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Cardio-Metabolic Risk Profile of a Diabetic Population in the Ho Municipality

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: This study aimed to determine the burden of cardio-metabolic risk factors among type 2 diabetes clients undergoing clinical management at the Ho Municipal Hospital in the Volta Region of Ghana.
Methodology: A hospital-based, cross-sectional study was conducted among sixty-two (62) individuals presenting with type 2 diabetes at the Diabetic Clinic from November 2017 to February 2018. The participants aged between 20-60 years were purposively recruited. Demographic data was captured using a semi-structured questionnaire. Anthropometric, haemodynamic and other laboratory variables were obtained using standard methods.
Results: The prevalence of hypertension and prehypertension was 16.1% and 51.6% respectively. About 33.8% of respondents were overweight and 17.7% obese. Raised Total Cholesterol (TC), Triglycerides (TG), Low-Density Lipoprotein cholesterol (LDL-C), Very Low-Density Lipoprotein cholesterol (VLDL-C), and low High-Density Lipoprotein cholesterol (HDL-C) were 69.4%, 35.5%, 72.6%, 3.2%, and 17.7% respectively. Obesity was higher among the females (24.3%) compared to their male counterparts (8.0%). There was a significant association of waist circumference (central adiposity) with systolic blood pressure and atherogenic lipid parameters among study participants.
Conclusion: The burden of cardio-metabolic risk factors is high among type 2 diabetes individuals at the Ho Municipal Hospital. Overweight, prehypertension and raised LDL-C were the predominant risk factors. The cardio-metabolic dysregulation may be mediated by adiposity and dyslipidaemia. We recommend that individuals with high risk profiles are identified for rigorous management to delay or prevent any fatal outcome.

Keywords: Type 2 diabetes, hypertension, cardiovascular risk, dyslipidaemia, obesity.

1. BACKGROUND

Type 2 diabetes also known as diabetes mellitus type 2 is a long-term metabolic disorder that is characterised by hyperglycaemia, insulin resistance, and relative lack of insulin [1]. The prevalence of diabetes mellitus in adults worldwide was estimated at 4.0% in 1995 and is predicted to rise to 5.4% by the year 2025 such that the number of adults diagnosed with diabetes mellitus in the world would rise from 135 million in 1995 to 300 million by 2025 [2].

In Africa, more than 16 million people were living with diabetes, with 518,400 cases diagnosed in Ghana alone, constituting a prevalence of 3.6% among the adult population in the year 2017 [3]. Type 2 diabetes is associated with certain cardio-metabolic risk profiles including obesity (especially central obesity), hyperglycaemia (or raised fasting glucose), hypertension and abnormal lipid metabolism (raised triglycerides and lowered High-Density Lipoprotein Cholesterol (HDL-C) [4, 5]. The clustering of these cardio-metabolic risk profiles known as Metabolic Syndrome (MetS) has been reported in various populations and its prevalence has increased over the past decades [6]. Cardiovascular complications contribute to morbidity and mortality in people suffering from type 2 diabetes mellitus [7].

With the increasing prevalence of type 2 diabetes, it is expected that the occurrence in the number of cardio-metabolic risk profiles without commensurate risk management would increase in the population. Undiagnosed and untreated risk factors could predispose individuals with type 2 diabetes mellitus to developing cardiovascular diseases in future [8]. Nonetheless, the understanding of the burden of cardio-metabolic risk profiles among chronically-ill type 2 diabetes individuals without clinical hypertension has not been adequately addressed in the current jurisdiction, Ho, in the Volta Region of Ghana. The purpose of this study is to estimate the prevalence of cardio-metabolic risk profiles among type 2 diabetes patients undergoing clinical management at the Ho Municipal Hospital. Adequate knowledge about the risk profiles among these patients would greatly influence their management and optimise care to prevent further complications.

2. MATERIALS AND METHODS

2.1 Study Design and Study Population

This was a hospital-based, cross-sectional study. A homogeneous population of sixty-two (62) non-hypertensive type 2 diabetes patients comprising 25 males and 37 females aged between 20-60 years were purposively sampled. Only registered

clients who reported at the Diabetic Clinic of the Ho Municipal Hospital for medical care and whose medical records were available for review during the study period (November 2017-February 2018) were recruited as participants.

2.2 Study Site Description

This study was carried out at the Diabetic Clinic of the Ho Municipal Hospital. It is located in the capital of the Volta Region, Ho. The health facility is the second major referral point of healthcare in the Ho Municipality.

2.3 Sample Size Determination

Using the average monthly attendance of diabetic patients (400), a total study population of 1,600 was generated for the four months study duration. Raosoft online sample size calculator (http://www.raosoft.com/samplesize.html) was used, and a recommended minimum sample of 60 participants was calculated at 95% confidence level, 10% margin of error, and a response distribution of 80%.

2.4 Socio-Demographic Data Capture (Questionnaire)

A semi-structured questionnaire was administered to obtain primary data from consented adult patients. Socio-demographic information of participants included age, gender, marital status, educational level, and occupation, personal and family history as well as lifestyle practices (alcohol consumption and smoking).

2.5 Blood Pressure Measurement

After participants had sat quietly for at least ten minutes, their blood pressures (BP) were measured using a digital fully-automated blood pressure monitor (OMRON Healthcare counting, Intelli-sense BP785, HEM-7222 counting, Australia) by a single qualified nurse.

2.6 Anthropometry Measurements

Weight was measured to the nearest 0.1 kilogram (kg) and height to the nearest 0.1 centimetres (cm) with participants standing erect back straight, heels together, and feet slightly apart using a dual purpose scale (Health O Meter Professional counting, United States). Waist circumference (to the nearest centimetre) was measured midway between the inferior angles of the ribs and the supra-iliac crests with a measuring tape. The hip circumference was measured at the level of the widest diameter around the gluteal protuberance to the nearest centimetres using a tape measure. Body mass index (BMI) was calculated by dividing weight (kg) by square of the height (m^2). The waist to hip ratio (WHR) was also calculated by dividing the waist circumference (cm) by the hip circumference (cm). Mid-Upper Arm Circumference (MUAC) was measured in centimetre (cm) at the mid-point between the tip of the shoulder and the tip of the elbow.

2.7 Other Calculated Adiposity Indices

I. Conicity Index (CI) [9]

$$\mathrm{CI} = \frac{\text{Waist Circumference (m)}}{\left[0.109 \times \sqrt{\frac{\text{Weight (Kg)}}{\text{Height (m)}}}\right]}$$

II. Abdominal Volume Index (AVI) [10]

$$\mathrm{AVI} = \frac{[2(\text{Waist C (cm)})^2 + 0.7(\text{Waist C (cm)} - \text{Hip C (cm)})^2]}{1000}$$

III. BAI-Body Adiposity Index [11]

$$\mathrm{BAI} = \frac{\text{Hip Circumference(cm)}}{[\text{Height (m)}]^{1.5}} - 18$$

IV. Visceral Adiposity Index (VAI) [12]

$$\text{Males: VAI} = \frac{\text{Waist Circumference(m)}}{36.58 + (1.89 \text{ x BMI})} X \frac{TG}{1.03} X \frac{1.31}{HDL-C}$$

$$\text{Females: VAI} = \frac{\text{Waist Circumference(m)}}{36.58 + (1.89 \text{ x BMI})} X \frac{TG}{0.81} X \frac{1.52}{HDL-C}$$

2.8 Laboratory Biochemical Assays

After an overnight fast, about 4 ml of venous blood samples were drawn from the antecubital vein with a syringe between 7 am and 10 am and about 2 ml was dispensed into a serum separator tube (yellow coloured stopper). The samples were allowed to clot at room temperature, centrifuged at 2500 revolutions per minute (rpm) for 5 minutes to obtain serum and stored at –20°C until analysis. The remaining 2 ml of whole blood samples was dispensed into a sodium fluoride tube and centrifuged also at 2500 rpm at room temperature to obtain plasma for glucose estimation. All assays were carried out at the Volta Regional Hospital Laboratory. Serum biochemistry including fasting blood glucose (FBG), total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) were measured using the Junior Selectra Pro S chemistry analyser (Netherlands). Very Low Density- Cholesterol (VLDL-C) and Low Density Lipoprotein-Cholesterol (LDL-C) were calculated using the pre-programmed Frederickson-Friedwald's formula according to the following: LDL=TC-HDL-TG/2.2, where VLDL= TG/2.2. The methods adopted for the determination of the biochemical profiles were predetermined by the reagent manufacturer (ELITech Clinical Systems, SAS, Zone Indusrrielle-61500 SEES, France).

2.10 Statistical Analysis

Data was analysed using the Statistical Package for Social Sciences version 22.0 software (SPSS Inc, Chicago, USA). Normality of all variables was tested. Continuous parametric data were expressed as mean and standard deviations. Non parametric data was expressed as median with minimum and maximum range. Categorical variables were expressed as figures and percentage in parenthesis. Gender comparisons of parameters were performed using unpaired t-tests, Mann–Whitney test, Chi square test, or Fisher exact test where appropriate. A $p < 0.05$ was considered as statistically significant for all analyses. IBM Statistical Package for the Social Sciences version 22.00 was used for data analysis (SPSS Inc., Chicago, USA; http://www.spss.com).

2.11 Ethical Issue

Ethical approval with the identification number UHAS-REC/A.5 [39] 17-18 was obtained from the Research and Ethics Committee of the University of Health and Allied Sciences. Written informed consent was also obtained from all participants.

3. RESULTS

The study comprised of 62 respondents of which 25(40.3%) were males and 37(59.7%) females. The average age of study respondents was 47.3±10.5 years with males 47.4±10.4 years and age-matched females (47.3±10.5 years). At the time of this study, majority of the respondents [37(59.7%)] were married, had attained basic education [22(35.5%)] and informally employed [36(58.1%)].

A self-report of social and moderate drinking was observed among 8(12.9%) and 3(4.8%) of study participants respectively. However, no report of heavy alcohol consumption and smoking was observed among study respondents. Dietary intakes of salt [45(72.6%)], sugar [44(71.0%)] and fat [35(56.5%)] were predominantly moderate among study participants. However, high intake of dietary salt, sugar and fat was observed among 1(1.6%), 1(1.6%), and 21(33.9%) respectively. Moreover, only 6(9.7%) of study participants were found to consume very high levels of dietary fat (Table 1).

The average weight of study participants was 67.1±12.7kg. However, the difference in the weight between the male and female participants was statistically comparable ($p=0.72$). The average WC was 84.1±12.0 cm, with the WC of the female participants (85.1±9.2 cm) insignificantly higher than their male counterparts (82.6±15.3 cm) ($p= 0.44$). Similarly, the average WHR, CI and AVI as well as VAI levels between both genders were statistically comparable. However, height, BMI, HC, and MUAC as well as BAI were significantly higher in the female respondents compared to their male counterparts (See Table 2).

As shown in Table 3, the average haemodynamic parameters systolic blood pressure (SBP) and diastolic blood pressure (DBP) were found to be insignificantly higher among the female population compared to the male population except for pulse levels where males presented a higher mean pulse than the females, though the difference was statistically comparable. Atherogenic dyslipidaemia indices were found to be higher in the female respondents than their male counterparts. However, there was no statistical difference between the two gender populations. Similarly,

statistically comparable average levels of fasting blood glucose was observed between the male and female participants (*p=0.52*), although the males presented with a higher mean fasting glucose levels (14.2±5.6 mmol/L) compared to the female participants (13.3±5.2 mmol/L) (Table 3)

Using the WHO BMI classification for obesity, 21(33.8%) of study respondents were classified as overweight and 11(17.7%) obese. Significantly, obesity was higher among the female [9 (24.3%)] compared to their male peers [2 (8.0%)] (See Table 4). Raised plasma fasting glucose levels were observed in 59(95.2%) of the study participants. Male population [24 (96.0%)] presents an increased fasting glucose level than the female counterparts [35 (94.6%)] though the difference was statistically similar (*p=0.52*). Participants who presented with dyslipidaemia were: 43(69.4%) with raised TC, 22(35.5%) with raised TG and 45(72.6%) with raised low LDL-C whiles low HDL-C as well as raised VLDL-C levels were observed among 11(17.7%) and 2(3.2%) of participants respectively. However, the difference in the proportion of male and female respondents with lipid abnormalities was statistically similar. In all, the study participants predominantly presented with at least two atherogenic scores (38.7%), followed by one

Table 1. Socio-demographic characteristics of the study population stratified by gender

Parameter	Total (%)	Male (%)	Female (%)
Total	62(100.0)	25(40.3)	37(59.7)
Age (Mean ± SD)	47.3±10.5	47.4±10.4	47.3±10.5
Marital status			
Married	37(59.7)	18(72.0)	19(51.4)
Widowed	3(4.8)	0(0.0)	3(8.1)
Single	22(35.5)	7(28.0)	15(40.5)
Educational background			
None	6(9.7)	1(4.0)	5(13.5)
Basic	22(35.5)	6(24.0)	16(43.2)
Secondary	16(25.8)	6(24.0)	10(27.0)
Tertiary	18(29.0)	12(48.0)	6(16.2)
Occupation			
None	9(14.5)	2(8.0)	7(18.9)
Formal	17(27.4)	11(44.0)	6(16.2)
Informal	36(58.1)	12(48.0)	24(64.9)
Alcohol consumption			
None	51(82.3)	17(68.0)	34(91.9)
Moderate	3(4.8)	3(12.0)	0(0.0)
Social	8(12.9)	5(20.0)	3(8.1)
Heavy	0(0.0)	0(0.0)	0(0.0)
Smoking			
No	62(100.0)	25(40.3)	37(59.7)
Sometimes	0(0.0)	0(0.0)	0(0.0)
Always	0(0.0)	0(0.0)	0(0.0)
Salt intake			
None	16(25.8)	6(24.0)	10(27.0)
Moderate	45(72.6)	18(72.0)	27(73.0)
High	1(1.6)	1(4.0)	0(0.0)
Sugar intake			
None	17(27.4)	7(28.0)	10(27.0)
Moderate	44(71.0)	18(72.0)	26(70.3)
High	1(1.6)	0(0.0)	1(2.7)
Fat intake			
Moderate	35(56.5)	18(72.0)	17(45.9)
High	21(33.9)	5(20.0)	16(43.2)
Very high	6(9.7)	2(8.0)	4(10.8)

Data is presented as figure and percentage in parenthesis

atherogenic score (22.6%), and three atherogenic scores (19.4%) as well as 5(8.1%) presented with four atherogenic scores. Only 1(1.6%) participant recorded five atherogenic scores. The difference in the atherogenic scores was significant between the male and female participants (*p=0.01*) (Table 4).

Using the Joint National Committee (JNC) VII criteria for the classification of blood pressure, 32(51.6%) and 10 (16.1%) of participants presented with prehypertension and hypertension respectively. Among participants presenting with hypertension, 8(12.9%) were classified as stage 1 hypertension and 2(3.2%) stage 2 hypertension, 2(3.2%) had hypertension with both high SBP & DBP, 3(4.8%) had hypertension with high SBP, 9(14.5%) had hypertension with high DBP, 1(1.6%) had hypertension with isolated SBP, and 7(11.3%) had hypertension with isolated DBP (Table 5).

Among the study respondents, after adjusting for gender, there was no significant association observed between the anthropometric parameters and the haemodynamic parameters except for WC which had a significant relationship with SBP. However, an inverse relationship of glycaemia was observed with anthropometric variables and adiposity indices (BMI, WC, HC, MUAC, AVI, and VAI). Also, an increase in WC was associated with a corresponding increase in the levels of TC/HDL ratio, LDL-C, and CR. Similarly, a significant additive trend was observed between VAI and TC/HDL ratio, HDL, VLDL, TG and CR (Table 6).

Table 2. Anthropometric parameters of study respondents stratified by gender

Parameter	Total	Male	Female	*p-value*
Measured anthropometry				
Weight(Kg)	67.1±12.7	66.4±9.7	67.6±14.0	0.72
Height(m)	1.6±0.1	1.7±0.1	1.6±0.1	<0.001
WC (cm)	84.1±12.0	82.6±15.3	85.1±9.2	0.44
HC (cm)	98.7±10.1	94.4±5.9	101.6±11.2	0.001
MUAC (cm)	30.3±4.0	29.1±3.4	31.1±4.2	0.04
Calculated anthropometry				
BMI(kg/m^2)	24.9±4.7	23.3±3.9	26.0±5.0	0.03
WHR	0.9±0.1	0.9±0.1	0.8±0.1	0.25
CI	1.2±0.1	1.2±0.2	1.2±0.1	0.85
AVI	7.7±2.5	8.2±2.8	7.4±2.3	0.21
BAI	29.1±5.8	25.1±4.1	31.8±5.3	<0.001
VAI	0.02 (0.01-0.03)	0.02 (0.01-0.03)	0.2 (0.14-0.26)	0.06

Data is presented as means ± standard deviation and as median with minimum and maximum range. WC– Waist Circumference, HC– Hip Circumference, WHR– Waist-to-hip ratio, BMI– Body Mass Index, MUAC– Mid Upper Arm Circumference, CI- Conicity Index, AVI- Abdominal Volume Index, BAI- Body Adiposity Index, VAI- Visceral Adiposity Index

Table 3. Haemodynamic and biochemical parameters of study population stratified by gender

Parameter	Total	Male	Female	*p-value*
SBP (mmHg)	117.6±14.7	116.4±14.4	118.3±15.1	0.62
DBP (mmHg)	74.3±10.3	74.0±10.4	74.5±10.3	0.84
Pulse (bpm)	79.5±7.4	80.7±8.8	78.6±6.4	0.32
FBG (mmol/L)	13.7±5.3	14.2±5.6	13.3±5.2	0.52
TC (mmol/L)	6.0±1.3	5.8±1.5	6.1±1.2	0.33
HDL-C (mmol/L)	1.5±0.5	1.5±0.4	1.6±0.5	0.55
LDL-C (mmol/L)	3.8±1.1	3.6±1.3	3.8±0.9	0.45
VLDL-C (mmol/L)	0.6 (0.3-2.2)	0.7(0.3-1.3)	0.6(0.3-2.2)	0.80
TG (mmol/L)	1.4(0.7-4.8)	1.5(0.3-2.9)	1.3(0.7-4.8)	0.79
TC/HDL-C	4.1±1.1	4.1±1.2	4.2±1.1	0.79
Coronary Risk	5.6±1.4	5.4±1.4	5.7±1.5	0.42

Data presented as mean ± standard deviation and as median with minimum and maximum range. TC- Total Cholesterol, TG- Triglycerides, HDL-C – High-Density Lipoprotein Cholesterol, LDL-C– Low-Density Lipoprotein Cholesterol, VLDL-C – Very Low-Density Lipoprotein Cholesterol, SBP- Systolic Blood Pressure, DBP- Diastolic Blood Pressure

Table 4. Prevalence of dyslipdaemia, obesity, and hyperglycaemia by gender

Variable	Total	Male n (%)	Female n (%)	*p-value*
Total respondents	62(100)	25(40.3)	37(59.7)	
Obesity classification				
Underweight	1(1.6)	0(0.0)	1(2.7)	
Normal	29(46.8)	15 (60.0)	14 (37.8)	
Overweight	21(33.8)	8 (32.0)	13 (35.1)	
Obesity	11(17.7)	2 (8.0)	9 (24.3)	0.03*
Raised FBG	59(95.2)	24 (96.0)	35 (94.6)	0.52
Atherogenic indices				
Raised TC	43(69.4)	14 (56.0)	29 (78.4)	0.33
Raised TG	22(35.5)	11(44.0)	11 (29.7)	0.79
Raised LDL-C	45(72.6)	15(60.0)	30(81.1)	0.41
Low HDL-C	11(17.7)	2(3.2)	9(14.5)	0.09
Raised VLDL-C	2(3.2)	0(0.0)	2(5.4)	0.80
Atherogenic scores				
None	6(9.7)	6(24.0)	0(0.0)	0.01*
One	14(22.6)	5(20.0)	9(24.3)	
Two	24(38.7)	6(24.0)	18(48.6)	
Three	12(19.4)	7(28.0)	5(13.5)	
Four	5(8.1)	1(4.0)	4(10.8)	
Five	1(1.6)	0(0.0)	1(2.7)	

*Data is presented as the frequency with the corresponding percentage in parenthesis; *indicates a significant difference. Body Mass Index (BMI), Waist Circumference (WC), Fasting Blood Glucose (FBG), Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C) Very Low-Density Lipoprotein Cholesterol (VLDL-C)*

Table 5. Prevalence of hypertension and haemodynamic presentation stratified by gender

Parameter	Total	Male	Female	*p-value*
Normal SBP < 120 and DBP < 80)	20(32.3)	8(32.0)	12(32.4)	0.92
Prehypertension (SBP 120–139 or DBP 80–89)	32(51.6)	14(56.0)	18(48.6)	
Stage 1 hypertension SBP 140–159 or DBP 90–99)	8(12.9)	2(8.0)	6(16.2)	
Stage 2 hypertension SBP ≥ 160 or DBP ≥ 100)	2(3.2)	1(4.0)	1(2.7)	
Hypertension (SBP ≥ 140 or DBP ≥ 90)	10(16.1)	3(12.0)	7(18.9)	0.73
Hypertension with both SBP & DBP (SBP ≥ 140 and DBP ≥ 90)	2(3.2)	1(4.0)	1(2.7)	1.00
Hypertension with high SBP (SBP ≥ 140)	3(4.8)	1(4.0)	2(5.4)	1.00
Hypertension with high DBP (DBP ≥ 90)	9(14.5)	3(12.0)	6(16.2)	0.73
Hypertension with isolated SBP (SBP ≥ 140 and DBP < 90)	1(1.6)	0(0.0)	1(2.7)	1.00
Hypertension with isolated DBP (SBP < 140 and DBP ≥ 90)	7(11.3)	2(8.0)	5(13.5)	0.69

Data is presented as the frequency with the corresponding percentage in parenthesis. p-value is significant at 0.05. SBP- Systolic Blood Pressure, DBP- Diastolic Blood Pressure

Table 6. Pearson bivariate correlation of cardiometabolic risk factors adjusted for gender

Parameter	BMI	WC	HC	MUAC	WHR	CI	AVI	BAI	VAI	SBP	DBP	PULSE	FBG	TC/HDL	TC	HDL	LDL	VLDL	TG	CR
BMI																				
WC	.677**																			
HC	.835**	.604**																		
MUAC	.779**	.581**	.669**																	
WHR	.151	.758**	-.058	.169																
CI	.067	.764**	.059	.106	.925**															
AVI	.548**	.827**	.360**	.428**	.705**	.642**														
BAI	.861**	.551**	.838**	.605**	.005	.060	.403**													
VAI	.147	.245	.025	.076	.263*	.179	.318*	-.003												
SBP	.159	.268*	.193	.202	.171	.204	.168	.171	-.039											
DBP	.122	.202	.135	.125	.147	.152	.091	.112	.113	.665**										
PULSE	-.158	-.192	-.222	-.088	-.078	-.143	-.216	-.189	-.062	.158	.258*									
FBG	-.326*	-.285*	-.379**	-.312*	-.057	-.094	-.288*	-.278*	-.046	-.090	-.212	.171								
TC/HDL	.243	.292*	.162	.135	.212	.159	.446**	.132	.783**	-.019	.046	-.193	-.099							
TC	.180	.276*	.086	.091	.256*	.238	.428**	.208	.057	.144	-.107	-.183	.042	.323*						
HDL	-.098	-.055	-.120	-.044	.030	.047	-.115	.011	-.605**	.147	-.053	.104	.105	-.692**	.381**					
LDL	.210	.283*	.162	.109	.205	.211	.443**	.237	.029	.109	-.133	-.259*	.004	.474**	.927**	.129				
VLDL	.147	.217	-.019	.062	.263*	.161	.351**	.027	.867**	.003	.070	-.021	.007	.618**	.332**	-.294*	.181			
TG	.147	.220	-.016	.062	.264*	.164	.351**	.028	.865**	.006	.074	-.016	.005	.614**	.332**	-.287*	.179	1.000**		
CR	.228	.281*	.135	.106	.223	.184	.402**	.188	.644**	.131	.202	-.206	-.176	.853**	.198	-.645**	.362**	.436**	.438**	

*Data are presented as the correlation coefficient of correlation. *p is significant at 0.05, **p is significant at 0.01. BMI (Body Mass Index), WC (Waist Circumference), HC (Hip Circumference), MUAC (Mid Upper Arm Circumference), WHR (waist-to-Hip Ratio), CI (Conicity Index), AVI (Abdominal Volume Index), BAI (Body Adiposity Index), VAI (Visceral Adiposity Index), SBP (Systolic Blood Pressure), DBP (Diastolic Blood Pressure), FBG (Fasting Blood Glucose), TC (Total Cholesterol), TG (Triglycerides), TC/HDL (Total Cholesterol: High-Density Lipoprotein ratio), HDL (High-Density Lipoprotein), LDL (Low-Density Lipoprotein), VLDL (Very Low-Density Lipoprotein), CR (Coronary Risk)*

5. DISCUSSION

Type 2 diabetes mellitus is a chronic and life-threatening disease, often accompanied by altered cardio-metabolic risk profiles such as abdominal obesity, hypertension and dyslipidaemia which contribute to morbidity and mortality [6]. Using the WHO BMI classification for obesity in this study, we found 33.8% of participants to be overweight and 17.7% obese. Significantly, obesity was higher among the female (24.3%) compared to their male peers (8.0%) (Table 4). One important feature of the current study is the analysis of anthropometric parameters to include less commonly used adiposity candidate markers including MUAC, CI, AVI, BAI, and VAI. Similarly, the female population significantly recorded higher mean levels of BMI, HC, MUAC, and BAI in comparison to the male population (Table 2). The 17.7% obesity recorded in our study is lower than those reported (48.15%, 69.14%, and 30.8%) in an earlier study in the same study area (Ho Municipality) using different definitive criteria [5]. Among a type 2 diabetes population in Northern Ghana, Mogre, Salifu [13] reported obesity prevalence of 77% in their study. The lower obesity rate observed in this study could be due to differences in population characteristics and the use of different definitive criteria for obesity estimation. Whiles our study employed the use of WHO BMI classification for obesity, the studies by Osei-Yeboah, Owiredu [5] and Mogre, Salifu [13] used the NCEP-ATP III, IDF and WHO classifications. Female preponderance to obesity as observed in this study has been reported among Ghanaians in previous studies [5,13,14]. The precise reason for female weight gain observed among the study participants cannot be ascertained from this study. However, it is thought to be influenced by acculturation through complex socio-cultural pathways that potentiate obesity in women in Sub-Saharan Africa [14].

In the present study, we determined the prevalence of hypertension and other haemodynamic presentations using the Joint National Committee (JNC VII) criteria [15]. Prehypertension and undiagnosed hypertension prevalence among study participants was estimated at 51.6% and 16.1% respectively. Among those presenting with hypertension, 12.9% were classified as having stage 1 hypertension and 3.2% having stage 2 hypertension (Table 5). The results of this study reveal that undiagnosed hypertension exists among type 2 diabetes population who were without symptoms consistent with clinical hypertension in the study area. Moreover, the high proportion of study participants presenting with prehypertension is equally alarming. Undiagnosed and untreated hypertension could lead to significant abnormalities in cardiac and vascular measures that can be identified as increased Left Ventricular Mass (LVM), carotid thickness, arterial stiffness and decreased diastolic function [16].

Dyslipidaemia is a well-recognised and modifiable risk factor that should be identified early to institute aggressive cardiovascular preventive measures [17]. Patients with type 2 diabetes are at greater risk of developing vascular diseases due to abnormalities in lipid parameters [18]. In the present study, 69.4% of the study respondents presented with raised TC, 35.5% with raised triglycerides and 72.6% with raised LDL-C whiles low HDL-C as well as raised VLDL-C levels were observed among 17.7% and 3.2% of participants respectively (Table 4). LDL-C, particularly, the small and dense LDL particles, are more susceptible to oxidation via mechanisms that may contribute to the formation of foam cells leading to atherosclerosis and subsequent cardiovascular disease [19].

In this study, we observed no significant association between anthropometric indices and haemodynamic parameters measured, except for WC which correlated significantly with SBP after adjusting for gender. This finding was previously described among type 2 diabetes clients at the outpatient department of a health facility in the Volta Region [20]. Among mechanisms proposed to link obesity to hypertension is the retention of sodium during the early phase of obesity resulting from increased tubular reabsorption leading to expansion of extracellular-fluid volume and resetting the kidney-fluid apparatus to a hypertensive level [21]. Visceral fat is a determinant of obesity which is directly linked with hyperglycaemia and dyslipidaemia [22]. This assertion was partly confirmed in the present study. Among the diabetic participants, the study revealed a direct association of WC (central adiposity) with levels of TC/HDL ratio, LDL-C and CR. Similarly, a significant additive trend was observed between VAI and TC/HDL ratio, HDL-C, VLDL-C, TG and CR (Table 6). The important link between obesity and dyslipidemia seems to be the development of insulin resistance in peripheral tissues leading to an enhanced hepatic flux of fatty acids from dietary sources, intravascular lipolysis and from adipose tissue

resistance to the antilipolytic effects of insulin [23].

The present study is limited by the cross-sectional nature of the study design and the relatively small sample size of study respondents, hence projections to establish any causality is limited. We recommend that future studies in the study area employ a larger sample size to authenticate the findings of this study.

6. CONCLUSION

The burden of cardio-metabolic risk factors is high among type 2 diabetes individuals at the Ho Municipal Hospital. Overweight, prehypertension and raised LDL-C were the predominant risk factors. The cardio-metabolic dysregulation may be mediated by adiposity and dyslipidaemia.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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