
Amanze N Ikwu¹, Anthony O Akintomide², Michael O Balogun², Tuoyo O Mene-Afejuku³

¹. Cardiology Department, University Hospitals Plymouth NHS Trust, United Kingdom.
². Cardiology Unit, Department of Medicine, Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Nigeria.
³. Department of Epidemiology, Emory University, Atlanta, USA.

Corresponding Author: DrAmanzeIkwu
Department of Cardiology, University Hospitals Plymouth NHS Trust Derriford Road, Plymouth PL6 8DH United Kingdom

Background
Cardiovascular disease (CVD) is a leading cause of death in women. Systemic hypertension is the commonest risk factor for CVD, and it frequently coexists with other risk factors in women, thereby increasing the absolute cardiovascular risk.

Objective
This study set out to study the spectrum of arrhythmia burden and CVD risk factors among hypertensive women.

Method
This is a cross-sectional study involving 300 hypertensive women and 150 age and sex-matched normotensive women as controls. Participants that met the inclusion criteria were recruited consecutively into the study. Prevalence and pattern of cardiovascular disease risk factors were sought by taking relevant history and examination. Blood was collected for glucose and lipid estimation. Patients underwent 24-hour Holter electrocardiography (ECG) and resting ECG study. Data were analysed using Statistical Package for Social Sciences (SPSS version 17.0 Chicago Illinois) software.

Results
The mean age of both groups was similar (57.15±11.58 years for hypertension group and 56.83±13.41 years for controls; p = 0.3967). The prevalence of generalized obesity was significantly higher in the hypertension group, as compared with controls (37.3% vs 22%, p=0.001). The overall prevalence of hypercholesterolemia was significantly higher in the hypertension group (12.33%), compared with controls (4%). 24-hour Holter ECG showed that 176 (59.7%) patients in the hypertension group and 50 (33.3%) control subjects had arrhythmia, with premature ventricular complex being the commonest arrhythmic pattern.

Conclusion
CVD risk factors and ventricular arrhythmia are more common in women with hypertension. Early risk factor detection and management in hypertensive women is desirable.

Keywords: hypertension, risk factors, cardiovascular disease, women

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I. Introduction
There is emerging evidence showing that the pattern of diseases in sub-Saharan Africa is changing, with cardiovascular disease alone accounting for 9.2% of the total mortality [World Health Organization (WHO) 2002]¹. Systemic hypertension is a major public health problem². It is the most frequent risk factor for cardiovascular disease, and frequently coexists with other risk factors, thereby increasing the absolute cardiovascular risk³.

Cardiovascular diseases have been described primarily as a ‘male disease’, and evidence-based clinical standards have been created on male pathophysiology and outcomes⁴. As a result, women are often misdiagnosed or under-diagnosed⁵.
Risk factors for cardiovascular disease in women may be modifiable or non-modifiable. Modifiable cardiovascular risk factors include systemic hypertension, type-2 diabetes mellitus (T2DM), dyslipidaemia, obesity, cigarette smoking, metabolic syndrome, lifestyle (lack of exercise, alcohol abuse and dietary habits) and psychosocial factors. Non-modifiable cardiovascular risk factors are age, gender, family history and post-menopausal state.

Arrhythmia is a common co-morbidity in patients with systemic hypertension and a manifestation of hypertensive heart disease. Underlying mechanisms are many, including left ventricular hypertrophy (LVH), myocardial ischaemia, impaired left ventricular function and left atrial enlargement. Any form of arrhythmia may be associated with LVH but ventricular arrhythmia is more common and could be life-threatening. Adebayo et al in a study on ‘Evaluation of Indications and Arrhythmic Pattern of 24-hour Holter ECG among Hypertensive and Diabetic patients at Ile-Ife, Nigeria’ reported palpitation as the commonest indication and premature ventricular contraction as the commonest arrhythmic pattern. The Atherosclerosis Risk in Communities (ARIC) study of more than 15,000 African American and White men and women reported that hypertension is associated with frequent or complex ventricular ectopic beats. Omotoso et al studied arrhythmias in 2017 Nigerian hypertensive heart disease patients and reported that premature ventricular complexes were the commonest arrhythmic pattern.

Only about 55% of women identified CVD as their greatest health risk in a 2006 survey conducted by the American Heart Association (AHA) despite estimates that a 40-year old woman has a lifetime risk of CVD of 32%. Studies over the last several decades from United States also indicate that despite an overall reduction in the death rate due to CVD, the rate of decline is less for women than men, and less for African- American women than White women. This suggests that women have little insight into their own risk of heart disease. Women also have a heightened mortality burden from cardiovascular disease than men.

1.1 What is already known:
Cardiovascular disease in women has to date, largely been under-recognized, under-diagnosed and under-treated and there are few data on the burden of cardiovascular disease risk factors in hypertensive women in Nigeria. However, cardiovascular disease is a leading cause of death in women around the world.

1.2 What this research adds:
The findings from this study are envisaged to elucidate CVD risk factors in hypertensive women, this will help to improve early detection, prompt diagnosis and treatment of these risk factors. Secondarily, it may promote advocacy and tools such as prevention campaigns that utilize understanding of gender to encourage heart-healthy behaviours and target risk behaviour.

II. Aim
The overall aim of this study is to determine the prevalence and pattern of cardiovascular disease risk factors associated with systemic hypertension in women. The occurrence and characterization of arrhythmias using resting electrocardiography (ECG) and 24-hour Holter ECG in hypertensive women will also be assessed.

III. Methods
3.1 STUDY LOCATION: The study was carried out at Obafemi Awolowo University Teaching Hospitals Complex (OAUTHC), Ile-Ife, Nigeria. Ile-Ife is a semi-urban ancient Yoruba city in Osun State, South-Western Nigeria.
3.2 STUDY POPULATION: This consisted of hypertensive women presenting at Cardiac Care Unit and Adult Accident and Emergency (A/E) unit of OAUTHC who satisfied the inclusion criteria.
3.3 STUDY DESIGN: This is a cross-sectional study involving hypertensive women with age and sex-matched normotensive women as control. Prevalence and pattern of cardiovascular disease risk factors was sought by taking relevant history with the use of interviewer administered proforma and physical examination including weight, height and office blood pressure measurement. Blood was collected for fasting blood glucose and 2-hour postprandial blood sugar, fasting lipid profile, packed cell volume, electrolytes, urea and creatinine. Patients underwent electrophysiological studies which include 24-hour Holter electrocardiography (ECG) and resting ECG.
3.4 SAMPLING TECHNIQUE: Subjects that fulfilled the inclusion criteria (aged 18-75 years) were consecutively recruited for this study until sample size is met. The control subjects were apparently healthy age and sex-matched volunteers with normal blood pressure recruited from among patients’ relatives, hospital staff members, medical students, and Ile-Ife community dwellers who responded to adverts placed at strategic positions within the hospital calling for research volunteers.
3.5 SAMPLE SIZE DETERMINATION
The sample size was determined using the formula.
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\[ N = \frac{(Z_i - a)^2 \cdot P(1-P)}{d^2} \]

N = Minimum sample size.
\( Z_i - a = 1.96 \) (from Z table)
\( P = \) best estimate of prevalence of hypertension in women from literature review = 25.4%.
\( d = \) absolute precision of 5%.

\[ N = \frac{1.96^2 \times 0.254 \times (1 - 0.254)}{0.05^2} = 291.2 \]

The estimated sample size of 291.2 was calculated. To accommodate for the dropouts from the study (approximately 3% drop-out rate), the sample size was made up to 300. Therefore, a total of 300 hypertensive women and 150 age and sex-matched apparently healthy normotensive women were recruited for this study.

3.6 INCLUSION CRITERIA
Women aged 18-75 years with systemic hypertension according to the WHO protocol or previously diagnosed hypertensive on medications.

3.7 EXCLUSION CRITERIA
Known type 2 diabetes mellitus patients.
Pregnant women.

3.8 ETHICAL CONSIDERATION
Approval of the Ethics and Research Committee of the OAUTHC was sought and obtained before the commencement of the study. Informed consent of the individuals for the study were obtained verbally and in written form.

3.9 DATA COLLECTION
3.9.1 PROTOCOL 1: HISTORY AND EXAMINATION
Data were obtained from participants using interviewer administered proforma and physical examination conducted by the investigator. Information on smoking habits, alcohol use, dietary habits, age at menopause, physical activity and family history of hypertension was recorded. Smoking was considered present if subjects reported smoking up to the day of the interview. Alcohol intake was calculated as the percentage of alcohol multiplied by volume (milliliters), divided by 1000. Low risk alcohol consumption was defined as a maximum of 3 units per day in females, with at least 2 days per week free of alcohol consumption; higher consumption was considered high risk.

3.9.2 PROTOCOL 2: BP MEASUREMENT
All participants had their blood pressure (BP) measured by the researcher only. BP was measured in the office using the left arm in the sitting position (after resting for 10 minutes) with mercury sphygmomanometer using standard procedures. The systolic BP was recorded at phase I Korokoff sounds while the diastolic BP was recorded at phase V Korokoff sounds. Hypertension was defined as a systolic BP ≥140mmHg and/or diastolic BP ≥90mmHg or the current use of anti-hypertensive medications.

3.9.3 PROTOCOL 3: ANTHROPOMETRIC MEASUREMENT
Anthropometric data was obtained by standard methods. Weight was taken with light clothing on with a weighing scale and measured to the nearest gram. A stadiometer was used for measurement of height. Body mass index (BMI) was calculated as weight in kilograms (kg), divided by the square of height in meters (m²).

WHO Classification of Obesity in Adults using BMI

<table>
<thead>
<tr>
<th>WHO Classification</th>
<th>BMI Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt;18.5kg/m²</td>
</tr>
<tr>
<td>Normal weight</td>
<td>18.5-24.9 kg/m²</td>
</tr>
<tr>
<td>Overweight</td>
<td>25-29.9 kg/m²</td>
</tr>
<tr>
<td>Class I or Mild Obesity</td>
<td>30-34.9 kg/m²</td>
</tr>
<tr>
<td>Class II or Severe Obesity</td>
<td>35-39.9 kg/m²</td>
</tr>
<tr>
<td>Class III or Extreme Obesity</td>
<td>≥40 kg/m²</td>
</tr>
</tbody>
</table>

DOI: 10.9790/0853-1908015463  www.iosrjournal.org  56 | Page
3.9.4 PROTOCOL 4: ELECTROCARDIOGRAPHY (ECG) - A conventional resting 12-lead ECG was performed. The recommendation of the American Heart Association (AHA) concerning standardization of leads and specification for instrument were followed\textsuperscript{18-19}. The 12-lead resting electrocardiogram (ECG) of the patient was obtained with the aid of a 3-channel electrocardiograph (Cardiofax YD-907D). The ECG was done with the machine set at standards with a paper speed of 25mm/s and amplitude of 10mm/mV. Lead II was used for long rhythm strip recording and evaluation. The ECG was analyzed to obtain the occurrence and pattern of arrhythmia.

3.9.5 PROTOCOL 5: 24-HOUR HOLTER ECG Monitoring: A 24-hour ambulatory Holter ECG was recorded using the Schiller type (MT-101) with a bipolar V1-V5 lead system. Patients were thoroughly educated about the test and handling of the recorder. The Holter monitor was strapped to the patient’s waist with the channel leads appropriately placed on the chest\textsuperscript{20}. Patients were asked to go home, continue their normal activities and record timing of any symptoms. After completion of the recording over a 24-hour period, the recorder was retrieved and analyzed with Schiller’s Cardiovit CS-200 digital ECG computer. The following data was recorded: heart rhythm, heart rate, heart rate variability and arrhythmias (multiple ventricular ectopic, ventricular tachycardia, premature atrial complexes, supraventricular tachycardia and atrial fibrillation).

3.9.6 PROTOCOL 6: LABORATORY TESTS

I. FASTING BLOOD GLUCOSE AND 2-HOUR POST-PRANDIAL: After 10-12 hours of overnight fast, venous blood was obtained from the subjects and analyzed for the fasting blood glucose. A second sample was collected 2 hours post-prandial for blood sugar measurement. All samples were quickly sent to Chemical Pathology unit and analyzed immediately using the glucose oxidase method. Diabetes mellitus and impaired fasting glucose was diagnosed according to the WHO criteria\textsuperscript{21}.

II. FASTING LIPID PROFILE: After 10-12 hours of overnight fast, venous blood was centrifuged and the serum immediately separated and the concentrations of triglycerides (TG), total cholesterol (TC) and its fractions [LDL-C, HDL-C] were ascertained. The atherogenic Index of plasma (TC/HDL-C and LDL/HDL-C) was calculated. For the purpose of this study, the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) cut off points was used to identify subjects with desirable, borderline high and high levels of lipoprotein risk factors\textsuperscript{22}. Electrolytes, packed cell volume (PCV) and creatinine are also estimated.

3.10 DATA ANALYSIS

Data were analysed using Statistical Package for Social Sciences (SPSS version 17.0 Chicago Illinois) software. Data were represented using descriptive statistics such as tables and bar charts where appropriate. Categorical variables were expressed as proportions and percentages while continuous variables were expressed as means and standard deviation Differences between 2 continuous variables were determined with the independent student t-test. Level of significance is P-value of $\leq 0.05$ and a confidence interval of 95% was used.

IV. Results

Three hundred (300) hypertensive women and one hundred and fifty (150) normotensive control subjects were consecutively recruited over the study period.

4.1 Demography and Clinical Characteristics of the Study Population

The demographic and clinical characteristics of the study population are shown in Table 1. The mean age of both groups was similar, (57.2±11.6 years for hypertension group and 56.8±13.4 years for controls; $p = 0.3967$). The mean duration of hypertension in the hypertension group was 5.6±6.7 years while the mean age at menopause of the study population was 50.5±2.7 years. The systemic hypertension group had significantly higher mean BMI (28.50±5.70 kg/m² for hypertension group and 25.57±3.70kg/m² for controls, $p=0.001$). The mean values of other variables including weight, waist circumference, waist-hip ratio, heart rate at rest, pressure-rate product at rest, systolic and diastolic blood pressure at rest were also significantly higher in the hypertension group. Family history of hypertension was detected in 87(29%) hypertensive patients and 29 subjects (9.66%) for the control group.
Table 1: Demographic and Clinical Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>HTN Subjects</th>
<th>Control</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex: Females (n)</td>
<td>300</td>
<td>150</td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>57.15±11.58</td>
<td>56.83±13.41</td>
<td>0.3967</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72.35±15.24</td>
<td>67.38±10.55</td>
<td>0.0002</td>
</tr>
<tr>
<td>Height (meters)</td>
<td>1.59±0.06</td>
<td>1.62±0.05</td>
<td>0.7933</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>90.59±14.61</td>
<td>84.43±10.75</td>
<td>0.001</td>
</tr>
<tr>
<td>Waist/Circ (cm)</td>
<td>99.83±11.55</td>
<td>95.57±8.10</td>
<td>0.001</td>
</tr>
<tr>
<td>Waist/hip Ratio</td>
<td>0.91±0.08</td>
<td>0.88±0.05</td>
<td>0.007</td>
</tr>
<tr>
<td>HRrest (b/min)</td>
<td>78.22±11.43</td>
<td>71.84±8.18</td>
<td>0.001</td>
</tr>
<tr>
<td>SBPrest (mmHg)</td>
<td>138.89±23.50</td>
<td>120.80±10.53</td>
<td>0.001</td>
</tr>
<tr>
<td>MAPrest (mmHg)</td>
<td>102.88±15.57</td>
<td>90.50±7.42</td>
<td>0.001</td>
</tr>
<tr>
<td>PRPrest (mmHg/min x 10^-7)</td>
<td>108.39±23.11</td>
<td>86.78±12.44</td>
<td>0.001</td>
</tr>
<tr>
<td>FH of HTN</td>
<td>87 (29)</td>
<td>29(9.66)</td>
<td>0.027</td>
</tr>
</tbody>
</table>

KEY: BMI= Body mass Index; BSA= Body surface area, HRrest= Heart rate at rest; SBPrest= Systolic blood pressure at rest; DBPrest= Diastolic blood pressure at rest; MAPrest= Mean arterial pressure at rest; PRPrest= Pressure-rate product at rest; FH of HTN= Family history of hypertension. Circ= Circumference.

4.2 Laboratory Findings of the Study Population

Table 2 shows the laboratory findings of the study population. The mean serum creatinine, total cholesterol, triglycerides, LDL-C and FBG were significantly higher in the hypertension group compared with controls. The systemic hypertension group had significantly lower mean PCV (38.55±3.14% for hypertension group and 39.59±2.26% for controls, p=0.0002) and serum sodium compared with control group. Sub-analysis of the hypertension group showed that hypertensive heart failure (HHF) patients had significantly lower PCV (37.40±4.18% vs 38.95±2.58%, P=0.001) and lower sodium (132.73±3.88mmol/L vs 136.04±3.32mmol/L, P=0.001), as compared with hypertension without heart failure sub-group. The HDL-C and serum urea were higher in control group, though this difference was not statistically significant.

Table 2: Laboratory Findings of the Study Population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>HTN Subjects</th>
<th>Control</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV (%)</td>
<td>38.55±3.14</td>
<td>39.59±2.26</td>
<td>0.001</td>
</tr>
<tr>
<td>Sodium (mmol/L)</td>
<td>135.19±3.75</td>
<td>138.61±2.63</td>
<td>0.001</td>
</tr>
<tr>
<td>Potassium (mmol/L)</td>
<td>4.06±0.46</td>
<td>4.19±0.48</td>
<td>0.7884</td>
</tr>
<tr>
<td>Creatinine (mmol/L)</td>
<td>94.79±28.84</td>
<td>79.60±13.23</td>
<td>0.001</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>4.22±0.45</td>
<td>4.05±0.76</td>
<td>0.9773</td>
</tr>
<tr>
<td>TC (mmol/L)</td>
<td>5.03±2.83</td>
<td>3.49±0.83</td>
<td>0.001</td>
</tr>
<tr>
<td>TG (mmol/L)</td>
<td>2.24±0.64</td>
<td>1.07±0.44</td>
<td>0.001</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>3.20±2.30</td>
<td>1.53±1.08</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>1.47±0.76</td>
<td>1.54±0.31</td>
<td>0.8819</td>
</tr>
<tr>
<td>FBG (mmol/L)</td>
<td>4.97±0.76</td>
<td>4.14±1.04</td>
<td>0.001</td>
</tr>
<tr>
<td>2HPP (mmol/L)</td>
<td>6.41±1.28</td>
<td>6.37±1.23</td>
<td>0.3777</td>
</tr>
<tr>
<td>TC/ HDL</td>
<td>3.29±1.85</td>
<td>2.38±0.89</td>
<td>0.001</td>
</tr>
<tr>
<td>LDL/HDL</td>
<td>2.16±2.01</td>
<td>1.09±1.05</td>
<td>0.001</td>
</tr>
</tbody>
</table>

KEY: HTN= hypertension, PCV= Packed cell volume; TC= Total cholesterol; TG= Triglycerides; LDL-C= Low density lipoprotein cholesterol; HDL-C= High density lipoprotein cholesterol; FBG= Fasting blood glucose; 2HPP= 2-hours post-prandial.

Figure 1 is a bar chart showing mean atherogenic index (using TC/HDL-C and LDL-C/HDL-C) in both study groups. The hypertension group had significantly higher mean TC/HDL-C (3.29±1.85 for hypertension group...
and 2.38±0.89 for controls, p=0.001) and LDL-C/HDL-C (2.16±2.01 for hypertension group and 1.09±1.05 for controls, p=0.001).

Figure 1 is a bar chart showing mean atherogenic index of plasma (using TC/HDL-C and LDL-C/HDL-C) in both study groups. Key: HTN= hypertensive patient group.

The prevalence of generalized obesity was significantly higher in the hypertension group, as compared with controls (37.3% vs 22%, p=0.001). Fifty-two-point three percent of the hypertension group had truncal obesity as compared to 30% of the control group. There was no statistical difference between the prevalence of IFG/DM in both groups. The overall prevalence of hypercholesterolemia was significantly higher in the hypertension group (12.33%), compared with controls (4%). The prevalence of other variables was significantly higher in the hypertension group: Raised LDL-C (9.7% for hypertension group and 3.3% for controls, p=0.017), triglyceride (7.33% vs 2.7%, p=0.045). The prevalence of low HDL-C and physical inactivity were higher in the hypertension group, though no statistical difference was observed in both groups. The percentage of post-menopausal women in both study groups is similar. Alcohol consumption was generally low and comparable in both groups. None of the study participants had a history of current or previous smoking. Sixty-seven (22.3%) hypertensive women had clinical and laboratory features of metabolic syndrome as compared with 9.3% of the control group, and this was statistically significant. These are as shown in Table 3 below.

Table 3: Frequency of cardiovascular disease risk factors and metabolic syndrome.

<table>
<thead>
<tr>
<th>CV risk factor</th>
<th>HTN subjects, N (%)</th>
<th>Control, N (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IFG/DM</td>
<td>25 (8.3)</td>
<td>7 (4.7)</td>
<td>0.154</td>
</tr>
<tr>
<td>BMI ≥30 kg/m²</td>
<td>112 (37.7)</td>
<td>33 (22)</td>
<td>0.001</td>
</tr>
<tr>
<td>Truncal Obesity</td>
<td>157 (52.3)</td>
<td>45 (30)</td>
<td>0.001</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>37 (12.33)</td>
<td>6 (4)</td>
<td>0.001</td>
</tr>
<tr>
<td>Raised LDL-C</td>
<td>29 (9.7)</td>
<td>5 (3.3)</td>
<td>0.017</td>
</tr>
<tr>
<td>Low HDL-C</td>
<td>24 (8)</td>
<td>8 (5.3)</td>
<td>0.299</td>
</tr>
<tr>
<td>Hypertriglyceridemia</td>
<td>22 (7.33)</td>
<td>4 (2.7)</td>
<td>0.045</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Alcohol &gt; 3 units/day</td>
<td>13 (4.3)</td>
<td>7 (4.7)</td>
<td>0.872</td>
</tr>
<tr>
<td>Physical Inactivity</td>
<td>8 (2.7)</td>
<td>3 (2)</td>
<td>0.666</td>
</tr>
<tr>
<td>Post-menopausal</td>
<td>197 (65.7)</td>
<td>95 (63.3)</td>
<td>0.625</td>
</tr>
<tr>
<td>Metabolic Syndrome</td>
<td>67 (22.3)</td>
<td>14 (9.3)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Key: HTN= hypertension, IFG/DM= impaired fasting glucose/ Diabetes Mellitus, TC= Total cholesterol; TG= Triglycerides; LDL-C= Low density lipoprotein cholesterol; HDL-C= High density
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lipoprotein cholesterol; N= number of persons, %= percentage.

4.3 24-hour Holter Electrocardiographic (ECG) findings

Evaluation of arrhythmias using 24-hour Holter ECG showed that 176 (59.7%) of hypertensive subjects and 50 (33.3%) of the controls had arrhythmias, as shown in Table 4.

Premature ventricular complex (PVC) was the commonest type of arrhythmias in both groups. PVC was significantly more prevalent in the hypertension group (32.3%) compared to 22% in the controls followed by premature atrial contractions (PAC). One hundred and twenty-four (41.3%) hypertensive patients had normal Holter ECG findings compared to 100 (66.7%) subjects in the control group. Fourteen (4.7%) hypertensive patients had ventricular tachycardia while none was recorded in the control group. Fifteen (5%) and 11 (3.7%) hypertensive patients had atrial fibrillation (AF) and supraventricular tachycardia while 2(1.3%) and 3 (2%) of control group had SVT and AF respectively. About 80% of the hypertensive patients with AF are HHF patients. Six hypertensive patients had severe bradycardia (HR less than 40 beats per minute). The hypertension cohort had a total of 2753 PVCs/24 hour compared with 1132 PVCs/24 hour in the control group.

The mean minimum and maximum heart rate (HR) were 48.22±11.43 and 143.30±24.38 beats/minute in the hypertensive patients and 46.84±8.19 and 141.99±18.70 beats/minute in the control group respectively. There was no statistical difference between the mean minimum and maximum heart rate of both groups.

Analysis of time domain heart rate variability (HRV) using standard deviation of normal to normal interval (SDNN) average (milliseconds) was normal in both groups but significantly lower in the hypertension group compared to the controls (104.99±34.45 vs 122.11±46.28, P<0.001).

Table 4: Distribution of the 24-hour Holter Electrocardiogram rhythm in Study group

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>HTN (n=300)</th>
<th>Controls (n=150)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Min HR (b/min)</td>
<td>48.22±11.43</td>
<td>46.84±8.19</td>
<td>0.0940</td>
</tr>
<tr>
<td>Mean Max HR (b/min)</td>
<td>143.30±24.38</td>
<td>141.99±18.70</td>
<td>0.0894</td>
</tr>
<tr>
<td>Mean SDNN</td>
<td>104.99±34.45</td>
<td>122.11±46.28</td>
<td>0.001</td>
</tr>
<tr>
<td>Total PVCs/24 hr</td>
<td>2753</td>
<td>1132</td>
<td></td>
</tr>
</tbody>
</table>

KEY: PVC= Premature ventricular complex; PAC= Premature atrial complex; NSVT= Non-sustained ventricular tachycardia; VT= Ventricular tachycardia; SVT= Supraventricular tachycardia; AF= Atrial fibrillation, Min=minimum, Max= maximum, HR= heart rate, b/min= beats per minute. SDNN= standard deviation of normal to normal interval

4.4 Resting Electrocardiographic (ECG) findings

Evaluation of arrhythmia using resting ECG showed that 77 (25.7%) hypertensive patients and 20 (13.3%) subjects in the control group had arrhythmia. Thirty-three (11%) and 10 (3.3%) hypertensive patients had PVC and PAC, while 10 (6.6%) and 4 (2.6%) subjects in the control group had PVC and PAC respectively. Other arrhythmic pattern observed during resting ECG in hypertensive subjects were sinus tachycardia 11 (3.7%), sinus bradycardia 6 (2%), atrial fibrillation 5 (1.7%), atrial flutter 1 (0.3%), multi atrial tachycardia 1 (0.3%) and junctional extrasystoles 10 (3.4%). The control group had lesser arrhythmic burden on resting ECG with sinus tachycardia in 3 (2%), sinus bradycardia 1 (0.7%), atrial fibrillation 1 (0.7%), and junctional extrasystoles 1 (0.7%). None of the control subjects had atrial flutter or multi atrial tachycardia. The variables of arrhythmia using resting ECG were more prevalent in the hypertension group, but no statistical difference was observed in both groups.

V. Discussion

5.1 Participants Characteristics

Patients with hypertension often have other major risk factors for CVD. The current study population consisted of 300 women with systemic hypertension and 150 age and sex-matched controls. The mean age of hypertensive patients was 57.15±11.58 years which is similar to the findings from another studies.
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The anthropometric characteristics including mean weight, BMI, waist circumference and waist-hip ratio were significantly higher in hypertensive women compared to the control group. There were high prevalence rates of generalized and abdominal obesity which is comparable with findings by Sani et al who reported generalized obesity of 29.2% and abdominal obesity of 67.3% in apparently healthy adult women in Katsina state. In the Heart of Soweto Study, 44% of patients with systemic hypertension were obese. In addition, Amira et al in a 5-year community-based screening in South-West Nigeria, reported that women had overweight and obesity rates of 31.9% and 29.5% respectively. The burden of obesity, especially in hypertensive women in both studies are similar to this study. Thakar et al in a study on effect of obesity on cardiovascular risk factors in an urban population of South India reported increasing cardiovascular abnormalities with increasing BMI.

Adebayo et al reported that the overall crude prevalence of overweight and obesity in 3 rural communities of Ile were 20.8% and 8.4% respectively. The lower prevalence reported by Adebayo and colleagues may be attributable to younger study population with a mean age of 36.3±14.3 years, the rural population and participation of men in the study. Adedoyin et al, in a study of Obesity in adult residents of Ile-Ife, reported a crude prevalence of overweight and obesity of 22.1% and 14.5% in women. Obesity prevalence in normotensive women in the current study is comparable to the findings by Adedoyin and colleagues.

Obesity is associated with increased prevalence of cardiometabolic risk. Cardiometabolic abnormalities associated with obesity are due to insulin resistance, glucose intolerance, dyslipidaemia, systemic hypertension and a prothrombotic-inflammatory profile. The INTERHEART global case-control study of 6787 women from 52 countries reported that abdominal obesity was more predictive of myocardial infarction than BMI alone.

The mean blood sugar, total cholesterol, LDL cholesterol, triglyceride, urea/creatinine and atherogenic index were significantly higher in the hypertension group, as observed in several previous studies.

Body fat distribution, especially visceral fat as observed to be more prevalent in hypertension group in current study is associated with obesity-related diseases such as DM, glucose intolerance, dyslipidaemia, CAD and systemic hypertension.

The hypertension group showed significantly lower mean PCV and serum sodium than control group. Sub-analysis of the hypertension subjects revealed that HHF had significantly lower mean sodium (132.73±3.87mmol/L) and PCV (37.40±4.18%) as compared with hypertension patients without heart failure. Hyponatremia was present in about 20% of patients admitted to hospital for heart failure and has been shown to increase mortality in the heart failure population. Hyponatremia in heart failure is due to inappropriate vasopressin activity despite hypoosmolality and volume overload as well as diuretic use.

Sixty-seven (22.3%) patients in the hypertension group had metabolic syndrome. Ogbaru et al. in a study on metabolic syndrome in hypertensive Nigerians reported a prevalence of 54% in hypertensive women. The higher prevalence in the study by Ogbaru and colleagues may be attributable to the fact that the cut-off age of the study participants was ≥ 35 years. Ramos et al reported 29% metabolic syndrome in non-Hispanic Black women of childbearing age in the National Health and Nutrition Examination Surveys (NHANES, 1999-2004). The higher prevalence reported in this study may be due to dietary lifestyle, physical inactivity and affluent economy of USA.

It is noteworthy that 52.3% of hypertensive women in current study had truncal obesity. Insulin resistance and increased visceral fat are the hallmarks of cardiometabolic syndrome, an assembly of risk factors for developing diabetes mellitus and cardiovascular disease. Visceral adiposity increases the degree of insulin resistance associated with obesity.

None of the study participants was a cigarette smoker. Ogumnola et al in a study of cardiovascular risk factors among adults in a rural community in Southwest Nigeria reported a 2.8% prevalence of current smokers in women.

5.2. Evaluation of arrhythmia in Hypertensive Subjects.

Cardiac arrhythmias secondary to hypertensive heart disease may be due to increase in systemic catecholamines, electrolyte derangements and associated heart failure. A significantly higher number (about 60 percent) of hypertensive patients in this study had arrhythmia on Holter ECG compared with 33.3% of control group. Premature ventricular complex (PVC) was the commonest type of arrhythmias in both groups but found more prevalent in the hypertension group (32.3% in hypertensive subjects and 22% in control group), followed by premature atrial contractions. Okeahialam reported ventricular ectopy as the commonest arrhythmia in a study of 1547 resting ECG tracings over a 5-year period in Jos, Nigeria. Fourteen (4.7%) hypertension subjects had ventricular tachycardia, while 15 (5%) had AF. About 80% of the hypertensive patients with AF are HHF patients. The high prevalence of AF in HHF subgroup is worrisome. Previous studies in Nigeria have documented similar arrhythmic findings. Familoni et al reported that AF was associated with increased mortality rates among patients with advanced heart failure.
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The HRV using SDNN average (millisecond) were significantly lower in the hypertension group. Previous studies in Nigeria have documented similar findings.6,47  

VI. Conclusion:  
This study has shown that hypertensive women had significantly higher cardiovascular disease risk factors. There is a greater arrhythmic burden in hypertensive women than in the control (59.7% vs 33.3%) with premature ventricular complex being the commonest arrhythmic pattern. Early risk factor detection and treatment by assessing CVD risk factors in women with hypertension, in primary care is recommended.  

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