



Association between Anthropometry, Dyslipidaemia and the Ten-Year Relative Risk of Cardiovascular Disease in Ghanaians with Type 2 Diabetes and Hypertension at the Battor Catholic Hospital

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Abstract

Disordered body fat distribution and plasma lipid levels promote atherosclerosis, a major risk factor for cardiovascular disease (CVD). The process of atherosclerosis is vigorous and rapid in diabetes and hypertension. This study sought to determine the relationship between anthropometric and dyslipidaemic parameters as well as ascertain using the Framingham percentage risk for heart disease, the ten-year relative risk of developing CVD among type 2 diabetes and hypertensive patients attending the Battor Catholic Hospital. This hospital-based case-control study involved 125 participants with hypertension, type 2 diabetes or both and 62 age-matched healthy individuals as controls. Socio-demographic data was captured using a semi-structured questionnaire; anthropometric and biochemical variables were obtained using standard methods. The anthropometric and atherogenic dyslipidaemic parameters of the case participants were found to be significantly higher compared to the controls. Among the case group, 49 (39.2%) were observed to have a high risk and 41 (32.8%) with a moderate risk of developing coronary heart disease in ten years. Among the control group, 11.3% presented with moderate risk with none scoring a high risk of developing coronary disease within ten years. The odds of developing coronary heart disease in ten years was 4.5 times higher among the case group with a higher female preponderance.

Subject Areas

Diabetes & Endocrinology

Keywords

Type 2 Diabetes, Hypertension, Cardiovascular Disease, Cardiovascular Risk, Framingham Risk Score, Dyslipidaemia

1. Introduction

In Ghana, type 2 diabetes and hypertension prevalence rates have recorded a steady increase over the years [1] [2]. Obesity and dyslipidaemia are mostly associated with people presenting with type 2 diabetes and hypertension [3] [4]. Disordered body fat distribution and plasma lipid levels promote atherosclerosis, a major risk factor for cardiovascular diseases (CVD) such as stroke, coronary artery disease and peripheral vascular disease [4] [5]. In diabetes mellitus and hypertension, the process of atherosclerosis is even more aggressive, thus rapidly predisposing these individuals to a greater risk for CVD [5] [6]. Studies in the Ghanaian populations regarding obesity and dyslipidaemia have mainly been carried out in urban and peri-urban settings [5] [6] [7] [8]. There is therefore a paucity of literature with respect to studies in rural settings like Battor in the Volta Region. This study therefore seeks to determine the relationship between anthropometric, dyslipidaemic parameters and ascertain using the Framingham percentage risk for heart disease, the relative risk of developing CVD in ten years among type 2 diabetes and hypertension patients under clinical management at the Battor Catholic Hospital.

2. Materials and Methods

2.1. Study Participants

This hospital-based case-control study was conducted between December 2012 and February 2013. A total of 187 participants were conveniently and purposively enrolled into the study. These consisted of 125 case participants, defined as persons previously diagnosed with hypertension and/or types 2 diabetes and were on medication. The control group consisted of 62 age-matched healthy individuals in the study area without a history of diabetes, hypertension or any inflammatory indications. The case participants were stratified into three groups: 1) known hypertension 43, 2) known diabetes 40 and 3) known hypertension and diabetes 42. The case participants were clients visiting the Out-Patient Department (OPD) of the Battor Catholic Hospital. The exclusive criteria were type 2 diabetes individuals who were not under management at the facility and those who did not consent to be part of this study.

Ethical Considerations

All procedures were approved by the Ethics Committee of the School of Medical Sciences, KNUST and KATH, Kumasi (CHRPE/RC/119/12). A written in-

formed consent form was completed by all the participants.

2.2. Socio-Demographic Data Capture (Questionnaire)

Data was captured using a self-reported semi-structured questionnaire. Socio-demographic variables included gender, educational background, duration of work, family history of diabetes and cardiovascular diseases. Self-reported rating of dietary intake of salt, sugar, fat, and alcohol, classified as none, moderate and high as well as physical activity rating (none, not often, very often) were qualitatively captured.

2.3. Blood Pressure Measurement

Mercury sphygmomanometer and stethoscope were used to measure the blood pressure of study participants after at least a 10-minute resting period in accordance with the recommendation of the American Heart Association [9]. Using an appropriate size blood pressure cuff, the blood pressure of each patient was taken twice by a single qualified nurse within an interval of 5 minutes and the average value taken.

2.4. Anthropometric Measurement

The anthropometric measurements were made using the methods described by Bannerman, *et al.* [10]. Measurements of height (to the nearest 0.1 cm) without shoes were made using a stadiometer (height meter) and body weight (to the nearest 0.1 kg) in light clothing using a portable weighing scale. Body mass index (BMI) was calculated by dividing weight (kg) by the square of the height (m²). The waist circumference was measured at the point yielding the smallest circumference between the lower rib margin and the iliac crest. Hip circumference was recorded at the point yielding the maximum circumference over the buttocks. The waist-to-hip ratio (WHR) was calculated by dividing the waist circumference (cm) by the hip circumference (cm).

2.5. Sample Collection, Preparation and Analysis

About 5 mL of fasting venous blood was drawn from each study participant after an overnight fast (12 - 14 hours) using standard phlebotomy procedures. Three (3) mL blood was dispensed into BD vacutainer® serum separator tubes for the estimation of different biochemical parameters whereas 2 mL was dispensed into sodium fluoride for blood glucose estimation. The samples were immediately transported to the clinical biochemistry laboratory of Battor Catholic Hospital, where clotted blood was centrifuged at 3000 revolutions per minute (rpm) for 5 minutes at room temperature to obtain serum/plasma. All laboratory assays were carried out at Battor Catholic Hospital clinical biochemistry laboratory. Sample for fasting blood glucose was analysed using ELITech reagents from Vital Scientific Co Ltd on a semi-automated biochemistry analyser (Maysun Company Limited, China). Lipid profile including Total Cholesterol (TC), High density lipoprotein-Cholesterol (HDL-C) and Triglycerides were assayed using rea-

gents from JASTM Diagnostics, Florida on BT 3000® (Biotenica Instruments, Italy) clinical chemistry auto analyser. Low density lipoprotein-Cholesterol (LDL-C) and Very low density lipoprotein-Cholesterol (VLDL-C) were automatically determined using in-built Friedewald, *et al.* [11] and De Long, *et al.* [12] equations respectively. The methods used in analysing the biochemical variables were predetermined by the reagent manufacturers.

2.6. Relative Risk of Cardiovascular Disease

The percentage risk of developing coronary heart disease over the next 10 years (Absolute Risk) was determined on the Framingham Risk Score [13] comparing the risk scores to the risk of others of the same age (Relative Risk). Those with percentage risk score equal or below the low risk population were classified as low risk, those with risk scores above the low risk group of same age but equal to the average risk group of the same age were classified as having moderate 10-year risk, while participants with percentage scores over and above the average risk of same group were considered as having high risk.

2.7. Statistical Analysis

Categorical data was presented as frequency and percentage. A Chi-square or Fisher exact test was used to test the difference between proportions. Continuous data was presented as means with standard deviation of the mean. Where appropriate, means of continuous data were compared using unpaired t-test and One-way ANOVA with a Bonferroni post-hoc test. Associations were evaluated using Pearson Correlation Coefficient. A *p*-value < 0.05 was taken as significant. Graph Pad Prism version 6.00 for windows (Graph pad software, San Diego California, USA, www.graphpad.com) and IBM Statistical Package for the Social Sciences (SPSS Inc, Chicago, USA; (www.spss.com)) version 22.00 were used for data analysis where appropriate.

3. Results

Out of the study population of 187, participants classified as control were 62 (33.2%) and the rest as case presenting with diabetes, hypertension or both 125 (66.8%). As can be seen from **Table 1**, majority of the participants were females 130 (69.5%). In general, significantly higher levels of education were reported among the controls, the inverse was however observed among the case group. Majority of the respondents worked between one (1) to eight (8) hours a day, though longer working hours were recorded among the case group in comparison to the control (*p*-0.0415). Dietary salt, sugar and fat intake as well as alcohol intake were predominantly moderate among the study group, with consumption significantly higher toward the controls. Casual participation in exercise was recorded among respondents; however, majority of the case group do not engage at all in exercise 67 (54.0%) (see **Table 1**).

The average age of the respondents in this study was 48.2 ± 14.2 years. In general, the anthropometric parameters of the case group were significantly

Table 1. General socio-demographic characteristics of respondents stratified by disease status.

Parameter	Total	Control	Case	P-value
	n-187	n-62	n-125	
Gender				
Female	130 (69.5)	43 (69.4)	87 (69.6)	0.5505
Male	57 (30.5)	19 (30.6)	38 (30.4)	
Educational Background				
None	45 (24.0)	6 (9.7)	39 (31.2)	< 0.0001
Basic	68 (36.4)	11 (17.7)	57 (45.6)	
Secondary	26 (13.9)	9 (14.5)	17 (13.6)	
Tertiary	48 (25.7)	36 (58.1)	12 (9.6)	
Working Hours				
None	37 (19.8)	8 (12.9)	29 (23.2)	0.0415
1-8 Hours	115 (61.5)	47 (75.8)	68 (54.4)	
>8 Hrs	35 (18.7)	7 (11.3)	28 (22.4)	
Dietary Salt				
Moderate	165 (88.2)	57 (91.9)	108 (86.4)	0.0134
High	9 (4.8)	5 (8.1)	4 (3.2)	
Dietary Sugar				
Moderate	107 (57.2)	55 (88.7)	52 (41.6)	< 0.0001
High	10 (5.3)	6 (9.7)	4 (3.6)	
Dietary Fat				
Moderate	147 (78.6)	52 (83.9)	95 (76.0)	0.0003
High	14 (7.5)	9 (14.5)	5 (4.0)	
Alcohol Intake				
Moderate	54 (28.9)	32 (51.6)	22 (17.6)	< 0.0001
High	11 (5.9)	3 (4.8)	8 (6.4)	
Exercise				
None	88 (47.1)	21 (33.9)	67 (53.6)	0.0272
Not Often	73 (39.0)	32 (51.6)	41 (32.8)	
Very Often	26 (13.9)	9 (14.5)	17 (13.6)	
Family History H,D,H/D				
One	81 (43.3)	32 (51.6)	49 (39.2.0)	0.1914
More Than 1	46 (24.6)	11 (17.7)	35 (28.0)	

Data is presented as figure with percentage in parenthesis. p is significant at 0.05. n: number, H: Hypertension, Diabetes, H/D: Both Hypertension and Diabetes.

higher compared to the controls, the exception though was found in the height of participants where though not statistically significant the controls were averagely 0.1 meter taller than the cases. Significantly higher systolic and diastolic blood pressures values were observed among the cases compared to the controls. Among the biochemical markers assayed, higher glycaemic and atherogenic lipid levels were observed among the cases (see **Table 2**).

Significant intra group age differences were observed among the cases, with the mean age increasing from those presenting with only diabetes [48.3 ± 13.9] through those presenting with only hypertension [57 ± 12.5] to participants presenting with both conditions [58.2 ± 12.3]. Significant differences were also observed among the case group with respect to both waist and hip circumference, presenting a trend as afore mentioned for age. The highest average fasting blood glucose concentration was reported among the diabetes only group [12.4 ± 4.4] followed by those with both chronic conditions [11.9 ± 2.7]. Lipid indices assayed in this study were found to be comparable across the case group with the exception of VLDL-C (see **Table 3**).

Table 2. General anthropometric, hemodynamic, and biochemical characteristics of study population stratified by disease status.

Parameter	Total n-187	Control n-62	Case n-125	p-value
Age (years)	48.2 ± 14.2	47.3 ± 15.0	50.4 ± 13.4	0.1541
<i>Anthropometric Parameters</i>				
Weight (kg)	71.6 ± 16.4	69.2 ± 15.7	73.0 ± 17.9	0.1393
Height (m)	1.6 ± 0.01	1.7 ± 0.01	1.6 ± 0.01	0.1049
BMI (kg/m ²)	26.9 ± 5.5	25.6 ± 5.5	27.7 ± 6.7	0.0181
WC (cm)	87.7 ± 14.1	79.2 ± 11.8	92.5 ± 12.3	<0.0001
HC (cm)	102.5 ± 15.5	98.1 ± 11.8	104.9 ± 16.8	0.0025
WHR	0.86 ± 0.01	0.81 ± 0.01	0.89 ± 0.01	<0.0001
<i>Hemodynamic Parameters</i>				
SBP (mmHg)	131 ± 26.0	113.9 ± 12.6	140.7 ± 26.8	<0.0001
DBP (mmHg)	80.9 ± 13.7	73.7 ± 8.7	85.0 ± 13.4	<0.0001
<i>Biochemical Assays</i>				
FBG (mmol/l)	8.1 ± 0.4	5.3 ± 0.1	9.7 ± 0.5	<0.0001
TC (mmol/l)	5.2 ± 0.1	4.5 ± 0.1	5.5 ± 0.1	<0.0001
TG (mmol/l)	1.4 ± 0.1	1.1 ± 0.1	1.6 ± 0.1	0.0004
HDL-C (mmol/l)	1.6 ± 0.04	1.4 ± 0.04	1.7 ± 0.1	0.0002
LDL-C (mmol/l)	2.9 ± 0.1	2.6 ± 0.1	3.1 ± 0.1	<0.0001
VLDL-C (mmol/l)	0.3 ± 0.01	0.2 ± 0.02	0.3 ± 0.01	0.0004

Continuous data is presented as means ± standard deviation of the mean. Continuous data were compared using unpaired t-test. p is significant at 0.05. VLDL-C: Very Low Density Lipoprotein-Cholesterol, LDL-C: Low Density Lipoprotein-Cholesterol, HDL-C: High Density Lipoprotein, TC: Total Cholesterol, TG: Triglycerides, FBG: Fasting Blood Glucose, WHR: Waist-to-Hip Ratio, BMI: Body Mass Index, WC: Waist Circumference, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure.

Table 3. General anthropometric, hemodynamic and biochemical characteristics of cases stratified by disease type.

Parameter	Diabetes	Hypertension	Diabetes/Ht	p-value
	n-40	n-43	n-42	
Age	48.3 ± 13.9	57.1 ± 12.5**	58.2 ± 12.3**	0.0009
Anthropometric Parameters				
Weight (kg)	67.5 ± 14.5	72.2 ± 16.4	75.4 ± 16.5	0.0910
Height (m)	1.6 ± 0.1	1.6 ± 0.0	1.6 ± 0.0	0.6341
BMI (kg/m ²)	25.4 ± 6.3	27.2 ± 5.2	28.7 ± 5.8*	0.0415
WC (cm)	85.2 ± 13.3	90.6 ± 11.1¥	97.8 ± 12.3***	<0.0001
HC (cm)	97.9 ± 13.9	103.9 ± 14.4	108.7 ± 17.5**	0.0080
WHR	0.9 ± 0.6	0.9 ± 0.0	0.9 ± 0.0	0.0669
Hemodynamic Parameters				
SBP (mmHg)	119.0 ± 15.2	149.8 ± 23.6***	143.8 ± 23.9***	<0.0001
DBP (mmHg)	74.9 ± 9.5	89.8 ± 11.1***	86.0 ± 12.3***	<0.0001
Biochemical Assays				
FBG (mmol/l)	12.4 ± 4.4	6.3 ± 2.6***¥¥	11.9 ± 2.7	<0.0001
TC (mmol/l)	5.1 ± 1.2	5.6 ± 1.3	5.6 ± 1.3	0.1030
TG (mmol/l)	1.5 ± 0.6	1.6 ± 0.7	1.5 ± 0.6	0.9148
HDL-C (mmol/l)	1.5 ± 0.6	1.7 ± 0.7	1.7 ± 0.6	0.0807
LDL-C (mmol/l)	3.0 ± 1.2	3.2 ± 0.6	3.2 ± 0.6	0.5243
VLDL-C (mmol/l)	0.4 ± 0.1	0.3 ± 0.0*	0.3 ± 0.0*	0.0069

Data is presented as means ± standard deviation of the mean. p is significant at 0.05. Continuous data were compared using one-way ANOVA with a Bonferroni posttest. Ht: Hypertensives, VLDL-C: Very Low Density Lipoprotein-Cholesterol, LDL-C: Low Density Lipoprotein-Cholesterol, HDL-C: High Density Lipoprotein, TC: Total Cholesterol, TG: Triglycerides, FBG: Fasting Blood Glucose, WHR: Waist-to-Hip Ratio, BMI: Body Mass Index, WC: Waist Circumference, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, p: compares all three, *compares control with any, **compares hypertensives or diabetic/hypertensive with diabetics, ¥compares hypertensives and diabetic/hypertensive.

In the control group, the study revealed no significant correlation among the anthropometric variables with the atherogenic indices, as well as glycaemic indices. An observed increase in waist circumference corresponded to an increase in both the systolic and diastolic blood pressure. Among the case group an increase in any of the anthropometric indices (BMI, WC, HC, and WHR) was associated with a corresponding increase in the haemodynamic parameters measured (SBP and DBP). Waist circumference was associated with all the atherogenic indices assayed. The haemodynamic parameters of the case group were found to be positively associated with glycaemia, TC and LDL. Also among the cases a positive relationship was found between glycaemic levels and TC as well as LDL (see **Table 4**).

Among the general study population, it was observed that 26.2% of the participants had a high risk of developing coronary heart disease over the next ten years. A further 26.2% were estimated to have a moderate risk of developing co-

Table 4. Pearson's correlation coefficients of Anthropometric variables, Haemodynamics and Atherogenic Indices for control group (upper right-hand side) and case group (lower left-hand side).

Parameter	BMI	WC	HC	WHR	SP	DP	FBG	TC	TG	HDL	LDL	VLDL
BMI		0.71**	0.66**	-0.03	0.11	0.18	0.014	0.08	0.14	-0.06	0.08	0.14
WC	0.68**		0.75**	0.29**	0.19*	0.21*	0.058	0.08	0.17	-0.11	0.09	0.17
HC	0.81**	0.82**		-0.41**	0.16	0.19	0.067	0.03	0.15	-0.14	0.06	0.15
WHR	0.07	0.63**	0.06		0.05	0.01	-0.017	0.06	-0.01	0.05	0.05	-0.01
SP	0.33**	0.39**	0.27*	0.31*		0.70**	-0.190	-0.01	0.03	-0.02	-0.01	0.03
DP	0.38**	0.43**	0.35**	0.26*	0.83**		-0.155	-0.11	0.05	-0.17	-0.05	0.05
FBG	0.15	0.11	0.23	-0.13	0.27*	0.27*		-0.03	0.10	-0.06	-0.04	0.10
TC	0.10	0.12*	0.16	0.15	0.40**	0.32*	0.276*		0.19*	0.51**	0.79**	0.19*
TG	-0.04	-0.23*	-0.05	0.03	-0.04	-0.02	0.031	0.30*		-0.10	-0.14	1**
HDL	-0.17	-0.37**	-0.06	-0.03*	0.14	0.01	0.038	0.52**	0.20		0.01	-0.10
LDL	0.21	0.29*	0.24	0.18	0.42**	0.38**	0.287*	0.78**	-0.24	0.06		-0.14
VLDL	0.04	0.01*	0.05	0.03	-0.04	-0.02	0.031	0.30*	1**	0.20	-0.24	

VLDL-C: Very Low Density Lipoprotein-Cholesterol, LDL-C: Low Density Lipoprotein-Cholesterol, BMI: Body Mass Index, WC: Waist Circumference, HC: Hip Circumference, WHR: Waist-to-Hip Ratio, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, FBG: Fasting Blood Glucose, TC: Total Cholesterol, TG: Triglycerides, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein-Cholesterol, VLDL: *Correlation is significant at the 0.05 level (2-tailed), **Correlation is significant at the 0.01 level (2-tailed), ***Correlation is significant at the 0.001 level (2-tailed).

ronary heart disease within the next ten years. As shown in **Figure 1(a)**, significantly all the study participants classified as having high risk of developing coronary heart disease were of the case group 49 (39.2%). Additionally, 41(32.8%) of the case group was estimated to carry a moderate risk of developing coronary heart disease compared to 11.3% of the control group. The odds of developing coronary heart disease in ten years was 4.5 times higher among the case group compared to the controls. Gender variations in percentage ten-year risk estimations was observed, with significantly greater number of female participants than male exhibiting high risk profile and the reverse observed at the moderate risk cluster (see **Figure 1(a)** & **Figure 1(b)**, **Figure 2(a)** & **Figure 2(b)**).

4. Discussion

Essentially, age is a risk factor for the development of type 2 diabetes and cardiovascular morbidity; the risk of heart disease is observed to increase about 3-fold with each advancing decade [14]. In developing countries, majority of people with diabetes are between the ages of 45 and 64 years [15]. Older age is reportedly associated with heart disease after age 55 and 45 years for women and men respectively [14]. In this study, the average age of the respondents was 48.2 ± 14.2 years. However, among the case group significant intra group age differences were observed, with the mean age increasing from persons presenting with diabetes [48.3 ± 13.9] through those presenting with hypertension [57.1 ± 12.5] to participants presenting with both conditions [58.2 ± 12.3] (see **Table 3**).

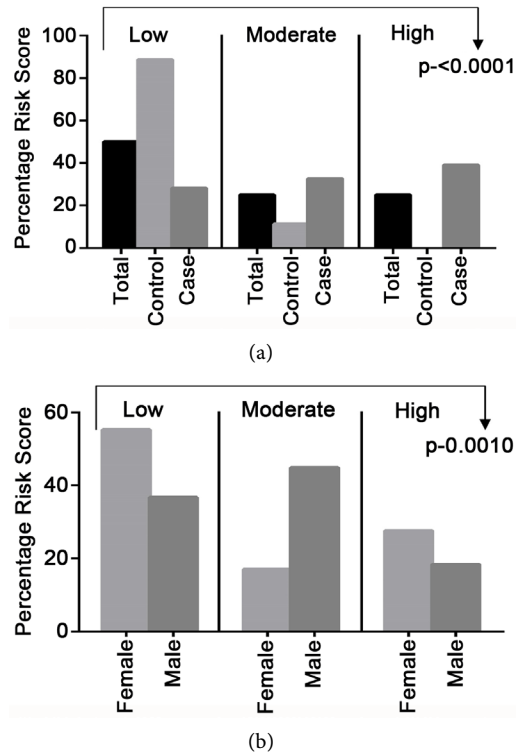


Figure 1. Relative risk scores of Framingham percentage risk of heart disease in ten years stratified by case, control and gender. (a) Case and Control; (b) Gender.

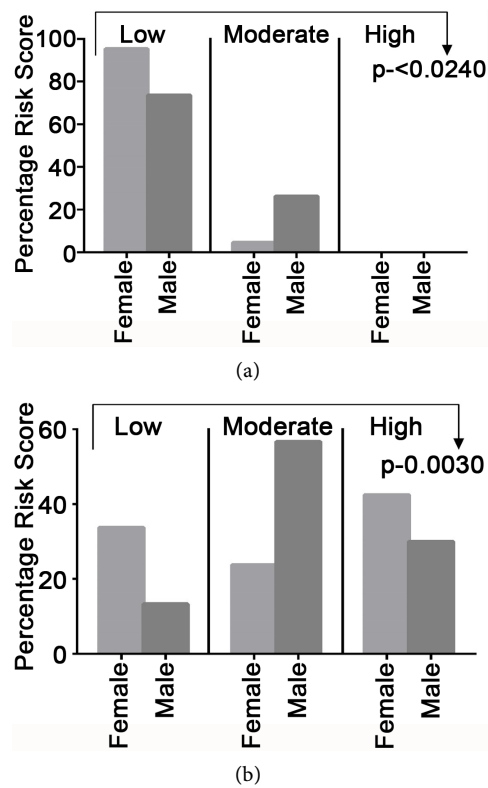


Figure 2. Relative risk scores of Framingham percentage risk of heart disease in ten years stratified by gender. (a) Control only; (b) Case only.

In general, the anthropometric parameters of the case group were significantly higher compared to the controls, the exception though was found in the height of participants where though not statistically significant the control group was averagely 0.1 meter taller than the cases. Significant intra group differences were observed among the case population with respect to both waist and hip circumferences, presenting a trend aforementioned for age. In conformity with the results of this study, earlier studies have reported significantly higher measures of obesity in type 2 diabetes and hypertension [4] [7] [16] [17] [18]. Among the case group an increase in any of the anthropometric indices (BMI, WC, HC, WHR) was associated with a corresponding increase in the haemodynamic parameters measured (SBP and DBP). Findings from previous studies have reported similar observations, a prospective study to examine the relationship between body fat distribution and 2-year incidence of hypertension and stroke demonstrated a relationship between hypertension and abdominal adiposity in a cohort of 41,837 study participants [19]. We [7], in a Pentecostal population in the Kumasi metropolis recorded a close association between hypertension and central obesity (waist circumference). Though mechanisms linking obesity to haemodynamic dysregulation are not fully understood, Kotsis, *et al.* [20] posited that during the early phases of obesity, there is sodium retention resulting from an increase in renal tubular re-absorption leading to expansion of extracellular-fluid volume; the kidney-fluid apparatus resets to a hypertensive level consistent with a model of hypertension due to volume overload.

Dyslipidaemia contributes to the process of arteriosclerosis, which develops even faster in the presence of hypertension and diabetes mellitus [6]. In the current study, with the exception of HDL-C, higher levels of unfavourable lipid profiles were recorded in type 2 diabetes and hypertension group. Our results conform to findings from studies conducted among different populations [21] [22] [23] [24] [25]. On the levels of HDL-C among the study population, the findings of the present study are consistent with three previous studies in the Ghanaian and Nigerian populations, which recorded significantly higher mean levels of HDL-C among the disease group in comparison to their control peers [8] [22] [26]. High density lipoprotein cholesterol had been considered to be cardio-protective, with efforts directed at increasing HDL-C levels to reduce the risk in those with increased risk of atherosclerosis [27]. However, emerging evidence shows that higher HDL-C levels may not necessarily result in decreased risk of cardiovascular disease but rather the functional quality of HDL-C [27] [28]. In diabetes, the functional quality of HDL-C has been found to be lower than that of healthy individuals, thus in diabetes HDL-C is unable to reverse-transport cholesterol, has impaired anti-inflammatory and anti-oxidative properties and may even be pro-arteriogenic [27] [29]. In explaining the occurrence of a better lipid profile (high HDL-C levels) among hypertensives, Owusu, *et al.* [8] suggested, higher mortality among hypertensives with unfavorable lipid profiles, leading to a lower percentage of diabetes survivors with dyslipidaemia.

The association between hypertensive parameters and glycaemia, TC and

LDL-C observed in this study were reported previously among Ghanaian diabetes mellitus patients under clinical management at the Tamale Teaching Hospital in the Northern Region of Ghana [30]. The fundamental defect in these patients is their resistance to insulin action which appears to cause hyperinsulinemia, enhanced hepatic gluconeogenesis and glucose output, reduced suppression of lipolysis in adipose tissue leading to a high free fatty acid influx and increased hepatic VLDL secretion causing hypertriglyceridemia and reduced plasma levels of HDL-C [31].

This study also sought to determine the relative risk of developing future cardiovascular events among different groups within the study population using the Framingham Risk Score [13]. All the study participants classified as having high risk of developing coronary heart disease were of the case group 49 (39.2%) (see **Figure 1(a)**). The odds of developing coronary heart disease in ten years was 4.5 times higher among the case group compared to the controls. In the STARNet study, Parchman, *et al.* [32] found 16.2% of type 2 diabetic population recording a high risk of coronary heart disease in ten years. The observed difference in the percentage ten-year coronary risk between the current study and that of the STARNet could be explained by the difference in population characteristics. Whereas the case participants in this study included type 2 diabetes and hypertension individuals, the STARNet study consisted of only persons presenting with type 2 diabetes.

The high coronary risk observed among the case group in this study could be attributable to the major unfavourable cardio-metabolic risk profiles (dyslipidaemia and hyperglycaemia) exhibited by these individuals compared to the controls [33]. The presence of these classic and putative risk factors may affect the heart (coronary artery disease), the central nervous system (cerebrovascular disease) and the lower limbs (peripheral vascular disease) [34].

Gender variations in percentage ten-year risk estimations were observed to be significantly tilted towards the female subpopulation ($p < 0.0030$) (**Figure 1(b)**, **Figure 2(a)** & **Figure 2(b)**). In a meta-analysis of 37 prospective cohort studies, Huxley, *et al.* [35] observed a significantly higher risk for fatal coronary heart disease associated with diabetes in women than in men. Women were more likely to exhibit unfavourable cardiovascular risk profiles than men [35] [36] [37]. Within the diabetes population, the female have been reported to exhibit significantly higher levels of blood pressure and lipids than their male counterparts, while the difference in these parameters among the female diabetes and non-diabetes population have been observed to be significantly greater than those observed for the male with diabetes and non-diabetes [35].

5. Conclusion

Our study demonstrates a significant association with obesity, dyslipidaemia with Ghanaian Type 2 diabetes and hypertensives. The risk of developing future cardiovascular event was high among the case participants. The preponderance of developing future cardiovascular events was higher among the female, a phe-

nomenon which may be mediated by body adiposity and dyslipidaemia.

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Conflict of Interests

Authors have declared that no conflict of interests exists.

Authors Contributions

This work was carried out in collaboration between all authors. Authors WKBAO, SYL, JOY, CO and MTAF conceptualized and designed the study. Authors JOY and SYL recruited the study participants. Authors JOY, SYL and CO generated the data. Authors JOY, SYL and WKBAO analyzed the data and drafted the manuscript. Authors WKBAO, SYL, JOY, CO and MTAF reviewed the manuscript for intellectual content and each author approved the final manuscript.

References

- [1] De-Graft, A.A. (2007) Ghana's Neglected Chronic Disease Epidemic: A Developmental Challenge. *Ghana Medical Journal*, **41**, 154-159.
- [2] Bosu, W.K. (2010) Epidemic of Hypertension in Ghana: A Systematic Review. *BMC Public Health*, **10**, 418. <https://doi.org/10.1186/1471-2458-10-418>
- [3] Halperin, R.O., Sesso, H.D., Ma, J., Buring, J.E., Stampfer, M.J. and Gaziano, J.M. (2006) Dyslipidemia and the Risk of Incident Hypertension in Men. *Hypertension*, **47**, 45-50. <https://doi.org/10.1161/01.HYP.0000196306.42418.0e>
- [4] Deshmukh, P., Gupta, S., Dongre, A., *et al.* (2006) Relationship of Anthropometric Indicators with Blood Pressure Levels in Rural Wardha. *Indian Journal of Medical Research*, **123**, 657.
- [5] Micah, F. and Nkum, B. (2012) Lipid Disorders in Hospital Attendants in Kumasi, Ghana. *Ghana Medical Journal*, **46**, 14-21.
- [6] Eghan, B. and Acheampong, J.W. (2003) Dyslipidemia in Outpatients at General Hospital in Kumasi, Ghana: Cross-Sectional Study. *Croatian Medical Journal*, **44**, 576-578.
- [7] Owiredu, W., Adamu, M., Amidu, N., *et al.* (2008) Obesity and Cardiovascular Risk Factors in a Pentecostal Population in Kumasi-Ghana. *Journal of Medical Science*, **8**, 682-690. <https://doi.org/10.3923/jms.2008.682.690>
- [8] Owusu, I., Aryee, C., Owiredu, W., Osei-Yeboah, J., Owusu-Dabo, E. and Laing, E. (2015) Analysis of Atherogenic and Anthropometric Profiles of Normotensive and Hypertensive Ghanaians in the Kumasi Metropolis. *British Journal of Medicine and Medical Research*, **7**, 378-397. <https://doi.org/10.9734/BJMMR/2015/14308>
- [9] Kirkendall, W.M., Burton, A.C., Epstein, F.H. and Freis, E.D. (1967) Recommendations for Human Blood Pressure Determination by Sphygmomanometers. *Circulation*, **36**, 980-988. <https://doi.org/10.1161/01.CIR.36.6.980>
- [10] Bannerman, E., Miller, M.D., Daniels, L.A., *et al.* (2002) Anthropometric Indices predict Physical Function and Mobility in Older Australians: The Australian Lon-

- itudinal Study of Ageing. *Public Health Nutrition*, **5**, 655-662.
<https://doi.org/10.1079/PHN2002336>
- [11] Friedewald, W.T., Levy, R.I. and Fredrickson, D.S. (1972) Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, without Use of the Preparative Ultracentrifuge. *Clinical Chemistry*, **18**, 499-502.
- [12] DeLong, D.M., DeLong, E.R., Wood, P.D., Lippel, K. and Rifkind, B.M. (1986) A Comparison of Methods for the Estimation of Plasma Low- and Very Low-Density Lipoprotein Cholesterol: The Lipid Research Clinics Prevalence Study. *JAMA*, **256**, 2372-2377. <https://doi.org/10.1001/jama.1986.03380170088024>
- [13] Brindle, P., Jonathan, E., Lampe, F., *et al.* (2003) Predictive Accuracy of the Framingham Coronary Risk Score in British Men: Prospective Cohort Study. *BMJ*, **327**, 1267. <https://doi.org/10.1136/bmj.327.7426.1267>
- [14] Lansky, A.J., Mintz, G.S., Popma, J.J., *et al.* (1998) Remodeling after Directional Coronary Atherectomy (with and without Adjunct Percutaneous Transluminal Coronary Angioplasty): A Serial Angiographic and Intravascular Ultrasound Analysis from the Optimal Atherectomy Restenosis Study. *Journal of the American College of Cardiology*, **32**, 329-337. [https://doi.org/10.1016/S0735-1097\(98\)00245-9](https://doi.org/10.1016/S0735-1097(98)00245-9)
- [15] The DECODA Study Group (2003) Age- and Sex-Specific Prevalence of Diabetes and Impaired Glucose Regulation in 11 Asian Cohorts. *Diabetes Care*, **26**, 1770-1780. <https://doi.org/10.2337/diacare.26.6.1770>
- [16] Pai, J.K., Pischon, T., Ma, J., *et al.* (2004) Inflammatory Markers and the Risk of Coronary Heart Disease in Men and Women. *New England Journal of Medicine*, **351**, 2599-2610. <https://doi.org/10.1056/NEJMoa040967>
- [17] Hu, F.B., Meigs, J.B., Li, T.Y., Rifai, N. and Manson, J.E. (2004) Inflammatory Markers and Risk of Developing Type 2 Diabetes in Women. *Diabetes*, **53**, 693-700. <https://doi.org/10.2337/diabetes.53.3.693>
- [18] Marques-Vidal, P., Schmid, R., Bochud, M., *et al.* (2012) Adipocytokines, Hepatic and Inflammatory Biomarkers and Incidence of Type 2 Diabetes. The CoLaus Study. *PLoS ONE*, **7**, e51768. <https://doi.org/10.1371/journal.pone.0051768>
- [19] Selby, J.V., Friedman, G.D. and Quesenberry, C.P. (1989) Precursors of Essential Hypertension: The Role of Body Fat Distribution Pattern. *American Journal of Epidemiology*, **129**, 43-53. <https://doi.org/10.1093/oxfordjournals.aje.a115123>
- [20] Kotsis, V., Stabouli, S., Papakatsika, S., Rizos, Z. and Parati, G. (2010) Mechanisms of Obesity-Induced Hypertension. *Hypertension Research*, **33**, 386-393.
- [21] Pitsavos, C., Tampourlou, M., Panagiotakos, D.B., *et al.* (2007) Association between Low-Grade Systemic Inflammation and Type 2 Diabetes Mellitus among Men and Women from the ATTICA Study. *The Review of Diabetic Studies*, **4**, 98. <https://doi.org/10.1900/RDS.2007.4.98>
- [22] Agrawal, Y., Goyal, V., Chugh, K., Shanker, V. and Singh, A.A. (2014) Types of Dyslipidemia in Type 2 Diabetic Patients of Haryana Region. *Scholars Journal of Applied Sciences*, **2**, 1385-1392.
- [23] Idogun, E., Unuigbo, E., Ogunro, P., Akinola, O. and Famodu, A. (2007) Assessment of Serum Lipids in Nigerians with Type 2 Diabetes Mellitus Complications. *Pakistan Journal of Medical Sciences*, **23**, 708.
- [24] Gordon, L., Ragoobirsingh, D., Morrison, E., McGrowder, D., Choo-Kang, E. and Martorell, E. (2010) Dyslipidaemia in Hypertensive Obese Type 2 Diabetic Patients in Jamaica. *Archives of Medical Science*, **6**, 701-708. <https://doi.org/10.5114/aoms.2010.17084>
- [25] Idemudia, J. (2014) Dyslipidaemia in Hypertensives in South-South Nigeria. *British*

Journal of Medicine and Medical Research, **4**, 4742-4750.

<https://doi.org/10.9734/BJMMR/2014/10678>

- [26] Nyarko, A., Adubofour, K., Ofei, F., Kpodonu, J. and Owusu, S. (1997) Serum Lipid and Lipoprotein Levels in Ghanaians with Diabetes Mellitus and Hypertension. *Journal of the National Medical Association*, **89**, 191.
- [27] Acquah, S., Boampong, J.N., Adusu, J., Achampong, E.K., Setorglo, J. and Obiri-Yeboah, D. (2012) Lipid and Lipoprotein Levels in Type 2 Diabetes Patients Attending the Central Regional Hospital in the Cape Coast Metropolis of Ghana. *International Journal of Health Research*, **4**, 75-82.
- [28] Rader, D.J. (2012) High-Density Lipoprotein Particle Number: A Better Measure to Quantify High-Density Lipoprotein? *Journal of the American College of Cardiology*, **60**, 517-520. <https://doi.org/10.1016/j.jacc.2012.03.058>
- [29] Kastelein, J.J., van Leuven, S.I., Burgess, L., *et al.* (2007) Effect of Torcetrapib on Carotid Atherosclerosis in Familial Hypercholesterolemia. *New England Journal of Medicine*, **356**, 1620-1630. <https://doi.org/10.1056/NEJMoa071359>
- [30] Titty, F. (2010) Glycaemic Control, Dyslipidaemia and Metabolic Syndrome among Recently Diagnosed Diabetes Mellitus Patients in Tamale Teaching Hospital, Ghana. *West African Journal of Medicine*, **29**, 8-11. <https://doi.org/10.4314/wajm.v29i1.55946>
- [31] Avramoglu, R.K., Basciano, H. and Adeli, K. (2006) Lipid and Lipoprotein Dysregulation in Insulin Resistant States. *Clinica Chimica Acta*, **368**, 1-19.
- [32] Parchman, M.L., Zeber, J.E., Romero, R.R. and Pugh, J.A. (2007) Risk of Coronary Artery Disease in Type 2 Diabetes and the Delivery of Care Consistent with the Chronic Care Model in Primary Care Settings: A STARNet Study. *Medical Care*, **45**, 1129-1134. <https://doi.org/10.1097/MLR.0b013e318148431e>
- [33] Grundy, S.M., Pasternak, R., Greenland, P., Smith, S. and Fuster, V. (1999) Assessment of Cardiovascular Risk by Use of Multiple-Risk-Factor Assessment Equations: A Statement for Healthcare Professionals from the American Heart Association and the American College of Cardiology. *Journal of the American College of Cardiology*, **34**, 1348-1359. [https://doi.org/10.1016/S0735-1097\(99\)00387-3](https://doi.org/10.1016/S0735-1097(99)00387-3)
- [34] Kengne, A.P., Amoah, A.G. and Mbanya, J.-C. (2005) Cardiovascular Complications of Diabetes Mellitus in Sub-Saharan Africa. *Circulation*, **112**, 3592-3601. <https://doi.org/10.1161/CIRCULATIONAHA.105.544312>
- [35] Huxley, R., Barzi, F. and Woodward, M. (2006) Excess Risk of Fatal Coronary Heart Disease Associated with Diabetes in Men and Women: Meta-Analysis of 37 Prospective Cohort Studies. *BMJ*, **332**, 73-78. <https://doi.org/10.1136/bmj.38678.389583.7C>
- [36] Wingard, D.L., Barrett-Connor, E.L. and Ferrara, A. (1995) Is Insulin Really a Heart Disease Risk Factor? *Diabetes Care*, **18**, 1299-1304. <https://doi.org/10.2337/diacare.18.9.1299>
- [37] Fuller, J., Keen, H., Jarrett, R., *et al.* (1979) Haemostatic Variables Associated with Diabetes and Its Complications. *British Medical Journal*, **2**, 964. <https://doi.org/10.1136/bmj.2.6196.964>



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