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Analysis of Atherogenic and Anthropometric Profiles of Normotensive and Hypertensive Ghanaians in the Kumasi Metropolis

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Authors' contributions

This work was carried out in collaboration between all authors. Authors WKBAO, IKO, EFL, EOD and JOY conceptualized and designed the study. Authors IKO, CA and EOD recruited the study participants. Authors IKO, CA, JOY, WKBAO and EOD generated the data. Authors CA, JOY, WKBAO and IKO analyzed the data. Authors IKO, CA, WKBAO and JOY drafted the manuscript. Authors IKO, CA, WKBAO, JOY, EOD and EFL reviewed the manuscript for intellectual content and each author approved the final manuscript. All authors read and approved the final manuscript.

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Original Research Article

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ABSTRACT

Ghana is undergoing a rapid epidemiological transition from solely communicable to a double burden of infectious and chronic disease such as hypertension.

Aims: We aimed to compare the association between different lifestyle practices, adiposity indices, atherogenic dyslipidaemic parameters and hypertension as well as the prognostic implications for the levels of these parameters on target cardiac organ damage among hypertensives. We also determined the optimal threshold points and the discriminative power of these parameters on this urban Ghanaian hypertensive population.

Study Design: A hospital-based case-control study was conducted.

Methodology: The study purposively recruited 241 Ghanaian indigenes in the Kumasi metropolis, with 180 hypertensives as cases and 61 normotensives as controls. In addition to socio-demographic data captured, all participants underwent standard haemodynamic, anthropometric, atherogenic lipid and cardiac organ damage assessment.

Results: In general, the case group presented with a significantly poorer atherogenic lipid profile compared to their counterparts in the control group. Participants presenting with significantly higher multiple atherogenic scores were found to cluster at the upper quartiles of systolic blood pressure, diastolic blood pressure and pulse rate. Population-specific threshold for waist circumference of >75 cm for females and >80 cm for male were the best adiposity indices for discriminating hypertension. Increasing atherogenic dyslipidaemia was more prevalent with the presence of cardiac target organ damage.

Conclusion: In this urban population, higher altered lipid scores and abdominal obesity aggravated by lifestyle choices including alcohol consumption, smoking and physical inactivity may constitute significant risk for cardiovascular complications among hypertensives.

Keywords: Hypertension; dyslipidaemia; anthropometry; atherogenic; cardiovascular.

1. INTRODUCTION

Cardiovascular disease accounts for approximately 30% of all deaths [1]. High blood pressure and its sequelae are strongly associated with overall cardiovascular risk and are a major health concern the world over [1,2]. Even though detection is said to be limited in Africa the prevalence of hypertension is increasing rapidly [3]. Major health budgetary drains in sub-Sahara countries are attributable to infectious diseases, with inadequate attention to the control and treatment of the upsurge of chronic non-communicable diseases like hypertension [4,5]. Evidence from previous works on the major causes of morbidity and mortality points to a rapid epidemiological transition from solely communicable to a double burden of infectious and chronic diseases among the Ghanaian population [4,6]. Among the list of non-communicable diseases plaguing the general Ghanaian population, hypertension is said to be the most prevalent [7-9]. Attributable factors for this change in disease burden trends in the population are among others, increasing life expectancy and lifestyle modification brought about by urbanization leading to a surge in the prevalence of contributing factors such as

obesity, physical inactivity, stress, unhealthy diet, dyslipidaemia, et cetera [6,8,10,11].

Duda R, Jumah N, Hill A, Seffah J and Biritwum R [12] report that most Ghanaian women are willing to reduce their body size in order to reduce the risk of obesity-linked illnesses, however due to limited knowledge and awareness of obesity related diseases [13], the cultural norms equating certain body images in particular overweight and obesity as a reflection of good living, wealth and prosperity is dominant among the general Ghanaian population [14]. There exists a direct relationship between such body images, adiposity and dyslipidaemia [15]. Earlier studies have reported high levels of dyslipidaemia among hypertensive populations in Kumasi [14,16]. Poor control of blood pressure has been implicated in the development of complications leading to prime target organ damage like the heart among Ghanaian hypertension groups [4,17]. To resolve the differing predictive thresholds of measured parameters for criteria definition of the clusters of risk factors for cardiovascular diseases, the general consensus is that components cutoff should be ethnic-specific adjusted to suit usage in different ethnic groups [18-20]. We [19] and Frank et al. [21] have reported different

population-specific adiposity cutoff point for increased health risk and diabetes respectively among sub-populations in Kumasi.

This study sought to compare the association between different lifestyle practices, adiposity indices, atherogenic dyslipidaemic parameters and hypertension as well as the predictive implications for levels of these parameters in target cardiac organ damage among an urban Ghanaian hypertensive population and as well determine the optimal threshold points and the discriminative power of these parameters.

2. MATERIALS AND METHODS

2.1 Subjects

A hospital-based case-control study was conducted between November 2012 and September 2013. Two hundred and forty one (241) participants were involved in this study. One hundred and eighty (180) non-diabetic hypertensive patients attending clinic at the Komfo Anokye Teaching Hospital (KATH) and the Precise Specialist clinic all in Kumasi Ghana and sixty one (61) age matched normotensives controls from the Kumasi metropolis. The study participants were recruited purposefully from a population of adult individuals between the ages of 22-87 years. Criteria for cases group were patients diagnosed with hypertension that were not suffering from diabetes and were of consent age. The control group were normotensive age matched healthy individuals with no past history of diabetes, cardiac, renal, hepatic dysfunction or dyslipidaemia, living in the Kumasi metropolis and consented to participation of this study.

2.2 Socio-Demographic Data Capture (Questionnaire)

Self-reported structured questionnaire were administered to determine duration of hypertension and treatment status, smoking status, alcohol intake, educational level, physical activity levels, occupation, the usage of non-prescribed orthodox and herbal medications, family history of hypertension, current and past symptoms of cerebrovascular disease, peripheral vascular and coronary heart diseases.

2.3 Blood Pressure (BP) Measurement

Blood pressure (BP) and pulse rate measurements were done using the Omron M5-I

digital fully automatic blood pressures monitor (OMRON Healthcare Europe BV Wegalaan 57 NL-2132 JD Hoofddorp). After participants had sat quietly for at least ten minutes, three measurements were taken at one minute interval on the right arm in a seated position, with arm supported at heart level and feet flat on the floor using an appropriate sized cuff. Hypertension was diagnosed when the mean of the second and third blood pressure (BP) measurements was equal or above 140/90 mmHg or when participants reported use of antihypertensive medication which was verified from their hospital files [22,23].

2.4 Anthropometric Variables

Anthropometric measurements included height to the nearest millimeter without shoes and weight to the nearest 0.1 kg in light clothing. Subjects were weighed on a bathroom scale (Zhongshan Camry Electronic Co. Ltd, Guangdong, China) and their height measured with a Seca stadiometer with the participant standing erect with back straight, heels together, and toes slightly apart at a 60 degree angle. Body mass index (BMI) was calculated by dividing weight (kg) by height squared (m^2). Waist circumference (to the nearest centimeter) was measured with a Gulick II spring-loaded measuring tape (Gay Mills, WI) midway between the inferior angle of the ribs and the suprailiac crest. The hip circumference was measured as the maximal circumference over the hip circumference (HC) at the level of the widest diameter around the gluteal protuberance in centimetres and the waist to hip ratio (WHR) calculated by dividing the waist circumference (cm) by the hip circumference (cm).

2.5 Biochemical Assays

Venous blood samples were collected after an overnight fast (12-16 hours) between 7 am and 10 am. About 5 ml of venous blood was drawn from the antecubital vein of which four (4) ml was dispensed into vacutainer® plain tubes and one (1) ml into fluoride oxalate tubes. After centrifugation at 500 g for 15 minutes, the serum and plasma were stored at $-80^{\circ}C$ until assayed. Parameters that were determined include: Fasting blood glucose (FBG) to exclude participants with diabetes, total cholesterol (TC), triglycerides (TG) and high density lipoprotein cholesterol (HDL-C). Serum low density lipoprotein cholesterol (LDL-C) was calculated using the Frederickson-Friedwald's formula

according to which $LDL-C = TC - TG/2 - HDL-C$. The methods adopted for the determination of the above parameters were predetermined by the reagent manufacturer – (Dialab GmbH, IZ-NÖ Süd, Hondastrasse, A-2351 Wiener Neudorf, Austria). All biochemical analytical investigations were carried out at the Kumasi Centre for Collaborative Research in Tropical Medicine (KCCR) located at Kwame Nkrumah University of Science and Technology.

2.6 Clinical Assessment

All the 241 participants underwent clinical assessment to determine target organ damage. Detailed history, physical examination, chest X-ray, 12-lead resting electrocardiogram (ECG) and transthoracic echocardiogram (ECHO) were done. All diagnosis and interpretation were performed by consultant Radiologist and Cardiologists.

2.7 Diagnosis of Heart Failure

Heart failure was diagnosed, using the modified Framingham criteria for diagnosis of heart failure [24].

Major criteria included: Paroxysmal nocturnal dyspnoea, raised jugular venous pressure, cardiomegaly, basal crepitation, S3 gallop and acute pulmonary oedema.

Minor criteria included: Tachycardia, orthopnea, exertional dyspnea, nocturnal cough, hepatomegaly and diuretic use. Heart failure was diagnosed if the participant had two major and one minor or one major and two minor criteria.

Other complications of hypertension included: Cardiomegaly (without heart failure), stroke or transient ischaemic attack and chronic kidney disease.

2.8 Statistical Analysis

Normality of all continuous variables was tested. All non-parametric variables were normalized by log transformation before analysis and results converted by antilog where appropriate. Continuous variables are expressed as their mean \pm SEM, whereas categorical variables were expressed as Figure and proportion. Comparisons of the general characteristics of the hypertensive group against the normotensive group were performed using unpaired t tests, chi

(χ^2) tests, or Fisher exact tests where appropriate. A posthoc linear contrast was used for trend analysis of continuous variables. Dyslipidaemic atherogenic scores were calculated as the additive score of the number of lipid abnormality recorded by an individual. The Youden Index was computed to identify population-specific cut-off points of selected parameters for the optimal differentiation between cases and controls. The Youden Index is derived from (sensitivity + specificity) - 1 and range from 0 to 1. Using the receiver operator characteristic curves (ROC), area under the curve (AUC), the discriminative power of the population-specific cut-off points for identifying hypertension cases was estimated [21]. A level of $p < 0.05$ was considered as statistically significant for all analysis. IBM Statistical Package for the Social Sciences (SPSS Inc, Chicago, USA; www.spss.com) version 20.00, GraphPad Prism version 6.00 GraphPad software, San Diego California USA, www.graphpad.com and MedCalc Software bv.ba, MedCalc Software, Acacialaan 22, B-8400 Ostend, Belgium, www.medcalc.org) version 12.3.2 for windows were used for statistical analysis where appropriate.

3. RESULTS AND DISCUSSION

Out of the 241 study participants involved in this study, 180 classified as cases were hypertensive patients, the remaining 61 who had no hypertension were classified as controls. The mean age of the participants was 49.57 ± 1.16 , the age of the cases were comparable to that of the controls. The female population (62.70%) was higher than that for male. Majority (53.94%) of the study participants indulged in alcohol intake. The percentage of participants taking in alcohol was significantly higher among the case group (60.00%) compared to the controls (36.06%). Similarly, among the 13.28% of the total study population who admitted to smoking, majority (90.63%) were found to belong to the case group.

Majority 134 (74.44%) of the people, who reported with hypertension belonged to the informal sector of employment; the converse was found among the control group. Even though a higher percentage of the case group were found to be engaged in some form of regular exercise compared to the controls, the percentage difference was not statistically significant. None of the controls presented with any of the complications associated with hypertension

including chronic kidney disease (CKD), cardiomegaly, pedal oedema et cetera. (Table 1).

Disparities in measured anthropometric markers were observed among the case and control group with the case group significantly weighing heavier than the control groups. The case group also presented with a significantly higher waist circumference, a proxy marker for central obesity compared to the control group (Table 2).

In general, the case group presented with a significantly poorer atherogenic lipid profile compared to their counterparts in the control group, the exception though was observed with HDL where the converse was the case (Table 2).

Among the controls there was no significant difference between the female and male anthropometric indices, haemodynamic indices and dyslipidaemia profile measured, recorded among the males compared to their female counterpart. As shown in Table 3, generally significant disparities were recorded in the levels of assayed markers between the females in the control group compared with the females presenting with hypertension, and also the males in the controls group against the hypertensive (Table 3).

Among the participants with hypertension, increasing age was significantly associated with the haemodynamic indices systolic blood pressure and pulse rate. After adjusting for age and gender pulse rate was found to be associated with increased levels of Body Mass Index (BMI), Waist Circumference (WC), Total Cholesterol (TC), Triglyceride (TG), and inversely associated with high density lipoprotein cholesterol (HDL). In general, central obesity measured by the proxy marker of WC was found to be positively correlated with glycaemia, TC, and TG but inversely correlated with HDL (Table 4).

The hypertensive risk age estimated in this study was above thirty-nine years (39 years) for the study population. The homeostatic diagnostic criteria for hypertension was greater than one hundred and thirty (>130 mmHg) for systolic blood pressure (SBP), greater than eighty (>80 mmHg) for diastolic blood pressure (DBP) and greater than sixty four (>64 bpm) for pulse with a significantly high discriminating power ranging from (0.97) for SBP through (0.968) for pulse to (0.995) for DBP on the scale of 0.5 to 1.0.

Among the anthropometric parameters that were measured WC >75 cm for female and >80cm for male as well as WHR of >0.97 for female were significant predictors of hypertension. All lipid atherogenic indicators measured were found to be significant predictors of hypertension among the study participants; however dyslipidaemia as indicated by all atherogenic parameters except HDL cholesterol had a stronger discriminating power (Table 5).

At a cut-off point of >64 bpm for pulse, this study established that 3.3% compared to 82.2% had abnormal pulse among the controls and the cases respectively. High level of abdominal obesity was recorded among the cases compared to the controls.

Dyslipidaemia was significantly higher among the hypertensive subjects compared to their control counterparts. Majority of the normotensive subjects presented with no atherogenic scores. The hypertensive patients were however found to cluster along increasing atherogenic scores. (Table 6).

Among the component atherogenic indices assessed between the study participants who take alcohol and those who do not take alcohol, significantly higher numbers of patients who take alcohol presented with hypertriglycylaemia (32%), reduced high density lipoprotein- HDL (45%), and raised low density lipoprotein- LDL (59%) compared to (22%, 27% and 45% respectively) among those who do not take alcohol. Majority of the participants who smoke presented with raised Total cholesterol – TC (87%) and Triglyceride – TG (56%).

Patients who did not partake in exercise in general presented with high levels of components of dyslipidaemia. No significant difference in terms of components of dyslipidaemia was found among users and non-users of non-prescribed medication, even though in most cases patients using non-prescribed medication presented with higher percentage of abnormalities (Fig. 1).

In general patients who consume alcohol showed increased cluster across increasing atherogenic scores and the opposite was observed in those who did not take alcohol. All the patients who smoked recorded at least one atherogenic abnormality with increasing percentage cluster across increasing atherogenic scores.

Table 1. Socio-demographic characteristic of the population under study stratified by hypertension status

Parameter	Total N=241	Control N=61	Case N=180	P-value
Age	49.57±1.16	49.63±0.58	50.88±0.89	0.82
Gender				
Female	151(62.70%)	45(73.80%)	106(58.90%)	0.046
Male	90(37.30%)	16(26.20%)	74(41.10%)	
Alcohol usage				
No	111(46.06%)	39(63.94%)	72(40.00%)	<0.001
Sometimes	29(12.03%)	12(19.67%)	17(9.44%)	
Always	101(41.91%)	10(16.39%)	91(50.56%)	
Smoking status				
No	209(86.72%)	58(95.08%)	151(83.89%)	0.04
Sometimes	8(3.32%)	2(3.28%)	6(3.33%)	
Always	24(9.96%)	1(1.64%)	23(12.78%)	
Employment status				
Formal	91(37.76%)	45(73.77%)	46(25.56%)	<0.001
Informal	150(62.24%)	16(26.23%)	134(74.44%)	
Family History of HPT	175(72.60%)	40(65.60%)	135(75.00%)	0.18
Herbal Usage	136(56.40%)	33(54.10%)	103(57.22%)	0.77
Non prescribed drugs	140(58.10%)	36(59.00%)	104(57.78%)	0.88
Exercise	152(63.10%)	36(59.00%)	116(64.40%)	0.45
Heart failure	153(63.50%)	0(0.00%)	153(85.00%)	<0.001
Other complications	176(73.00%)	0(0.00%)	176(97.80%)	<0.001

Data is presented as figure with percentage in parenthesis, mean ± SEM

Table 2. Anthropometric, haemodynamic and dyslipidaemia atherogenic parameters of study population stratified by hypertension status

Parameter	Total	Control	Case	P-value
Haemodynamics indices				
SBP(mmHg)	145.24±1.60	117.38±1.04	154.69±1.59	<.001
DBP(mmHg)	94.17±0.10	73.28±0.77	101.25±0.79	<.001
MAP	73.25±1.39	51.41±0.91	80.65±1.46	<.001
Adiposity indices				
Weight(Kg)	78.45±1.0	71.90±1.64	80.68±1.18	<.001
Height(cm)	160.96±0.79	155.24±1.81	162.90±0.82	<.001
BMI(Kg/m ²)	29.52±0.30	29.36±0.65	29.58±0.34	.75
WC(cm)	89.28±1.34	69.098±1.57	96.13±1.37	<.001
HC(cm)	0.94±0.02	0.72±0.02	1.016±0.02	<.001
WHR	0.96±0.00	0.96±0.07	0.97±0.04	.72
Glycaemic index				
Glucose(mmol/L)	4.78±0.06	4.57±0.09	4.85±0.07	.04
Atherogenic indices				
TG(mmol/L)	1.88±0.51	1.67±0.51	1.95±0.52	.001
HDL(mmol/L)	2.09±0.51	1.71±0.52	2.24±0.52	<.001
TC(mmol/L)	6.13±1.54	4.58±1.6	6.77±1.54	<.001
LDL(mmol/L)	3.30±0.68	2.40±0.70	3.68±0.68	<.001

Data is presented as mean±standard error of the mean, SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure, BMI-Body Mass Index, WC- Waist Circumference, HC-Hip Circumference, WHR- Waist-to-Hip Ratio, TG-Triglyceride, HDL-High density lipoprotein, TC-Total Cholesterol, LDL-Low density lipoprotein.

Table 3. Anthropometric, haemodynamic and dyslipidaemia atherogenic parameters of study population stratified by hypertension status and gender

Parameter	Control			Case			Male	Female
	Male	Female	p-value	Male	Female	p-value	p-value	p-value
Haemodynamics indices								
SBP	116.25±1.55	117.77±1.31	.52	156.49±2.68	153.44±1.95	.35	<.001	<.001
DBP	73.125±1.51	73.33±.89	.91	103.45±1.56	99.72±0.75	.02	<.001	<.001
Pulse	50.88±2.00	51.6±1.01	.73	79.88±1.99	81.19±2.06	.66	<.001	<.001
Adiposity indices								
Wgt	72.0±3.16	71.86±1.94	.97	81.49±1.95	80.11±1.46	.56	.04	0.002
BMI	29.73±1.41	29.22±0.73	.74	28.87±0.53	30.08±0.44	.08	.51	.31
WC	69.56±3.28	68.93±1.79	.86	95.16±2.05	96.80±1.85	.56	<.001	<.001
WHR	0.97±0.01	0.96±0.01	.72	0.96±0.06	0.97±0.01	.67	.82	0.57
Glycaemic index								
FBS	4.19±.19	4.71±.09	.01	4.96±0.11	4.78±0.09	.20	.002	.66
Atherogenic indices								
TG	1.67±0.53	1.68±0.52	.91	2.02±0.52	1.90±0.52	.25	.04	.02
HDL	1.83±0.52	1.67±0.52	.17	2.31±0.52	2.19±0.52	.35	.02	<.001
TC	4.68±1.6	4.54±1.55	.65	7.02±1.6	6.60±1.55	.25	<.001	<.001
LDL	2.29±0.71	2.44±0.71	.53	3.86±0.69	3.56±0.69	.09	<.001	<.001

Data is presented as mean ± standard error of the mean, SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure, BMI-Body Mass Index, WC- Waist Circumference, HC-Hip Circumference, WHR- Waist-to-Hip Ratio, TG-Triglyceride, HDL-High density lipoprotein, TC-Total Cholesterol, LDL-Low density lipoprotein. FBS – Fasting blood sugar

Table 4. Partial correlation coefficients of anthropometric variables, haemodynamic, atherogenic indices of control group (upper right sided), indices of case group (Lower left sided). Adjusted for age, gender and medication

Parameter	Age	SBP	DBP	Pulse	BMI	WC	WHR	RBS	TG	HDL	TC	LDL
Age		.09	0.09	-.08	-.06	-.06	.02	-.06	.04	.07	-.04	.11
SBP	.26*		.080	.06	.10	.02	.07	.02	.10	-.02	.05	-.16
DBP	.05	.39**		.06	.15	.11	.00	-.12	-.04	.03	.03	.08
Pulse	.29*	.01	0.0		.22	.21	-.03	.27*	.19	.08	.02	.08
BMI	.15	-.07	.01	.18*		.54**	.11	-.01	-.01	.07	-.18	.16
WC	.17	.04	-.08	.60**	.37**		.30*	.09	.08	.16	-.17	.07
WHR	-.10	.04	-.02	.08	-.00	.07		-.03	-.03	.03	-.02	-.12
FBS	.06	.03	-.06	.09	-.08	.20**	.08		.06	-.09	.07	.08
TG	-.12	.07	.1	.16*	-.06	.29**	-.11	-.14		.31*	.32*	.06
HDL	.15	-.04	.1	-.16*	-.04	-.19*	-.05	-.07	.35**		.11	.07
TC	-.16	-.01	-.12	.15*	.020	.32**	-.11	-.01	.24**	.11		.30*
LDL	-.13	-.00	0	-.07	-.02	.07	-.10	.04	.16**	.37**	.38**	

SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure, BMI-Body Mass Index, WC- Waist Circumference, HC-Hip Circumference, WHR- Waist-to-Hip Ratio, TG-Triglyceride, HDL-High density lipoprotein, TC-Total Cholesterol, LDL-Low density lipoprotein, FBS - Fasting blood sugar. *p* is significant at *0.05 ; **0.01

In general, patients who exercised actively showed lower atherogenic scores compared to those who did not take part in physical exercise. The hypertensive population presenting with left ventricular hypertrophy observed by both electrocardiograph (ECG) and echocardiogram (ECHO) exhibited an increased trend of clustering across increasing atherogenic scores, with the converse observed with those without an abnormal electrocardiograph (ECG) and echocardiogram (ECHO). Patients presenting with cardiomegaly observed by X-ray significantly showed a pattern of increased clustering with increasing atherogenic scores (Fig. 2).

Table 5. Receiver operative characteristics threshold cut-off values of selected variables and their ability to predict hypertension

Parameter	Cutoff	Sensitivity	Specificity	(AUC)	P -value	Youden J
Age	>39	95(90.7 - 97.7)	100(94.1 - 100.0)	0.995	<.001	0.9500
SBP	>130	87.22(81.4 - 91.7)	98.36(91.2 -100.0)	0.970	<.001	0.8558
DBP	>80	98.89(96.0 - 99.9)	100(94.1 - 100.0)	0.995	<.001	0.9889
Pulse	>64	82.22(75.8 - 87.5)	96.72(88.7 - 99.6)	0.968	<.001	0.7894
WC _F	>75	90.57(83.3 - 95.4)	77.78(62.9 - 88.8)	0.911	<.001	0.6834
WC _M	>80	81.08(70.3 - 89.3)	86.67(59.5 - 98.3)	0.897	<.001	0.6775
WHR _F	>0.97	45.28(35.6 - 55.2)	77.78(62.9 - 88.8)	0.616	.02	0.2306
WHR _M	>0.91	85.14(75.0 - 92.3)	10.2(7.0 - 21.8)	0.517	.84	0.1486
BMI	>30.23	96.11(92.2 - 98.4)	11.48(4.7 - 22.2)	0.507	.88	0.0759
FBS	>5.7	22.22(16.4 - 29.0)	100(94.1 - 100.0)	0.563	.11	0.2222
TG	> 2.08	39.44(32.3 - 47.0)	91.80(81.9 - 97.3)	0.646	<.001	0.3125
HDL	< 2.18	49.44(41.9 - 57.0)	90.16(79.8 - 96.3)	0.271	<.001	0.3961
TC	>6.75	39.44(32.3 - 47.0)	73.77(60.9 - 84.2)	0.548	.26	0.1321
LDL	> 4.37	77.78(71.0 - 83.6)	86.89(75.8 - 94.2)	0.860	<.001	0.6466

SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure, BMI-Body Mass Index, WCF- Female Waist Circumference, WCM Male Waist Circumference, WHRF- Female Waist-to-Hip Ratio, WHRM- Male Waist-to-Hip Ratio, TG-Triglyceride, HDL-High density lipoprotein, TC-Total Cholesterol, LDL-Low density lipoprotein. FBS – Fasting

Table 6. Prevalence of obesity, hypertension, dyslipidaemia indices of study population stratified by hypertension status

Parameter	Total	Control	Case	p-value
Haemodynamic indices				
Pulse rate	150(62.2%)	2(3.3%)	148(82.2%)	<.001
Adiposity Indices				
Abdominal Obesity	169(70.1%)	13(21.3%)	156(86.7%)	<.001
Obesity	122(50.6%)	31(50.8%)	91(50.6%)	.55
Central Obesity	1137(56.8%)	26(42.6%)	111(61.7%)	.01
Dyslipidaemia parameters				
Raised TC	148(61.4%)	14(23%)	134(74.4%)	<.001
Raised TG	76(31.5%)	5(8.2%)	71(39.4%)	<.001
Reduced HDL	96(39.8%)	7(11.5%)	89(49.4%)	<.001
Raised LDL	127(52.7%)	2(3.30%)	125(52.7%)	<.001
Atherogenic scores				
None	49(20.3%)	39(63.9%)	10(5.6%)	<.001
One	53(22.0%)	17(27.9%)	36(20.0%)	
Two	54(22.4%)	4(6.6%)	50(27.8%)	
Three	54(22.4%)	1(1.6%)	53(29.4%)	
Four	31(12.4%)	0(0.0%)	31(17.2%)	

SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, TG-Triglyceride, HDL-High density lipoprotein, TC-Total Cholesterol, LDL-Low density lipoprotein

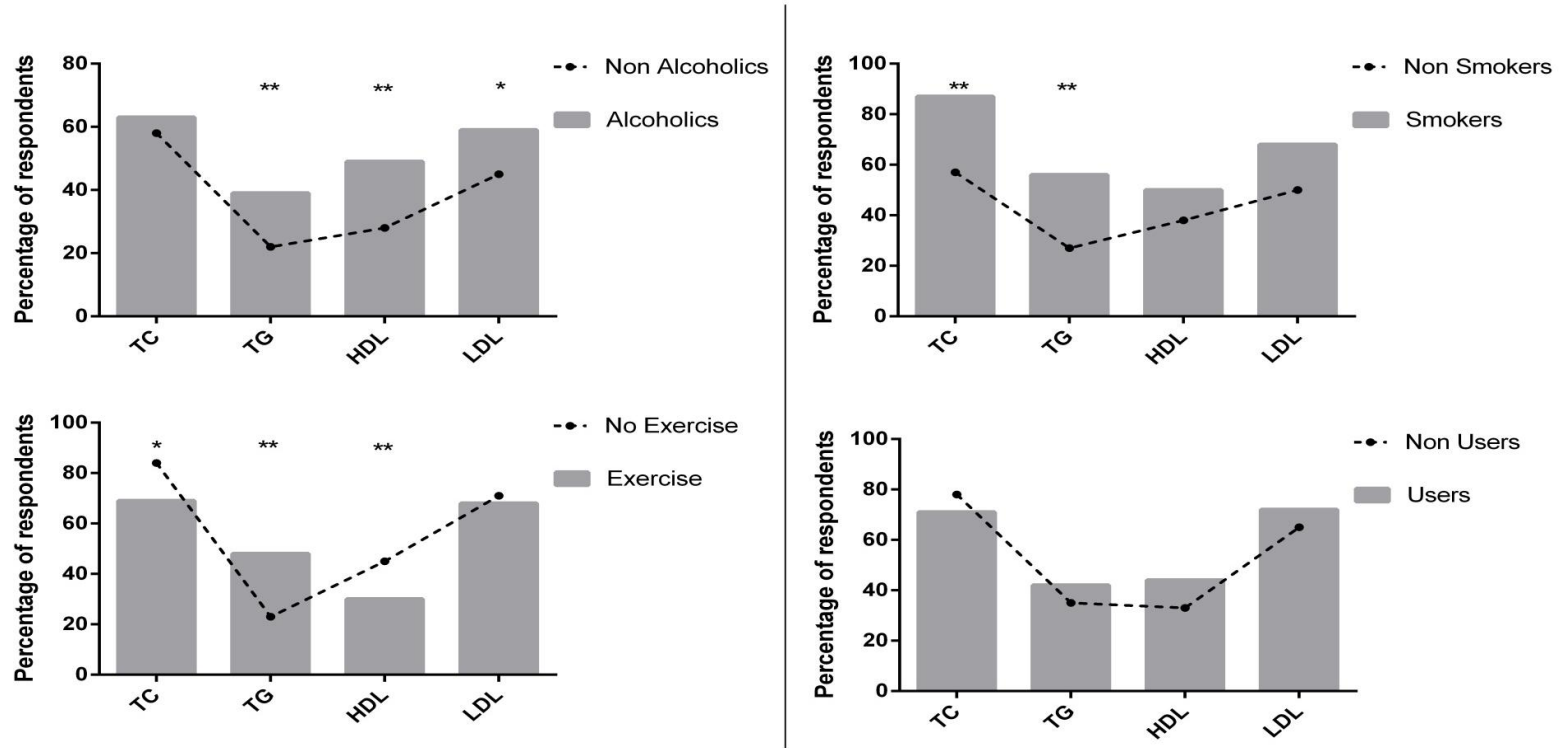


Fig. 1. Component dyslipidaemia stratified by socio-demographic characteristic among hypertension population: TC-Total Cholesterol, TG-Triglyceride, HDL-High density lipoprotein, , LDL-Low density lipoprotein, Non Users & Users of non-prescribed medication. P is significant at 0.05*, 0.01**, 0.001***

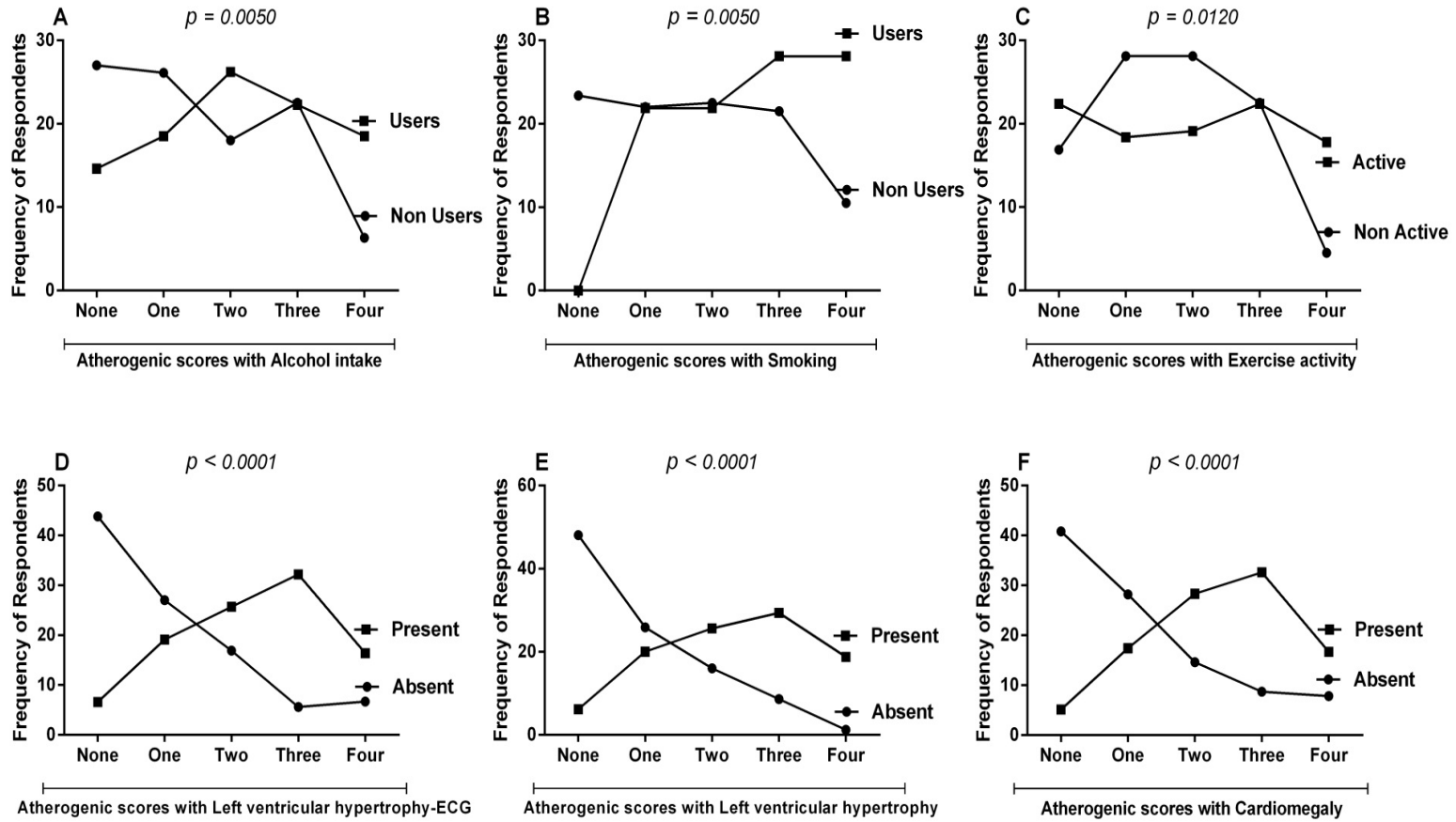


Fig. 2. Atherogenic scores stratified by socio-demographic characteristics and clinical disease characteristics

After assessing selected variables for progressive linear increment or decline from the first through to the fourth quartile of the haemodynamic parameters using linear contrast analysis, a significant additive incremental linear relationship was observed for age, waist circumference and all component atherogenic lipid indices measured with increasing quartile levels for both systolic and diastolic blood pressure. In the case of quartile cluster distribution for pulse rate, significant additive linear relationship was observed for age, all anthropometric indices and all component atherogenic lipid parameters measured except for waist to hip ratio and triglyceride levels respectively (Table 7).

In general, percentage cluster distributions by dyslipaemia were observed to significantly increase progressively from the first to the fourth quartile of all haemodynamic parameters

evaluated in this study. Categorization by systolic blood pressure observed in participants presenting with at least one lipid abnormality were 39.3%, 77.8%, 95.0% and 93.6% at the first, second, third and fourth quartile respectively. The percentage population clusters recorded for diastolic blood pressure were (39.5%, 72.3%, 94.5%, 93.6%) and pulse rate were (37.0%, 87.5%, 92.5%, 95.3%) for the first, second, third and fourth quartile respectively.

As seen in Fig. 3, participants presenting with significantly higher multiple atherogenic scores were found to cluster at the upper quartiles of systolic blood pressure, diastolic blood pressure and pulse rate. Whilst majority of subjects with no or single lipid atherogenicity clustered at the first and second quartiles of the haemodynamic parameters measured (SBP, DBP and pulse rate) (Fig. 3).

Table 7. Linear contrast analysis of quartile cluster distributions of Haemodynamic indices stratified by Age, Adiposity and Atherogenic parameters

Parameter	Q-1	Q-2	Q-3	Q-4	<i>p- lineartrends</i>	
Systolic Blood Pressure	Age	28.82±1.80	41.19±3.13	56.60±1.38	59.92±1.26	<.001
	WC	72.20±2.02	85.19±3.80	97.13±2.18	94.94±2.09	<.001
	BMI	29.10±0.67	30.16±0.83	29.51±0.52	29.62±0.53	0.73
	WHR	0.97±0.01	0.97±0.01	0.96±0.01	0.97±0.01	0.89
	TG	1.84±0.57	1.90±0.58	2.21±0.58	2.20±0.58	<.001
	HDL	2.06±0.61	2.33±0.63	2.63±0.612	2.65±0.61	<.001
	TC	4.67±1.55	5.58±1.59	7.16±1.56	6.58±1.56	<.001
	LDL	2.06±0.61	2.33±0.63	2.63±0.612	2.65±0.61	<.001
Diastolic Blood Pressure	Age	24.45±0.63	45.96±2.30	58.40±1.17	57.40±1.93	<.001
	WC	68.03±1.99	93.10±2.94	93.29±1.97	93.45±2.06	<.001
	BMI	28.74±0.85	29.55±0.62	29.36±0.46	30.44±0.64	0.12
	WHR	0.97±0.01	0.97±0.01	0.96±0.01	0.97±0.01	0.97
	TG	1.87±0.57	1.91±0.57	2.22±0.58	2.25±0.58	.001
	HDL	1.90±0.57	2.04±0.57	2.54±0.58	2.74±0.59	<.001
	TC	4.59±1.56	6.28±1.58	6.74±1.56	6.24±1.57	<.001
	LDL	1.96±0.59	2.62±0.58	3.07±0.58	3.14±0.58	<.001
Pulse Rate	Age	28.48±1.88	52.45±2.42	57.09±1.34	56.69±1.51	<.001
	WC	70.56±2.02	84.88±1.79	92.82±1.82	105.25±2.71	<.001
	BMI	28.82±0.72	29.18±0.58	29.12±0.57	30.84±0.54	.03
	WHR	0.96±0.01	0.96±0.01	0.97±0.07	0.97±0.01	.33
	TG	1.85±0.57	2.21±0.58	2.16±0.58	2.10±0.58	.05
	HDL	2.04±0.61	2.49±0.62	2.84±0.62	2.44±0.61	.001
	TC	4.58±1.56	6.46±1.58	6.57±1.56	6.97±1.57	<.001
	LDL	2.14±0.62	3.08±0.62	3.25±0.61	3.21±0.61	<.001

Data is presented as mean ± standard error of the mean, SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, BMI-Body Mass Index, WC- Waist Circumference, WHR- Waist-to-Hip Ratio, TG-Triglyceride, HDL-High density lipoprotein, TC-Total Cholesterol, LDL-Low density lipoprotein. Q-1 - First quartile, Q-2 - Second quartile, Q-3 - Third quartile, Q-4 - Fourth quartile

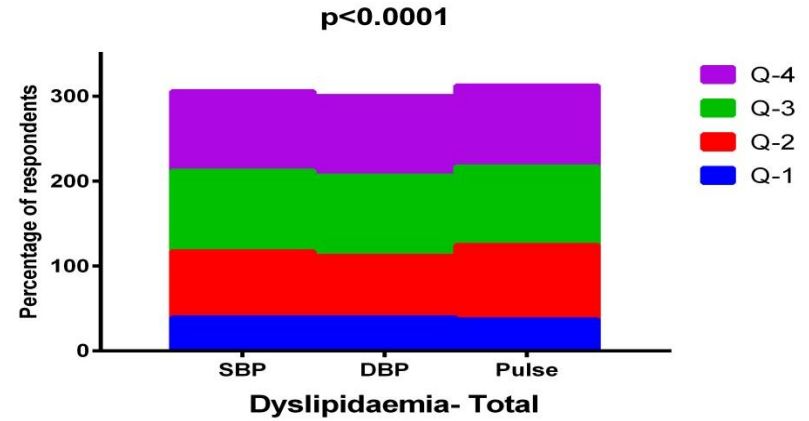
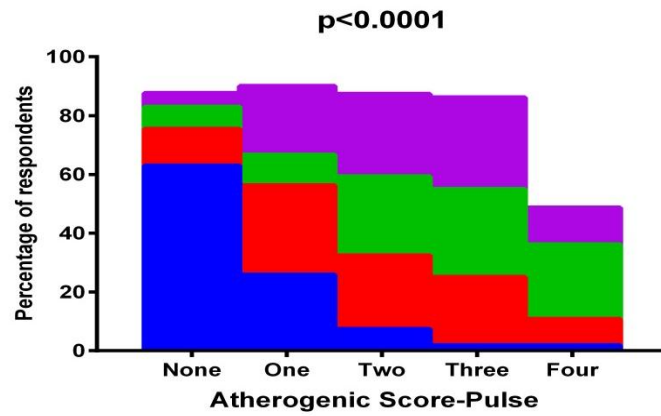
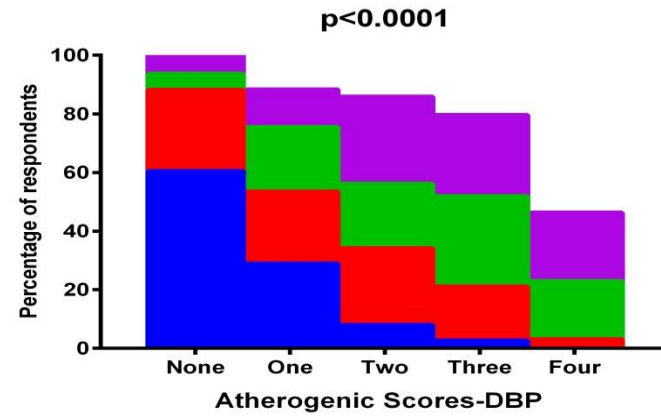
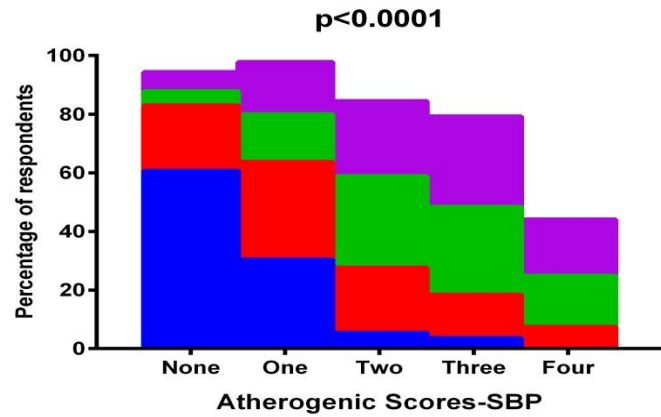


Fig. 3. Atherogenic dyslipidaemia quartile cluster distributions with Haemodynamic parameters. SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure Q-1 - First quartile, Q-2 - Second quartile, Q-3 - Third quartile, Q-4 - Fourth quartile

3.1 Discussion

Hypertension is regarded as a major public health problem [25] and the leading aetiology of cardiovascular diseases [1,26], cardiomegaly [9] and heart failure [27] among the Ghanaian population. Majority of the hypertensive (74.44%) patients were employed in the informal sector and this validates the assertion by Bosu W [7] in a comprehensive review of hypertension that the phenomenon affects every thrust of society rather than the prevailing myth that hypertension is a major problem in only the affluent population. Though increasing age was found to be associated with hypertension the critical age threshold of 39 years was observed as the diagnostic age of hypertension in this study population. This finding, affirms reports of various studies that have reported that the onset of hypertension and its complications occur in a relatively young population among Ghanaians [7,9,28,29].

Alcohol consumption was significantly more profound among the hypertensives than the normotensives. Among the hypertensive population unfavourable lipid outcome as well as a greater cluster of subjects with multiple atherogenic abnormalities was observed with self-reported alcohol intake. Various studies in different sub populations in Ghana have found positive association of alcohol consumption with hypertension [7,8,28]

Various studies have reported varied association between body adiposity and hypertension [30-34]. In Ghana positive association has been reported among various populations between body adiposity and hypertension [7,8,19,35]. In the present study, visceral adiposity measured by the proxy marker of waist circumference [36] was found to be significantly higher among the cases than the control. Even though the body mass index of the cases was higher than the controls, the difference was not statistically significant (Table 2). Interestingly the number of participants presenting with total body obesity was found to be comparable among the two groups, however significantly greater numbers of hypertensive patients presented with abdominal/central obesity compared to their control counterparts, Table 6. These findings are consistent with the results of our earlier study [19], where we found a close association between hypertension (SBP, DBP) and central

obesity (waist circumference) but not with other adiposity indices (BMI, WHR, WHtR) among a Pentecostal population in the Kumasi metropolis. According to Kotchen et al. [37] although the measurement of waist circumference includes both subcutaneous and visceral adipose tissue, visceral adipose tissue independent of total body fat has been found to be the most consistent correlate of hypertension [38,39] and this seems to be the case in this present study. Although the causal relationship between adiposity and hypertension is yet to be clearly delineated, in the view of Reddy K, Reddy K and Sudha G [40] the mechanism of obesity-associated hypertension appears to be associated with inadequate vasodilatation in the face of the increased blood volume and cardiac output, which are the natural consequences of an increased body mass [39]. Obesity may lead to hypertension via pathways that stimulate sympathetic nerve activity (SNA), the renin-angiotensin-aldosterone system, insulin resistance, altered vascular function and physical compression of the kidneys, all of which can cause sodium retention [37,40-43]. These mechanisms underlying the blood pressure – adiposity relationship in hypertensive individuals is suggested by some authors to be modulated by a combination of environmental and genetic factors [37,44,45]. After adjusting for age, gender and hypertension treatment status, no association was found between the adiposity and haemodynamic indices among the normotensives, however among the hypertensives increasing waist circumference as well as body mass index was associated with increasing pulse in this study. There is also an increasing linear trend of average waist circumference with a progressive quartile clustering of the study population with all haemodynamic indices (SBP, DBP and Pulse) assessed in this study. This finding is more consistent with the concept of accentuated association of hypertension with regional body fat distribution rather than the extent of total body fat accumulation [46,47]. Fat tissues have been identified as an important endocrine organ engaged in the secretion of many bioactive molecules such as angiotensinogen, angiotensin II, and adipokines [47]. Overweight and obesity are associated with adipose tissue dysfunction, characterized by enlarged hypertrophied adipocytes, increased infiltration by macrophages and marked changes in secretion of adipokines and free fatty acids resulting in chronic vascular inflammation, oxidative stress,

activation of the renin-angiotensin-aldosterone system and sympathetic overdrive, eventually leading to hypertension [48]. However in the pathogenicity of adiposity, android (mainly visceral) and renal sinus fat depots have been viewed to be more pathologic than gynoid (mainly subcutaneous) depots [49-51]. Visceral adiposity (mesenteric and omentum fat) induces excess free fatty acids into the liver through accelerated lipogenesis and lipolysis which eventually cause the enhancement of hypertension through atherosclerosis [52,53]. Among the hypertensive population, this study has established an association between central obesity as indicated by waist circumference (WC) with raised TC, TG and reduced HDL through a positive correlation with TC and TG but an inverse correlation with HDL (Table 4).

Consistently high prevalence of dyslipidaemia has been reported in previous studies which evaluated lipid levels among both the hypertensive and normotensive populations in the Kumasi metropolis [14,16]. The finding of the current study confirms the prevalence of high atherogenic dyslipidaemia among the population as reported by the earlier reports. With the exception of the HDL level, unfavorable lipid profile was significantly higher among the hypertensives compared to their normotensive counterparts. Thus a discrepancy arises in this report compared with the former [14] where higher HDL levels were recorded in the normotensives group, but concordance was established with similar findings of higher HDL level in hypertensives [16]. Disordered reduced HDL recorded a lower percentage among the normotensive group in contrast to the earlier observation in the latter study. This suggests that the attributed factors which were mainly socio-demographic transformation through life-style modification to affluence and sedentary attitudes coupled with psychosocial distress brought about by increasing urbanization of the population in Kumasi is still persistent or has probably worsened [14,16,19]. Among the possibilities put forward by Abdulle AM [54] in accounting for the occurrence of a better lipid profile among hypertensives were excess mortality among hypertensives with unfavorable lipid profiles which may infer lower levels among survivors and the effects of lipid lowering medications. However the latter suggestion was found not to be plausible from this study since significantly higher levels of HDL and lower percentage of reduced HDL abnormality was exhibited in

treatment naïve, newly diagnosed hypertensive patients compared to normotensives controls.

In the present study independent of age, gender, medications, TG and TC were positively associated with pulse whilst HDL was inversely associated with pulse among the hypertensives. Multiple dyslipidaemia atherogenic scores were more profound among the cases (74.4%) with only 8.2% of the normotensives recording multiple atherogenic scores. Though no significant relationship was observed in the total population between the lipid parameters and the primary haemodynamic measures (SBP, DBP), component atherogenic lipid indices measured was found to increase with quartile levels for both systolic and diastolic blood pressure. Increasing percentage cluster distribution by atherogenic dyslipidaemia was observed to significantly increase in magnitude with progressive quartile grading of haemodynamic readings (SBP, DBP and Pulse rate). Also the upper quartile of SBP, DBP and Pulse rate recorded significantly higher multiple atherogenic abnormalities (Fig. 3). This finding is consistent with that of Halperin RO, Sesso HD, Ma J, Buring JE, Stampfer MJ and Michael Gaziano J [55] who reported an association of the higher quintile of TC, non-HDL-C, and TC/HDL-C ratio with increased risks of developing hypertension, compared with participants in the lowest quintile.

Though the biological mechanism through which atherogenic lipids influence hypertension is still a subject of intense scientific research, the critical role played by lipids in the induction of endothelial dysfunction as well as alterations in endothelin-1 and endothelin A and B receptor expression are the most important event in the pathogenesis of hypertension [55,56]. Experimental studies suggest a direct role for HDL-C in promoting cholesterol efflux (reverse cholesterol transport) from foam cells in the atherosclerotic plaque depots in blood vessels to the liver for excretion. HDL-C also exhibits potent anti-inflammatory and antioxidant effects that inhibit the atherogenic process and thus lower HDL-C which has much influence on hypertension development [57]. The physiological balance between the potent vasodilator endothelium derived nitric oxide (NO) and various endothelium derived vasoconstrictors and the sympathetic nervous system is associated with vascular homeostasis [58,59]. Endothelium derived nitric oxide among its' anti-atherogenic functions has been reported

to maintain blood vessel tone through the suppression of platelet aggregation, leucocyte migration, and cellular adhesion to the endothelium, vascular smooth muscle cell proliferation and migration, activation and expression of certain adhesion molecules, and influence production of superoxide anion [58,60,61]. Pro-atherogenic lipids, such as oxidized low-density lipoprotein (oxLDL) inhibit signal transduction from receptor activation to endothelial nitric oxide synthase (eNOS) activation and therefore reduce the bioavailability of NO [58,60,61]. Links between dyslipidaemia and hypertension has been established through mechanisms such as angiotensin I over expression, insulin resistance and sympathetic hyperfunction [55].

Higher dyslipidaemic atherogenic scores were found to significantly cluster with poor prognostic presentation of clinical cardiac target organ damage among the hypertensives (nonspecific sinuses or left ventricular hypertrophy, cardiomegaly). According to Gulati A, Vasawala H, Dalal J, Jain P, Patil S and Padmanabhan TNC [62] who termed the coexistence of dyslipidaemia and hypertension 'LIPITENSION' the co-existence of the two risk factors has more than an additive adverse impact on the vascular endothelium and CVD development. Left ventricular hypertrophy is an ominous harbinger of coronary disease [63] and Sundström J, Lind L, Vessby B, Andrén B, Aro A and Lithell HO [64] in a 20 years prospective longitudinal cohort study found lipids as an important predictor of the onset of left ventricular hypertrophy (LVH). Among a Ghanaian population, hypertension has been found to account for 78.4% cases of cardiomegaly in a pathological review by Akosa A and Armah H [9]. To the best of our knowledge no known study has evaluated dyslipidaemic atherogenic abnormalities among Ghanaian hypertensive subjects with target cardiac organ damage, thus the characterization of the hypertensive participants in the present study by lipid abnormalities vis-à-vis target cardiac organ damage evaluated by the use of X-ray, echocardiogram and electrocardiograph does not only throw light on the role of dyslipidaemia in worsening prognosis among hypertensives in Ghana, but also is a novelty in its setting.

In recent times the World Health Organisation/International Association for the Study of Obesity/International Obesity Task Force (WHO/IASO/IOTF) have suggested

consideration of using lower obesity measurements in Asian and other places to guide health care professionals to promote healthy life and weight control [19,65-67]. The International Diabetes Federation (IDF) task force on epidemiology and prevention in an attempt to harmonizing the definition of metabolic syndrome also proposed the use of ethnic based anthropometric threshold cutoffs [20]. In this study a waist circumference of greater than 80cm and 75 cm for men and women respectively was found to be significant predictors of hypertension. A waist to hip ratio of greater than 0.97 for women and not men was also a predictor of hypertension. Compared with a waist circumference of ≥ 94 cm for men and ≥ 80 for women for sub-Sahara African population proposed by the IDF [20] this study buttresses our earlier finding that lower cutoff of obesity indices are needed for association with increased health risks [19].

4. CONCLUSION

In this urban population, higher altered lipid scores and abdominal obesity aggravated by lifestyle choices including alcohol consumption, smoking and physical inactivity may constitute significant risk for cardiovascular complications among hypertensives.

ETHICAL APPROVAL

The participation of the respondents who are all indigenes of Ghana was voluntary and informed consent was obtained from each of them after thorough explanation of what the study entailed. This study was approved by the School of Medical Sciences and KATH Committee on Human Research Publications and Ethics (CHRPE/08/11).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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