Obesity and Hypertension: A Product of BMI and WHR is a Better Predictor of Hypertension

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Abstract

Obesity is a modifiable risk factor for hypertension. Waist-hip-ratio is favored over body mass index for assessing truncal obesity and cardometabolic risk. Is a product of body mass index and waist-hip-ratio a better predictor of hypertension than waist-hip-ratio?

The objective of this study was to determine the measure of obesity that best predicted hypertension. The study was a cross-sectional survey of 3013 participants across the 36 states of Nigeria and Abuja. The census sampling technique was used to collect the data. The data collecting instruments included measuring tape, stadiometer, weighing scale, and Amron blood pressure monitor. The data was analyzed using X² test and correlation.

The mean body mass index of the participants was 26.99 ± 4.89kg/m², waist circumference 79.13 ± 26.72m, hip circumference 87.24 ± 28.57cm, waist-hip-ratio 0.91 ± 0.07, systolic blood pressure 129 ± 18mmHg, and diastolic blood pressure 80 ± 12mmHg.

Measures of obesity had statistically significant positive correlation with systolic and diastolic blood pressures. The best predictor of hypertension was a product of body mass index and waist-hip-ratio (r .228 and .200), followed by body mass index (r .191 and .180), then waist-hip-ratio (r .187 and .135), waist circumference (r .082 and .089), and lastly hip circumference (r .040 and .060).

A product of body mass index and waist-hip-ratio should be used to assess obesity since it predicts hypertension better than either of the two alone. This study should also be extended to other risk factors of cardiovascular disease like diabetes and dyslipidemia.

Keyword: Body Mass Index, Waist Hip Ratio, Waist Circumference, Hip Circumference, Hypertension.

Introduction

Obesity is a modifiable risk factor of cardiovascular disease (CVD) (Chrysant, 2019). It is also a risk factor of other diseases associated with CVD such as dyslipidemia, type 2 diabetes, and hypertension (Hall et al., 2019). CVD is the leading cause of death globally (Kotsis et al., 2019).

Despite varying criticisms over the years, body mass index (BMI), as shown in Table 1, remains the most acceptable and widely used tool for determining abnormal body weight in adults globally (Shams-White et al., 2019). Critics have highlighted that BMI does not indicate the proportion of body weight attributable to fat, and an increase in body weight due to muscle mass reflects as abnormal BMI (Friibbeck et al., 2019). A person with visceral obesity can have a normal BMI and vice versa (Gurunathan and Myles, 2016). Authors have also critiqued BMI for not taking into account the age, racial, and geographical variations seen across the globe (Stanford et al., 2019; Weaver et al., 2019). Recent research has shown that BMI does not indicate truncal obesity and is therefore, not a good predictor of cardio-metabolic outcomes (Mastroeni et al., 2019). What constitutes the best predictor of CVD, the leading cause of death globally remains a subject of research.

Emerging technologies are becoming favorable allowing for better measures of obesity (Heckman et al., 2019). These methods include dilution methods (hydrometry), sophisticated computerized tomography (CT), magnetic resonance imaging (MRI), dual-energy x-ray absorptiometry (DEXA), bioelectrical impedance, and ultrasound (Jones et al., 2019; Kutâê et al., 2019; Woo and OH, 2019). Adab et al. (2018) asserted that DEXA and imaging techniques are the most accurate direct measures of obesity.
However, Fang et al. (2018) maintained that waist circumference (WC) remains a simple, inexpensive, and better method of measuring abdominal obesity than recent advances such as CT, MRI, DEXA, bioelectrical impedance analysis, ultrasound, or 3D body scanning that may not be readily available in developing countries. In contrast to BMI, waist circumference (WC) has been reported to measure visceral obesity better than waist hip ratio (WHR), and it is associated with increased cardiometabolic risks irrespective of BMI (Gurunathan and Myles, 2016). WHR is faulted for failing to address the racial differences of buttock and hip fat distribution (Lalazar, 2015). The limitation of WC is its inability to differentiate subcutaneous fat from visceral fat (Freisling et al., 2017).

Therefore, there is a need for a globally acceptable and reliable method for determining what constitutes an abnormal body weight and abnormal body composition. There is also the need for such a measurement to significantly predict cardiometabolic risks such as dyslipidemia, diabetes, hypertension, and ultimate cardiovascular morbidity and mortality. This study focused on determining if a product of BMI and WHR (BMI*WHR) is a better predictor of hypertension than BMI, WHR, WC, or hip circumference (HC) alone.

Methods

The dataset used in this study was from a cross-sectional survey of 3013 bank employees during a healthy lifestyle program across the 36 states of Nigeria and the capital city Abuja in 2018. The survey was a census of all central bank employees where every staff was invited to participate. Participation was voluntary but excluded employees on sick, annual, or study leave and duty tour, and those excused because of work exigency. The aim of this secondary study was to determine the best obesity measure predictor of hypertension. The obesity measure predictors studied were BMI, WC, HC, and WHR. A product of BMI and WHR (BMI*WHR) was also computed and tested against BMI, WC, HC, and WHR to determine the best predictor of hypertension. Personal data was deleted in this study for ethical reasons. Approval was obtained from the Medical Services Department to use the dataset after the research proposal was reviewed by the in-house ethics committee. Approval was also given by the Health Research Ethics Committee of the Federal Capital Territory, Abuja.

The primary data was collected by qualified medical personnel through interviews and measurement of anthropometric parameters, blood pressure (BP), and calculation of BMI manually using calculators as weight in kilogram divided by height squared in centimeters. Other data collecting instruments included sewing tape, weighing scales, stadiometers, and electronic sphygmomanometers. The instruments were checked, calibrated, and validated by technicians before the commencement of the program. Additionally, electronic sphygmomanometers and weighing scales were also tested against similar instruments known to be accurate. This dual check ensured that the instruments used had proper calibration for accurate measurements. The participants were first registered, during which personal information was taken and informed consent obtained by signing the attendance register. Then, a team of doctors and nurses took vital signs and anthropometric measurements of BP, height, and weight using standard procedures.

Weight was measured with the participants wearing their exercise kit consisting of a tracksuit and program shirt. Mobile phones, keys, and other heavy items were kept aside before weight measurement. WC was measured above the hipbones (iliac crests) with the participant standing up straight and after normal expiration while HC was measured at the widest part of the buttocks in straight standing position. Table 2 summarized the adopted procedure used for BP measurement. BP was recorded as systolic BP over diastolic BP in mmHg.

The primary data in Excel was exported to SPSS. BMI, WHR, and BP were also transformed and grouped as ordinal variables based on the WHO criteria to allow for cross-tabulation and X² tests. The ordinal variables were BMI groups, WHR groups, and BP groups. BMI groups and WHR groups were used as measures of obesity. The BP groups were normal (<140/90mmHg) and hypertension (>140/90mmHg). Obesity was defined as BMI >30 kg/m² or >0.85 in women and >0.9 in men and hypertension was defined as BP >140/90mmHg.

The predictor variable was obesity assessed by BMI, WC, HC, WHR, and BMI*WHR while the dependent variables were systolic BP, diastolic BP, and hypertension. BMI was measured in kg/m², and
WC and HC in cm. The unit of BMI*WHR remained the same as BMI since WHR was a ratio and had no unit.

The analysis involved cross-tabulation and $X^2$ tests and correlations to test associations between obesity and hypertension. A confidence level (CL) of 95% and a level of significance of 0.05 were used for the analysis. When p-value was < 0.05, the observed association was reported as statistically significant.

The strength of the association was used to determine the best predictor of hypertension. Cramer’s V and Phi or odds ratio (OR) were used for asymmetrical and two by two tables respectively for categorical variables in $X^2$ test. Similarly, correlation coefficient ($r$) was used for continuous variables in Pearson’s correlation. The predictor with the highest strength was reported as the best predictor of the outcome.

**Results**

The BMI of the 3013 participants ranged from 14kg/m$^2$ to 53 kg/m$^2$, WC from 24cm to 142cm, HC from 28cm to 152cm, WHR from 0.56 to 1.43, BMI*WHR from 11.95 to 51.69kg/m$^2$, systolic BP from 79mmHg to 214mmHg, and diastolic BP from 50mmHg to 150mmHg (Table 3). The mean BMI, WC, HC, WHR, BMI*WHR, systolic BP, and diastolic BP of the participants were 26.99 ± 4.89kg/m$^2$, 79.13 ± 26.72cm, 87.24 ± 28.57cm, 0.91 ± 0.07, 24.54 ± 5.30kg/m$^2$, 129 ± 18mmHg, and 80 ± 12mmHg respectively (Table 3).

BMI, WHR, and diastolic BP had almost identical means, medians, and modes of 27kg/m$^2$, 0.9, and 80mmHg respectively suggesting a near perfect normal distribution as depicted in the respective histograms with normality curves (Figure 1). However, BMI*WHR had trimodal distribution with the average mode (23.9kg/m$^2$) tallying roughly with mean (24.54 kg/m$^2$) and median (23.94 kg/m$^2$).

Bivariate analysis using cross-tabulation and $X^2$ (Table 4) showed a statistically significant association between hypertension and obesity measured by both BMI ($X^2$ (1) = 50.34, p < .001) and WHR ($X^2$ (1) = 32.62, p < .001). The effect size of both associations was small but stronger with BMI (Phi 129, p < .001) than with WHR (Phi .104, p < .001). Similarly, the odds of being obese and hypertensive (OR 1.9, 95% CI 1.59 - 2.27) was more than the odds of having abnormal WHR and hypertension (OR 1.64, 95% CI 1.38 - 1.94).

Furthermore, the prevalence of hypertension was higher among obese participants (38%) than among those with abnormal WHR (31.3%), and both figures were in excess of the overall prevalence of 27.6% (Table 5). Therefore, obesity, as measured by BMI is a better predictor of hypertension than obesity defined by abnormal WHR.

Pearson’s correlation demonstrated a statistically significant positive correlation between obesity and hypertension with most p values < 0.001(Table 6). The strength of the association as measured by the correlation coefficient ($r$) was different for the various measures of obesity (BMI, WHR, WC, HC, and BMI*WHR). A product of BMI and WHR (BMI*WHR) had the strongest positive correlation with both systolic BP ($r$ .228, p < .001) and diastolic BP ($r$ .200, p < .001). This was followed by BMI having a systolic BP relationship with r .191, p < .001 and diastolic BP with r .180, p < .001. WHR was next with systolic BP coefficient of r = .187, p < .001 and diastolic BP coefficient of r = .135, p < .001. WC had systolic r .082, p < .001 and diastolic r .089, p < .001. HC had the weakest strength (systolic: r = .040, p = .029 and diastolic r = .060, p = .001).

A plot of systolic and diastolic r against BMI*WHR, BMI, WHR, WC, and HC revealed that BMI*WHR, BMI, and WHR predicted systolic BP more than diastolic BP while WC and HC predicted diastolic BP more than systolic BP (Figure 2). Among the better predictors of systolic BP, WHR was the best, followed by BMI*WHR, and BMI was the least. Similarly, HC predicted diastolic BP better than WC.

$X^2$ test suggested that BMI was a better predictor of hypertension than WHR while Pearson’s correlation suggested that BMI*WHR was the best predictor of hypertension while others followed in this descending order: BMI, WHR, WC, and HC. Therefore, BMI*WHR is a better predictor of hypertension than either BMI WHR, WC, or HC.
**Discussion**

Obesity is defined as excessive body weight due to fat (Garvey, 2019). This study employed the use of common non-invasive anthropometric measurements to assess obesity (Mastroeni et al., 2019). These anthropometric parameters include BMI, WC, HC, and WHR. In addition, the study used a new parameter, BMI*WHR, to predict hypertension alongside the benchmarks of BMI, WHR, WC, and HC.

This study demonstrated the known association between obesity and hypertension based on findings from X² tests as well as Pearson’s correlation. Obesity as measured by BMI was a better predictor of hypertension than obesity measured by abnormal WHR. The strength of the association with BMI was higher than that of abnormal WHR as determined by both Phi (.129, p < .001 vs .104, p < .001) and OR (1.9, 95% CI: 1.59 - 2.27 vs 1.64, 95% CI: 1.38 - 1.94). This finding contradicts arguments that BMI does not assess visceral fat or differentiate fat from muscle mass (Gurunathan and Myles, 2016) and that BMI lacks gender, racial, and geographical variation (Agbim et al., 2019), though the role of visceral or central or truncal obesity in the etiopathogenesis of cardiovascular disease (CVD) has long been recognized (Barroso et al., 2017).

However, in another study using area under the curve, Adejumo et al. (2019) demonstrated that WC was the best predictor of cardio-metabolic risk when compared to ten other anthropometric measures: abdominal volume index, ‘A’ body shape index, BMI, body adiposity, body roundness index, conicity index, lipid accumulation product, visceral adiposity index, waist circumference-triglyceride index, and weight height ratio (WHR) in both men (0.814, 95% CI: 0.721 - 0.907) and women (0.819, 95% CI: 0.771 - 0.867).

Using Pearson’s correlation, BMI*WHR appears to be a better predictor of hypertension than the other tested parameters (r .228/200, p <.001), followed by BMI (r .191, p <.001), then WHR (r .187, p <.001), WC (r .082, p <.001) and lastly, HC (r .040, p .029). This finding is in contrast to other findings. Atkins (2019) reported WHR as a better predictor of CVD mortality than BMI, but studies in sub-Saharan Africa and Turkey suggest that waist-to-height ratio (WtHr) is a better predictor of central obesity, CVD, type 2 diabetes, and oxidative stress than other anthropometric parameters (Ashwell et al. 2012; Meseri et al., 2014; Ware et al., 2014). Ashwell et al. (2012) using area under the curve (AUC) showed WtHr to be significantly better than BMI and WC for predicting Hypertension, CVD, diabetes, and all outcomes (p < 0.005) in adults.

This new parameter, BMI*WHR, has not been studied before now. The only parameter close to it is a combination of BMI and WHR. A study in Singapore demonstrated this combination as the best predictor of CVD risks when compared with BMI alone, body adiposity index, WC, and WHR (Lam et al., 2015).

**Conclusion**

The objective of this study of determining the measure of obesity that best predicts hypertension has been met largely. The study has proved that BMI*WHR is a better predictor of hypertension than BMI, WHR, WC, or HC alone. The study has also provided supporting evidence on the association between obesity and hypertension.

This new tool, BMI*WHR, needs further studies to explore its potential in assessing obesity and in predicting hypertension as well as other cardiometabolic risks such as dyslipidemia and type 2 diabetes. It is potentially, an important public health tool in developing countries where newer and emerging sophisticated imaging modalities involving CT and IMR are not readily available.

The study needs to be replicated with cardiometabolic outcome variables such as fasting blood lipids, fasting blood sugar, and glycosylated hemoglobin. This approach will provide a wider review of metabolic syndrome and cardiovascular risks, and establish the increasing role of obesity in predicting those risks.

Healthy eating, regular exercise, and weight loss are established interventions that reduce hypertension and other cardiometabolic diseases. These intrapersonal strategies will go a long way in discouraging sedentary lifestyle and curbing the escalating menace of cardiovascular morbidity and mortality globally.
Tables and figures

Table 1. Classification of BMI by WHO

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI Score (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt; 18.5</td>
</tr>
<tr>
<td>Normal</td>
<td>18.5 – 24.9</td>
</tr>
<tr>
<td>Overweight</td>
<td>25.0 – 29.9</td>
</tr>
<tr>
<td>Obesity type 1</td>
<td>30.0 – 34.9</td>
</tr>
<tr>
<td>Obesity type 2</td>
<td>35.0 – 39.9</td>
</tr>
<tr>
<td>Obesity type 3 (morbid obesity)</td>
<td>&gt; 40.0</td>
</tr>
</tbody>
</table>

Table 2. Adopted procedure for using electronic BP measurement

1. The participant was seated comfortably with his forearm resting on the table.
2. An appropriate cuff size that matched to the size of the arm was chosen.
3. The cuff was applied firmly to the bare upper arm so that the lower edge was 3 cm above the elbow crease and the bladder was centered over the brachial artery.
4. There was no rest period before measurement.
5. The arm with the BP cuff was maintained at heart level.
6. There was no talking or movements of any parts of the body, and legs were not be crossed.

Table 3: Univariate descriptive statistics of the variables

<table>
<thead>
<tr>
<th>Statistics/Variable</th>
<th>BMI</th>
<th>WC</th>
<th>HC</th>
<th>WHR</th>
<th>BMI*WHR</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>26.98</td>
<td>79.13</td>
<td>87.24</td>
<td>.91</td>
<td>24.54</td>
<td>128.63</td>
<td>79.91</td>
</tr>
<tr>
<td>Median</td>
<td>26.50</td>
<td>88.00</td>
<td>98.00</td>
<td>.90</td>
<td>23.94</td>
<td>126.00</td>
<td>79.00</td>
</tr>
<tr>
<td>Mode</td>
<td>27.00</td>
<td>92.00</td>
<td>100.00</td>
<td>.90</td>
<td>23.90</td>
<td>120</td>
<td>80</td>
</tr>
<tr>
<td>Std. Deviation</td>
<td>4.85</td>
<td>26.72</td>
<td>28.57</td>
<td>.07</td>
<td>5.30</td>
<td>18.14</td>
<td>12.38</td>
</tr>
<tr>
<td>Minimum</td>
<td>14.04</td>
<td>24.00</td>
<td>28.00</td>
<td>.56</td>
<td>11.95</td>
<td>79</td>
<td>50</td>
</tr>
<tr>
<td>Maximum</td>
<td>52.80</td>
<td>142.00</td>
<td>152.00</td>
<td>1.43</td>
<td>51.69</td>
<td>214</td>
<td>151</td>
</tr>
</tbody>
</table>

Figure 1. Histograms with normality curves
Table 4. Association between obesity and hypertension

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Outcome</th>
<th>Test</th>
<th>X²</th>
<th>df</th>
<th>P-value</th>
<th>Effect size (Φ)</th>
<th>OR 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity by BMI</td>
<td>Hypertension</td>
<td>X²</td>
<td>50.34</td>
<td>1</td>
<td>&lt; .001</td>
<td>.129 (p &lt; .001)</td>
<td>1.9 (1.59 - 2.27)</td>
</tr>
<tr>
<td>Obesity by WHR</td>
<td>Hypertension</td>
<td>X²</td>
<td>32.62</td>
<td>1</td>
<td>&lt; .001</td>
<td>.104 (p &lt; .001)</td>
<td>1.64 (1.38 - 1.94)</td>
</tr>
</tbody>
</table>

Table 5. Obesity by BMI and abnormal WHR as predictors of hypertension

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Significant Predictor</th>
<th>SR</th>
<th>% Within Predictor</th>
<th>% Total Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>Obesity by BMI</td>
<td>5.3</td>
<td>38%</td>
<td>27.6%</td>
</tr>
<tr>
<td></td>
<td>Obesity by abnormal WHR</td>
<td>3.0</td>
<td>31.3%</td>
<td></td>
</tr>
</tbody>
</table>

Table 6. Correlations between measures of obesity and hypertension

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Systolic BP</th>
<th>Diastolic BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI*WHR</td>
<td>r .228, p &lt; .001</td>
<td>r .200, p &lt; .001</td>
</tr>
<tr>
<td>BMI</td>
<td>r .191, p &lt; .001</td>
<td>r .180, p &lt; .001</td>
</tr>
<tr>
<td>WHR</td>
<td>r .187, p &lt; .001</td>
<td>r .135, p &lt; .001</td>
</tr>
<tr>
<td>WC</td>
<td>r .082, p &lt; .001</td>
<td>r .089, p &lt; .001</td>
</tr>
<tr>
<td>HC</td>
<td>r .040, p .029</td>
<td>r .060, p .001</td>
</tr>
</tbody>
</table>

Figure 2. A plot of correlation coefficients against predictor variables

Acknowledgement

I wish to express my gratitude to the management of the banks in the 36 states of Nigeria and Abuja for giving me the permission and support to conduct this study.

References


