Mbadiwe et al www.iaajournals.org IAA Journal of Applied Sciences 7(1):95-105, 2021. ©IAAJOURNALS

ISSN: 2636-7246

Evaluation of the anthropometric measures of obesity with Cardiometabolic Risk factors in relation to gender and age

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ABSTRACT

Despite the rising global epidemic of obesity, and increasing number of studies on the association of the anthropometric measures of obesity with obesity. cardiometabolic risk factors namely blood pressure, fasting blood glucose and lipid profile in relation to gender and age have not been fully explored up to date. The aim of this study was to evaluate the anthropometric variables with blood pressure, fasting blood glucose and lipid profile in relation to gender and age. A total of 224 subjects participated in the study. 144 obese and 80 non-obese (control). The weight, height, waist circumference and hip circumference were measured, BMI and WHR were calculated, and B.P measured. Fasting blood glucose (FBG) test, fasting lipid profile test, chest x-ray and ECG were carried out on all subjects, while echocardiography was performed on the first 144 eligible subjects (84 obese and 60 non-obese) who turned up for the echocardiography. BMI showed significant correlation with both systolic and diastolic BP in females. Total cholesterol (TC) had significant correlation with both BMI. In conclusion BMI has a good correlation with blood pressure and lipids and therefore is a good predictor of hypertension and hyperlipidemia.

Keywords: Anthropometric variables, blood pressure, fasting blood glucose and lipid profile, gender and age.

INTRODUCTION

adolescence Obesity during is associated with cardiovascular mortality in adulthood. The adverse obesity-related cardiometabolic risk profile already is observed in adolescence. We aimed to examine possible gender differences in cardiometabolic risk factors and lifestyle behaviors among adolescents with severe obesity, hypothesizing that have both a higher bovs would prevalence of the metabolic syndrome as well as less healthy lifestyle behaviors than girls. Obesity is becoming a global epidemic, [1,2] and in the past two decades in the United States of America, dramatic increases in obesity have occurred in both children and adults [3, 4] The word Obesity is the nominal form of 'Obese' which comes from the latin word Obesus which means "Stout, fat or plump [5].

Obesity is a condition in which the natural energy reserve, stored in the fatty tissue of humans and other animals is increased to a point where it is a risk factor for certain health condition or increased mortality [6]. It is the normal physiological response to an environment in which energy intake exceeds energy output [7]. It is an mechanism. adaptive Major environmental changes that support this adaptive mechanism are the greater availability of foods especially in the developed countries, and the increase in sedentary life-style [8]. The social environment has moved from being obesity retardant to being obesity conducive. This important has implications on patients with certain genotypes. Metabolic rates differ between persons and may be important in determining who becomes obese [9].

Excessive body weight has been shown to predispose to various diseases, particularly cardiovascular diseases. diabetes mellitus type 2, sleep apnoea, and osteoarthritis [10]. The presence of cardiovascular diseases such as coronary heart disease, hypertension, and other risk factors including smoking, age and family history of obesity, are not only used to establish a clinical diagnosis but also they may indicate treatment [11, 12,13]. Certain causative factors have been suggested to contribute to the development of obesity, namely:

- Genetic factors and some genetic disorders (e.g. Prader-Willi syndrome)
- Underlying illness (e.g. hypothyroidism)
- Certain medications (e. g. atypical antipsychotic)
- Sedentary life style.

In the developed countries of Europe and America, studies on obesity abound. But in the developing nations of Africa, and particularly, the West African sub region, only a few studies have been reported [15]. Therefore it is needful to carry out more studies in order to enrich our obesity data base in this part of the world. Secondly, considering that we live in an environment with scarce resources and

- A high glycaemic diet (i.e. a diet that consists of meals that give high post prandial blood sugar).
- Weight cycling caused by repeated attempts to lose weight by dieting.
- Eating disorder (such as binge eating disorder)
- Stressful mentality
- Insufficient sleep
- Smoking ceasation [14].

As with many medical conditions, the caloric imbalance that results in obesity often develops from a combination of genetic and environmental factors. Polymorphism in various genes controlling appetite, metabolism, and adipokine release predispose to obesity, but the condition requires availability of sufficient calories, and possibly other factors to develop fully [15].

RATIONALE FOR THE STUDY

limited manpower, it would be useful to know which of the anthropometric measures of obesity best indicates risk of cardiovascular disease, so that emphasis could be rightly placed on the particular measure(s) in the evaluation and management of obesity. This will ultimately help in the reduction of the global burden of cardiovascular diseases.

AIM OF THE RESEARCH

factors (blood pressure, fasting blood glucose and lipid profile) in relation to gender and age.

MATERIALS AND METHODOLOGY

Design

The study was cross-sectional, descriptive and hospital based.

The aim of this research was to evaluate

obesity with some cardiometabolic risk

the anthropometric measures of

Setting

Place of study

This was at the University of Nigeria Teaching Hospital, UNTH, Enugu, South Eastern Nigeria, a 760 bed tertiary

Eligible and consenting patients were drawn from the medical out-patient department of the UNTH from March 2006 to November 2006. Most of the institution serving Enugu and neighbouring states.

Sample population

patients were Ibos, a tribe in the South Eastern part of Nigeria, with a relatively high literacy level, although majority of them were traders.

Ethical cl						
Ethical clearance was obtained from the Ethics committee of Basis of D	-					
• Subjects with BMI of 30kg/m ² and above were taken as obese; those with BMI of less than 30kg/m ² were taken as non obese.	 Waist-hip ratio of >0.85 for women and >0.9 for men were also taken as central/visceral obesity. 					
 Abdominal (waist) circumference of 88cm for women and >102 for men were taken as central/visceral obesity. Hyperte 	ension					
A mean blood pressure of \geq 140 mmHg (systolic) and/or \geq 90 mmHg	(diastolic) was regarded as Hypertension.					
Hypergly	vcaemia					
Using capillary whole blood sample: Fasting blood glucose level of: • 3.3 mmol/L - 5.6 mmol/L was normal	 >5.6 mmol/L and < 6.1 mmol/L was impaired fasting glycaemia (IFG). ≥ 6.1 mMol/L was Diabetes Mellitus. 					
Hyperlipidaemia						
• Total cholesterol level of > 6.2	• HDL level of <0.9 (<35 mg/dl) is low					
mmol/L	and regarded as dyslipidaemia					
(>240 mg/dl) is high	 Triglyceride level of > 1.69 mmol/L (150mg/dl) is hunortriglycerideemia 					
 LDL level of > 3.36 (>130 mg/dl) is high VLDL level of > 1.3 (50mg/dl) is high 	(150mg/dl) is hypertriglyceridaemia					
Sample Size						
Patient (sampl The prevalence of obesity in adult	Where $n = the$					
Nigerians is estimated at 10.5% Using	minimum sample size					
the WHO formula for sample size	P= prevalence rate (in a previous					
determination in a finite population (Fischer's formula):	study) Z = Standard deviation value at 95%					
$\mathbf{n} = 7^2 \mathbf{D} \mathbf{r}$	confidence interval					
$n = \frac{Z^2 Pq}{d^2}$	d = Sample error tolerated (5.0%)					
ŭ	The sample size is thus calculated to be 144.					
Control S	-					
Eighty (80) non-obese patients were recruited consecutively along side the Cons	obese patients. These served as a control for the study.					
Informed consent was obtained from subjects, both sample and control groups. Those who were not literate Study Cri	enough to sign their signature were free to use thumb printing.					
Inclusion	l Criteria					
The study included adult patients of both sexes, up to 18 years and above but not more than 75 years, who were	not pregnant and had no chronic debilitating diseases like tuberculosis,					

metastastic cancers and other diseases **Exclusion Criteria**

- Age below 18 years and above 75 • vears
- Smokers
- Pregnant women
- Women on oral or parenteral contraception.
- AIDS patients.
- **Tuberculosis** patients •
- Patients who have metastatic cancer and patients on cytotoxic therapy.
 - Verbal withdrawal of consent

- mentioned in the exclusion criteria.
 - Patients with chronic renal failure, • nephrotic syndrome. chronic glomerulo-nephritis etc.
- **Oedematous** patients
- Patients with congestive cardiac failure, cardiomyopathies and structural heart diseases including valvular heart diseases.
- Patients who have kyphoscoliosis.
- Amputees
- Patients who could not stand.

Withdrawal Criteria

Failure to turn up for laboratory tests.

Materials Equipment for study

- Stadiometer (Hospitex brand) (a)
- Weighing balance (incorporated (b) in the stadiometer)
- (c) Tape measure.

(d)

- The mercury sphygmomanometer (Accoson brand) with standard cuff size 15cm x 55xm, and Lithmans stethoscope.
- Standard 3 channel (e) electrocardiograph (ECG) machine (cardiette authoruler) with 12 leads.
- (f) Glucometer (Accutest)
- (g) Autoanalyzer
- (h) 2 Dimensional Echocardiography machine (Hewlett Packard m2406A Ultrasonic system)

METHODOLOGY

Procedure

Anthropometric data (weight, height, waist circumference and hip circumference) of all eligible patients who attended the medical outpatient clinic during the period of study were collected. To obtain the anthropometric measurements, each subject or control was asked to stand on the stadiometer bare foot and with minimal clothing, without shoes in a special room set out for the purpose. The weight and height were thus measured. Then the waist circumference (WC) was measured using the tape measure, with the patient standing bare foot on the floor. The WC was taken at the level of the iliac crests [16], passing along the umbilical level of the unclothed abdomen; and the hip circumference was measured at the level of the external margins of the anterior superior iliac spines [17]. The mean of two readings was taken. Patients whose BMI were up to 30kg/m^2

and above were recruited for the study, and those with BMI $<30 \text{kg/m}^2$ were recruited as controls, if they satisfied the inclusion criteria, and gave their consent. Data collection sheet was pretested on about 10 consecutive patients. These were reviewed and then applied to consenting subjects. Blood pressure was measured in the sitting position, with the patient's index arm resting on the consulting table, after patient must have relaxed for at least 10 minutes. The right arm was used for every patient for the purpose of uniformity. The 1st Korotkoff sound was used to determine the systolic blood pressure, and the disappearance of the sound (or muffling if the sound does not disappear) was used to determine the diastolic blood pressure. Two readings were taken at 10 minutes interval, and the mean of the two reading was recorded as the blood pressure. About 8

am, after an overnight fast: a drop of capillary blood was used for fasting blood glucose test, by means of Analysis of data was done using SPSS version 11, while statistical calculations were carried out with the computer software PEPI (programme for Epidemiologists), version 4.0. Categorical variables were compared using the non parametric chi-square (χ^2), while parametric variables were compared using the student t-test. The relationship between anthropometric variables and the cardiovascular risk RESULTS

Table 1: Correlation of the anthropometric variables with blood pressure, fasting

glucometer. 5 millilitres of blood sample was also drawn for laboratory tests [18].

Data Analysis

factors was analyzed by Pearson's correlation coefficient test, while the relationship between anthropometric variables and echocardiographic parameters was evaluated bv Spearman's rho coefficient of correlation test, Pearson's correlation coefficient and Stepwise regression analysis. Partial correlation was used to correct for differences in age and to control for blood pressure.

blood glucose and lipid profile in all the subjects in relation to gender

Risk factor	Gender	BMI	WC	WHR
		r (p-value)	r (p-value)	r (p-value)
Systolic BP (mmHg)	M (n = 90)	0.596 (0.000)***	0.643 (0.000)***	0.383 (0.000)***
	F (n = 134)	0.536 (0.000)***	0.408 (0.000)***	0.185 (0.033)*
Diastolic BP (mmHg)	M (n = 90)	-0.636 (0.000)***	-0.571 (0.000)***	-0.346 (0.001)***
	F (n = 134)	-0.499 (0.000)***	-0.421 (0.000)***	0.102 (0.243)
Fasting blood glucose	M(n = 90)	0.135 (0.209)	0.153 (0.151)	0.122 (0.256)
(mmol/l)	F (n = 134)	0.191 (0.028)*	0.322 (0.000)***	0.156 (0.073)
Lipid profile:				
Total cholesterol	M(n = 90)	0.013 (0.903)	0.040 (0.707)	0.022 (0.835)
(mmol/l)	F(n = 134)	0.293 (0.001)***	0.225 (0.009)**	0.151 (0.082)
HDL (mmol/l)	M (n = 90)	0.042 (0.697)	-0.053 (0.621)	-0.070 (0.516)
	F(n = 134)	0.106 (0.224)	-0.037 (0.677)	-0.015 (0.862)
LDL (mmol/l)	M (n = 90)	0.097 (0.364)	-0.042 (0.693)	-0.017 (0.872)
	F(n = 134)	0.136 (0.118)	0.186 (0.032)*	0.107 (0.219)
VLDL (mmol/l)	M (n = 90)	0.374 (0.000)***	0.251 (0.017)	0.307 (0.003)**
	F(n = 134)	0.149 (0.086)	0.130 (0.136)	0.188 (0.031)*
TG (mmol/l)	M (n = 90)	0.234 (0.028)*	0.111 (0.299)	0.183 (0.086)
	F(n = 134)	0.130 (0.137)	0.190 (0.029)*	0.175 (0.044)*

***P =< 0.001 * P< 0.05, **P < 0.02,

M = male

F = female

After correcting for difference in age, blood pressure systolic showed significant correlation with BMI ((m) r =0.596; p = 0.000, (f) r = 0.536; p = 0.000), WC ((m) r = 0.643; p = 0.000, (f) r= 0.408; p = 0.000) and WHR ((M) r = 0.383; p = 0.000, (f) r = 0.185; p = 0.033)) in both males and females. Diastolic blood pressure showed significant correlation with BMI ((m) r =-0.636; p = 0.000, (f) r = 0.499; p = 0.000) and WC ((m) r = -0.571; p = 0.000, (f) r = -0.421; p = 0.000) in males and females. When these were subjected to further analysis only BMI showed significant correlation with both systolic and diastolic BP in females (p =0.000 and p = 0.000 respectively, while in males WC had the best correlation with systolic BP (p = 0.003), and BMI had the best correlation with diastolic BP (p = 0.000). Fasting blood glucose (FBC) in females showed correlation with WC (r =0.322; p = 0.000) and BMI (r = 0.191; p =0.028) only. No correlations were found in males. Further analysis however WC indicated that had stronger correlation with FBG (p = 0.000) than

BMI (p = 0.235). Total cholesterol (TC) had significant correlation with both BMI (r = 0.293; p = 0.001) and WC (r =0.225; p = 0.009) in females only, but further analysis yielded BMI as having the best correlation with TC (p = 0.027). No correlation was found in males. HDL had no correlation with BMI, WC and WHR. LDL had significant correlation with WC only, and in females only (r =0.186; p = 0.032). VLDL had significant correlation with WHR only in females, (r = 0.188; p = 0.031), but in males it had correlation with BMI (r = 0.374; p = 0.000), WHR (r = 0.307; p = 0.003) and WC (r = 0.251; p = 0.017). Further analysis of this result showed BMI to have the best correlation with VLDL cholesterol (p = 0.003). Triglyceride had significant correlation with BMI only in males, (r = 0.234; p = 0.028), but with WC and WHR in females (r = 0.190; p =0.029 and r = 0.175; p = 0.044respectively). Further analysis however did not show any significant correlation between the WC and WHR and TG (p =0.116 and p = 0.305 respectively).

Risk factor		BMI	WC	WHR
	Age	r (p – value)	$\mathbf{R} = (\mathbf{p} - \mathbf{value})$	r (p – value)
Systolic BP	<40 yrs (n = 25)	0.266 (0.198)	0.211 (0.312)	0.307 (0.135)
(mmHg)	≥40 yrs (n = 199)	0.015 (0.833)	0.032 (0.651)	-0.002 (0.975)
Diastolic BP	<40 yrs (n = 25)	0.371 (0.068)	0.364 (0.073)	0.250 (0.228)
(MmHg)	<u>≥</u> 40 yrs (n = 199)	0.067 (0.345)	0.096 (0.179)	0.044 (0.539)
F (1111	40 (25)	0 1 4 4 (0 4 0 0)	0.000 (0.01 ()	0.004 (0.070)
Fasting blood	<40 yrs (n = 25)	0.144 (0.492)	0.209 (0.316)	0.034 (0.870)
glucose	\geq 40 yrs (n = 199)	0.192 (0.006)**	0.299 (0.000)***	0.168 (0.018)**
(mmol/L))				
Lipid profile:				
Total Cholesterol	<40 yrs (n = 25)	0.479 (0.015)**	0.453 (0.023)*	0.479 (0.015)**
(mmol/L)	>40 yrs (n = 199)	0.115 (0.105)	0.102 (0.153)	0.005 (0.948)
HDL (mmol/L)	<40 yrs (n = 25)	0.036 (0.866)	-0.098 (0.642)	-0.120 (0.568)
	\geq 40 yrs (n = 199)	0.162 (0.023)*	0.006 (0.937)	-0.041 (0.564)
LDL (mmol/L)	<40 yrs (n = 25)	0.011 (0.959)	0.060 (0.776)	0.404 (0.045)*
	<u>></u> 40 yrs (n = 199)	-0.027 (0.702)	0.055 (0.438)	-0.004 (0.956)
	10 (25)			0.450.00.015
VLDL (mmol/L)	<40 yrs (n = 25)	0.096 (0.646)	0.161 (0.441)	0.479 (0.015)**
	≥40 yrs (n = 199)	0.320 (0.000)***	0.221 (0.002)**	0.166 (0.019)**
TG (mmol/L)	<40 yrs (n = 25)	0.448 (0.025)*	0.238 (0.251)	0.470 (0.018)**
10 (IIIII0I/L)	<40 yrs (n = 23) \geq 40 yrs (n = 199)	0.175 (0.014)**	0.190 (0.007)**	0.129 (0.070)
	2 + 0 yrs (11 - 199)	$0.173(0.014)^{-1}$	$0.190(0.007)^{11}$	0.129(0.070)

Table 2: Correlation of the anthropometric variables with blood pressure, fasting blood glucose and lipid profile in relation to age

* P< 0.05, **P < 0.02, ***P < 0.001 In relation to age <40 years and \geq 40 years, no correlation existed between the BMI, WC and WHR and the systolic and diastolic blood pressures. But fasting blood glucose had stronger correlation with WC (r = 0.299; p =0.000) than BMI (r = 0.192; p = 0.006) and WHR (r = 0.168; p = 0.018) in the older age group. No correlation was found in the younger age group. The BMI and WHR have similar correlation with total cholesterol (r = 0.479; p = 0.015 each), and stronger than that of WC (r = 0.453; p = 0.023) in the younger subjects, but no correlation was found for the older age group. HDL was correlated with BMI only, in the older age group (r = 0.162; p = 0.023), and there was no other correlation in both

the younger and older age groups. LDL had significant correlation with WHR only in the younger age group (r =0.404; p = 0.045). VLDL was highly correlated with BMI in the older subjects (r = 0.320; p = 0.000), followed by WC (r = 0.221; p = 0.002) and WHR 0.166 (0.019). But WHR alone had significant correlation with VLDL in the younger age group (r = 0.479; p = 0.015). Triglyceride was more strongly correlated with WC (r = 0.190; p = 0.007) than BMI (r = 0.175; p = 0.014) in older subjects, and not correlated with WHR. But in the younger subjects, WHR had highest correlation, (r = 0.470; p =0.018), followed by BMI (r = 0.448; p =0.025), and none for WC.

BMI was found in this study to have the strongest correlation with systolic and diastolic blood pressure in females, and diastolic blood pressure in males. This is supported by the result of study by [19] who also found BMI to have the strongest correlation with high systolic and diastolic blood pressure in females, after adjusting for age differences, in Australian adults. Amodu and colleagues using BMI as the measure of obesity also found very high association between obesity and Hypertension, with 71.6% of the hypertensive women being obese, and 50.5% of the hypertensive men being obese [20]. The relationship between obesity and hypertension can be explained: generalized obesity. reflected in high BMI has been shown to give rise to increment in total blood volume and cardiac output partly due to increased metabolic demand induced by excess body weight [21,22]. Also in generalized obesity there is stimulation of renin release and promotion of renal tabular absorption of sodium [23]. These mechanisms are responsible for increase in blood pressure in obese individuals. In this study also, waist circumference was found to have the strongest correlation with fasting blood glucose levels in the females, but no significant correlation in males. The reason for the gender difference is not clear, but obesity is more prevalent in females than males [24] and this may contribute to the relationship being better portraved in females than in males. Abdominal adiposity which is increased reflected in waist circumference, is more lipo-active than the adipose tissue in other parts of the body, and are easily mobilized into circulating free fatty acids, which have been found to enhance the development of insulin resistance and hence hyperglycaemia. This may explain why waist circumference is found to be strongly correlated with fasting blood glucose level. The finding in this study, of strong correlation between fasting glucose level blood and waist circumference is similar to the result of a study by [25] in which they also found the strongest correlation between fasting glycaemic levels and waist circumference in females (but closer relationship between BMI in males) and weakest correlation with WHR; and that by [26] in which WHR was also found to have the weakest association with diabetes and other cardiovascular risk conditions. It however differs from the result of study by [27] in which they found waist-hip ratio to have the strongest relationship with type 2 diabetes. This divergence in findings may be as a result of difference in study designs.

The finding of very strong correlation between total cholesterol and BMI in females in this study is supported by the result of study by [28] in which they found a better correlation between total cholesterol and BMI than WHR in female hypertensive patients. However, unlike in this study where no correlation was found in males, they found significant correlation between WHR and total in the male cholesterol subjects. although waist circumference was not considered in their study. It is therefore possible that gender has some effect on the relationship between serum lipids and the anthropometric measurements. The strong correlation between BMI and lipids found in this study is, however, at variance with the results of the study by [29] in which they found WHR to be most closely related plasma lipids in Canadian men and women, and that by [30]. in which WC and WHR were found to be more closely correlated with plasma lipids than BMI in Hindu men. Again differences in study designs may have contributed to the differing results: the latter two studies were population based with fairly large sample sizes, unlike this study and that by [30] which are hospital-based with relatively small sample sizes. The hospital subjects had one ailment or the other which may have exerted some confounding effects on the outcome of the lipid profile and the correlation with anthropometric measurements. Due to rising global epidemic of obesity, there are speculations that the adverse health

consequences of excess adiposity will speescalate in the future. In this context, rang several investigators have described the circ adverse effects of obesity on the heart stru [31,32,33]. Obesity has been linked to a faile CONCLUSION

In conclusion BMI has good correlation with blood pressure and lipids therefore

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spectrum of cardiovascular changes ranging from a hyperdynamic circulation, through sub-clinical cardiac structural changes to eventual heart failure [34].

is a good predictor of hypertension and hyperlipidemia.

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