ejpmr, 2022, 9(4), 34-44

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Research Article

ISSN 2394-3211

EJPMR

PREVALENCE OF METABOLIC SYNDROME COMPONENTS AND PREDICTORS AMONG HUMAN IMMUNODEFICIENCY VIRUS POSITIVE HIGHLY ACTIVE ANTIRETROVIRAL THERAPY NAÏVE PATIENTS

[1,2]*Bello Aminu, [1,2]Anaja Peter O., [2]Rasheed Yusuf, [1]Abdul Rahman Muhammad Bashir, [1,2]Abubakar Abdullahi Fakku and [1]Umar Aminu Abdullahi

[1]Department of Chemical Pathology and Immunology, Faculty of Basic Clinical Sciences, College of Health Sciences Usmanu Danfodiyo University, Sokoto. Sokoto State, Nigeria.
[2]Department of Chemical Pathology, Faculty of Basic Clinical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria. Kaduna State, Nigeria.

***Corresponding Author: Bello Aminu**
Department of Chemical Pathology and Immunology, Faculty of Basic Clinical Sciences, College of Health Sciences Usmanu Danfodiyo University, Sokoto. Sokoto State, Nigeria.

Article Received on 22/01/2022 **Article Revised on 12/02/2022** **Article Accepted on 05/03/2022**

ABSTRACT

Background: The current study aims to determine the prevalence of metabolic syndrome (MetS) components and their predictors among HIV seropositive Highly Active Antiretroviral therapy (HAART) Naïve patients and controls in Sokoto. **Materials and Method:** Fasting blood glucose, lipid profile anthropometric parameters as well as urine creatinine, micro-albumin and blood pressure were measured in 86 HIV positive HAART naïve patients aged 18years and above and 86 HIV negative age and sex-matched controls. The receiver operating characteristic (ROC) curve was deployed to calculate the cut-off value for the predictor of MetS, Spearman correlation was used to determine the association between the development of MetS and its predictors. **Results:** Among 86 HIV Seropositive patients 15(17.4%) had MetS of which 14(16.3%) fulfils the NCEP ATP III Criteria and 11(12.8%) fulfils the IDF criteria and 10(11.6%) satisfied both NCEP ATP III and IDF criteria. There are more women with MetS 11(12.8%) than men 4(4.7%). When compared to healthy controls, 16(18.6%) were confirmed with MetS, of which 9(10%) and 12(13.9%) fulfil the NCEP ATP III and IDF criteria respectively. Male constitute 5(5.8%) and female make up to 11(12.8%) respectively. There is a static significant difference in anthropometric parameters and systolic blood pressure between MetS and non-MetS groups ($p<0.0001$) but not diastolic blood pressure and also between male and female patients, increase TG ($p<0.001$) decrease HDL ($p=0.002$) with no significant difference in FBG. Urinary creatinine and albumin creatinine ratio positively correlate the development of MetS in case but not in controls ($p= 0.017$, $p= 0.016$) and ($p=0.467$,$p=0.288$)respectively. **Conclusion:** The prevalence of MetS was higher among controls than the HAART naïve patients, Female Gender, and advanced age were found to be significantly associated with MetS. Anthropometric parameters and Systolic Blood Pressure were better predictors of MetS than lipid profile parameters and FBG.

KEYWORDS: Metabolic Syndrome, Predictors, Prevalence, HIV, HAART, Naïve.

INTRODUCTION

Metabolic syndrome (MetS) is an imminent problem and the prevalence of overweight and obesity is increasing rapidly among HIV subjects.[1] MetS is a group of cardiovascular disease and type 2 diabetes-associated disorders with prevalence among human immunodeficiency virus (HIV) infected patients extremely increasing in epidemic proportion globally.[2] The prevalence of MetS among HIV infected individuals is lower than that reported among the general population.[3] A spectrum of metabolic disorders occurs in patients with HIV infections on highly active antiretroviral therapy (HAART) such as dyslipidaemias, changes in body fat, insulin resistance and peripheral lipoatrophy.[3] The report on the prevalence of MetS in HIV infected patients is very scarce in North-western Nigeria, Sokoto in particular. However, the prevalence of MetS and its components is on the increase, mirroring its rise as a consequence of changes in lifestyle. Besides, urbanization and industrialization are also implicated, as indicated by a study on the trend of MetS in the US which shows a marginal reduction in its prevalence.[4] Moreover, in the study by Dutra, de Carvalho[5] among the Brazilian population they report the prevalence of 32.0%, Gyakobo, Amoah[6] also reported a MetS prevalence of 35.9% in a rural population of Ghana. The prevalence of MetS was found to be 6.8% among female students in United Arab Emirates University.[1] Equally, among urban dwellers in Nigerian cities, Ojji, Ajayi[7] reported a low prevalence of MetS of 13%,

Siminialayi,[8] reported a high prevalence of 35.4% in Abuja and River state respectively. Furthermore, the Sokoto community base study reported MetS prevalence of 35.1%[9], 17.8% among civil servants.[10] A prevalence of 59.1% was reported among type 2 diabetic native Africa.[11] Furthermore, the prevalence of metabolic syndrome among HIV infected people was reported to be 14%-18%[3], 15.6% was reported in south-western Cameroon[12], 24%-48% in Ghana.[13] In Nigeria Uwanuruochi, Michael[14] reported the prevalence of 24.3% in the south eastern part of the country, in Kano north-western Nigeria Muhammad, Gezawa[15] reported the prevalence of 19.3% and 5.3% in north-western city of Kano among ART and ART naïve patients respectively. However, the prevalence of MetS was shown to be elevated among HAART than HAART naïve individuals.[15,16] HIV patients not on HAART have shown evidence of dyslipidaemia with decreasing high-density lipoprotein cholesterol (HDL) and increasing triglyceride (TG). It has been postulated HIV infected individuals experience low-level LDL-C and HDL-C and elevated serum concentration of triglyceride years before developed AIDS.[17] Treatment with PIs and NNRTIs is associated with many metabolic disorders such as dyslipidaemia that may increase the risk of coronary heart disease. Furthermore, HIV infected persons are subject to dyslipidaemia and other complication due to HAART which are called HIV MetS.[18] Studies among HIV infected individuals revealed a range of lipids disorders, even before ART is readily available. Reports have indicated a low concentration of various lipid components among HIV infected persons.[19] Lipid values and glucose homeostasis were influenced by the disease and demographic characteristics even in the absence of ART, with less favourable lipid and glucose homeostasis in more advanced diseases. Lipids and HIV RNA levels have an independent association, that suggest viral replication have effect on lipid concentrations.[20] Elevated total cholesterol and decrease HDL cholesterol increase the risk of cardiovascular disease independent of hs-CRP.[21] Reports show increased concern about metabolic complications related to HIV infection and HAART use, which may lead to an increase in cardiovascular (CVD) events as a major cause of morbidity and mortality among HIV infected patients.[13, 22, 23] The diagnosis of MetS is based on International Diabetic Foundation (IDF) and National Cholesterol Education programme adult treatment program III (NCEP ATP III) classifications 2001 definition, which was later updated by American Heart Association and National Heart, Lung and Blood Institute(AHA/NHLBI) in 2005.[2, 3] MetS represent a constellation of biochemical and physiologic factors that are linked directly with a high risk of type 2 DM and hypertension.[2] These risk factors are hypertriglyceridaemia, low HDL-C, hypertension and abdominal obesity.[24] MetS are considered as a clinical and public health crisis and its incidence has burst to epidemic proportions all over the world.[2] Individuals with MetS in the general population have two-time the risk of developing CVD and five times more risk of developing DM[25], the impact of which has now extended to PLWH.[2]

There are limited data available on the association between MetS and human Immunodeficiency virus disease in Sokoto, Nigeria.

MATERIALS AND METHODS

This cross-sectional study was carried out at the Institute of Human Virology of Nigeria (IHVN) centres of Specialist Hospital Sokoto and Usmanu Danfodiyo University Teaching Hospital Sokoto.

Eighty-six (86) HIV positive HAART naïve patients aged 18years and above, (86) HIV negative age and sex-matched controls were recruited for the study.

All participants signed the written informed consent form. A pretested questionnaire was also completed which contained the bio data of participants such as socio-demographic, duration of infection, smoking status, lifestyle, Blood pressure measurement, weight, height, waist circumference and hip circumference etc. The ethical approval for the study was sort and granted by the ethic and research committees of Usmanu Danfodiyo University Teaching Hospital and Specialist Hospital Sokoto.

Subjects with a documented medical history of comorbidities such as tuberculosis, hepatitis B virus infection, known diabetes and hypertension were excluded from the study.

The guideline of the study was expounded to the successful participants and instructed to fast for 8hrs to 10hrs overnight. Furthermore, about 5mls of blood were collected and 2mls transmitted into fluoride oxalate container for fasting glucose analysis and 3mls into plain container for fasting lipid profile. The sample was allowed to clot before separation. Both glucose and lipid were measured by oxidase methods with Mindray BA 88 semi-auto Chemistry analyser using Random reagent. The fasting blood glucose was assessed enzymatically using the method of Trinder.[26] The serum triglyceride (TG) concentrations were estimated using the method of Trinder[27], serum cholesterol was estimated by the enzymatic method of Allain, Poon[28] and serum HDL-C Concentration was estimated by the method of Burstein.[29] The commercial kits from random laboratory England were used for the analysis. Micro albumin was quantitatively measured in urine by turbid metric method. Determination of urine Creatinine was by the method of Jaffe.[30] Blood pressure was measured using a mercury sphygmomanometer, two readings were taken at interval of five minutes after patients was seated for 10mins and the average was taken and recorded. the weight of the subjects was measured with minimal clothing at nearest o.5kg using a weighing scale (Seca, Hamburg Deutschland, Germany) height was measured

using a stadiometer with the subject standing erect without shoes at nearest 0.1cm (Seca Hamburg Deutschland, Germany). Body mass index (BMI) was calculated by dividing weight in kilogram by the square of height in meters (kg/m^2). Waist circumference and hip perimeters were measured using an inflexible tape measure. The waist is a measure to the nearest 0.1cm at midpoint between the lower rib cage and the iliac crest, while the hip circumference was measured at the nearest 0.1cm at the widest diameter between the two greater trochanters. The waist to hip ratio was calculated from the waist and hip circumference.

BMI were categorized base on WHO definition for adults as underweight (<18.5kg/M^2), Normal (18.5-24.9kg/M^2), overweight (25 -29.9kg/M^2) and obese (>30kg/M^2)

Female subjects with a waist circumference of <80cm, 80cm-87.9cm, and ≥88cm were classified as normal, overweight and obese respectively, whereas, male subjects were categorized in the same obesity classification as <94cm, 94cm-101.9cm and ≥102cm respectively. WHR were grouped as <0.90cm, 0.90cm-0.99cm, and ≥1.0 for male subjects and <0.80cm, 0.80cm-0.84cm and ≥0. 85cm for female subjects as normal weight, overweight and obese respectively.

Statistical analysis

The data obtained were entered in Microsoft excel 2016 and checked for double entry. The statistical analyses were performed using statistical software Analyse-it for Microsoft Excel version 5.802 (Analyse-it Software Ltd. The Tannery, 91Kirkstall Road, Lead LS3 1HS, United Kingdom, www.analyse-it.com) and IBM Statistical Package for Social Sciences (SPSS) version 23 statistical software. Frequency distribution tables were constructed, the independent student's t-test was used to compare the mean difference between the two diagnostic criteria. ROC analysis was used to determine the variables that Predict MetS among participants. All values were significant at $p<0.05$.

RESULTS

The results obtained in the present study are presented in Tables 1 to 7 and figures 1 and 2. Table 1: Socio-demographic and clinical characteristics of study subjects by MetS Status. The mean age of subjects with and without MetS is (38.2±2.1) and (31.4±0.8) (Mean±SE) respectively. The anthropometric parameters were statistically significantly higher among subjects with MetS than in those without MetS, ($p < 0.0001$). Systolic and Diastolic blood pressure were also higher in the MetS group than those without MetS. Table 2 show the clinical characteristics of study subjects by gender. The mean age is higher among male subjects than females, BMI is significantly higher among females than males ($p <0.0001$) systolic blood pressure was also significantly high in females ($p= 0.0260$) but not diastolic blood pressure. The increase in waist circumference is higher in females than males ($p <0.0001$). Decrease in high-density lipoprotein cholesterol (HDL-C) is more significant among males than females ($p= 0.0028$), triglyceride is higher among males than in females ($p <0.0001$) with no statistical difference in fasting blood glucose (FBG) among the groups. Table 3: Show the NCEP ATPIII (AHA/NHLBI) 2005 and IDF 2005 criteria used to determine the prevalence of MetS in this study. However, out of 86 HIV Seropositive patients, 15(17.4%) had MetS out of which 14(16.3%) fulfils the NCEP ATP III Criterion (Thomas *et al.*, 2005) and 11(12.8%) fulfils the IDF criterion[31] and 10(11.6%) satisfied both NCEP ATP III and IDF criteria. There are more women 11(13.1%) than men 4(1.8%). When compared to healthy controls 16(18.6%) were confirmed with MetS, of which 9(10.5%) and 12(14.0%) fulfil the NCEP ATP III and IDF criteria respectively. Male constitute 5(5.8%) and females make up 11(12.8%). The prevalence of MetS components among cases and controls with elevated triglycerides above 1.7mmol/L 42(48.8%), and 28(32.6%) respectively, decrease high-density lipoprotein cholesterol were seen in 34(39.5%) and 32(37.2%) in cases and control respectively, elevated fasting blood glucose 5(5.8%) cases and 6(6.9%) as identified by ATP III and by IDF it was 6(6.9%) in cases and 8(9.3%) among controls respectively. Furthermore, elevated systolic blood pressure was 15(17.4%) in cases and 12(13.9%) among controls, elevated diastolic blood pressure was 18(20.9%) in cases and it was 14(16.3) among controls, by both criteria. An increase in waist circumference is seen in 7(8.1%) and 9(10.5%) among cases and controls by NCEP ATP III criterion while 11(12.8%) and 18(20.9%) in cases and controls fulfils the IDF criterion respectively.

Table 4 shows the cut-off value AUC of Anthropometric parameters, systolic and diastolic blood pressure for a good Prediction power of MetS among participants, with weight, waist circumference, hip circumference, waist to hip ratio and body mass index showed a very good prediction power ($p<0.0001$) and high sensitivity and specificity. Systolic blood pressure is a better predictor of MetS ($p<0.0001$) than diastolic blood pressure ($p>0.05$). The area under the curve (AUC) with value 0.7 to 0.8 was very good and any value less than 0.6 have no prediction power. Figure 1 shows the ROC analysis of anthropometric and clinical parameters and AUC as predictors of MetS.

Table 5 shows the cut-off values of AUC sensitivity and specificity for lipid profile components and other parameters. However, only TG show a good prediction value with an AUC of 0.6980 ($p<0.001$). Urine micro-albumin had a high sensitivity of 76.7 and specificity of 38.0 which indicated that it could be good for screening but still its power as a predictor of MetS AUC (0.503) is poor. Even though, LDL and urine creatinine show a significant power for detection of Mets the AUC remain power predictors of MetS. Furthermore, the Lipid profile

shows weak prediction power when compared with other components of MetS, Table 4 and Table 5 respectively. Figure 2a and 2b show the AUC of lipid with other parameters and HDL cholesterol respectively.

Table 6 show the correlation between age and component of metabolic syndrome, WHR TG and DBP a positive correlation with age (p< 0.000, 0.001 and 0.003) and also height (HT), SBP and Albumin creatinine ratio (ACR) show positive correlation with age (p< 0.029,0.051 and 0.032) respectively. Conversely HDL cholesterol shows a negative correlation with age (p<0.05) and other parameters show no significant correlation with age. Table 7 compares the correlation between the development of MetS and its component among cases and controls. There significant negative correlation in age p<=0.042, weight p<0.000, BMI p<0.001, waist circumference (WC) p<0.0001, hip circumference (HC) p<0.0001 and SBP (p=0.002) respectively. However, lipid profiles show no correlation among both cases and controls. Furthermore, FBG shows no correlation among all groups. WHR show a positive correlation among cases (p=0.009) but a negative correlation among controls (p=0.005). However, urine creatinine shows a positive correlation with MetS among cases but in controls (p= 0.017) and (p= 0.016) respectively.

Table 1: Clinical and Demographic characteristic of study subjects by MetS distinction.

	With MetS (n=32) mean± SE	Without MetS (n=138) mean± SE	p- Value
Age(years)	38.2±2.1	31.4±0.8	0.0001
WT(cm)	69.9±2.5	57.4±1.1	0.0001
BMI(kg/M^2)	25.5±1.0	20.6±0.4	0.0001
WC(cm)	89.4±2.5	72.6±0.8	0.0001
HC(cm)	101.5±2.7	87.5±0.9	0.0001
WHR(cm)	0.88±0.02	0.83±0.01	0.0001
SBP(mmHg)	123.7±3.2	110.8±1.3	0.0001
DBP(mmHg)	81.8±2.5	74.8±0.8	0.0001

MetS Metabolic syndrome, SE standard Error of mean, WT Weight, BMI Body Mass Index, WC Waist Circumference, HC Hip Circumference, WHR Waist to Hip Ratio, SBP Systolic Blood Pressure and DBP Diastolic Blood Pressure. P <0.05. Determine by student t test.

Table 2: Clinical Characteristic of the study subjects showing mean value of components of MetS by group and gender. (Mean ±SE)

Parameters	Group A	Group B	P value	Male	Female
	86	86		A=59 B=38	A=27 B=48
Age(yrs.)	30.8±1.09	35.0±1.1	0.0060	31.7±1.40* 36.2±1.73	28.7±1.60 34.0±1.30
BMI(kg/m^2)	23.9±0.4	19.2±0.5	<0.0001	23.1±0.37** 18.7±0.54	25.64±1.02 19.07±0.77**
SBP(mmHg)	116.0±1.3	110.6±2.0	0.0260	115.6±1.6* 107.6±2.65	116.93±2.2 112.95±2.98
DBP(mmHg)	76.93±1.1	75.3±1.3	0.3401	76.7±1.47 72.6±1.45	77.44±1.98 77.45±1.89
WC(cm)	79.5±1.3	72.2±1.3	0.0001	77.0±1.28* 70.8±1.66	85.0±2.85* 73.3±1.97
HDL(mmol/l)	1.17±0.05	0.98±0.04	0.0028	1.15±0.05 0.99±0.07†	1.22±0.09* 0.97±0.05
TG(mmol/l)	1.37±0.06	1.88±0.10	0.005	1.34±0.08* 2.06±0.16	1.44±0.12 1.74±0.13*
FBS(mm0l/l)	4.73±0.12	4.64±0.12	0.571	4.86±0.12 4.74±0.22	4.44±0.19 4.55±0.12

Group A. HIV Negative controls, Group B Positive HAART Naïve patients, Data are (mean± SE) **P <0.0001, *P <0.05, between groups, determine by independent sample t test.

Table 3: Prevalence of metabolic syndrome component among study subjects by NCEP-ATP III and IDF Criteria.

		Diagnostic	Criteria			
MetS Prevalence	**NCEP-ATP III**	**Definition**		**IDF**	**Definition**	
15(17.4%)		14(16.3%)	Cases	11(12.8%)		
Variables	Males n (%)	Females n(%)	Total n (%)	Males n (%)	Females n(%)	Total n (%)
↑WC(cm)	1(1.2)	6(6.9)	7(8.1)	4(4.7)	11(12.8)	15(17.4)
↓HDL(mmol/l)	34(39.5)	44(51.2)	78(90.7)	28(32.6)	41(47.7)	69(80.2)
↑TG(mmol/l)	22(25.5)	20(23.3)	42(48.8)	22(25.2)	20(23.3)	42(48.8)
↑FBS(mmol/l)	2(2.3)	3(3.5)	5(5.8)	5(5.8)	1(1.2)	6(6.9)
↑BP(mmHg)	6(6.9)	9(10.5)	15(17.4)	7(8.1)	12(13.9)	19(22.1)
MetS Prevalence	NCEP-ATP III	Definition		IDF	Definition	
16(18.6%)		9(10.5%)	Controls	12(14.0%)		
↑WC(cm)	2(2.3)	9(10.5)	11(12.8)	3(3.5)	15(17.4)	18(20.9)
↓HDL(mmol/l)	13(15.)	19(22.1)	32(37.2)	17(19.8)	13(15.1)	30(34.9)
↑TG(mmol/l)	15(17.4)	13(15.1)	28(32.6)	15(17.4)	13(15.1)	28(32.6)
↑FBS(mmol/l)	5(5.8)	1(2.1)	6(6.7)	5(5.8)	3(3.5)	8(9.3)
↑BP(mmHg)	8(9.3)	14(16.3)	22(25.6)	12(13.9)	6(6.9)	18(20.9)

↑WC, increase waist circumference, ↓HDL reduced high density lipoprotein cholesterol, ↑TG increase triglycerides, ↑FBS increase fasting blood sugar, and ↑BP elevated blood pressure. By descriptive Statistics.

Table 4: Shows the cut-off value, AUC, Sensitivity and Specificity of weight, height, BMI, Waist Circumference, Hip Circumference, waist to ratio, Systolic Blood Pressure and Diastolic Blood Pressure for prediction of MetS in study subjects.

Parameter	Value	AUC	95%CI	Sensitivity (%)	Specificity (%)
WT(kg)	62.5kg	0.768**	0.617-0.862	81.3	71.4
HT(cm)	1.735cm	0.492	0.394-0.590	94	78.6
BMI(kg/M^2)	21.7kg/m^2	0.768**	0.669-0.867	78.1	60.7
WC(cm)	79.5cm	0.835**	0.747-0.924	81.3	78.4
HC(cm)	88.5cm	0.796**	0.701-0.890	78.1	67.3
WHR(cm)	0.846cm	0.651**	0.546-0.756	62.5	52.9
SBP(mmHg)	110mmHg	0.716**	0.609-0.823	75	52.9
DBP(mmHg)	73mmHg	0.650*	0.533-0.764	68.8	52.1

WT, Weight, HT, Height, BMI, Body Mass Index, WC, Waist Circumference, HC, Hip Circumference, WHR, Waist to Hip Ratio, SBP, Systolic Blood Pressure, DBP, Diastolic Blood Pressure, AUC, Area under Curve. **very good sensitivity P <0.0001,* good sensitivity P <0.05 statistics by ROC Analysis.

Table 5: Shows the cut-off value, AUC, Sensitivity and Specificity for Total cholesterol, triglycerides, High density lipoprotein, low density lipoprotein, fasting blood glucose, urine micro albumin, urine creatinine, albumin creatinine ratio and viral load for prediction of MetS Among study subjects.

Parameter	Value	AUC	95% CI	Sensitivity (%)	Specificity (%)
TC(mmol/l)	3.39	0.581	0.476-0.685	60	58.6
TG(mmol/l)	1.72	0.677**	0.589-0.764	76.7	66.8
HDL9(mmol/l)	1.54	0.539	0.429-0.622	56.7	52.6
LDL(mmol/l)	0.98	0.580	0.474-0.685	59.4	51.4
FBG(mmol/l)	4.6	0.563	0.441-0.693	56.7	50.4
UMALB(mg/l)	83.4	0.484*	0.379-0.588	76.7	38.0
U CRT(mg/dl)	83.4	0.588	0.457-0.671	63.3	51.1
ACR(mg/g)	0.25	0.444	0.341-0.547	60	42.1
VL(copies/ml)	22.5	0.507	0.391-0.624	50	52.8

TC, Total cholesterol, TG, Triglycerides, HDL, High density lipoprotein cholesterol, low density lipoprotein cholesterol, UM ALB; Urine Micro-albumin, U. CRT Urine creatinine and Albumin creatinine ratio, AUC Area under the curve [** very good sensitivity (p <0.05),

* good sensitivity(p >0.05) but poor prediction power] Statistic by ROC Analysis.

Table 6: Shows the association between Age and MetS components among study subjects.

Parameter	Correlation coefficient	P value
HT(cm)	0.117*	0.029
BMI(kg/M^2)	0.013	0.803
HC(cm)	0.013	0.800
WHR(cm)	0.244**	0.0001
SBP(mmHg)	0.102*	0.057
DBP(mmHg)	0.160**	0.003
TC(mmol/l)	-0.039	0.460
TG(mmol/l)	0.175**	0.001
HDL(mmol/l)	-0.104*	0.051
LDL(mmol/l)	-0.045	0.394
UMALB(mg/ml)	0.100	0.075
UCREAT(mg/dl)	-0. 036	0.333
ACR(mg/g)	0.114*	0.032

HT, height, BMI, Body mass Index, HC, hip circumference, SBP, systolic blood pressure, DBP, diastolic blood pressure, TC, total cholesterol, TG, triglycerides, HDL =, high density lipoprotein, LDL, low density lipoproteins, UMALB, Urine micro-albumin, UCREAT, urine creatinine and ACR, Albumin creatinine ratio. * P value <0.05, ** p value <0.01. Statistic by spearman correlation.

Table 7: Compares the Correlation between developments of MetS and its component among Patients and controls.

Parameter	Correlation coefficient (r) Cases	P value	Correlation coefficient(r) Controls	P value
Age(years)	-0.186*	0.042	-0.334**	0.0001
WT(kg)	-0.324**	0.000	-0.294**	0.001
HT(cm)	-0.048	0.592	0.085	0.350
BMI(kg/M^2)	-0.306**	0.001	-0.301**	0.001
WC(cm)	-0.444**	0.000	-0.347**	0.0001
HC(cm)	-0.357**	0.000	-0.304**	0.001
WHR(cm)	0.240**	0.009	-0.191*	0.033
SBP(mmHg)	-0.283**	0.002	-0.254**	0.006
DBP(mmHg)	-0.257**	0.005	-0.157	0.087
TC(mmol/l)	-0.067	0.465	-0.107	0.239
TG(mmol/l)	0.005	0.959	-0.087	0.337
HDL(mmol/l)	0.013	0.886	-0.096	0.293
LDL(mmol/l)	0.081	0.568	0.123	0.169
FBG(mmol/l)	0.133	0.144	-0.040	0.660
UMALB9mg/ml)	0.053	0.573	-0.041	0.666
UCREAT(mg/dl)	0.233**	0.017	-0.071	0.467
ACR(mg/g)	0.217**	0.016	-0.097	0.288

HT, height, BMI, Body mass Index, HC, hip circumference, SBP, systolic blood pressure, DBP, diastolic blood pressure, TC, total cholesterol, TG, triglycerides, HDL =, high density lipoprotein, LDL, low density lipoproteins, UMALB, Urine micro-albumin, UCREAT, urine creatinine and ACR, Albumin creatinine ratio. * P value <0.05, ** p value <0.01. Statistic by spearman correlation.

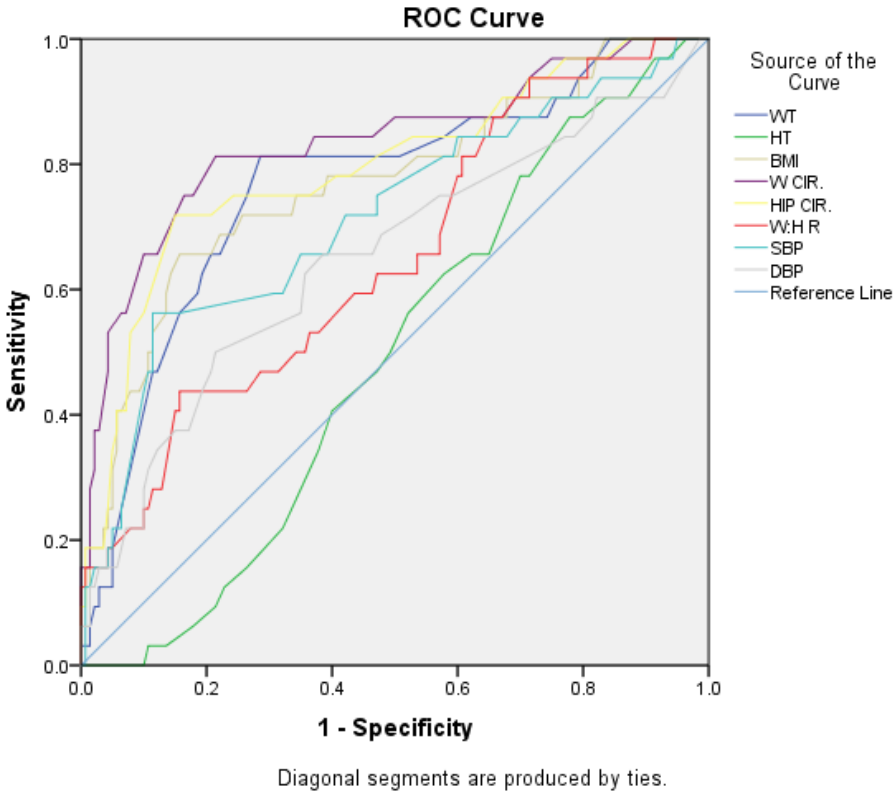

Figure 1: ROC Analysis of anthropometric and clinical Parameters showing AUC as predictors of MetS.

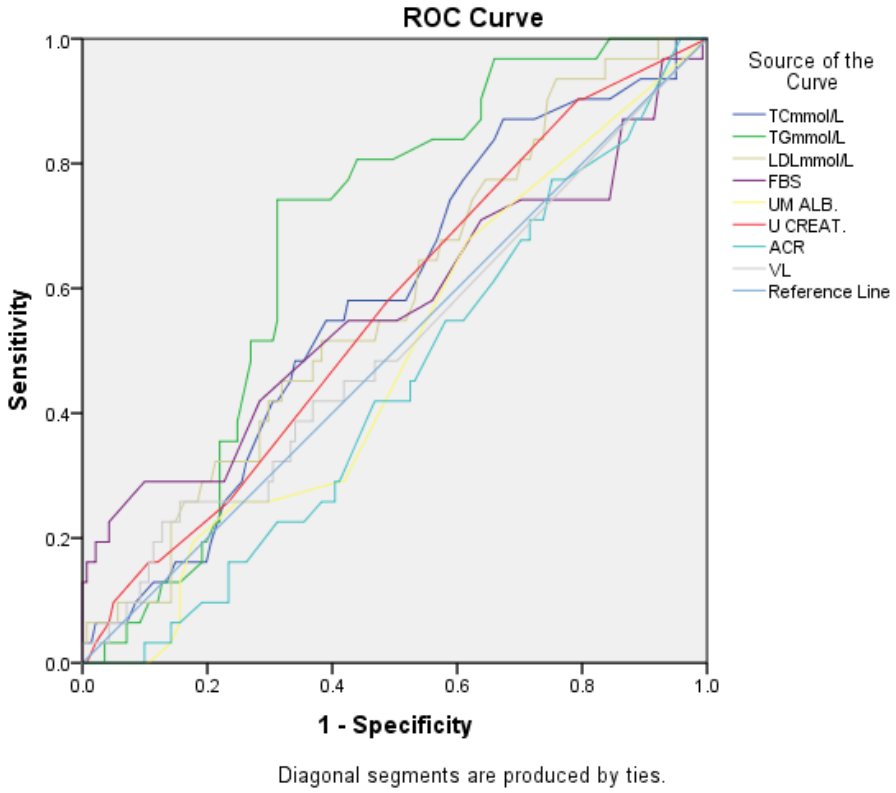

Figure 2a: ROC Analysis of lipid profile, fasting blood glucose, urine micro albumin and creatinine and albumin creatinine ratio and viral load showing AUC as predictors of MetS.

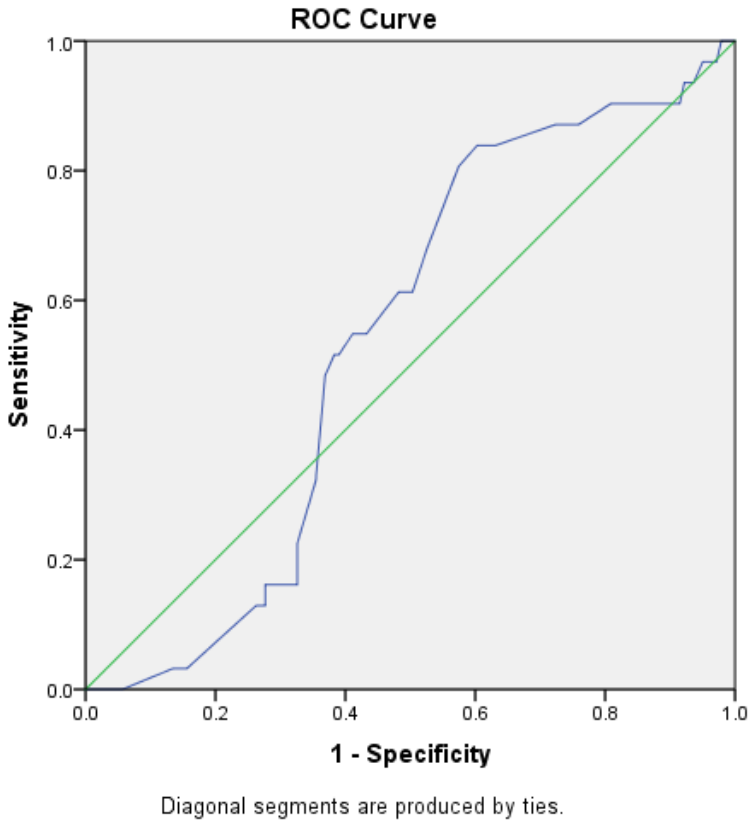

Figure 2b: ROC Analysis of HDL component lipid profile, showing AUC as predictor of MetS.

DISCUSSION

Although there have been many works on MetS in Sokoto none is done in HIV seropositive patients. The current study was conducted among HIV seropositive HAART Naïve patients. The prevalence of MetS is higher among healthy controls than in HIV seropositive patients in the present study [16(18.6%) vs. 15(17.4%)]. These findings are Similar previous report by[10] who reported 17.8% among civil servants but contrary to[9] who reported 34.1%. However, the high prevalence was reported among HAART naïve patients reported by.[15] More patients with MetS among HIV infected subjects were identified by NCEP ATP criteria than the IDF criteria, while in normal controls IDF reports having identified more subjects with MetS than NCEP ATP. This is in agreement with the report of[3], which may not be unconnected with the peripheral fat wasting that tends to increase waist to hip ratio in HIV patients due to lipodystrophy. Our findings also show that females are more prone to MetS than their male counterparts. this is in agreement with the report of[16, 32, 33], but it is contrary to the findings of.[18] Similarly, WC was significantly higher among females than males (p= 0.0001) comparable to the report by.[16] However, it was contrary to the report of[34] who show males have a high risk of developing MetS than females. The mean age among those with and without MetS is Mean ±SE (38.2±2.1 vs. 31.4±0.8) respectively. This agrees with several reports, that advancing age plays a role in developing MetS as reported by[35] that unveils the HAART unexperienced patients 35years of age and above have a greater risk of emergent with MetS than younger age. Equally,[3] reported 45±10 vs. 41±9 among HIV patients on ART and[36] reported 21±2 vs. 20±2 among young adults, Mean (±SD) for those with and without MetS respectively. Moreover, de carvalho vidigal et al.,[37] also reported those over the age of 40 years among health professionals. Furthermore, the anthropometric assessment shows high values among patients with MetS than those without the syndromes. Hirigo and Tesfaye (16) Also reported that the risk of developing MetS is higher among HIV individuals above 45 years than in those below 34 years. 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 ($p< 0.0001$). 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 seen 72.1% in HIV positive HAART naïve patients and controls below 1.04mmol/l and 1.29mmol//L for males and females respectively. This agrees with the findings of[38] of 59.3% among HIV sero-negative subjects. NCEP ATP III criteria identified more of these components than IDF Criteria 98.8% and 90.7% vs. 97.7% and 80.2% for TG and HDL cholesterol respectively. Our finding also contradict the study of[39] who reported a decrease in HDL as the most frequent component in their study, followed by an increase in total cholesterol (TC).

Elevated fasting blood glucose (FBG) is the least component seen in the present study with only 5.8% and 6.7% in patients and controls using NCEP criteria and 6.9% and 9.3% by IDF criteria respectively. This agrees with the findings of[40] who reported only a 5.5% elevation in FBG among HIV positive patients. Factors associated with MetS in the present study are age, sex, weight, Body Mass Index, waist circumference, hip circumference, waist to ratio and blood pressure. BMI and Age were reported by[41, 42] to be associated with MetS among HIV patients. Weight, BMI, Waist Circumference, Hip Circumference, Waist to Hip Ratio and Systolic Blood Pressure were better predictors of MetS than lipid profile parameters and FBG.

CONCLUSION

The prevalence of metabolic syndrome is high among HIV HAART naïve patients but higher among non-HIV controls. Female Gender and advanced age were found to be significantly associated with MetS. Weight, BMI, Waist Circumference, Hip Circumference, Waist to Hip Ratio and Systolic Blood Pressure were better predictors of MetS than lipid profile parameters and FBG.

ACKNOWLEDGEMENT

Wish to acknowledge the management and staff of the IHVN laboratory of Usmanu Danfodiyo University teaching hospital and Specialist Hospital Sokoto for allowing us to use their facilities for the research periods and also the staff of Chemical Pathology Laboratory UDUTH for their support in handling the samples, and to all participant for the consent to partake in the study.

Source of funding: Nil.

Competing interests: Authors declare no competing interests.

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 the manuscript.

REFERENCES

1. Al Dhaheri AS, Mohamad MN, Jarrar AH, Ohuma EO, Ismail LC, Al Meqbaali FT, et al. A Cross-Sectional Study of the Prevalence of Metabolic Syndrome among Young Female Emirati Adults. PloS one., 2016; 11(7): e0159378.
2. Naidu S, Ponnampalvanar S, Kamaruzzaman SB, Kamarulzaman A. Prevalence of metabolic syndrome among people living with HIV in developing countries: a systematic review. AIDS patient care and STDs., 2017; 31(1): 1-13.
3. Samaras K, Wand H, Law M, Emery S, Cooper D, Carr A. Prevalence of metabolic syndrome in HIV-infected patients receiving highly active antiretroviral therapy using International Diabetes Foundation and Adult Treatment Panel III criteria: associations with insulin resistance, disturbed body fat compartmentalization, elevated C-reactive protein, and hypoadiponectinemia. Diabetes care., 2007; 30(1): 113-9.
4. Beltrán-Sánchez H, Harhay MO, Harhay MM, McElligott S. Prevalence and trends of metabolic syndrome in the adult US population, 1999–2010. Journal of the American College of Cardiology, 2013; 62(8): 697-703.
5. Dutra ES, de Carvalho KM, Miyazaki É, Merchán-Hamann E, Ito MK. Metabolic syndrome in central Brazil: prevalence and correlates in the adult population. Diabetology & metabolic syndrome, 2012; 4(1): 20.
6. Gyakobo M, Amoah AG, Martey-Marbell D-A, Snow RC. Prevalence of the metabolic syndrome in a rural population in Ghana. BMC endocrine disorders, 2012; 12(1): 25.
7. Ojji DB, Ajayi SO, Mamven MH, Alabi P. Prevalence of metabolic syndrome among hypertensive patients in Abuja, Nigeria. Ethnicity & disease, 2012; 22(1): 1-4.
8. Siminialayi I, Emem-Chioma P, Odia O. Prevalence of metabolic syndrome in urban and suburban Rivers State, Nigeria: International Diabetes Federation and Adult Treatment Panel III definitions. The Nigerian postgraduate medical journal, 2010; 17(2): 147-53.
9. Sabir AA, Jimoh A, Iwuala SO, Isezuo SA, Bilbis LS, Aminu KU, et al. Metabolic syndrome in urban city of North-Western Nigeria: prevalence and determinants. Pan African Medical Journal, 2016; 23(1).
10. Awosan K, Ibrahim M, Arisegi S, Ejimadu S, Erhiano E, Aderahman A. Prevalence of metabolic syndrome and its components among civil servants in a metropolitan city in Northern Nigeria. Glob Adv Res J Med MedSci., 2013; 2(11): 238-46.
11. Isezuo S, Ezunu E. Demographic and clinical correlates of metabolic syndrome in Native African type-2 diabetic patients. Journal of the National Medical Association, 2005; 97(4): 557.
12. Herbert AM, Henry DM, Anthony TK, Omarine N, Theresa N-A. Prevalence of metabolic syndrome in human immunodeficiency virus - infected patients from the South-West region of Cameroon, using the adult treatment panel III criteria. Diabetol Metab Syndr, 2014; 6(92).
13. Obirikorang C, Quaye L, Osei-Yeboah J, Odame EA, Asare I. Prevalence of metabolic syndrome among HIV-infected patients in Ghana: A cross-sectional study. Nigerian medical journal: journal of the Nigeria Medical Association, 2016; 57(2): 86.
14. Uwanuruochi VN, Michael FS, Uwanuruochi K, Okafor C, Ofoegbu EN, Onwubere BJ, et al. Assessment of Metabolic syndrome among adult human immunodeficiency virus/acquired immunodeficiency syndrome patients in a tertiary health facility in Southeast Nigeria. Journal of HIV and Human Reproduction, 2015; 3(2): 41.

15. Muhammad F, Gezawa I, Uloko A, Yakasai A, Habib A, Iliyasu G. Metabolic syndrome among HIV infected patients: A comparative cross sectional study in northwestern Nigeria. Diabetol Metab Syndr, 2017; 1: 523-9.
16. Hirigo AT, Tesfaye DY. Influences of gender in metabolic syndrome and its components among people living with HIV virus using antiretroviral treatment in Hawassa, southern Ethiopia. BMC research notes, 2016; 9(1): 145.
17. Fontas E, Van Leth F, Sabin C, Friis-Møller N, Rickenbach M, d'Arminio Monforte A, et al. Lipid profiles in HIV-infected patients receiving combination antiretroviral therapy: are different antiretroviral drugs associated with different lipid profiles? The Journal of infectious diseases, 2004; 189(6): 1056-74.
18. Carr A, Miller J, Law M, Cooper DA. A syndrome of lipoatrophy, lactic acidaemia and liver dysfunction associated with HIV nucleoside analogue therapy: contribution to protease inhibitor-related lipodystrophy syndrome. Aids., 2000; 14(3): F25-F32.
19. Riddler SA, Smit E, Cole SR, Li R, Chmiel JS, Dobs A, et al. Impact of HIV infection and HAART on serum lipids in men. Jama, 2003; 289(22): 2978-82.
20. El-Sadr W, Mullin C, Carr A, Gibert C, Rappoport C, Visnegarwala F, et al. Effects of HIV disease on lipid, glucose and insulin levels: results from a large antiretroviral-naïve cohort. HIV medicine, 2005; 6(2): 114-21.
21. De Luca A, de Gaetano Donati K, Colafigli M, Cozzi-Lepri A, De Curtis A, Gori A, et al. The association of high-sensitivity c-reactive protein and other biomarkers with cardiovascular disease in patients treated for HIV: a nested case–control study. BMC infectious diseases, 2013; 13(1): 414.
22. Biron A, Bobin-Dubigeon C, Volteau C, Piroth L, Perré P, Leport C, et al. Metabolic syndrome in French HIV-infected patients: prevalence and predictive factors after 3 years of antiretroviral therapy. AIDS research and human retroviruses, 2012; 28(12): 1672-8.
23. Feleke Y, Fekade D, Mezegebu Y. Prevalence of highly active antiretroviral therapy associated metabolic abnormalities and lipodystrophy in HIV infected patients. Ethiopian medical journal, 2012; 50(3): 221-30.
24. Després J-P, Poirier P, Bergeron J, Tremblay A, Lemieux I, Alméras N. From individual risk factors and the metabolic syndrome to global cardiometabolic risk. European Heart Journal Supplements, 2008; 10(suppl_B): B24-B33.
25. Grundy SM. Metabolic syndrome pandemic. Arteriosclerosis, thrombosis, and vascular biology, 2008; 28(4): 629-36.
26. Trinder P. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Annals of clinical biochemistry, 1969; 6(1): 24-7.
27. Trinder P. Quantitative determination of triglyceride using GPO-PAP method. Ann Biochem, 1969; 6: 24-7.
28. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. Clinical chemistry, 1974; 20(4): 470-5.
29. Burstein M. A fully enzymatic colorimetric determination of HDL cholesterol in the serum. Lipid Res., 1970; 11: 583-95.
30. Jaffe M. About the precipitation caused by pikrinic acid in normal urine and about a new reaction of creatinine. Physiol Chem., 1886; 10: 391-400.
31. Alberti G, Zimmet P, Shaw J, Grundy SM. The IDF consensus worldwide definition of the metabolic syndrome. Brussels: International Diabetes Federation, 2006; 23(5): 469-80.
32. Muhammad S, Sani MU, Okeahialam BN. Cardiovascular disease risk factors among HIV-infected Nigerians receiving highly active antiretroviral therapy. Nigerian medical journal: journal of the Nigeria Medical Association, 2013; 54(3): 185.
33. Tachebele B, Abebe M, Addis Z, Mesfin N. Metabolic syndrome among hypertensive patients at University of Gondar Hospital, North West Ethiopia: a cross sectional study. BMC cardiovascular disorders, 2014; 14(1): 177.
34. Awotedu K, Ekpebegh C, Longo-Mbenza B, Iputo J. Prevalence of metabolic syndrome assessed by IDF and NCEP ATP 111 criteria and determinants of insulin resistance among HIV patients in the Eastern Cape Province of South Africa. Diabetes & Metabolic Syndrome: Clinical Research & Reviews, 2010; 4(4): 210-4.
35. Jantarapakde J, Phanuphak N, Chaturawit C, Pengnonyang S, Mathajittiphan P, Takamtha P, et al. Prevalence of metabolic syndrome among antiretroviral-naive and antiretroviral-experienced HIV-1 infected Thai adults. AIDS patient care and STDs., 2014; 28(7): 331-40.
36. Manjunath D, Uthappa CK, Kattula SR, Allam RR, Chava N, Oruganti G. Metabolic syndrome among urban Indian young adults: prevalence and associated risk factors. Metabolic syndrome and related disorders, 2014; 12(7): 381-9.
37. de Carvalho Vidigal F, Ribeiro AQ, Babio N, Salas-Salvadó J, Bressan J. Prevalence of metabolic syndrome and pre-metabolic syndrome in health professionals: LATINMETS Brazil study. Diabetology & metabolic syndrome, 2015; 7(1): 6.
38. Sani MU, Wahab KW, Yusuf BO, Gbadamosi M, Johnson OV, Gbadamosi A. Modifiable cardiovascular risk factors among apparently healthy adult Nigerian population-a cross sectional study. BMC research notes, 2010; 3(1): 1-7.
39. Muhammad S, Sani MU, Okeahialam BN. Prevalence of dyslipidemia among human immunodeficiency virus infected Nigerians. Annals of African medicine, 2013; 12(1): 24.

40. Carr DB, Utzschneider KM, Hull RL, Kodama K, Retzlaff BM, Brunzell JD, et al. Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. Diabetes, 2004; 53(8): 2087-94.
41. Signorini DJHP, Monteiro MCM, Andrade MdFCd, Signorini DH, Eyer-Silva WdA. What should we know about metabolic syndrome and lipodystrophy in AIDS? Revista da Associação Médica Brasileira, 2012; 58: 70-5.
42. Malangu N. Factors associated with metabolic syndrome among HIV-positive patients at a health facility in Botswana. Journal of Advances in Medicine and Medical Research, 2014: 2352-61.