The Locus of Fat Over-Accumulation as Predictor of Cardiometabolic Risks among Non Obese Normotensive Adults

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Authors' contributions

This work was carried out in collaboration between both authors. Author KOS designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author EIE managed the analyses of the study and in the literature searches. Both authors read and approved the final manuscript.

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ABSTRACT

Studies on Metabolic Syndrome (MS) have recently been narrowed down to fat over-accumulation but there is no consistency in the obesity requirements for any of the health risk scores and definitions, thus, the utility of anthropometric measures in cardiometabolic risks prediction among non-obese requires additional research. This cross sectional study assessed the relationships and predictabilities of visceral adiposity index (VAI) and Lipid accumulation product (LAP) for cardiometabolic risks among 174 non obese adults (91 males and 83 females) 35 - 70 years of age that was randomly selected for this study after their consent. Anthropometric evaluation included weight, height, hip and waist circumferences. BMI, Waist-height ratio and waist-hip ratio were calculated. Serum Triglyceride and High Density Lipoprotein measured in a 12 hour fasting condition (mmol/L) using standard laboratory techniques were recorded. The VAI and LAP scores were calculated using the gender-specific equations. Systolic blood pressure and Diastolic blood pressure were measured and MABP was calculated as: DBP + 1/3 Pulse Pressure (mm/Hg). The results showed significant correlations among LAP, VAI, WHTR, WHPR and BMI (p<0.01). Multivariate correlations and regression analysis of measures of adiposity and MS features also
showed that LAP had a greater impact on features of MS and superior to other adiposity measures in male and female (R²: 0.956; p≤0.01). In conclusion, this study shows that LAP is superior in predicting risks of lipid and visceral adipose over-accumulations than other measures of adiposity among non-obese adults. The use of LAP as an assessment tool for Risks of fat over-accumulations and its intervention among non-obese adults was suggested.

Keywords: Lipid adiposity product; visceral adiposity index; metabolic syndrome; non-obese adults.

ABBREVIATIONS

- BMI : Body Mass Index (kg/m²)
- CVD : Cardiovascular Disease
- DM : Diabetes mellitus
- DBP : Diastolic Blood Pressure (mmHg)
- FBG : Fasting blood glucose (mmol/l)
- HC : Hip Circumference (cm)
- HDL : High Density Lipoprotein (mmol/l)
- HR : Heart Rate
- PR : Pulse Rate (/min)
- Ht : Height (cm)
- IDF : International Diabetes Federation
- IR : Insulin Resistance
- LAP : Lipid accumulation product
- MABP : Mean Arterial Blood Pressure
- MS : Metabolic syndrome
- SBP : Systolic Blood Pressure (mmHg)
- TC : Total Cholesterol (mmol/l)
- TG : Triglyceride (mmol/l)
- VAI : Visceral adipose index
- WC : Waist Circumference (cm)
- WHpR : Waist-to-Hip Ratio
- WHtR : Waist-to-Height Ratio
- Wt : Body Weight (kg)

1. INTRODUCTION

The concept of the metabolically obese normal weight individual is based on the observation that the clustering of multiple metabolic abnormalities which include abdominal obesity, hypertension, dyslipidemia, insulin resistance, and impaired glucose tolerance; as the major risk factors for both DM and CVD, often associated with obesity may be found in normal weight individuals [1,2]. Studies have shown that indices of fat over-accumulation, as well as subcutaneous abdominal adipose tissue, visceral abdominal fat and several others are associated with blood pressure [3,4]. The documented association of excess body fat with hypertension and high cardiometabolic risks among individuals with central obesity are due to metabolic and hemodynamic alterations as well as Sympathetic over-activation and reduced parasympathetic tone that are seen in overweight and obese individuals [5,6]. Other studies have demonstrated strong associations of visceral obesity with increased adipocytokine production, proinflammatory activity, deterioration of insulin sensitivity, increased risk of developing diabetes, high-triglyceride/low HDL cholesterol dyslipidemia, hypertension, atherosclerosis, and higher mortality rate [7,8]. The role of body fat distributions, particularly visceral obesity and their association with cardiometabolic risks among non-obese individuals has received limited attention.

Recently, gender specific equations, Visceral Adipose Index (VAI) and Lipid Accumulation Product (LAP) have been identified as surrogate marker of adipose tissue function and distribution and routinely applicable indicators for the evaluation of visceral adipose function and metabolic syndrome and have higher sensitivity and specificity than classical parameters for cardiometabolic risk assessment [9,10,11]. The VAI has shown to be independently associated with cardiovascular and cerebrovascular events and inversely related to insulin sensitivity [12,13]. It indirectly reflects other non-classical risk factors, such as altered production of adipocytokines, increased lipolysis, and plasma free fatty acids which are not signified by BMI, WC, TGs, and HDL separately [9]. It has also showed a strong association with both the rate of peripheral glucose utilization and with visceral adipose tissue measured with MRI and independently associated with both steatosis and necro-inflammatory activity and directly correlated with viral load among patients with genotype 1 chronic hepatitis C [14]. The association of the VAI with hyperuricemia was also reported to be significant and this association was independent of metabolic health and obesity phenotypes in the studied population [15]. The LAP was initially described [10] as a novel index of central lipid accumulation that is based on WC and the fasting TG level and it is strongly associated with CVD [10,16]. Like VAI, Previous studies indicated that the LAP reflects certain vascular risk factors, including DM [17], IR [18,19] and hypertension [20], and also associated with the mortality of non-diabetic patients with cardiovascular risks [17,21].
Likewise, other studies have identified remarkable associations of VAI and LAP with hypertension [22], DM [23], IR [24], increased risk of intracranial atherosclerotic stenosis [25], atherosclerosis, increased arterial stiffness and age-related testosterone deficiency syndrome[10,26]. Unhealthy sedentary lifestyles, ethnic and genetic variations, more visceral fat accumulation than general body fats seen among the non-obese [27,28] have also been mentioned to explain the existence of these constellation of illnesses among non-obese but investigation and diagnosis remains a cumbersome and an expensive task. However, the potential association of these indicators with cardiometabolic risks among non-obese has not been previously investigated in this population. Therefore, in this study we evaluated whether the VAI and LAP can be used as indicators of cardiometabolic risks, able to diagnose metabolic syndrome defined by IDF and also compare their strength of correlation with other anthropometric indicators among non-obese adults male and female.

2. MATERIALS AND METHODS

The participants are one hundred and seventy four (174) non-obese adults (91 males and 83 females) mean age 47.13 ±8.10 years and 44.96 ± 9.58 years, male and female respectively. A sample size determination equation for a correlation study that the primary outcome is the mean of all the values obtained from the measurements was used: Least number of participants, M = \[2 \times \left[\frac{(1-\alpha/2) + (1-\beta)}{\Delta^2}\right] + 1\] [29]. The participants for this study were randomly selected from different wards of the University village in Samaru, a suburb of Zaria in Kaduna state, Northern Nigeria. The Samaru town is the fourth and the most recent addition to the Zaria suburban area. It evolves from a small colonial farming settlement to become a large community, a melting-pot, often referred to as ‘the University village’. It is cosmopolitan in nature, drawing and fusing people of diverse national and international backgrounds. The following were excluded: Obesity, pendulum abdomen, untreated cases of hypertension or diabetes, or non-compliance to anthropometric measurements.

With participants barefooted and in light clothing, Wt and Ht were measured using standardized procedures to the nearest 0.1 kg and 0.1 cm, respectively. WC was measured with a non-elastic flexible tape in standing position at the level of umbilicus. Hp was measured around the pelvis at the point of maximal protrusion of the buttocks. The mean of two measurements to the nearest 0.1 cm were recorded. WHpR was calculated by dividing the WC by the Hp and WHTR was calculated by dividing the WC by the Ht. The BMI was calculated as Wt/Ht (kgm\(^2\)). The VAI and LAP scores were calculated using the following sex-specific equations, when TG and HDL levels were expressed in mmol/l.

\[
VAI \ [9]: \quad \text{Male: } \frac{(WC + [39.68 + (1.88 \times BMI)]) \times (TG/1.03)}{(TG/1.31) \times (1.52/HDL)} \\
\text{Female: } \frac{(WC + [36.58 + (1.89 \times BMI)]) \times (TG/0.81)}{(TG/1.31) \times (1.52/HDL)}
\]

\[
LAP \ [10]: \quad \text{Male: } \frac{(WC - 65) \times TG}{X} \\
\text{Female: } \frac{(WC - 58) \times TG}{X}
\]

The SBP and DBP were obtained with a mercury sphygmomanometer using auscultatory methods. Measurements were carried out in triplicate at intervals of 10 mins seated rest, and their means were used in the analyses. The MABP, the average arterial pressure throughout the cardiac cycle was calculated as follows: MABP = DBP + 1/3 Pulse Pressure (mmHg); Pulse Pressure = Difference between SBP and DBP. Fasting blood samples were collected for biochemical analyses of FBG, TC, TG and HDL using standard and established laboratory techniques.

2.1 Definitions of Cut-off Values, Protocol Criteria, Metabolic Health and Obesity Phenotypes

In this study, The BMI was used to define non-obese and obese status, using a cut-off point of 25 kg/m\(^2\), and this BMI threshold is based on the definition advocated by Western Pacific Regional Office of WHO (WPRO) for obesity. We adopted the IDF, 2005 published criteria to define metabolically unhealthy status with ethnic specific WC for Sub-Sahara Africans: SBP/DBP ≥130/85 mmHg or use of antihypertensive drugs, TG ≥1.7 mmol/l or use of lipid-lowering drugs, FPG ≥5.6 mmol/l or use of medications for diabetes, HDL-C ≥1.0/1.3 mmol/l for men/women. The WC of ≥ 94 cm (male) and ≥ 80 cm (female) i.e., Metabolic syndrome defined as central obesity plus at least 2 of the stated criteria.

2.2 Statistical Analysis

Data was analyzed using statistical package for social sciences (SPSS Inc, version 22.0;
Chicago). Descriptive statistics of mean and standard deviation was computed by gender for age, Wt, Ht, BMI, VAI, LAP, TG, HDL, WC, WHtR, WHpR, PR, MABP, SBP and DBP for the purpose of data interpretation. Partial correlation analysis was used to examine the relationships among these variables after controlling for age. Correlations were considered significant at P ≤ 0.05 with critical values located at 0.2050 (male), 0.2172 (female). A multiple regression analysis, adjusted for age was used to examine the influence of WC, VAI, LAP, WHtR, WHpR and BMI on the cardiometabolic risks of male and female. Differences were considered significant at P ≤ 0.05.

3. RESULTS

The gender-specific baseline characteristics of study subjects are shown in Table 1. Our study included 174 subjects, 91 males and 83 females. Overall, males had significant higher average Wt**, Ht**, WHpR**, and TG* compared with females (P ≤0.05; 0.01), HDL levels were lower in males than in females (P≤0.01), while females had a significant higher VIA* and pulse rate levels compared with males (P≤0.05).

There was no significant difference between males and females for the following variables: Age, WC, SBP, DBP, MABP, BMI, HDL, FPG and LAP.

Table 2 Shows relationships among the obesity measures, Pearson’s correlation analysis showed that, in males and females, LAP correlated positively with WC, BMI, WHpR, and VAI (p≤0.01). VAI on the other hand showed no correlation with BMI in both male and female but correlated with WC, WHpR and WHtR (p≤0.01) in male and only WHtR (p≤0.01) in female. In Table 3, Multivariate correlations of measures of adiposity and MS features in male and female participants using IDF, 2005 criteria, showed that in the male group, LAP versus (WC, TG, HDL and FPG) was superior to VAI versus (WC, TG, HDL and FPG); WHtR versus (WC, SBP, DBP, TG);

WHPR versus (WC, SBP, TG), BMI versus (WC, SBP, DBP); Also, in females, LAP versus (WC, SBP, TG, FPG) was superior to VAI versus (TG, HDL, FPG), WHtR versus (WC, SBP, DBP) WHPR versus (WC) BMI (WC). These data suggested that in both males and females, LAP had a greater impact on features of MS than other adiposity measures.

Table 1. Mean (SD) of Baseline characteristics and cardiometabolic risk factors of the study population

<table>
<thead>
<tr>
<th>Variables</th>
<th>Male (n=91)</th>
<th>Female (n=83)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>47.1 ± 8.1</td>
<td>45.0 ± 9.6</td>
<td>0.11</td>
</tr>
<tr>
<td>Weight (kg)**</td>
<td>69.6 ± 9.6</td>
<td>62.9 ± 7.6</td>
<td>0.00</td>
</tr>
<tr>
<td>Height (m)**</td>
<td>173.3 ± 6.4</td>
<td>162.6 ± 6.1</td>
<td>0.00</td>
</tr>
<tr>
<td>Systolic BP (mmhg)</td>
<td>127.0 ± 20.1</td>
<td>132.8 ± 26.1</td>
<td>0.10</td>
</tr>
<tr>
<td>Diastolic BP (mmhg)</td>
<td>79.0 ± 10.8</td>
<td>82.7 ± 14.7</td>
<td>0.06</td>
</tr>
<tr>
<td>Mean arterial BP (mmhg)</td>
<td>95.0 ± 12.8</td>
<td>99.4 ± 17.9</td>
<td>0.06</td>
</tr>
<tr>
<td>Pulse Rate (beats/min)**</td>
<td>68.7 ± 8.9</td>
<td>75.8 ± 11.8</td>
<td>0.00</td>
</tr>
<tr>
<td>Waist Circumference(cm)</td>
<td>87.9 ± 8.2</td>
<td>86.9 ± 7.7</td>
<td>0.39</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.1 ± 2.7</td>
<td>23.8 ± 2.4</td>
<td>0.10</td>
</tr>
<tr>
<td>WHpR**</td>
<td>0.94 ± 0.1</td>
<td>0.88 ± 0.1</td>
<td>0.00</td>
</tr>
<tr>
<td>WHtR**</td>
<td>0.51 ± 0.05</td>
<td>0.54 ± 0.05</td>
<td>0.00</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)*</td>
<td>1.32 ± 0.99</td>
<td>1.07 ± 0.49</td>
<td>0.03</td>
</tr>
<tr>
<td>HDL (mmol/L)</td>
<td>0.94 ± 0.33</td>
<td>0.96 ± 0.37</td>
<td>0.83</td>
</tr>
<tr>
<td>FPG (mmol/L)</td>
<td>4.0 ± 1.4</td>
<td>4.2 ± 1.3</td>
<td>0.39</td>
</tr>
<tr>
<td>VAI*</td>
<td>2.02 ± 1.4</td>
<td>2.58 ± 1.6</td>
<td>0.02</td>
</tr>
<tr>
<td>LAP</td>
<td>32.5 ± 30.1</td>
<td>30.7 ± 16.5</td>
<td>0.63</td>
</tr>
</tbody>
</table>

(*P s .05; ** P ≤ .01; Df: 174)

Table 2. Association of measures of adiposity

<table>
<thead>
<tr>
<th></th>
<th>WC</th>
<th>BMI</th>
<th>WHpR</th>
<th>WHtR</th>
<th>VAI</th>
<th>WC</th>
<th>BMI</th>
<th>WHtR</th>
<th>WHpR</th>
<th>VAI</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAP</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.01*</td>
<td>0.00**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VAI</td>
<td>0.00**</td>
<td>0.07</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.00**</td>
<td>0.88</td>
<td>0.84</td>
<td>0.00**</td>
<td>0.17</td>
<td>-</td>
</tr>
</tbody>
</table>

(*P s .05; ** P ≤ .01; Df: 174)
Using the metabolic syndrome features as independent variables, the multiple regressions were used in Table 4 to show the predictability of measures of adiposity for MS among non-obese participants. In male, the value of R: 0.976 for LAP indicates a high level of predictability with independent variable explaining 95.6% (R^2 0.956; adj R^2: 0.950) of its strength followed by VAI (R: 0.944; R^2: 0.891; Adj R^2: 0.883); WHtR (R: 0.936; R^2: 0.877; Adj R^2: 0.868); BMI (R: 0.855; R^2: 0.730; Adj R^2: 0.711) and WHpR (R: 0.777; R^2: 0.603; Adj R^2: 0.575) as the lowest.

Similarly in female, the value of R: 0.979 for LAP indicates a high level of predictability with independent variable explaining 95.9% (R^2 0.959; adj R^2: 0.956) of its strength followed by VAI (R: 0.850; R^2: 0.722; Adj R^2: 0.700); WHtR (R: 0.930; R^2: 0.865; Adj R^2: 0.855); BMI (R: 0.789; R^2: 0.622; Adj R^2: 0.592) and WHpR (R: 0.747; R^2: 0.559; Adj R^2: 0.524) as the lowest.

4. DISCUSSION

The LAP and VAI were developed with the view of reflecting the combined anatomic and physiologic changes associated with lipid and visceral adipose over-accumulation in adults. The present study assessed the association of measures of adiposity with the features of MS among non-obese adults. The results showed significant correlations among LAP, VAI, WHtR, WHpR and BMI. Compared with BMI, WHtR and WHpR; LAP and VAI exhibited significant correlations with features of MS but their correlations with blood pressure were inconsistent. The regression analysis confirmed the predictive value of measures of adiposity in discriminating features of MS. After adjustment for age and potential risk factors, the LAP was independently associated with features of MS, followed by VAI, WHtR, BMI and WHpR. This is in agreement with other studies that showed that LAP has a strong and reliable diagnostic accuracy for MS-IDF and MS-NCEP/ATP III especially among non-diabetic adults [30,31]. LAP is also considered a reliable marker of IR and a risk factor to cardiovascular disease [21, 32,33,34], it is associated with fatty liver disease [35] and superior to other conventional obesity indices [36]. Studies have also shown that VAI is strongly associated with adipocytokine synthesis, proinflammatory activity, IR, dyslipidemia, hypertension and atherosclerosis [13,37] as well as a reliable indicator of visceral fat function associated with cardiometabolic risk [9] and VAI has proven to be a good predictor of MS components in the elderly [38]. But in this study, LAP is superior in predicting risks of lipid and visceral adipose over-accumulations than other measures of adiposity among non-obese adults, this is similar to findings from other studies carried out in different populations that showed the LAP index obtained better precision for MS screening and cardiovascular disease predictor than other indices of adiposity, regardless of gender differences [32,36,39,40].

Table 3. Multivariate Correlations of measures of adiposity and Metabolic Syndrome Features in male and female participants

<table>
<thead>
<tr>
<th></th>
<th>WC</th>
<th>SBP</th>
<th>DBP</th>
<th>TG</th>
<th>HDL</th>
<th>FPG</th>
<th>WC</th>
<th>SBP</th>
<th>DBP</th>
<th>TG</th>
<th>HDL</th>
<th>FPG</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAP</td>
<td>0.00*</td>
<td>0.23</td>
<td>0.32</td>
<td>0.00*</td>
<td>0.03*</td>
<td>0.00*</td>
<td>0.00*</td>
<td>0.03*</td>
<td>0.08</td>
<td>0.00*</td>
<td>0.56</td>
<td>0.03*</td>
</tr>
<tr>
<td>VAI</td>
<td>0.01*</td>
<td>0.82</td>
<td>0.72</td>
<td>0.00*</td>
<td>0.02*</td>
<td>0.02*</td>
<td>0.88</td>
<td>0.53</td>
<td>0.83</td>
<td>0.00*</td>
<td>0.00**</td>
<td>0.00**</td>
</tr>
<tr>
<td>WHtR</td>
<td>0.00*</td>
<td>0.03*</td>
<td>0.04*</td>
<td>0.01*</td>
<td>0.19</td>
<td>0.33</td>
<td>0.00*</td>
<td>0.02*</td>
<td>0.03*</td>
<td>0.40</td>
<td>0.70</td>
<td>0.80</td>
</tr>
<tr>
<td>WHpR</td>
<td>0.00*</td>
<td>0.01*</td>
<td>0.22</td>
<td>0.00*</td>
<td>0.68</td>
<td>0.80</td>
<td>0.00*</td>
<td>0.30</td>
<td>0.87</td>
<td>0.35</td>
<td>0.91</td>
<td>0.90</td>
</tr>
<tr>
<td>BMI</td>
<td>0.00*</td>
<td>0.02*</td>
<td>0.02*</td>
<td>0.08</td>
<td>0.32</td>
<td>0.15</td>
<td>0.00*</td>
<td>0.15</td>
<td>0.09</td>
<td>0.54</td>
<td>0.81</td>
<td>0.84</td>
</tr>
</tbody>
</table>

(* P ≤ .05; ** P ≤ .01; Df: 174)

Table 4. Association of measures of adiposity and risk factor accumulations in logistic regression analysis

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>R</th>
<th>R^2</th>
<th>Adj R^2</th>
<th>p</th>
<th>R</th>
<th>R^2</th>
<th>Adj R^2</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAP</td>
<td>0.976</td>
<td>0.956</td>
<td>0.950</td>
<td>0.000**</td>
<td>0.979</td>
<td>0.959</td>
<td>0.956</td>
<td>0.000**</td>
</tr>
<tr>
<td>VAI</td>
<td>0.944</td>
<td>0.891</td>
<td>0.883</td>
<td>0.000**</td>
<td>0.850</td>
<td>0.722</td>
<td>0.700</td>
<td>0.000**</td>
</tr>
<tr>
<td>WHtR</td>
<td>0.936</td>
<td>0.877</td>
<td>0.868</td>
<td>0.000**</td>
<td>0.930</td>
<td>0.866</td>
<td>0.855</td>
<td>0.000**</td>
</tr>
<tr>
<td>BMI</td>
<td>0.855</td>
<td>0.730</td>
<td>0.711</td>
<td>0.000**</td>
<td>0.789</td>
<td>0.622</td>
<td>0.592</td>
<td>0.000**</td>
</tr>
<tr>
<td>WHpR</td>
<td>0.777</td>
<td>0.603</td>
<td>0.575</td>
<td>0.000**</td>
<td>0.747</td>
<td>0.559</td>
<td>0.524</td>
<td>0.000**</td>
</tr>
</tbody>
</table>

Predictors: (Constant), WC, SBP, DBP, TG, HDL and FPG (* P ≤ .05; ** P ≤ .01; Df: 174)
It was speculated that the two common components of LAP and VAI, that is, enlarged abdominal fat depots and increased TG concentration are each an indication that available lipid fuels have exceeded the individual's capacity to buffer and safely store this major form of acquired energy which could also be dictated in part by individual genes or features of environmental circumstances [10]. By implication, excess lipid material will increasingly be deposited in non-adipose "ectopic" tissues (e.g., liver, skeletal muscle, heart, blood vessels, kidneys, and pancreas) where it may adversely modify cellular metabolism, accelerate apoptosis (cell death), and interfere with cardiovascular control [41,42]. These deposits are difficult and expensive to quantify directly, but a simple and easy method of measure of adiposity like LAP may indicate that various tissues or organs have become more vulnerable to injury from lipid and visceral adipose over-accumulation.

BMI and WHTR values, which are functions of height, are less specific in their anatomic or physiologic implications. The BMI for instance might represent enhancement of lean tissues and enlargement of the protective subcutaneous adipose depots in the lower extremities or systemic overload of fluid [43,3,44,45] which may be compensatory, salutary or simply secondary consequences of other disease processes and could also explain why BMI cannot be efficiently used to precisely predict CVD risk in comparison with other indicators of central adiposity that have more capacity to predict obesity-related cardiovascular risk and criteria for clinical diagnosis of MS [10,46].

The relative delay in lipid over-accumulation in females compare to males which is consistent with their greater amount of lower body adipose tissue and extends to increased buffering and storage capacity [38]; and the fact that obese individuals have significantly more blood pressure (both systolic and diastolic) and increased heart rate compared to non-obese individuals [6]; and that subcutaneous abdominal adipose tissue is a better parameter than visceral abdominal adipose tissue that correlates with blood pressure in normal subjects [47]; may explain the inconsistencies with blood pressure relationships and gender differences in the predictability of some of the measures of adiposity for MS; In our study however, LAP was more consistent and behaved as the best predictor for MS than other measures of adiposity in both gender. More data collected prospectively in a variety of populations would help to confirm that the LAP assessment and laboratory cost is cheaper compared with the costs of multiple assays for extensive biochemical panel. LAP is also better and has more advantages over other simple indices for predicting the incidence of major diseases and mortality, and in assessments of fat over-accumulations and its intervention.

5. CONCLUSION

Complications of fat over-accumulation have reached epidemic proportion and continue to be a growing problem worldwide. The potential mechanisms involve insulin resistance, inflammation, renal (Renin Angiotensin-Aldosterone System) hyperactivity, Sympathetic Nervous System hyperactivity and perhaps other unknown mechanisms. Increased awareness is needed in non-obese individuals for early diagnosis and implementation of prevention and treatment measures. The results obtained from this study suggested that the LAP index demonstrated better precision for screening and identifying features of MS among non-obese adults compared with other measured variables. We recommend the utility of this easily obtainable, accurate, convenient and cost-effective index for the screening of features of MS among non-obese adults. LAP may therefore be a useful tool in daily clinical practice and in population studies for the assessment of cardiometabolic risks associated with fat over-accumulation.

CONSENT AND ETHICAL APPROVAL

This cross sectional study was approved by the Ethical committee of the Ahmadu Bello University Teaching Hospital, Shika, Nigeria. At the time of observation all participants signed an informed consent for the scientific use of their data.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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