Adiposity Measures in Metabolic Syndrome among Hausas in Kano, Northern Nigeria

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Summary: Ethnic variations exist in the relationship of adiposity indices with metabolic syndrome (MetS). There are however, limited studies on the usefulness of body adiposity index (BAI) and visceral adiposity index (VAI) among Hausas of Kano, Northern Nigeria. The aim of the study was to determine the relationship of measures of adiposity to the components of MetS in Hausas of Kano. The study included 465 (266 males and 199 females) subjects, with mean age of 34.4 years and 32.0 years for males and females respectively. Anthropometric measures were obtained using standard protocols. Visceral adiposity was estimated using sex specific VAI. Fasting blood sample was obtained for serum analyses of lipid profiles, glucose, protein and uric acid. Pearson’s correlation was used to test the association between adiposity measures with MetS indices while Student’s t test was used for group comparison. The results of the study showed that the adiposity indices significantly correlate with metabolic syndrome indices. Visceral adiposity index was superior to other adiposity measures and Waist to hip ratio was the strongest anthropometric correlate of MetS components. In conclusion, WHR is the strongest anthropometric correlate of MetS components. Body adiposity index, NC and HC are weaker adiposity tools. Visceral adiposity index is superior to all other adiposity tools.

Keywords: Adiposity measures, metabolic syndrome, metabolic syndrome biomarkers, Hausas, Northern Nigeria.

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INTRODUCTION

Body adiposity is documented to be tightly linked with cardio-metabolic risk factors and metabolic syndrome (Akuyam et al., 2009) which are leading causes of death in both developed and developing countries (Mahmoud et al., 2010). The metabolic syndrome is a cluster of interrelated common clinical disorders, including hypertension, hyperglycemia, glucose intolerance and dyslipidaemia in addition to obesity (Moller and Kaufman, 2005). It is defined based on the presence of three or more of the following criteria: abdominal obesity with waist circumference >94cm for men or > 80cm for women (Grundy et al., 2005), triglycerides >150 mg/dl, high density lipoprotein cholesterol (HDL-cholesterol) < 40 mg/dl for men or <50 mg/dl for women (Bergman et al., 2006), blood pressure >130/85 mmHg (Tremblay et al., 2004) and fasting glucose >100 mg/dl (Grundy et al., 2005).

Rapidly rising prevalence levels of the MetS in developed and developing countries and the associated high mortality and morbidity are forcing scientists to review promising therapeutic agents and population specific anthropometric criteria for defining its phenotype (Matsuzawa, 2005). Robust evidences in the literature indicate that the various anatomic reserve of adipose tissues donot carry the same burden of metabolic risk (Bergman et al., 2011; Amato et al., 2014). It is also documented that that race and ethnicity affects both adiposity measures (Tulloch-Reid et al., 2003) and pattern of relationship with metabolic parameters (Duerenberg et al., 1998). There is currently an ongoing controversy on the particular adiposity measure which has the best discriminatory strength and confers the highest metabolic risk (Bergman et al., 2011; Mbanya et al., 2015). Conflicting results have been reported from different races and ethnic groups showing variation in the strength of correlation of the various adiposity measures with the different components of the MetS (Bergman et al., 2011; Mbanya et al., 2015). It is becoming a popular notion in recent time that adiposity and metabolic risk do not follow a universal trend and therefore MetS risk prediction must take into account ethnicity and population peculiarities. On this
note, many ethnic groups or populations such as South Asians, Chinese, and Aboriginals among others have identified adiposity measures that are germane to their population in terms of correlation and prediction of metabolic syndrome indices (Razak et al., 2007). Similarly, the validity of adiposity measures in various disease conditions and their population and ethnic variability have been reported among Nigerians (Charles-Davies et al., 2012).

Visceral adiposity index (VAI) is a recently derived index to measure visceral fat based on the knowledge of waist circumference (WC), plasma HDL, triglycerides and BMI (Amato and Giordano 2014). VAI has been adjusted for gender and is based on the formula proposed by Amato and Giordano (2014). The body adiposity index (BAI) is also a relatively new adiposity measure which was described and subsequently validated (Bergman et al., 2011). It estimates percentage of body adipose tissue in both sexes without numerical correction and has the advantage of not requiring a gender-specific calculation making this surrogate index very convenient for practical use. The usefulness of this adiposity measure has been tested in other populations and revealed varying degree of correlation with MetS components (Amato et al., 2010; Bergman et al., 2011; Amato and Giordano, 2014). Such studies are scarce in the Nigeria literature, we therefore seek to investigate the usefulness of BAI and VAI and compare with other measures of body adiposity in terms of their relationship with the different components of MetS, uric acid and adiponectin. The aim of this study is to identify the adiposity markers that are most germane to the Hausas of Kano in MetS.

MATERIALS AND METHODS

Study Location and participants

Systematic random sampling technique was employed in selecting 465 original Hausas of Kano based on a history of at least two parental generation being Hausas from Kano. Participants were recruited from outpatient units of Murtala Muhammad Specialist Hospital, Khadija Memorial Hospital, Shehu-Uran clinic, General Hospital Dawakin-Tofa and the old campus of Bayero University, Kano. The study included only subjects in the age range of 18 years to 68 years. Subjects with pregnancy, abdominal or pelvic space occupying lesions, congenital and / or acquired spinal deformity were however excluded. Subjects that were on medications that could interfere with any component of metabolic syndrome were also excluded. Ethical approval was obtained from Kano State Hospitals Management Board and written informed consent obtained from the subjects. The study was conducted commenced October, 2016 and ended September, 2017.

Anthropometry

Height was measured to the nearest 0.1cm as the vertical distance between the standing surface and the vertex of the head while the subject was standing erect in the frank forth plane and without shoes using a stadiometer. The weight was measured in kilograms using a digital weighing scale while the subject is in light clothes. The body mass index was be calculated by dividing the weight in kilograms by the square of the height in meters and the result expressed in kg/m². Waist circumference was measured in centimeter with a non- stretchable plastic tape horizontally placed over the unclothed abdomen at the narrowest point between the lowest rib and the iliac crest. Hip circumference: was measured while the subject is standing erect with the feet fairly close together; pockets emptied and the tape passed around the point with the maximum circumference over the bottom (Lean et al., 1995). WHR and WHtR were obtained by dividing waist circumference by hip circumference and height respectively. Neck circumference: was measured in centimeter with a non- stretchable plastic tape horizontally placed over the unclothed neck at the level of the thyroid cartilage (Lean et al., 1995).

Estimation of Visceral and Body Adiposity Index

Visceral adiposity was estimated using sex specific visceral adiposity index (Amato and Giordano, 2014):

\[ VAI(Male) = \frac{WC}{39.68 + (1.88 \times BMI)} \times \frac{1.03 \times TG}{1.31} \]

\[ VAI(Female) = \frac{WC}{36.58 + (1.89 \times BMI)} \times \frac{0.81 \times TG}{1.31} \]

Where WC is waist circumference, TG is triglyceride, HDL is high density lipoprotein and BMI is body mass index

Body adiposity index was obtained using the formula proposed by Bergman et al. (2011).

\[ Body \ Adiposity \ Index \ (BAI) = \frac{Hip \ Circumference \ (cm)}{Height \ (m)^{1.5}} - 18 \]

Measurement of Blood Pressure

A mercury sphygmomanometer was used for measuring blood pressure. Two measurements were taken, and at least 2 minutes was allowed between readings. While the diastolic reading was taken at the level when sounds disappear (Korotkoff phase V), the systolic was taken at the level when it appears (Prisant et al., 1995). The brachial artery was the site of auscultation. Subjects were asked to refrain from smoking or ingesting caffeine for 30 minutes before measurement and the Measurement was taken after at least 5 minutes of rest (Haffner et al., 2005).

Estimation of Serum Parameters

For the estimation of serum total cholesterol(TC), triglyceride(TG), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), and fasting blood glucose (FBG), uric acid and adiponectin, blood specimen was collected from 161 of the subjects after 10 to 12 hours of fasting via
superficial veins of the upper limb. From each selected subject, 5ml of venous blood sample was collected using a sterile 21G needle fitted with syringe. Blood collection was done at the morning hours before 8 a.m to avoid the effect of diurnal variation or circadian rhythm in the blood parameters to be measured. Standard technique of venepuncture and universal safety precaution was employed. Blood sample was transferred into a plain blood specimen bottle and allowed to clot. Sample was then centrifuged at 300rpm for 5 minutes after which serum was separated and immediately used for analysis. Serum glucose was measured using the enzymatic method of Trinder (1969). Serum TC, TG and HDL concentrations were measured using enzymatic method by Wybenga et al. (1970). LDL-cholesterol was calculated from measured values of total cholesterol, triglycerides and HDL-cholesterol according to the Friedewald’s equation. (Friedewald et al., 1972)

\[
\text{LDL-Cholesterol} = \text{TC} - (\text{HDL-C} + \text{Triglycerides}/2.2) \text{mmol/L.}
\]

Serum uric acid concentration was measured using Caraway method (1955). Serum adiponectin concentration was determined using the Solid - Phase ELIZA method (Pischon et al., 2003).

**Statistical Analysis**
The data were expressed as mean ± standard deviation. Pearson’s correlation was used to determine the relationship between each adiposity measure and the metabolic parameters. Student’s t test was used to compare between-group parameters of males and females. SPSS version 20 (IBM Corporation, NY) software was used for statistical analyses and P < 0.05 was set as level of significance.

**RESULTS**
A total of 465 subjects were studied, 266 males (57%) and 199 females (43%). The subjects had a mean age of 34.45 years and 32.06 years for males and females respectively. Table 1 and 2 showed descriptive statistics of age, anthropometric indices of adiposity, blood pressure and serum biomarkers metabolic syndrome of participants.

Table 2 showed correlation of anthropometric and visceral adiposity markers with MetS components in the studied population, males and females respectively. Among the anthropometric indices of adiposity in the general population, BMI showed significant positive correlation with all the serum components of MetS (r = 0.31, 0.34, 0.39 0.42) for FBG, TC, TG and LDL respectively, except HDL with significant negative correlation (r = - 0.28). Its correlation with the biomarkers of MetS was positive for serum uric acid (r = 0.31) and negative for adiponectin (r = - 0.39). However, both were statistically significant. BMI correlated positively and significantly with both SBP (r = 0.42) and DBP (r = 0.46). The Pearson coefficient (r) showed that, among the serum parameters of MetS, BMI had the strongest correlation with TC (r = 0.43) and LDL-C (r = 0.42) and weakest correlation with HDL-C. Its strength of correlation was similar for both components of BP and for SUA and Adiponectin. BMI also correlated positively with VAI (r = 0.38).

### Table 1: Descriptive statistic of age, anthropometric indices of adiposity and blood pressure of participants

<table>
<thead>
<tr>
<th>Variables</th>
<th>Male (n=260)</th>
<th>Female (n=199)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.45 ± 13.52</td>
<td>32.06 ± 15.18</td>
</tr>
<tr>
<td>Height(cm)</td>
<td>169.15 ± 6.27</td>
<td>158.53 ± 6.83</td>
</tr>
<tr>
<td>Weight(Kg)</td>
<td>63.03 ± 12.28</td>
<td>55.86 ± 12.99</td>
</tr>
<tr>
<td>BMI</td>
<td>21.98 ± 3.93</td>
<td>22.19 ± 4.7</td>
</tr>
<tr>
<td>WC(cm)</td>
<td>77.28 ± 11.17</td>
<td>76.02 ± 13</td>
</tr>
<tr>
<td>HC(cm)</td>
<td>87.01 ± 7.8</td>
<td>88.96 ± 9.86</td>
</tr>
<tr>
<td>NC(cm)</td>
<td>34.99 ± 2.29</td>
<td>31.58 ± 2.46</td>
</tr>
<tr>
<td>W/H</td>
<td>0.89 ± 0.08</td>
<td>0.85 ± 0.11</td>
</tr>
<tr>
<td>W/HT</td>
<td>0.46 ± 0.06</td>
<td>0.48 ± 0.08</td>
</tr>
<tr>
<td>BAII</td>
<td>21.6 ± 3.71</td>
<td>26.61 ± 4.62</td>
</tr>
<tr>
<td>DBP</td>
<td>82.59 ± 12.37</td>
<td>84.5 ± 12.99</td>
</tr>
<tr>
<td>SBP</td>
<td>128.07 ± 20.09</td>
<td>130.66 ± 21.87</td>
</tr>
</tbody>
</table>

Validity of Adiposity measures in metabolic syndrome among Hausas

Table 2: Descriptive statistic of serum biomarkers and indices of metabolic syndrome

<table>
<thead>
<tr>
<th>Variables</th>
<th>Male (n=120)</th>
<th>Female (n=41)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Urinary Acid</td>
<td>5.51</td>
<td>1.95</td>
</tr>
<tr>
<td>Adiponectin</td>
<td>23.28</td>
<td>5.96</td>
</tr>
<tr>
<td>FBG</td>
<td>84.67</td>
<td>24.73</td>
</tr>
<tr>
<td>TC</td>
<td>174.35</td>
<td>32.31</td>
</tr>
<tr>
<td>HDL-C</td>
<td>44.1</td>
<td>6.32</td>
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<tr>
<td>TG</td>
<td>117.18</td>
<td>31.76</td>
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<tr>
<td>LDL-C</td>
<td>106.81</td>
<td>32.44</td>
</tr>
</tbody>
</table>

Table 3: Correlation of anthropometric and visceral adiposity markers with MetS components in the general population

<table>
<thead>
<tr>
<th>DBP</th>
<th>SBP</th>
<th>SUA</th>
<th>Adiponectin</th>
<th>FBG</th>
<th>TC</th>
<th>HDL-C</th>
<th>TGR</th>
<th>LDL-C</th>
<th>VAI</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>0.465** 0.427** 0.314** -0.397** 0.318** 0.434** -0.287** 0.396** 0.420** 0.360**</td>
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<tr>
<td>WC (cm)</td>
<td>0.573** 0.578** 0.540** -0.582** 0.502** 0.638** -0.482** 0.586** 0.615** 0.644**</td>
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<tr>
<td>HC (cm)</td>
<td>0.208** 0.111** 0.016** -0.114** 0.061 0.166** 0.006** 0.138 0.141 0.109**</td>
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<tr>
<td>NC (cm)</td>
<td>0.303** 0.270** 0.294** -0.364** 0.177** 0.317** -0.502** 0.392** 0.381** 0.318**</td>
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<tr>
<td>W/H</td>
<td>0.439** 0.759** 0.841** -0.834** 0.760** 0.837** -0.701** 0.805** 0.845** 0.834**</td>
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<tr>
<td>W/Ht</td>
<td>0.561** 0.609** 0.558** -0.593** 0.553** 0.643** -0.442** 0.602** 0.627** 0.675**</td>
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<tr>
<td>BAI</td>
<td>0.142** 0.139** 0.097** -0.142** 0.172** 0.205** 0.058 0.178 0.164* 0.261**</td>
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<tr>
<td>VAI</td>
<td>0.795** 0.869** 0.888** -0.854** 0.860** 0.901** -0.808** 0.937** 0.891** 1</td>
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</table>

Table 4: Correlation of anthropometric and visceral adiposity markers with MetS components in male participants

<table>
<thead>
<tr>
<th>DBP</th>
<th>SBP</th>
<th>SUA</th>
<th>Adiponectin</th>
<th>FBG</th>
<th>TC</th>
<th>HDL-C</th>
<th>TGR</th>
<th>LDL-C</th>
<th>VAI</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>0.331** 0.356** 0.289** -0.300** 0.247** 0.372** -0.332** 0.379** 0.361** 0.339**</td>
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<tr>
<td>WC (cm)</td>
<td>0.543** 0.611** 0.552** -0.575** 0.522** 0.590** -0.600** 0.600** 0.587** 0.635**</td>
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<tr>
<td>HC (cm)</td>
<td>0.229** 0.259** 0.069 -0.129 0.071 0.133 -0.142 0.173 0.127 0.176</td>
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<tr>
<td>NC (cm)</td>
<td>0.402** 0.396** 0.446** -0.474** 0.443** 0.503** -0.480** 0.505** 0.497** 0.512**</td>
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<tr>
<td>W/H</td>
<td>0.657** 0.777** 0.830** -0.802** 0.774** 0.816** -0.823** 0.789** 0.819** 0.844**</td>
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<tr>
<td>W/Ht</td>
<td>0.486** 0.595** 0.573** -0.591** 0.524** 0.615** -0.615** 0.631** 0.609** 0.648**</td>
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<tr>
<td>BAI</td>
<td>0.061 0.138* 0.107 -0.158 0.068 0.181* -0.167 0.230* 0.168 0.193*</td>
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</tr>
<tr>
<td>VAI</td>
<td>0.777** 0.877** 0.905** -0.870** 0.910** 0.908** -0.917** 0.944** 0.898** 1</td>
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</table>

Compared to BMI, WC showed a stronger but similar pattern of correlation with all MetS Components. It had a strong positive and significant correlation with all the serum components of MetS. (r = 0.5, 0.62, 0.58, 0.61) for FBG, TC, TG and LDL respectively, but HDL showed a negative and significant correlation (r = -0.48). Its correlation with the biomarkers of MetS was positive for serum uric acid (r = 0.54) and negative for adiponectin (r = -0.58). WC correlated positively and significantly with both SBP (r = 0.57) and DBP (r = 0.57). For the serum parameters of MetS, WC also showed the strongest correlation with TC while the weakest correlation was with HDL-C. Pearson’s correlation coefficient of WC with BP was similar for SBP and DBP and was also similar for SUA and adiponectin. WC also had a positive correlation with VAI (r = 0.64). BAI, HC and NC relatively showed weaker correlation with MetS components. The weakest was HC which showed very weak correlations with DBP (r = 0.20), SBP (r = 0.14) and TC (r = 0.16). No significant correlation was observed between HC and SUA, adiponectin, FBG, HDL, TG, and LDL. The correlation of HC with VAI was also weak (r = 0.19). BAI had no significant correlation with serum biomarkers and HDL. Its correlation with FBG, TC, LDL and TG were relatively weak, with its highest coefficient of correlation observed for TC (r = 0.20). Its correlation with VAI (r = 0.26) was also weak when compared with BMI and WC. NC like HC and BAI showed a weak correlation with MetS parameters, but
its correlation coefficient with all the components of MetS was higher than observed for BAI and HC. WHtR correlated positively and strongly with DBP, SBP and all serum parameters except HDL and adiponectin with which it showed significant negative correlation. The correlation coefficient of WHtR with MetS Components was slightly higher than that of WC except for HDL and DBP where the correlation of WC was stronger. WHtR showed a positive and significant correlation with VAI and its strength of correlation with VAI (r = 0.67) was similar to that of WC (r = 0.64).

Putting all the anthropometric adiposity indices together, WHR showed the strongest correlation with blood pressure and serum components of MetS. Higher correlations of WHR were observed for SUA (r = 0.84), adiponectin (r = -0.83), TC (r = 0.83), LDL (r = 0.84) and VAI (r = 0.83). Comparing the index of visceral adiposity with all the anthropometric indexes, higher correlation coefficients were observed between VAI and all the parameters of MetS. However, the correlation strength of WHR was close to that of VAI. In males and females, the anthropometric adiposity markers correlated with MetS Components in a similar pattern of varying strength. In that, while all the indices correlated positively with DBP, SBP, FBG, SUA, TC, TG and LDL, they showed a negative correlation with adiponectin and HDL. HC showed only a weak correlation with both SBP and DBP in males and with only DBP in females. Its correlation with DBP is stronger in males. The correlation of BMI with all MetS parameters was stronger in females. Also, in female subjects, the WC had higher correlation with TC, TG and LDL while in males, it had higher correlation with SUA, adiponectin, FBG and HDL. The correlation of WC with VAI was similar in both sexes. The correlation coefficient of NC with the MetS indicators was similar for males and females. However, it had a slightly higher correlation with the serum biomarkers in males. Also, NC had no significant correlation with FBG in female subjects. While WHR showed comparable powers of correlation with MetS in both sexes, WHtR showed higher correlation among females. In both sexes, BAI correlated weakly with some of the MetS components. Correlating only with DBP in females, in males it correlated with SBP, TC, and TG. VAI showed weak and similar correlation with BAI in both sexes.

**DISCUSSION**

The positive correlation between the indices of body adiposity and MetS parameters observed in this study is in keeping with many studies (Eckel et al., 2010; Okampka et al., 2016). Similarly, the correlation between body adiposity measures and SUA and their negative correlation with adiponectin as observed in this study is also in conformity with previous findings (Hotta et al., 2001; Stefan et al., 2002). The positive correlation of SUA with MetS as observed in this study agrees with previous studies (Billiet et al., 2014), and is believed to have an evolutionary basis resulting from uricase mutation in order to confer a survival advantage by helping to maintain blood pressure (BP), stimulate salt-sensitivity, induce insulin resistance (IR) and obesity, thereby helping promote survival during a period of famine or stress (Johnson et al., 2008). Studies have also showed that hyperuricemia is an independent predictor of MetS (Kadiri and Salako, 1997; Billiet et al., 2014). Also, since studies have demonstrated the protective effect of adiponectin against MetS (Hotta et al., 2000; Weyer et al., 2001), it therefore means that as obtained in this study, all adverse metabolic indicators are expected to correlate inversely with adiponectin and positively with SUA.

The antagonist effect of adiponectin against MetS which may be the basis for the inverse correlation observed in this study is reported to result from its antiatherogenic (Ouchi et al., 2001; Okamoto et al., 2002), anti-diabetic (Yamauchi et al., 2002; Stefan et al., 2003) and anti-inflammatory (Engeli et al., 2003) effects. Therefore, similar to the result obtained in this study, low plasma levels of adiponectin is reported to characterize higher measures of body adiposity and adverse metabolic parameters (Engeli et al., 2003). In this study, one of the serum components of MetS, HDL correlated negatively with body adiposity measures. This finding is previously (Bergman et al., 2006)
Consequent to this inverse relationship, unlike other serum components, lower levels of HDL characterize obesity and MetS (Bergman et al., 2006). Also, the significant correlation between anthropometric measures of adiposity and VAI observed in this study is in line with documented reports showing positive correlation between various measures of visceral adiposity and anthropometric measures (Després et al., 2000; Lara-Castro et al., 2002).

Comparing the pattern of correlations observed in this study to those of previous studies, while close similarities were observed for some of the indices, wide variations were noted in others. These variations are not unexpected as there is currently an ongoing controversy on the adiposity measure with the highest discriminatory power for MetS because of conflicting reports from different ethnicity and populations (Tulloch-Reid et al., 2003; Shao et al., 2010). The relatively weak correlation of BMI with MetS indices and VAI when compared with indices of centripetal adiposity as found in this study is supported by many other studies (Grundy et al., 2005; Pischon et al., 2008; MacKay et al., 2009). There is increasing number of reports pointing at the probable superiority of central measures of adiposity compared to BMI (Pischon et al., 2008; MacKay et al., 2009). This is mainly because of its reported tight association with intra–abdominal visceral fat which is a critical determinant of MetS (Adiels et al., 2008; Korenblat et al., 2008). Also, the unique anatomic location of visceral adipose tissue (Kraegen et al., 1991), difference in structural and functional characteristics between visceral and subcutaneous adipocytes (Mathieu et al., 2009; Browning et al., 2010), difference in pattern of vascularisation (Bergman et al., 2011; Bélanger et al., 2002) are additional theories that have been put forward to explain these findings of central adiposity measures correlating with MetS better than BMI. Additionally, in the case of this study which included adults of advanced age, since elderly people are more likely to be physically inactive and physical inactivity has been shown to preferentially increase visceral adipose reserve (Ross and Janiszewski, 2008) manifesting as increased central adiposity measure, this factor may further contribute to the superiority of central indices over BMI as observed in this particular study.

Contrarily, there are some studies which either showed both to be equivalent or found BMI to be superior in its discriminatory power for all or some components of MetS (Ford et al., 2003 and Wang et al., 2005).

These wide variation and conflicting reports on the comparison of generalized and central adiposity measure may suggest that there are probably population specific factors that determine the interrelationship between body adiposity measures and MetS. These factors may include race, ethnicity, diet and physical activity level. For example, in the case of race, it is documented that blacks have lower body fat content for the same adiposity measure when compared to whites (Deurenberg et al., 1998). Since adipose tissue reserve is the main consideration, this has implication on the interrelationship between adiposity and metabolic parameters and this also means that subjects belonging to different races although may have similar adiposity measures, the MetS parameters and their pattern of relationship with adiposity may differ. In case of physical activity, individuals with similar body adiposity measures but different levels of physical activity may have different metabolic profile since PA has been shown to correlate negatively with metabolic parameters independent of adiposity measures (Andersen, 2006; Butte et al., 2007). In any case, the difference between the results of this study compared to those obtained from different populations on this issue further strengthen the current recommendation that anthropometric criteria for metabolic risk assessment should be population specific (Lear et al., 2010; Katzmarzyk et al., 2011).

Interestingly, the result of this study shows that even the indices of central adiposity do not exhibit the same strength of relationship with MetS indices. WHR in both males and females had the highest correlation with all the components of MetS. This relationship was further validated by WHR showing the strongest relationship with SUA and adiponectin which are serum biomarkers that could test the validity of relationships between body adiposity measures and MetS parameters. The finding in the present study that BAI is a relatively weak adiposity tool is very similar to that demonstrated by Melmer et al. (2013) who conducted one of the first studies after the discovery of BAI.

The superiority of VAI over all the anthropometric measures obtained in this study is similar to reports from different populations (Amato et al., 2010; Amato and Giordano, 2014). However, this index, according to the present study differed from some studies in terms of its predilection for certain components of MetS. Deviating slightly from this study which shows the highest predilection of VAI for TC and TG, the study of Knowles et al. (2011) found significant association of VAI with all MetS components, but with a stronger predilection for triglyceride and HDL-C in both genders. The study of Heloisa et al. (2015) differs from the present study in that, even though VAI showed superior correlation with MetS components compared to anthropometric measures of adiposity like this study, unlike this study, the superiority of VAI did not cut across all the components of MetS because according to Heloisa et al. (2015), BMI in the general population and in females showed a higher correlation with serum glycemia. From the result of the present study showing the weakest correlation of VAI with DBP and SBP compared to other MetS parameters, it
may be speculated that the relationship between BP and visceral adiposity may be weaker compared to other MetS components. This may be due to larger number of factors that come into play in the regulation of BP compared to other MetS components, making the contribution of visceral adipose tissue deposit less in the pathogenesis of hypertension. Further, the higher mRNA concentrations for angiotensinogen reported for visceral compared to abdominal subcutaneous adipose tissue is thought to be a major pathophysioslogic mechanism linking hypertension with visceral adipose tissue as well as adipocyte differentiation (Dusserre et al., 2000). This pathogenic pathway may seem to be longer than those linking visceral adiposity with serum lipids and glucose which often involves direct release of lipid products into the circulation (lipidemia) or glucose release via hepatic glycogenolysis (Matsuzawa, 2008; Browning et al., 2010).

There are speculations that the superior discriminatory ability of visceral adipose tissue over other adiposity measures may not be a unanimous contention and may not follow a uniform trend in all population suggesting that factors such as ethnicity may influence the interrelationships between visceral adipose tissue and MetS. Moreover, Goh et al. (2014) has reported that ethnicity is a principal determinant of the extent of impact of a particular adiposity measure on MetS components. This means that ethnic specific factors may either up regulate or down regulate the relationship. Overall, the superior performance of visceral adipose measure observed in this study may have its explanation rooted to the fact that visceral adipose tissue is tied to overproduction of triglyceride-rich lipoproteins and glucose, leading to the dysglycaemic and dyslipidaemast state found in viscerally obese subjects (Adiels et al., 2008; Korenblat et al., 2008).

This study reveals that, for the Hausas of Kano, visceral adiposity index is better correlated with metabolic syndrome indices when compared to all anthropometric adiposity measures. Waist to hip ratio is superior to other anthropometric markers. Body adiposity index, neck circumference and hip circumference are weak correlates of metabolic parameters.

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