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## Original Article

## Association between high blood pressure with risk of type 2 diabetes, metabolic syndrome and its predictors: A cross-sectional study

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## 1. Introduction

Cardiovascular heart disease (CHD) is the leading cause of death globally in men and women [1,2]. A constellation of risk factors known as the metabolic syndrome (MetS) is an increasingly prevalent predisposing factor for CHD [3–5]. Previous studies show positive association between hypertension and CHD [6–8]. The global burden of hypertension and other non-communicable diseases (NCDs) is staggering and African continent may be affected the most [9]. It has been estimated that global prevalence of hypertension is about 1 billion [10–13]. Its prevalence in Nigeria may constitute significant proportion of the total burden in Africa due to its large population (giant of Africa) and its increasing adult population [10,13,14]. The prevalence of NCDs and hypertension [15–20] in Nigeria may be very high because most studies are done in urban settings and are hospital-based than population-based studies. In addition, there are lots of undiagnosed cases in the population. Empirical data on the prevalence of hypertension is essential to the government and other stakeholders of health policy and for intervention. However, systematic reviews of the various prevalence studies on hypertension have been conducted [10,15,21].

In a large-scale randomized trials of blood pressure (BP) lowering drugs, all classes of BP lowering drugs have similar effects in reducing CHD and stroke; there is a 22% reduction in risk of CHD and a 41% in stroke for every 10 mmHg decrease in BP [22]. Furthermore, in trials of BP lowering against control, a greater BP

reductions was achieved via multitherapies than in those for monotherapy [22]. Finally, trials comparing different BP lowering substances, therapies with greatest achieved BP lowering also has resultant greater differences in effects on clinical signs [23].

T2DM (also known as non-insulin-dependent diabetes mellitus) is a heterogeneous metabolic disorder associated with hyperglycemia resulting from impaired insulin utilization coupled with the body's inability to compensate with increased insulin production. It is diagnosed either when symptoms of hyperglycemia (such as polydipsia, polyuria and weight loss) occurred simultaneously by a fasting plasma glucose (FPG)  $\geq 126$  mg/dL (7.0 mmol/L) or 2-h plasma glucose  $\geq 200$  mg/dL (11.1 mmol/L) during oral glucose tolerance test or glycated hemoglobin (HbA1c)  $\geq 6.5\%$  (48 mmol/L) or in patients with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose  $\geq 200$  mg/dL (11.1 mmol/L) according to the National Glycohemoglobin Standardization Program [www.ngsp.org] [24].

In Nigeria, “the giant of Africa” with population well over 180 million, the prevalence of T2DM has been on the increase with the country commonly reported as having the highest burden in Africa [25,26]. Unfortunately, there are no known country-wide studies or any reported effort within Nigeria in recent times to specifically estimate the burden of diabetes or even more its association to other cardiometabolic diseases in the country. The previous national survey of non- NCDs was conducted more than two decades ago with a prevalence of 2.2% reported for diabetes while the results of the 2003 national NCDs survey in the Southwest region was inconclusive [27]. In the 2013 IDF global study, a prevalence of 5% was estimated for Nigeria, accounting for 3.9 million cases among persons 20–79 years old [28]. In 2018, the researchers specifically noted that Nigeria was without up-to-date data on diabetes; as a result, the Nigerian estimate was regressed and extrapolated from pooled estimates in Cameroon, due to relatively homogenous sociodemographic patterns with Nigeria [28].

Metabolic syndrome is a constellation of hypertension, glucose intolerance, abdominal obesity, dyslipidemia and prothrombotic and proinflammatory states. The definition of MetS is controversial and generated a heated debate for a long time. More than two decades ago, several researchers and organizations have suggested

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different definitions. However, all accepted that the characteristics of MetS include hypertension, central obesity, insulin resistance, dyslipidemia, prothrombotic state and microalbuminuria as well as elevated fibrinogen, decreased plasminogen activator-1 (PA-1), increased uric acid and increased blood viscosity [29]. Each anomaly is a comorbidity for the development of atherosclerosis, morbidity and mortality. In addition, MetS has been associated with the development of CHD, stroke, T2DM, retinopathy, diabetic nephropathy and distal neuropathy.

According to the updated National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), the criteria for diagnosing MetS are as follows: 1) fasting plasma glucose  $\geq 110$  mg/dL ( $5.6$  mmolL<sup>-1</sup>), 2) abdominal obesity defined as waist circumference, men  $> 102$  cm and women  $> 88$  cm, 3) blood pressure  $\geq 130/85$  mmHg, 4) triglycerol  $\geq 150$  mg/dL ( $1.7$  mmolL<sup>-1</sup>) and 5) HDL-cholesterol  $<40$  mg/dL ( $<1.03$  mmolL<sup>-1</sup> in men) or  $<50$  mg/dL ( $1.30$  mmolL<sup>-1</sup> in women) [30,31]. MetS was defined as the presence of at least two of the above five abnormalities. Each of these components is a known risk factor for the development of CHD, T2DM and atherosclerosis.

Individuals with MetS are at increased risk for developing hypertension, which is a major risk factor for CHD incidence. It is still a matter of speculation whether treatment of hypertension in isolation among people with MetS will reduce CHD mortality. Hypertension is a major risk factor for CHD and occurs approximately in 50–60% of individuals with T2DM, more than two times common in prediabetic individuals compared to nondiabetic individuals [32,33]. Increasing body of evidence has associated the renin-angiotensin-aldosterone system (RAAS) with hypertension and obesity. Increased plasma aldosterone levels are found in people with MetS, resistant high blood pressure and are linked with the development of left ventricular hypertrophy and increased cardiac fibrosis [34]. Pharmacologic therapies that block RAAS (ACE inhibitors, ARB inhibitors) are considered treatment of choice in hypertensive patients with MetS and T2DM.

Hypertension destroys the vascular wall by endothelial dysfunction, which is the first cascade of events in the atherosclerotic event [35] and can predict subsequent clinical vascular process [36,37]. It has been reported that an increase in BP by small factor can predispose to clinical risks [38]. For instance, epidemiologic analyses revealed that BP  $> 115/75$  mmHg is associated with elevated CVD and mortality among diabetic patients [39–41].

The purpose of this study is to evaluate the association between high blood pressure with T2DM, MetS and their predictors among patients attending the general outpatient department (GOPD) of Enugu State University Teaching Hospital.

## 2. Materials and methods

This is a cross-sectional study conducted from September 2017 to August 2018 at the GOPD of Enugu State University Teaching Hospital. Only subjects 18 years and above, and of Igbo ethnicity were considered for this study. Data from a total of 4202 eligible participants (2772 males and 1430 females) were collected using a standardized questionnaire. Only participants who have fasted overnight ( $\geq 7$  h) before visiting the hospital were included. Physical examination was by trained staff and physician of the hospital using standard protocols. Participants who met the inclusion criteria were required to complete a questionnaire to determine their demographic characteristics. Smokers were defined as those who were currently smoking either regularly or occasionally and regular exercise was defined as 150 min of moderate-intense aerobic activity a week or 60 min of vigorous-intense aerobic activity a week. The study protocol was reviewed and approved by the Enugu State Ministry of Health Research Ethics Committee and only

participants who gave their informed consent were enrolled.

All anthropometric measurements were by well-trained research assistant, with participants wearing only minimal clothing with no footwear during measurements. Height and weight were measured with the subject standing, to the nearest 0.1 cm and 0.2 kg respectively, using a stadiometer and portable weighing scale. BMI was calculated as weight (kg)/square of height (m). Waist and hip circumferences were measured to the nearest 0.1 cm using a flexible measuring tape with the subject standing in the Frankfurt position. Waist circumference (WC) was measured by placing the tape around the abdomen at the level of the umbilicus. Hip circumference (HC) was measured at the level of maximum extension of the buttocks posteriorly in a horizontal plane. Waist height ratio was calculated by dividing waist circumference (cm) by height (cm). Body adiposity index (BAI) was calculated as proposed by Bergman et al. [42]: hip circumference (cm) divided by height (m)<sup>1.5</sup> minus 18. Resting blood pressure (BP) was measured after five minutes in a seated position with a standard mercury sphygmomanometer. BP was grouped into four categories;  $<120/75$ ,  $120\text{--}129/75\text{--}84$ ,  $130\text{--}139/85\text{--}89$  and  $\geq 140/90$  mmHg.

Blood samples were collected on-site, then sent to a laboratory located within the hospital. Fasting blood glucose (FBG) was quantitatively by the enzymatic reference technique with hexokinase [43] while total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C) and triglycerides (TRG) were measured using standard enzymatic colorimetric technique [44,45]. Low-density lipoprotein cholesterol (LDL-C) was assessed indirectly with the aid of the Friedwald formula [46]:  $LDL-C = TC - (HDL-C + (TRG/5))$  for subjects with TRG  $<4.516$  mmol/L (400 mg/dl).

Hypertension was defined as having one of the following: a systolic BP  $\geq 140$  mm Hg, a diastolic BP  $\geq 90$  mm Hg, physician-diagnosed hypertension or use of hypertensive medication. Diabetes was defined as having one of the following: FBG  $\geq 7$  mmol/dl (126 mg/dL), physician-diagnosed diabetes mellitus or use of oral hypoglycaemic medications. Metabolic syndrome (MetS) was defined as the presence of at least three of the following five abnormalities: 1) fasting plasma glucose  $\geq 110$  mg/dL ( $5.6$  mmolL<sup>-1</sup>), 2) abdominal obesity defined as waist circumference, men  $> 102$  cm and women  $> 88$  cm, 3) blood pressure  $\geq 130/85$  mmHg, 4) triglycerol  $\geq 150$  mg/dL ( $1.7$  mmolL<sup>-1</sup>) and 5) HDL-cholesterol  $<40$  mg/dL ( $<1.03$  mmolL<sup>-1</sup> in men) or  $<50$  mg/dL ( $1.30$  mmolL<sup>-1</sup> in women). According to the updated National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria, MetS should be identified as at least three of the five component abnormalities.

## 3. Statistical analysis

Results were expressed as mean  $\pm$  SD for continuous variables and for categorical variables by frequency and percentage. Differences between males and females and health status (MetS-/MetS+) in baseline characteristics were tested using independent samples *t*-tests for continuous variables and by chi-squares or Fisher's exact tests for categorical variables. One-way analysis of variance was used to test for differences in continuous across various blood pressure categories. Multinomial logistic model was used to evaluate the association between blood pressure and diabetes without adjustments. Model A was adjusted for age and sex, and model B: adjusted for model A as well as BMI, smoking status, exercise habits, alcohol intake; model C: adjusted for model B as well as fasting glucose, high density lipoprotein and triglycerides; model D: adjusted for model C as well as waist circumference and the presence or absence of hypercholesterolemia.

Principal Component Analysis was used to reduce the covariates into a single factor. Receiver Operating Characteristics (ROC)

analysis was used to calculate the area under ROC curves (AUC) between each MetS risk factor based on sex. The ROC was also used to identify thresholds that best balanced sensitivity and specificity of the MetS risk factors. Statistics were performed using IBM SPSS version 25 (IBM SPSS Inc., Chicago, IL, USA) and GraphPad Prism 6. All statistics were two-tailed and a *P*-value <0.05 was considered statistically significant.

#### 4. Results

Table 1 shows the general characteristics of the study population, stratified by blood pressure. All anthropometric variables and lipid profile increase significantly with increase in blood pressure. Fasting blood glucose of hypertensive subjects was comparatively higher than that of any other group. This is an indicative that blood pressure is significantly associated with diabetes. The results also revealed significant association between lipid profile, blood pressure and anthropometric variables. No significant difference was observed in FBG between males and females (*P*=0.133). The prevalence of MetS in these subjects was 26.5% (1114/4202; data not shown); but higher among females than males and increased across the BP categories.

The odds ratios of having diabetes mellitus significantly increased across the various BP categories (Table 2). Blood pressure <120/75 was used as the reference category. No significant increase in OR was observed in the group 120–129/75–84 mmHg. The association between high BP with the risk of diabetes was consistent even after adjustment for age, sex, BMI, smoking status, exercise habits, alcohol intake, FBG, HDL-C, TRG, WC and presence or absence of hypercholesterolemia. Finally, the results from the table revealed that for every 1 mmHg increase in BP, subjects with BP 130–139/85–89 and hypertensive patients are more than two times likely to have diabetes than those in control and lower BP groups.

In females, all measures were significantly higher in subjects with MetS (Table 3). In males, all measures are higher among MetS group but were less significantly different. ROC curve analyses are detailed in Table 4. Because MetS was higher among females, their ROC AUC for the five predictors of MetS was significantly higher than that of males.

#### 5. Discussion

In the present cross-sectional study of young adults from southeastern Nigeria, we found that blood pressure categories 130–139/85–89 and hypertensive groups were associated with 2.01- and 2.41-fold higher risks of the development of diabetes after controlling for various factors that are known to either affect metabolism of glucose or be associated with its subsequent risk, such as adiposity. Various studies have investigated the association between association between diabetes and blood pressure. However, most of such studies were not with Africans [47–49]. In one of such studies [50] conducted in a Japanese gas company based on health check-up of employees revealed that hypertension and high normotensive blood pressure are associated with an increased risk of onset of diabetes; a relative risk of 1.39 among men with high normotensive blood pressure (95% CI 1.14–1.69) and 1.76 in men with hypertension (1.43–2.16). Similar finding was observed in another study in Taiwan [51] in which a positive association was found between high blood pressure and onset of diabetes. However, these studies were not population-based cohort studies and oral glucose tolerance tests were not performed in all participants. Even more, significant indicators of insulin resistance or  $\beta$ -cell function were not accounted for in their analyses. Our study is perhaps one of the most comprehensive in Nigeria that reports the association between high blood pressure and diabetes, MetS and its predictors. Various mechanisms have been proposed as underlying the association between high blood pressure and diabetes [52], however, it worth mentioning that no direct causal nexus has been established. The dysfunctional endothelial permeability and decreased peripheral vascular flow caused by high blood pressure may limit insulin delivery and enhance insulin resistance in metabolically active tissues [53]. It has been hypothesized that the oxidative stress associated with high blood pressure plays a critical role in pancreatic  $\beta$ -cell malfunction [54,55]. Another hypothesis is that cytokines associated to oxidative stress (interleukin-1, interleukin-6 and tumor necrosis factor- $\alpha$ ), are capable of modifying lipid and glucose metabolism [56]. Studies investigating the effects of statin therapy on incident diabetes revealed that statin therapy especially at high doses increases the risk of incident

**Table 1**  
Participant characteristics according to blood pressure categories<sup>a</sup>.

Characteristics	<120/75 mmHg	120-129/75–84 mmHg	130-139/85–89 mmHg	Hypertension mmHg	<i>P</i> -value <sup>b</sup>	Male	Female	<i>P</i> -value <sup>c</sup>
n	723	1779	1278	422		2772	1430	
Age	40.97 ± 1.09	42.11 ± 1.72	43.17 ± 1.32	45.59 ± 2.67	<b>0.023</b>	46.17 ± 1.73	44.19 ± 1.43	<b>0.01</b>
Weight (kg)	67.43 ± 10.48 <sup>a</sup>	69.02 ± 10.73 <sup>b</sup>	70.32 ± 11.75 <sup>b</sup>	72.73 ± 10.94 <sup>c</sup>	<b>0.002</b>	69.05 ± 10.77	70.42 ± 11.72	< <b>0.001</b>
BMI (kgm <sup>-2</sup> )	25.93 ± 4.54 <sup>a</sup>	26.63 ± 4.65 <sup>b</sup>	26.79 ± 5.54 <sup>b</sup>	27.65 ± 4.73 <sup>c</sup>	< <b>0.001</b>	26.54 ± 4.93	26.88 ± 4.98	<b>0.035</b>
WC (cm)	80.16 ± 8.75 <sup>a</sup>	81.69 ± 10.11 <sup>b</sup>	81.77 ± 11.10 <sup>bc</sup>	84.41 ± 11.23 <sup>c</sup>	< <b>0.001</b>	80.57 ± 9.73	83.96 ± 11.19	< <b>0.001</b>
HC (cm)	91.74 ± 11.22 <sup>a</sup>	93.50 ± 11.99 <sup>b</sup>	94.83 ± 13.68 <sup>ab</sup>	95.81 ± 12.76 <sup>c</sup>	< <b>0.001</b>	92.52 ± 12.14	96.37 ± 12.91	< <b>0.001</b>
WHR	0.50 ± 0.06 <sup>a</sup>	0.51 ± 0.06 <sup>b</sup>	0.50 ± 0.08 <sup>ab</sup>	0.52 ± 0.07 <sup>c</sup>	< <b>0.001</b>	0.50 ± 0.07	0.52 ± 0.07	< <b>0.001</b>
BAI	26.86 ± 6.56 <sup>a</sup>	27.76 ± 6.61 <sup>a</sup>	27.81 ± 8.45 <sup>ab</sup>	28.33 ± 7.00 <sup>b</sup>	<b>0.004</b>	27.14 ± 7.34	28.73 ± 6.97	< <b>0.001</b>
Triglycerides	1.17 ± 0.83 <sup>a</sup>	1.30 ± 0.93 <sup>b</sup>	1.37 ± 1.03 <sup>bc</sup>	1.43 ± 1.00 <sup>c</sup>	< <b>0.001</b>	1.33 ± 0.97	1.27 ± 0.92	<b>0.039</b>
HDL-C	2.85 ± 1.42 <sup>a</sup>	2.91 ± 1.74 <sup>a</sup>	3.01 ± 1.62 <sup>ab</sup>	3.17 ± 1.44 <sup>b</sup>	< <b>0.001</b>	2.97 ± 1.76	2.89 ± 1.33	0.169
Cholesterol	5.34 ± 1.67 <sup>a</sup>	4.97 ± 1.64 <sup>a</sup>	4.85 ± 1.62 <sup>b</sup>	5.28 ± 1.59 <sup>b</sup>	< <b>0.001</b>	4.99 ± 1.62	5.11 ± 1.68	<b>0.025</b>
Hypercholesterolemia	342 (47.3)	643 (36.6)	430 (33.8)	175 (41.5)	< <b>0.001</b>	1005 (36.5)	585 (41.1)	<b>0.004</b>
MetS (%)	116 (16)	339 (19.1)	496 (38.8)	163 (38.6)	< <b>0.001</b>	309 (11.1)	805 (56.3)	< <b>0.001</b>
Fasting glucose	6.51 ± 1.46 <sup>a</sup>	6.55 ± 1.72 <sup>a</sup>	7.06 ± 1.75 <sup>b</sup>	7.21 ± 5.90 <sup>b</sup>	< <b>0.001</b>	6.74 ± 2.45	6.91 ± 5.04	0.133
SBP (mmHg)	108.25 ± 3.97 <sup>a</sup>	120.15 ± 1.02 <sup>b</sup>	130.35 ± 1.55 <sup>c</sup>	141.22 ± 3.93 <sup>d</sup>	< <b>0.001</b>	123.64 ± 9.99	122.71 ± 9.39	<b>0.004</b>
DBP (mmHg)	77.18 ± 6.69 <sup>a</sup>	81.10 ± 6.58 <sup>b</sup>	89.10 ± 6.21 <sup>c</sup>	91.94 ± 5.16 <sup>d</sup>	< <b>0.001</b>	84.13 ± 8.20	83.58 ± 8.01	<b>0.038</b>
Smoking (kg)	493 (68.2)	1247 (70.1)	933 (73)	279 (66.1)	<b>0.022</b>	1972 (71.1)	980 (68.5)	0.081
Alcohol (%)	409 (56.6)	875 (49.2)	656 (51.3)	217 (51.4)	<b>0.044</b>	1440 (51.9)	717 (50.1)	0.372
Level of exercise	537 (74.3)	1339 (75.3)	947 (74.1)	327 (77.5)	0.055	2045 (73.8)	1105 (77.3)	<b>0.041</b>

<sup>abcd</sup>Denotes Tukey's multiple comparison tests, applied to assess significance difference (*P* < 0.05) in means across blood pressure groups.

<sup>a</sup> Plus–minus values are Means ± SD. To convert the values for glucose to milligram per deciliter, divide by 0.055551. To convert the values for cholesterol to milligram per deciliter, divide by 0.02586. To convert the values for triglycerides to milligram per deciliter, divide by 0.01129.

<sup>b</sup> *P* values were calculated by Fisher's exact test for categorical variables and one-way analysis of variance for continuous variables.

<sup>c</sup> *P* values were calculated by Fisher's exact test for categorical variables and independent sample *t*-tests for continuous variables.

**Table 2**  
Association of blood pressure with the incidence of diabetes in multinomial logistic models.

	<120/75 mmHg (n = 723)	120-129/75–84 mmHg (n = 1779)	P	130-139/85–89 mmHg (n = 1278)	P	Hypertension mmHg (n = 422)	P
		OR (95% CI)		OR (95% CI)		OR (95% CI)	
Crude	1.0	1.28 (0.84, 1.97)	NS	<b>2.20 (1.45, 3.32)</b>	<0.001	<b>2.57 (1.59, 4.15)</b>	<0.001
Model A	1.0	1.27 (0.80, 1.90)	NS	<b>2.16 (1.42, 3.27)</b>	<0.001	<b>2.56 (1.54, 4.03)</b>	<0.001
Model B	1.0	1.25 (0.82, 1.92)	NS	<b>2.14 (1.41, 3.23)</b>	<0.001	<b>2.53 (1.56, 4.10)</b>	<0.001
Model C	1.0	1.23 (0.80, 1.88)	NS	<b>2.07 (1.37, 3.14)</b>	<0.001	<b>2.48 (1.53, 4.01)</b>	<0.001
Model D	1.0	1.17 (0.76, 1.43)	NS	<b>2.01 (1.39, 3.19)</b>	<0.001	<b>2.41 (1.49, 3.91)</b>	<0.001

Model A: adjusted for age and sex, and model B: adjusted for model A as well as BMI, smoking status, exercise habits, alcohol intake; model C: adjusted for model B as well as fasting glucose, high density lipoprotein and triglycerides; model D: adjusted for model C as well as waist circumference and the presence or absence of hypercholesterolemia.

**Table 3**  
Participant characteristics according to health status.<sup>a</sup>

n	Male (n = 2772) <sup>b</sup>			Female (n = 1430) <sup>b</sup>		
	MetS-	MetS+	P	MetS-	MetS+	P
	2463	309		625	805	
Weight, kg	68.15 ± 10.71	69.24 ± 11.25	0.161	64.49 ± 9.46	75.03 ± 11.22	< 0.001
BMI, kgm <sup>-2</sup>	26.48 ± 5.02	27.03 ± 4.12	0.067	25.73 ± 4.30	27.78 ± 5.28	< 0.001
Waist circumference, cm	79.98 ± 9.51	80.65 ± 11.37	0.255	75.60 ± 7.16	90.45 ± 9.28	< 0.001
Hip circumference, cm	92.08 ± 11.96	92.58 ± 13.52	0.499	87.79 ± 10.03	103.02 ± 10.79	< 0.001
Waist-height ratio	0.50 ± 0.07	0.50 ± 0.06	0.216	0.48 ± 0.05	0.55 ± 0.07	< 0.001
Body adiposity index	27.03 ± 7.51	27.95 ± 5.82	<b>0.039</b>	26.05 ± 5.78	30.82 ± 7.10	< 0.001
Triglycerides, mmol/L	1.20 ± 0.87	2.40 ± 1.06	< 0.001	1.11 ± 0.81	1.39 ± 0.99	<b>0.008</b>
HDL-Cholesterol, mmol/L	2.95 ± 1.79	2.96 ± 1.54	0.908	2.79 ± 1.39	2.98 ± 1.27	<b>0.009</b>
Cholesterol, mmol/L	4.65 ± 1.66	5.03 ± 1.30	< 0.001	4.85 ± 1.72	5.30 ± 1.62	< 0.001
Fasting glucose, mmol/L	6.61 ± 2.50	7.71 ± 1.65	< 0.001	6.21 ± 1.70	7.45 ± 2.56	< 0.001
SBP, mmHg	122.47 ± 9.72	132.95 ± 6.69	< 0.001	120.83 ± 8.71	124.18 ± 9.64	< 0.001
DBP, mmHg	83.18 ± 8.00	91.74 ± 5.21	< 0.001	82.71 ± 8.00	84.26 ± 7.96	< 0.001

<sup>a</sup> All values are means ± SDs. Group differences were analyzed with the use of Student's independent *t*-test. MetS+, metabolic syndrome present, MetS-, metabolic syndrome absent.

<sup>b</sup> Sample sizes varied depending on metabolic outcomes.

**Table 4**  
Results of ROC curve analyses to detect metabolic syndrome.<sup>a</sup>

	Threshold	Sensitivity <sup>b</sup>	Specificity <sup>b</sup>	Likelihood ratio	ROC AUC <sup>c</sup>
Male (n = 2772)					
BMI	>26.28	52 (47, 58)	52 (50, 54)	1.1	<b>0.54 (0.51, 0.57)<sup>†</sup></b>
Mid upper arm circumference, cm	>28.5	54 (48, 59)	52 (50, 54)	1.12	0.51 (0.48, 0.55)
Waist-height ratio	>0.5	49 (43, 55)	55 (53, 57)	1.09	0.52 (0.48, 0.55)
Body adiposity index	>26.82	53 (47, 59)	53 (51, 55)	1.14	<b>0.54 (0.51, 0.58)<sup>†</sup></b>
Triglycerides, mmol/L	>1.76	84 (79, 88)	84 (83, 86)	5.29	<b>0.84 (0.81, 0.87)<sup>‡</sup></b>
High density lipoprotein, mmol/L	>3.11	53 (47, 59)	56 (54, 58)	1.2	0.52 (0.48, 0.56)
Cholesterol, mmol/L	>4.81	59 (53, 64)	58 (56, 60)	1.4	<b>0.58 (0.55, 0.61)<sup>‡</sup></b>
Combined <sup>d</sup>	>-0.07	53 (47, 58)	53 (51, 55)	1.11	0.51 (0.47, 0.54)
Female (n = 1430)					
BMI	>26.45	59 (55, 62)	59 (55, 63)	1.44	<b>0.63 (0.60, 0.66)<sup>‡</sup></b>
Mid upper arm circumference, cm	>29.5	66 (63, 70)	66 (62, 70)	1.96	<b>0.72 (0.70, 0.75)<sup>‡</sup></b>
Waist-height ratio	>0.51	76 (72, 78)	74 (70, 77)	2.88	<b>0.83 (0.81, 0.85)<sup>‡</sup></b>
Body adiposity index	>28.04	66 (63, 69)	66 (62, 69)	1.92	<b>0.73 (0.70, 0.75)<sup>‡</sup></b>
Triglycerides, mmol/L	>1.11	54 (51, 58)	56 (52, 60)	1.22	<b>0.60 (0.57, 0.62)<sup>‡</sup></b>
High density lipoprotein, mmol/L	>2.95	56 (53, 60)	53 (49, 57)	1.19	<b>0.55 (0.52, 0.58)<sup>‡</sup></b>
Cholesterol, mmol/L	>5.22	55 (51, 58)	57 (53, 61)	1.27	<b>0.58 (0.55, 0.61)<sup>‡</sup></b>
Combined <sup>d</sup>	>-0.14	70 (67, 73)	70 (66, 74)	2.34	<b>0.78 (0.75, 0.80)<sup>‡</sup></b>

<sup>†</sup>P < 0.01.

<sup>‡</sup>P < 0.001.

ROC; receiver operating characteristic.

<sup>a</sup> Threshold values with highest sensitivity and specificity to detect metabolic syndrome.

<sup>b</sup> Values are percentages; 95% CIs in parentheses.

<sup>c</sup> Values in parentheses are 95% CIs.

<sup>d</sup> Combined predictive strength from principal component analysis.

diabetes. In contrast, lipid-lowering therapies were not associated with the incidence of diabetes [57].

The prevalence of MetS of 26.5% found in this study is quite high. Females account for even higher than males given the age category

of the study population. This sample was meant to represent adults at mid-age group. Our findings on MetS was consistent with prior study in Kenya that reported a prevalence of 25.6% [58] among subject slightly above those in the present study. Torriani and

associates (2014) however reported a slightly higher prevalence (50%) among patients attending Massachusetts General Hospital [59].

There is a growing need for affordable diagnostic tool that can supplement biochemical test for MetS. Anthropometric variables have gained significant interest due to its non-invasive, affordability and simplicity for optimal diagnosis of MetS and other cardiovascular risk factors. Results from present cross-sectional study reveal gender disparity in the prevalence and predictors of MetS. Our data show significant association between MetS and all its components in females. Weight, BMI, HC, waist-height-ratio, BAI along with all five components of MetS are significantly associated with MetS in females while only BAI was significantly associated with MetS in males. Paradoxically, WC, a component of MetS did not show significant association with MetS among males. It stands to reason that MetS among males is mainly due to other four components of MetS while in females, all five components significantly contribute to MetS. Furthermore, the presence of lipid stores in skeletal muscle has been reported to be associated with risk factors of MetS [60,61]. Therefore, triglycerides, cholesterol, fasting glucose level and systolic BP are screening tools in identifying development of MetS among males unlike females in which both anthropometric and lipid profile are indicators. In contrast, Pavanello et al. [62] found significant association between WHtR and MetS regardless of gender. Furthermore, previous studies have also shown significant association between WC and MetS irrespective of gender [63–67]. The high prevalence of MetS observed in females than males calls for the need for more awareness of MetS among females to prevent or reduce the risk of MetS and CHD. The high prevalence and predisposing factors seen in females comparatively to male may be due to high fat accumulation especially abdominal obesity in females. Although it has been reported that central obesity measured by MRI is more reliable than that assessed anthropometric parameters or indices [68], availability, cost, technical know-how surrounding MRI makes it rather impossible for assessing central obesity in Nigeria.

The association between WC and other components of MetS have been well reported; increase in WC have been linked to increase in TRG and glucose levels as well as decrease in HDL-C level among healthy mid-aged males and females [69]. A correlation of 0.73 and 0.78 ( $P < 0.001$ ) respectively between MetS and WC were obtained for males and females and a significantly high correlation between HDL-C, visceral adiposity, systolic BP and FBG [70].

The present study provides evidence that WHtR is the strongest predictor of MetS among females. Other powerful tools that predict MetS are MUAC, WHtR, BAI, HDL-C and total cholesterol while TRG level is the best predictor of MetS among males. Unlike in females, MUAC, WHtR, HDL-C are not predictors of MetS among males. In order of superiority in predicting MetS in females, our study suggests WHtR > BAI > MUAC > BMI > TRG level > TG > HDL-C. Previous study has also reported that WHtR as the best predictor of MetS in women [71]. In males we found that BMI and HDL-C are weak predictors of MetS. Our sensitivity and specificity analyses indicated that combining all measures rather than screening for each one separately in females may increase the reliability for prediction of MetS. Though this is not the case for male subjects.

In conclusion, the present study provides additional evidence that high BP is a risk factor to the onset of diabetes mellitus. For subjects in the high BP groups, for every 1 mmHg increase in BP, the subjects are more than two times more likely to have diabetes mellitus. The study also provides evidence that WHtR is among the most powerful tools to predict MetS among females while TRG was the best predictive value of MetS among males.

## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.dsx.2018.11.065>.

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