000

HC, Hip circumference; WHR, Waist to hip ratio; BMI, Body mass index; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; TC, Total cholesterol; TG, Triglycerides; HDL, High density lipoprotein cholesterol; LDL, Low density lipoprotein cholesterol; FBG, Fasting blood glucose. Statistic by one way ANOVA

Table 3: Baseline values and lipids levels changes from baseline to six months, twelve months, eighteen months and twenty-four months among HIV positive patients who commence HAART between June 2019 to May 2021. Total cholesterol has significantly elevated from baseline 3.02(2.89-3.16) 95% IC. To 3.42(3.13-3.72) 95% CI at six months and proportionally increasing at twelve, eighteen and twenty-four months respectively. TG concentrations is irregularly decreasing between baseline and twenty-four months 1.88(1.68-2.08) 95% CI and 1.73(1.53-1.92) 95% CI at baseline and twenty-four months respectively. Decrease HDL-c is significantly higher at baseline than six months post HAART but gradually increases at twelve months to twenty-four months mean (95% CI) 0.98(0.89-1.07), 0.95(0.83-1.07), 1.24(1.12-1.136), 1.50 (1.391-1.62) and 1.23(1.12-1.34) respectively. Furthermore LDL-c is statistically increasing from pre-HAART at baseline to twenty-four months post HAART 1.26(1.12-1.42 and 2.08(1.84-2.03) respectively. However, FBG has increase from 4.6(4.40-4.86) Mean (95% CI) at baseline to 6.2(5.94-6.51) six months post HAART.

Table 3: Mean values and changes in lipids profile levels from baseline to six months, twelve months, eighteen months and twenty-four months among HIV positive patients who commence HAART between June, 2019 to May 2021.

	Baseline	6 months	12 months	18 months	24 months
Lipids (mmol/L)		**Mean(95%CI)**			
TC	3.02(2.82-3.16)	3.42(3.12-3.72)	3.62(3.38-3.86)	4.11(3.88-4.35)	4.11(3.81-4.43)
TG	1.88(1.68-1.07)	1.32(1.14-1.17)	1.24(1.13-1.34)	1.37(1.22-1.51)	1.73(1.53-1.92)
HDL-c	0.98(0.89-1.07)	0.95(0.83-1.07)	1.24(1.12-1.36)	1.50(1.39-1.62)	1.23(1.12-1.34)
LDL-c	1.26(1.12-1.42)	2.12(1.88-2.35)	1.92(1.75-2.08)	2.14(1.94-2.35)	2.08(1.84-2.03)
FBG	4.60(4.40-4.86)	6.22(5.94-6.51)	6.3(5.92-6.76)	5.96(5.65-6.27)	6.35(6.09-6.61)

TC, Total cholesterol; TG, Triglycerides; HDL-c High density lipoprotein cholesterol; LDL-c Low density lipoprotein cholesterol; FBG, Fasting blood glucose statistic one way ANOVA.

Table 4: Shows the correlation of viral load with lipid profile, anthropometric and clinical component of MetS among study subjects over twenty-four months. Triglycerides were positively correlated with viral load (r= 0.260 p= 0.000). While TC, HDL-C and LDL-C were negatively correlated with viral load (r= -0.194, -0.168, -0.216 p=0.000) respectively. Furthermore, the anthropometric parameters were all negatively correlated with viral load except waist to hip ratio that has no statistical correlation with viral load. Both systolic and diastolic blood pressure were inversely correlated with viral load (r= -0.283 and -0.185 p=0.000) respectively.

Table 4: Correlation of viral load with lipid profile, anthropometric and Blood pressure among study subjects.

Parameter	Correlation coefficient (r)	P value
TC (mmol/l)	-0.194	0.000
TG (mmol/l)	0.260	0.000
HDL (mmol/l)	-0.168	0.000
LDL (mmol/l)	-0.216	0.000
FBG (mmol/l)	-0.391	0.000
HC (cm)	-0.220	0.000
WC (cm)	-0.216	0.000
WHR (cm)	-0.071	0.148
WT (kg)	-0.144	0.003
BMI (kg/m^2)	-0.170	0.000
SBP (mmHg)	-0.283	0.000
DBP (mmHg)	-0.185	0.000

TC, Total cholesterol; TG, Triglycerides; HDL, High density lipoprotein; LDL, Low density lipoprotein; HC, Hip Circumference; WC, Waist circumference; WHR, Waist to hip ratio; WT, Weight; BMI, Body mass index; SBP, Systolic blood pressure and DSP, Diastolic blood pressure. Statistics by Spearman correlation.

DISCUSSION

In our study, the population of patients with HIV infection there is sporadic elevation of lipid at base line and six months through twenty-four months. This was reported by Butchaz et al [12] that report infrequent elevation of lipid among rural Ugandan HIV population at baseline and at 24 months.

However, our study also reports reduced HDL-C as the most common lipid derangements at base line up to 55.8% and gradually increasing after commencing HAART. Which agrees with the report of [23] that report during the course of HIV disease serum lipid concentration naturally changes, with early decrease in HDL-C and total cholesterol and increase in triglycerides concentration later in HIV infection. However, TC gradually increase among patients after commencing HAART from 0% at baseline to 29.8% at twenty-four months. This agree with report of [2,9,23] that HAART is linked to increase in total cholesterol. Furthermore, there is increase in triglyceride and reduced high density lipoprotein that shows statistically significant difference (higher or lower) among HIV patients with MetS than those without MetS ($p < 0.005$). This agrees with work of [9,13,23] whose reports indicate an increase in triglycerides later in HIV Patients on HAART. Though, there was significant decrease in viral load in all the patients but there was significant change in lipid at different level of the study. Metabolic irregularities are associated with prolong HAART use among HIV positive patients such as glucose and lipid abnormalities as reported by Gallagher [24]. This was proved by our study that shows high increase in FBG level within six months of commencing HAART from 5.8% to 61.9% respectively. Studies among young healthy adult[25,26], both reports significantly high

The Mean (±SE) systolic and diastolic BP, WC, HC, BMI and WHR were seen to be higher in patients with than in those without the MetS ($p < 0.0001$). High body mass index is reported to significantly associated with MetS[27,28]. This agrees with the current study that shows higher BMI among patients with MetS than those without MetS. Fluctuation in BMI is noted in the current study over twenty-four-month period from 9.3% at baseline to 13.3% at 12month and later drop to 10.7% at 18 and 24 months respectively. This was reported in early studies that indicate change in nutritional profile with increase in obesity and decrease on thinness after introduction of HAART [28,29]. Likewise, WC component is significantly higher among female than male ($p<0.001$). The current study shows increase in hypertension with increasing exposure to HAART from (22.1% at baseline to 47.6% at 24months) in diastolic blood pressure, while it was 17.4% at baseline to 34.5% at 24months in systolic blood pressure. This agrees with report of [30] that report the link between hypertension and increasing exposure to HAART. Furthermore, some studies report high prevalence of hypertension among HIV positive patients[28,31], this correspond with the result of current study that report up to 58% prevalence of systolic hypertension and 47% diastolic hypertension respectively. Increase in the triglyceride (TG) component of MetS seen in 48.8% among HIV positive patients with a value above 1.7mmol/L in the present study, and 32.6% among controls. Likewise, a decrease in High-density lipoprotein cholesterol (HDL-C) is reported in 72.1% among HIV positive HAART naïve patients and controls, with value below 1.04mmol/l and 1.29mmol//L for males and females respectively. This agrees with the findings of Sani, Wahab, Yusuf, Gbadamosi, Johnson and Gbadamosi [32] who reports decrease HDL-C of 59.3% among HIV negative subjects. In the current study NCEP ATP III criteria identified more of increase in TG and decrease in HDL-C components of MetS than IDF Criteria (98.8% and 90.7% vs. 97.7% and 80.2%) for TG vs. HDL cholesterol respectively. This finding of current study also agrees with the study of [33,34], who reported a decrease in HDL-C as the most frequent component in their study, but not an increase in total cholesterol (TC). Even though the combination HAART used in the current study contained no Protease inhibitor (PI) there is statistically significant increase ($p =00001$) in concentration of all lipid components at various levels of

measurement. This agrees with Some early studies that suggested exposure to some class of drugs in HAART combination such as protease inhibitors may be linked to lipid abnormalities [14,15]. Elevated fasting blood glucose (FBG) is the least component seen in the present study with only 5.8% at baseline. This agrees with the findings of Carr, Utzschneider, Hull, Kodama, Retzlaff, Brunzell, Shofer, Fish, Knopp and Kahn [35] who reported only a 5.5% elevation in FBG among HIV positive patients.

However, the prevalence of FBG statistically increase after commencing HAART up to 82.1%, 78.6%, 57.1% and 76.2% at six months, twelve months, eighteen months and twenty-four months respectively. This agrees with the report of Castaneda-Sceppa, Bermudez, Wanke and Forrester [36] who report HARRT may affect insulin resistance independent of HIV infection with no association with Protease inhibitors. Metabolic irregularities are associated with prolong HAART use among HIV positive patients such as glucose and lipid abnormalities as reported by Gallagher [24]. This was substantiated by the current study that shows high increase in FBG level within six months of commencing HAART from 5.8% to 61.9% respectively. Though, there was significant decrease in viral load in all the patients in our study, but there was significant change in lipid at different level of the study, and both lipid profile parameters, FBG, anthropometric and blood pressure were inversely correlated to the viral load among study subjects in our study except for TG which positively correlated to the viral load ($p = 0.0001$). This agree with study of Tort et al, [37] who reported the effect of viral load on cholesterol efflux was initially positive but later turn negative.

CONCLUSION

In this study of newly diagnosed HIV positive patients' lipids were irregularly elevated at baseline through 24 months, HDL-C is the most derange lipid component seen among HIV positive patients at baseline and post HAART exposure. Likewise, FBG is significantly elevated after HAART exposure than pre-HAART period. Furthermore, viral load is negatively correlated with lipid profile parameters and FBG.

Limitation: lack of ample time to follow up the patients to properly document the lipid changes is a big constrain in this study. There is also need for a large cohort study that will allow for proper evaluation of statistically significant of the outcome in our study.

Author's contribution: All mentioned authors were fully involved in the research. At conception and design of the study, performed data analysis and interpretation of data and manuscript preparation. They all read and accepted the final draft of manuscript.

Declaration by Authors

Ethical Approval: Approved

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