

## Evaluation of risk factors/prevalence of insulin resistance, hyperglycemia, hypertension and central-obesity in people living with HIV/AIDS at the Bamenda Regional Hospital

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### ABSTRACT

Despite the advancement and benefits of antiretroviral therapy in recent years since its introduction, it has also resulted in complications such as metabolic syndrome like insulin resistance. This study is aimed at determining the prevalence and risk factors of metabolic syndrome in people living with HIV/AIDS (on Highly Active Antiretroviral Therapy (HAART) and HAART-naïve) at the Bamenda Regional Hospital. 273 participants were consecutively recruited; 191 were HAART-exposed, and 82 were HAART-naïve. Anthropometric and blood pressure measurements were conducted under standard conditions. Fasting blood sugar, triglyceride, and High-density-lipoprotein cholesterol (HDLc), were determined spectrophotometrically. Chi-square ( $\chi^2$ ) and odd ratios were used to compare the prevalence of the metabolic syndromes between cases and controls at a 99% confidence interval. The study recorded a prevalence of 24.2%, 3.66%, 38.1% and 39.56% for insulin resistance, hyperglycemia, hypertension, and central obesity, respectively. HAART-naïve were significantly about twice at risk of developing decreased HDL compared to the HAART-exposed group [OR 2.04; 95% CI 1.14-3.66; P=0.015]. However, HAART-treatment duration  $\geq 10$  years was significantly associated with hyperglycemia. The type of HAART regimen also showed a significant association with increased central obesity. It is evident from this study that the following risk factors of metabolic syndrome are common among HIV/AIDS patients: lower HDL, central obesity, alcohol consumption, smoking habits, and age  $\geq 50$  years. This, therefore, highlights the importance of these patients regulating their lifestyle since there is a rising risk of developing metabolic syndromes among them.

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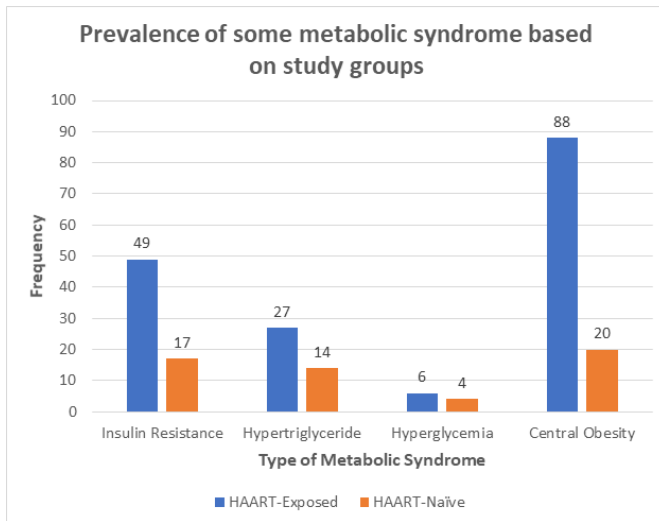
### 1. INTRODUCTION

Heart attacks and strokes are frequently the first indications of an underlying metabolic disease, which is why cardiovascular dis-

eases (CVDs) are sometimes referred to as "silent killers". About 20 million people in the African region are thought to be affected by metabolic syndromes [1]. Life expectancy of HIV/AIDS patients has been on the rise since the advent of Highly active antiretroviral therapy (HAART) in the mid-1990s because of a decrease in their immunodeficiency events and causes of death [2].

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**Figure 1.** Prevalence of Metabolic syndrome based on study groups.

Even if HIV/AIDS patients' health conditions have improved, they are still susceptible to risk factors found in the general population, which can lead to obesity, diabetes mellitus, and cardiovascular illnesses [2].

However, while the patient's prognosis improved with ARV/HAART medication, long-term problems followed, including changes in metabolic parameters such as insulin sensitivity (insulin resistance), dyslipidemia, and hypertension [2, 3]. The strong antiretroviral medication has decreased HIV-related morbidity and death, but concerns regarding metabolic side effects and cardiovascular disorders have surfaced because of the treatment [2, 3].

Metabolic syndrome has been identified by Adult Treatment Panel III (ATP III) report as a group of metabolic abnormalities. These abnormalities include; increased fasting glucose, abdominal obesity, increased triglycerides, decreased high-density lipoproteins (HDLs), and high blood pressure [4, 5].

The risk of CVDs can be altered by HAART as well as HIV infection itself. As previously stated, HAART regimens block the growth of viruses by utilising various drug combinations to act at different phases of the infection [6]. Globally, public health experts continue to prioritise the eradication of HIV/AIDS, which has been focused on trying to reduce the viral load, but the presence of some of these side effects has increasingly led to non-adherence to treatment and the complexity of managing the diseases [7]. The current treatment guidelines for the management of HIV/AIDS in Cameroon call for the determination of fasting blood glucose levels before instituting antiretroviral therapy (ART). However, these guidelines are not respected more often, and data regarding the prevalence of these metabolic abnormalities are scarce. The study determines the prevalence and factors associated with insulin resistance among HAART-naïve patients and patients on HAART.

## 2. METHODOLOGY

### 2.1. ETHICS CLEARANCES AND ADMINISTRATION OF QUESTIONNAIRES

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). The participants were educated on the procedures regarding specimen collection and analysis, as well as the benefits (the free checks of the study parameters) and potential risks of the study. The participants were told their participation was voluntary, and they were free to withdraw from the study at any time without any justification. Samples were collected only after the participants gave their consent to participate in the study by signing an informed consent form.

Participants' information was coded to ensure restricted access to data by unauthorised persons. The source data, test results, and enrolment questionnaires were kept safe in closed cabinets for future reference until the time when they would be disposed of following standard procedures.

### 2.2. STUDY AREA

This was a hospital-based cross-sectional study conducted between May-August 2018 at the treatment centre of People Living With HIV/AIDS (PLWHA) in the Bamenda Regional, in Mezam Division, NorthWest Region of Cameroon. The centre receives patients referred from all the District Hospitals and Clinics in the region and its main referral site for the region. The patients attend the clinic once a month for clinical evaluation and refill of ART.

### 2.3. STUDY POPULATION AND SAMPLE SIZE

The expected sample size was 288 using a 25.5% prevalence of total cholesterol  $\geq 200$  mg/dl in First-line antiretroviral therapy and dyslipidemia in people living with HIV-1 in Cameroon [8], 95% confidence interval, and a margin of error of 0.05. The consecutive sampling technique was used to collect the data from patients. The study population included HIV-infected individuals who came to the hospital for routine health evaluation and were classified into two groups. Group 1 was individuals with HIV infection who had been receiving ART for at least 6 months (ART group). Participants who had had their treatment regimens changed during follow-up were noted. The second group was made up of HIV-positive individuals newly or previously diagnosed who were not yet receiving ART (ART-naïve group) attending the Regional Hospital Bamenda Treatment Center. The inclusion criteria: HIV-positive patients aged at least 21 years at enrollment. The exclusion criteria: Patients on other hepatotoxic, nephrotoxic drugs or diagnosed with other co-morbidities that markedly affect the blood Lipid profile, glucose, and pressure (patients with renal failure on dialysis, Thyroid disease, and liver disease); acutely ill patients that require medical/surgical treatment or admission; those with documented hypertension, diabetes, and dyslipidemia before beginning HAART; and as well as pregnant and lactating women.

### 2.4. DATA COLLECTION

Pre-designed and validated questionnaires were administered by the principal investigator to each participant. The questions

**Table 1. Socio-demographic characteristics of the study population (n=273).**

Socio - demographic Characteristics		HAART exposed Group (n=191) (%)	HAART naïve Group (n=82) (%)	Total (n=273) No (%)
Gender	Male	54(28.27)	26(31.71)	80(29.20)
	Female	137(71.73)	56(68.29)	193(70.70)
Age groups	21-30 years	19(9.95)	20(24.29)	39(14.29)
	31-41 years	64(33.51)	28(34.15)	92(33.70)
	41-50 years	65(34.53)	29(28.05)	88(32.23)
	51- 60 years	35(18.33)	08(09.76)	43(15.75)
	≥61 years	08(4.19)	03(3.66)	11(04.03)
Age (years)	[Mean±SD]	42.82±9.98	38.62±10.47	41.56±10.29
Education	None	10(5.24)	00(0.00)	10(3.66)
	Primary	90(47.12)	40(48.78)	130(47.62)
	Secondary	64(33.51)	29(35.37)	93(34.07)
	Tertiary	27(14.13)	13(15.85)	40(14.56)
Marital Status	Married	97(50.78)	48(58.54)	66(24.18)
	Single	49(25.66)	17(34.6)	145(53.11)
	Widowed	45(23.56)	17(20.73)	62(22.71)
Occupation	Salary employed	27(??)	9(10.98)	36(13.19)
	Self-employed	142(74.35)	61(74.39)	203(74.36)
	Unemployed	45(11.52)	12(14.63)	34(12.45)
Alcohol	No	85(44.50)	53(64.63)	138(59.55)
	Yes	106(55.50)	29(35.37)	135(49.55)
Exercise	No	111(58.12)	52(63.41)	163(59.71)
	Yes	80(41.48)	30(36.59)	110(40.29)
Smoking	No	162(84.82)	79(96.34)	241(88.28)
	Yes	29(15.18)	3(3.66)	32(11.72)

Key: HAART-Highly Active Antiretroviral Therapy

sought information on socio-demographic characteristics, history of HIV disease and treatment, histories of high blood pressure, diabetes, cardiovascular events, and their treatments. The weight of each participant was then measured using a digital weighing scale in a standing position, with light clothing and without shoes. Next, in the standing position, the waist circumference was measured midway between the costal margins and the iliac crest using a meter tape, and the height of the patient was measured using a stadiometer. Body mass index (BMI) was calculated from the formula: weight (kg)/height<sup>2</sup> (m<sup>2</sup>). After five minutes of rest in a sitting position, the blood pressure was measured using a Medical Rossmax Blood Pressure Monitor (Model Number; LC150). The 1st blood pressure reading was measured on the right arm and the 2nd on the left, and the average of both readings was used for analysis. The ARV types and duration data were obtained and confirmed from participants' hospital records. The dates that participants were diagnosed to be HIV-positive, the date of commencement of antiretroviral therapy, the duration of antiretroviral therapy, and the recent CD4<sup>+</sup> T cell count were obtained and confirmed from the participants' hospital folders.

## 2.5. SPECIMEN COLLECTION AND ANALYSES

About 4ml of overnight fasting venous blood specimens (8–12 hour fast) were obtained by venipunctures under sterile conditions into sodium fluoride + potassium oxalate and dry test tubes (about 2ml each). Blood specimens in the dry tubes were allowed to coagulate and then centrifuged with the anticoagulated

tube at 3000 rpm for 5 minutes (using Heraeus Sepatech Centrifuge) to separate the serum and plasma from the cells respectively, stored at – 8°C. All laboratory techniques and procedures were safe and of acceptable standards as all analytical procedures were done following standard operating procedures (SOPs) prepared according to the manufacturer's instruction for each type of reagent kit and measured using a spectrophotometer. Quality control samples (Chronolab Pathological and Normal Human Control Sera) were analysed alongside study samples during analytical procedures. Results of each analytical run were only recorded when quality control samples gave results that were within range as indicated by the manufacturer.

Serum TC, TG, LDL, and HDL were determined by the Enzymatic-spectrophotometric method (Jaffe reaction, using kits from Cypress Diagnostic, Belgium. Fasting blood Glucose levels were measured using the Glucose oxidase enzymatic method according to the manufacturer's instructions for the kit (HUMAN, Germany).

The National Cholesterol Education Program Adult Treatment Panel III Guidelines (NCEP ATP III) were used to evaluate the insulin resistance profile of the study participants [9]. The ATP III criteria define insulin resistance (metabolic syndrome) as the presence of three or more of the following criteria: Abdominal obesity (WC ≥ 102 cm for men and ≥ 88 cm for women), increased fasting serum TG levels (≥ 150 mg/dL or 1.70 mmol/L), reduced HDL-C (< 40 mg/dL or 1.03 mmol/L in men and < 50 mg/dL or 1.29 mmol/L in women), FPG ≥ 110 mg/dL or 6.1 mmol/L), and

**Table 2. Clinical and Biochemical characteristics of the study population (n=273).**

Parameter	Overall n=273 No (%)	HAART experienced n=191 No (%)	exposed Group (%)	HAART naïve group n=82No (%)	OR	95% CI	p-value
SBP (mmHg) Mean±SD	127.41±24.83	128.97±27.38		123.78±17.14	1.38	0.80-2.37	0.011
DBP (mmHg)Mean±SD	83.68±15.25	84.97±16.58		80.67±11.11	1.33	0.78-2.28	0.032
CD <sub>4</sub> + countMean±SD	439.93±250.9	451.18±257.78		413.73±233.49	1.11	0.52-2.35	0.788
<200	39 (14.29)	28(14.66)		11(13.41)			
200-499	140 (51.28)	95(49.74)		45(54.88)			
≥500	94(34.43)	68(35.60)		26(31.71)			
TG (mg/dL)Mean±SD	104.79±87.78	105.91±97.39		102.18±60.11	0.80	0.40-1.62	0.534
Normal	232(84.98)	164(85.86)		68(82.93)			
High	41(15.02)	27(14.14)		14(17.07)			
FBS(mg/DL)Mean±SD	84.07±13.43	83.67±13.19		84.99±13.99	0.63	0.17-2.30	0.487
Normal	263(96.34)	185(96.86)		78(95.12)			
High	10(3.66)	6(3.14)		4(4.88)			
HDL-C					2.04	1.14-3.66	0.015
Normal	209(76.56)	154(80.63)		55(67.07)			
Low	64(23.44)	37(19.37)		27(32.93)			
Central obesity					0.38	0.21-0.67	0.001
No	165(60.44)	103(53.93)		42(75.6)1			
Yes	108(39.56)	88(46.07)		20(24.39)			
Age					2.18	1.09-4.35	0.027
<50 years	209(76.56)	139(72.77)		70(85.37)			
≥50 years	64(23.44)	52(27.23)		12(14.63)			
Alcohol Intake					2.23	1.34-3.89	0.003
No	138(50.55)	85(44.50)		53(64.63)			
Yes	135(49.45)	106(55.50)		29(35.37)			
Current Smoker					4.71	1.39-15.9	0.027
No	243(88.28)	162(84.82)		79(96.34)			
Yes	32(11.72)	29(15.18)		3(3.66)			
Physical inactivity					1.23	0.73-2.13	0.414
No	173(59.71)	111(58.12)		49(59.76)			
Yes	110(40.29)	80(41.88)		33(40.24)			
BMI					1.11	0.55-2.24	0.773
<30kg/m <sup>2</sup>	227(83.15)	158(82.72)		69(84.15)			
≥30kg/m <sup>2</sup>	46 (16.85)	33(17.28)		13(15.85)			

OR-Odd ratio; CI-Confidence interval; HAART-Highly Active Antiretroviral Therapy; IR- Insulin Resistance; HDL-High density lipoprotein; CD<sub>4</sub> +-cluster of differentiation 4; SBp-systolic blood pressure; DBp- diastolic blood pressure

systolic blood pressure (SBP) ≥130 mmHg and/or diastolic blood pressure (DBP) ≥85 mmHg [9].

## 2.6. DATA MANAGEMENT AND ANALYSIS

Questionnaire and laboratory data (initially entered into a paper questionnaire) were entered into Microsoft Excel 2010 and imported into Epi info for statistical analysis. Continuous variables were summarised as mean ± standard deviation (SD) and categorical variables were expressed as percentages.

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS), version 16.0, and Statistics/Data Analysis (STATA) version 10.1 at a 95% confidence interval. Continuous variables were analysed using descriptive statistics procedures; later, we used the student's "t" test to identify any differences. Continuous variables were analyzed with descriptive statistics and compared using Student's *t*-tests. Categorical variables were analyzed using contingency tables involving Chi-

square ( $\chi^2$ ) tests to identify statistical differences between the groups. The relationship between HAART and insulin resistance profile was determined using logistic regression analysis. All p-values were two-tailed, and values less than 0.05 were considered statistically significant. Variables such as gender, age, BMI, educational status, employment status, smoking history, alcohol intake, current CD<sub>4</sub><sup>+</sup> T cell count, HAART use, HAART type, and HAART duration were investigated to determine factors associated with insulin resistance.

## 3. RESULTS

### 3.1. DEMOGRAPHIC CHARACTERISTICS OF STUDY PARTICIPANTS

A total of 273 were enrolled; 70% (191/273) HAART exposed, and 30% (82/273) naïve HIV positive patients. The study participants were dominated by females (193/273; 70.70%), and the majority 47.62% (130/273) of the participants had attained a

**Table 3. Distribution of risk factors of Insulin Resistance by duration of HAART used among HAART exposed participants (n=191).**

Duration of HAART (months)	No (%)	Risk factors for (IR)	OR	95% CI	p-value
<b>Hyperglycaemia</b>					
6 - 59*	97 (50.79)	2(2.06)	1.00	-	-
60 - 119	69(36.13)	1(1.44)	0.70	0.06-7.86	0.77.1
≥120	25(13.09)	3(12.0)	6.48	1.02-41.12	0.048
<b>Hypertriglyceridemia</b>					
6 - 59*	97 (50.79)	14(14.43)	1.00	-	-
60 - 119	69(36.13)	10(14.49)	1.00	0.42-2.42	0.991
≥120	25(13.09)	3(12.00)	0.81	0.21-3.06	0.751
<b>Decreased HDL</b>					
6 - 59*	97 (50.79)	19(19.59)	1.00	-	-
60 - 119	69(36.13)	16(23.19)	1.24	0.58-2.63	0.575
≥120	25(13.09)	2(8.00)	0.36	0.08-1.65	0.187
<b>SBp</b>					
6 - 59*	97 (50.79)	35(36.08)	1.00	-	-
60 - 119	69(36.13)	31(44.93)	1.45	0.77-2.71	0.252
≥120	25(13.09)	11(44.00)	0.39	0.57-3.40	0.467
<b>DBp</b>					
6 - 59*	97 (50.79)	39(40.21)	1.00	-	-
60 - 119	69(36.13)	28(40.58)	1.02	0.54-1.91	0.961
≥120	25(13.09)	11(44.00)	1.17	0.48-2.84	0.731
<b>BMI</b>					
6 - 59*	97 (50.79)	17(17.53)	1.00	-	-
60 - 119	69(36.13)	13(18.84)	1.01	0.49-2.43	0.828
≥120	25(13.09)	03(12.00)	1.17	0.17- 2.39	0.508
<b>WC</b>					
6 - 59*	97 (50.79)	44(45.36)	1.00	-	-
60 - 119	69(36.13)	32(46.38)	1.04	0.56-1.94	0.897
≥120	25(13.09)	12(48.00)	1.11	0.46-2.68	0.813

\*-Reference group; OR-Odd ratio; CI-Confidence interval; HAART-Highly Active Antiretroviral Therapy; IR- Insulin Resistance; HDL-High density lipoprotein; WC- Waist circumference; SBp-systolic blood pressure; DBp- diastolic blood pressure; BMI body mass index.

primary education, followed by 34.07% (93/273) for secondary education, 14.56% (40/273) for tertiary education, and 3.66% (10/273) without any formal education. The ages ranged from 21-73 years, with the overall mean age being  $41.56 \pm 10.29$  years. The majority, 33.70% (92/273) and 32.70% (88/273) of study participants were between 31- 40 and 41-50 years, respectively. Married people constituted a larger proportion of the study participants (145/273; 53.11%), followed by single participants (66/273; 24.18%), whereas widowed participants (62/273; 22.71%) contributed to the minority (Table 1). Most participants did not take alcohol (138/273; 50.55%) nor smoked cigarettes (241/273; 88.28%), while a majority of the participants were not exercising [163(59.71%)].

### 3.2. CLINICAL AND BIOCHEMICAL CHARACTERISTICS OF THE STUDY POPULATION

The overall prevalence of insulin resistance in the study using the NCEP-ATP III Criteria (where they had at least any three abnormalities in the following parameters: waist circumference, systolic blood pressure, diastolic blood pressure, fasting blood sugar, triglyceride or HDL- cholesterol) was 24.2% (66/273) [95 CI: 19.1% -29.3%], with the prevalence higher in HAART pa-

tients (25%(49/191)) as compared to naïve patients (20%(17/82)) (Figure 1).

The mean systolic blood pressure of HAART naïve participants ( $123.78 \pm 17.14$  mmHg) was significantly lower than that of HAART-experienced participants ( $128.97 \pm 27.38$  mmHg) with a P-Value less than 0.05 ( $p=0.01134$ ). Abdominal obesity was significantly higher in the HAART exposed group (46.07%) than in the HAART naïve group (24.39%) [OR 0.38; 95% CI 0.21-0.67;  $P=0.001$ ] (Table 2).

The HAART-expose study participants had high levels of systolic blood pressure, diastolic blood pressure,  $BMI \geq 30.0$  kg/m<sup>2</sup>, central obesity, alcohol intake, smoking habit, physical inactivity, low CD4+ and age  $\geq 50$  years when compared with HAART-naïve participants.

### 3.3. DISTRIBUTION OF RISK FACTORS OF INSULIN RESISTANCE BY DURATION OF HAART USED AMONG HAART EXPOSED PARTICIPANTS

Hyperglycemia was about six times significantly higher in participants on HAART above 120 months than those on HAART under 60 months [OR 6.48; 95% CI 1.02-41.12;  $P=0.048$ ]. Hypertriglyceridemia was not significantly higher with any HAART

**Table 4. Association of HAART regimen used with risk factors for Insulin Resistance (IR) (n=191).**

Type of HAART	No (%)	Risk factor for IR	OR	95% CI	p-value
		Hyperglycaemia			
HAART naïve*	82 (30.04)	4(4.88)	1.00	-	-
NRTI+NNRTI	159(58.24)	5(3.14)	0.63	0.17-2.42	0.505
NRTI+PI	32(11.72)	1(3.13)	0.63	0.07-5.85	0.684
		Hypertriglyceridemia			
HAART naïve*	82 (30.04)	14(17.07)	1.00	-	-
NRTI+NNRTI	159(58.24)	17(10.69)	0.58	0.27-1.25	0.164
NRTI+PI	32(11.72)	10(31.25)	2.21	0.86-5.67	0.100
		Decreased HDL			
HAART naïve*	82 (30.04)	27(32.93)	1.00	-	-
NRTI+NNRTI	159(58.24)	24(15.09)	0.36	0.19- 0.68	0.002
NRTI+PI	32(11.72)	13(40.63)	1.39	0.60-3.24	0.440
		SBp			
HAART naïve*	82 (30.04)	27(33.93)	1.00	-	-
NRTI+NNRTI	159(58.24)	72(45.28)	1.69	0.97-2.94	0.066
NRTI+PI	32(11.72)	5(15.63)	0.38	0.13-1.09	0.071
		DBp			
HAART naïve*	82 (30.04)	28(34.15)	1.00	-	-
NRTI+NNRTI	159(58.24)	67(42.14)	1.41	0.81-2.45	0.230
NRTI+PI	32(11.72)	11(34.38)	1.01	0.43-2.34	0.982
		BMI			
HAART naïve*	82 (30.04)	13(15.85)	1.00	-	-
NRTI+NNRTI	159(58.24)	27(16.98)	1.04	0.50-1.22	0.100
NRTI+PI	32(11.72)	07(21.88)	1.49	0.53- 4.15	0.760
		WC			
HAART naïve*	82 (30.04)	20(24.39)	1.00	-	-
NRTI+NNRTI	159(58.24)	68(42.77)	2.31	1.28-4.20	0.006
NRTI+PI	32(11.72)	20(62.50)	5.17	2.15.-12.40	<0.001

\*-Reference group; OR-Odd ratio; CI-Confidence interval; HAART-Highly Active Antiretroviral Therapy; IR- Insulin Resistance; HDL-High density lipoprotein; WC- Waist circumference; SBp-systolic blood pressure; DBp- diastolic blood pressure; PI-protease inhibitors; NNRTI- nonnucleoside reverse transcriptase inhibitors; NRTI-nucleoside reverse transcriptase; BMI body mass index.

duration categories compared to those under 60 months. Decreased HDL was also not significantly higher in any HAART treatment durations (Table 3). Obesity was about twice as high in participants on HAART above 120 months compared to those under 60 months on treatment, but this was insignificant [OR 1.17; 95% CI 0.17-2.39; P=0.508](Table 3).

### 3.4. ASSOCIATION OF HAART REGIMEN USED (NRTIS + NNRTIS AND NRTIS + PIS) WITH RISK FACTORS FOR INSULIN RESISTANCE

The risk of developing hyperglycemia was similar in the treatment groups compared to the untreated group. Hypertriglyceridemia was more than two times higher among participants treated with Protease inhibitors and about half time as higher among participants treated with non-protease inhibitors, and this was not significant [OR 2.21; 95% CI 0.86-5.67; P=0.100 and OR 0.58; 95% CI 0.27-1.25; P=0.164 respectively]. The risk of developing decreased HDL in the non-protease treatment group was significantly higher compared to the naïve group [OR 0.36; 95% CI 0.19-0.68; P=0.002]. The risk of developing high systolic blood pressure was higher in the treatment group than in the naïve group, though not significant (Table 4).

The risk of having increased diastolic blood pressure was

higher in both the protease treatment group and the non-protease treatment compared to the naïve group, but this was not significantly different [OR 1.01; 95% CI 0.43-2.34; P=0.071 and OR 1.41; 95% CI 0.81-2.45; P=0.0230 respectively]. The risk of having body mass index was higher in both the protease treatment group and the non-protease treatment compared to the naïve group, but this was not significantly different [OR 1.49; 95% CI 0.53-4.15; P=0.760 and OR 1.04; 95% CI 0.50-1.11; P=0.100 respectively]. Having central obesity was about 5 and 2 times higher in protease and non-protease inhibitors treated participants compared to HAART naïve participants, and this was significant. The risk of having increased blood pressure was higher in both the protease treatment group and the non-protease treatment compared to the naïve group, but this was not significantly different [OR 5.17; 95% CI 2.15-12.40; P<0.001 and OR 2.31; 95% CI 1.28-4.20; P=0.006 respectively] (Table 4).

### 3.5. DISCUSSION

The prevalence of metabolic syndrome (insulin resistance) using the NCEP-ATP III Criteria was 24.2% (66/273), where these participants had at least any three metabolic abnormalities in the following six parameters: waist circumference, systolic blood pressure, diastolic blood pressure, fasting blood sugar, triglyc-

eride or HDL- cholesterol. These findings were similar to 20%-37% reported by several other studies [2, 5, 10] with slight differences. This difference could be related to the methodology, especially the metabolic syndrome classification. The prevalence of metabolic syndrome among HAART-exposed (25%) was slightly higher than that of HAART-naïve (20%), but the difference was not significant, which adds more weight to the difficulty of attributing metabolic syndrome either to the infection or the treatment since they both could lead to it [2, 5].

Several reports have shown that HIV can directly block the ATP-binding cassette transporter A1 (ABCA-1) mediated cholesterol efflux to HDL particles, resulting in the intracellular accumulation of lipids and enhanced foam cell formation [12]. So, this could be the reason why we observed a higher prevalence of hypertriglyceridemia, decreased HDL, and hyperglycemia in HAART-naïve participants than in HAART-exposed. The HAART effectively suppresses HIV-1 replication, which may imply a better lipid profile. Still, some combinations of ART drugs have been reported to have varying effects on lipid metabolism, suggesting that HAART plays an additive role in increasing the risk of Metabolic syndrome [11].

The prevalence of hyperglycemia in this study was 3.66%, which was smaller compared to 7-10% reported by previous studies [12–14]; this could be because of the difference in diagnostic and research methods with some longer study periods. This hyperglycemia could be a result of HIV infection, which is known to cause chronic inflammation, or its medications, which have a negative impact on sugar metabolism and could potentially cause insulin resistance [15].

The prevalence of hypertension in HIV/AIDS patients in the Bamenda Regional Hospital was about 38%, which was higher than the 25% of Anastase *et al.* [16] This difference could be due to differences in our smaller sample and blood pressure calculation method. Several factors could be related to the hypertension observed, such as chronic inflammation (Induced by the HIV infection), immune reconstruction due to the immune system's response to ART, which can impact blood, and the changes in fat distribution, which affects the sympathetic and renin-angiotensin-aldosterone systems (RAAS) [17–19].

The risk of abdominal obesity was significantly higher in the HAART-exposed group (46.07%) than in the HAART naïve group (24.39%) [OR 0.38; 95% CI 0.21-0.67; P=0.001]. This could be a result of the PI and NRTIs, which the participants were on, which have been reported to be related to HIV-associated adipose redistribution syndrome [20, 21].

### 3.6. LIMITATIONS AND STRENGTHS OF STUDY

Firstly, the study's small timeline makes it difficult to make causal inferences since only a snapshot of the population was studied. A small number of male participants and a lack of HIV-negative controls were also among the limiting factors. Secondly, confounders such as genotype and diet weren't measured or assessed, which are some risk factors associated with metabolic syndrome. Thirdly, the sampling frame for the study only included HIV-infected persons attending this public hospital, which may imply the study might give just a tiny inference to the general population. As a strength, we excluded all HIV infected with chronic medical illnesses, such as diabetes and

chronic hypertension, as these diseases can have a measurable impact on lipid levels.

## 4. CONCLUSION

It is evident from this study that the following risk factors of metabolic syndrome are common among HIV/AIDS patients: lower HDL, central obesity, alcohol consumption, smoking habits, and age $\geq$ 50 years. This, therefore, highlights the importance of these patients regulating their lifestyle since there is a rising risk of developing metabolic syndromes among them. This work adds to the mapping of epidemiologic data of HIV/AIDS patients in the region and Cameroon at large.

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