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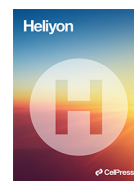
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## Research article

## The pattern of dyslipidaemia and factors associated with elevated levels of non-HDL-cholesterol among patients with type 2 diabetes mellitus in the Ho municipality: A cross sectional study



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## ABSTRACT

**Background:** Dyslipidaemia is a key comorbid condition of type 2 diabetes mellitus that increases the risk of cardiovascular disease. This study describes the pattern of dyslipidaemia and factors associated with elevated levels of non-high density lipoprotein cholesterol (HDL-C) among patients with type 2 diabetes mellitus in Ho.

**Methods:** This hospital-based cross-sectional study enrolled 210 patients with type 2 diabetes mellitus from Ho municipality. A semi-structured questionnaire was used to obtain demographic and other relevant parameters. Anthropometric, haemodynamic, and biochemical variables were obtained using standard methods. Dyslipidaemia was defined according to the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) criteria while elevated levels of non-HDL-C was defined as non-HDL-C level  $\geq 3.37$  mmol/L. A Chi-square test and multivariate logistic regression analyses were performed to determine factors associated with elevated non-HDL-C levels.

**Results:** Overall, dyslipidaemia and elevated levels of non-HDL-C prevalence was 67.1% and 64.3%, respectively. The frequency of atherogenic, isolated, and mixed dyslipidaemias were 10.5%, 58.09% and 53.33 %, respectively. Females were four times more likely to develop elevated levels of non-HDL-C after adjustment for age (AOR: 4.07; CI: 2.20–7.51;  $p < 0.0001$ ). Likewise, overweight (AOR: 3.1; CI: 1.45–6.61;  $p = 0.0035$ ), grade 1 obesity (AOR: 2.8; CI: 1.20–6.49;  $p = 0.0168$ ), and truncal obesity (AOR: 3.09; CI: 1.54–6.19;  $p < 0.0001$ ) were three times each more likely to develop elevated levels of non HDL-C after adjustment for age and gender. However, alcohol intake was 66% unlikely to develop elevated levels of non-HDL-C (COR: 0.34; CI: 0.16–0.73;  $p = 0.006$ ).

**Conclusion:** Dyslipidaemia and elevated levels of non-HDL-C were common in our study participants. Hypercholesterolaemia and co-occurrence of high TG and high LDL-C levels were the most prevalent isolated and mixed dyslipidaemias, respectively. The female gender, overweight, grade 1 obesity and truncal obesity, as well as alcohol intake were significant predictors of elevated levels of non-HDL-C.

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## 1. Introduction

Dyslipidaemia is a metabolic disorder which reflects abnormalities in plasma lipid levels. These include either one or a combination of elevated levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) or low high-density lipoprotein cholesterol (HDL-C) [1]. According to a recent report by the WHO, dyslipidaemia accounts for approximately 60% ischaemic heart disease and 40% cases of stroke worldwide [2]. Dyslipidaemia is also responsible for an estimated 4.5% of the total global mortality and 2% of the total disability-adjusted life years worldwide [3]. Currently, the African continent is experiencing a rapid increase in the prevalence of dyslipidaemia, with a pooled prevalence of 25.5% among the general population [4].

Dyslipidaemia is a common complication of diabetes mellitus. Thus, in type 2 diabetes mellitus, there are changes in lipids and lipoprotein parameters that contribute to oxidative stress, endothelial dysfunction, and ultimately leading to atherosclerosis [5, 6]. Although dyslipidaemia is such a devastating complication of type 2 diabetes mellitus, different populations may exhibit different changes in their lipid profiles owing to the variations in genetic predisposition and environmental factors [7]. For instance, the incidence of high LDL-C levels was found to be lower in non-Hispanic white men (29.4%) compared to non-Hispanic black men (30.7%) and Mexican Americans (38.8%) based on the National Health and Nutrition Examination Survey data (2011–2012). Moreover, the mean serum TG was lower whereas HDL-C and apolipoprotein A1 (Apo A1) were significantly higher among African-American women compared to white American women with prediabetes [8].

Meanwhile, the proportion of people with diabetes mellitus is increasing worldwide, including Ghana. According to the International Diabetes Federation (IDF), about 463 million adults lived with diabetes globally in 2019, and approximately, 79% of adults with diabetes mellitus were living in low- and middle-income countries [9]. In Ghana, the total number of cases of diabetes mellitus in adults aged 20–79 years was 281,100 in 2019, with a prevalence of 1.8%, while higher rates have been reported among different subpopulations, including older adults (3.95%) [10], homeless and slum dwellers (5.4%) [11] as well as peri-urban dwellers (25.2%) [12]. People with type 2 diabetes mellitus are prone to developing pro-atherogenic cardiovascular risk factors as a consequence of the inability to control blood glucose and the presence of atherogenic dyslipidemia [13]. Moreover, non-HDL-C which represents all cholesterol present in potentially atherogenic lipoprotein particles namely, LDL-C, Lipoprotein a, very-low density lipoprotein-cholesterol (VLDL-C), VLDL remnant and intermediate-density lipoprotein (IDL-C) in serum is fast gaining reputation as a predictor of cardiovascular disease in comparison with other lipid parameters [14]. Previous reports have indicated that patients who achieved the LDL-C management target had residual risk of recurrent coronary artery disease [15] suggesting that there is more to achieving optimal LDL-C levels. Thus, the World Health Organization recommends strategies to reduce the incidence of CVD, including reducing the risk factors, developing standards of care, and improving the capacity of the health system to care for patients, as well as monitor disease patterns [16]. However, health system resource constraints and inadequate surveillance capacity to identify high-risk populations remain barriers to effective CVD control in low-and middle-income countries [17].

Till date, there is no comprehensive literature on the characteristic pattern of dyslipidaemia among Ghanaians, and in particular, patients with type 2 diabetes mellitus in the Volta Region, although a few reports of high dyslipidaemia prevalence have been documented previously in the Ho municipality [18, 19]. Besides, understanding the dynamics in dyslipidaemia patterns could inform appropriate clinical interventions in mitigating adverse events of cardiovascular origin and associated complications. Hence, in this study, we provide a comprehensive analysis on the frequency and pattern of dyslipidaemia as well as possible risk factors associated with elevated levels of non-HDL-C among patients with type 2 diabetes mellitus in the Ho municipality.

## 2. Materials and methods

### 2.1. Study design and study site

This hospital-based cross sectional study was carried out at the diabetic clinic of the Ho Municipal Hospital located in Ho, the capital of Volta Region.

### 2.2. Study population and sampling technique

The study conveniently sampled previously diagnosed adult patients with type 2 diabetes mellitus aged 20 years and above, on anti-diabetic medications for at least three (3) months, those ambulatory, without chronic complications such as kidney failure, stroke and heart failure etc., without microvascular complications including nephropathy, retinopathy etc as well as participants who had the will to participate in the study. However, patients with type 2 diabetes mellitus who were too ill to participate, those who were pregnant, less than 3 months on antidiabetic medication, or those on statins or other lipid-lowering therapy were excluded from the study.

### 2.3. Sample size calculation

Using the Raosoft online sample calculator ([http://www.raosoft.com/sample\\_size.html](http://www.raosoft.com/sample_size.html)), a minimum recommended sample size of 197 was calculated from a population of 400 patients with type 2 diabetes mellitus who regularly attended the Diabetic Clinic, at 95% confidence interval, with a 5% margin of error. However, a total of 210 respondents were recruited into this study.

### 2.4. Data collection

A semi-structured questionnaire was administered to participants to obtain information on demographic profile (age, sex), socioeconomic profile (educational level) and cardio-metabolic risk factors (smoking, level of physical activity, salt, fat and sugar, intake). Blood pressure was measured under standard conditions using a mercury sphygmomanometer and a stethoscope with the subject in a sitting position. Two measurements of blood pressure were made at 5-minute intervals by trained personnel to ensure accuracy. The average of the measurements was used for the analysis. A portable scale was used to measure the weight of the participants in light clothing and without shoes. Height was measured with a stadiometer with participant standing erect, back straight, heels together with feet slightly spread. Hip circumference (HC) was measured as the maximal circumference over the hip at the level of the widest diameter around the gluteal protuberance in centimeters. Body mass index (BMI) was calculated as weight in kilograms divided by height in meter squared. The waist-hip ratio (WHR) was calculated by dividing the value of the waist circumference (cm) by the hip circumference (cm). The waist-height ratio was calculated by dividing the waist circumference (cm) by the height (cm).

About 5 ml venous blood samples were drawn from the antecubital vein after an overnight fast (8–12 h) in the morning between the hours of 7am to 10am. Two (2) ml of each sample were dispensed into fluoride oxalate tubes and the remaining 3 ml in a gel separator tube. The blood samples were centrifuged at 3000 revolutions per minutes (rpm) for 5 min at room temperature to obtain plasma and serum. Plasma was used to estimate fasting blood glucose (FBG) levels and serum lipid profile (Total Cholesterol, HDL-C, and TG). All assays were carried out on ELItech Selectra Pro Chemistry analyzer at the Clinical Chemistry Unit of the Ho Teaching Hospital Laboratory.

#### 2.4.1. Estimation of LDL-C and VLDL-C

VLDL-C and LDL-C levels were calculated based on the pre-programmed Frederickson-Friedwald's formula according to the following:  $LDL-C = TC - HDL - [TG]/2.2$ , where  $VLDL-C = [TG]/2.2$  [20].

## 2.4.2. Definition of dyslipidaemia phenotypes

**2.4.2.1. Atherogenic dyslipidaemia.** Atherogenic dyslipidemia was defined as a combination of high serum TG  $\geq 1.7$  mmol/L, high serum LDL-C  $\geq 2.6$  mmol/L and low serum HDL-C  $< 1$  mmol/L for men and  $< 1.30$  mmol/L for women [21–22].

**2.4.2.2. Mixed dyslipidaemia.** Mixed dyslipidemia was defined as a combination of the following: high TG, low LDL-C; high TG, high LDL-C; high LDL-C and low HDL-C [21].

**2.4.2.3. Isolated dyslipidaemia.** Isolated dyslipidemia was defined as isolated hypercholesterolaemia - combination of high TC and normal/low TG and LDL-C; isolated hypertriglyceridemia - combination of high TG and normal/low TC and LDL-C; isolated high LDL-C combination high LDL-C and normal/low TG, TC while isolated low HDL-C was defined as a combination of low HDL-C with normal LDL-C, TG and TC [21].

Dyslipidaemia was defined according to the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) criteria which includes raised TC levels  $\geq 5.2$  mmol/L and/or high serum TG levels  $\geq 1.7$  mmol/L and/or high serum LDL-C levels  $\geq 2.6$  mmol/L and/or low serum HDL-C levels  $< 1$  mmol/L for men and  $< 1.3$  mmol/L for women [23].

### 2.4.3. Coronary artery risk

TC/HDL-C ratio  $\leq 3.5$  was defined as low coronary risk, whereas levels  $\geq 3.5$  was defined as high coronary risk [24].

### 2.4.4. Elevated non-HDL-cholesterol

Non-HDL-C was calculated by subtracting HDL-C from the TC while elevated levels of non-HDL-C was defined as levels  $\geq 3.37$  mmol/L [23].

### 2.4.5. Body mass index

The BMI was classified according to the cut-off points proposed by the World Health Organization. Normal weight was defined as BMI values 18–24.9 kg/m<sup>2</sup>, overweight: 25–29.9 kg/m<sup>2</sup>, obesity grade 1: 30.0–34.9 kg/m<sup>2</sup>, obesity grade 2: 35.0–39.9 kg/m<sup>2</sup> and morbid obesity:  $\geq 40.0$  kg/m<sup>2</sup> [25].

### 2.4.6. Truncal obesity

Truncal obesity was defined as WC  $\geq 94$ cm for men and  $\geq 80$ cm for women [25].

### 2.4.7. Alcohol intake and smoking status

Alcohol intake and smoking status were defined as a 'yes' response to having been current or previous alcohol drinker or smoker.

### 2.4.8. Dietary salt, sugar and fat intakes

The diet intakes of salt, sugar and fat were defined as a 'yes' response to consuming moderate or high salt, sugar, and red meat which was used as a proxy of fat consumption.

## 2.5. Data analysis

Data were entered into Microsoft Excel 2016 and exported into Statistical Package for Social Science (SPSS) version 26. Normality of continuous data was tested using Kolmogorov-Smirnov test. Descriptive statistics was used to summarise continuous and categorical data. A Chi-square test analysis was used to determine variables (demographic, anthropometric, clinical, and biochemical) associated with the main outcome of the study (elevated non-HDL-C). Multiple logistic regression analysis was used to determine risk factors associated with elevated levels of non-HDL-C.

## 2.6. Ethical consideration

Ethical clearance was sought from the University of Health and Allied Sciences Research Ethics Committee (UHAS REC) with protocol identification number: UHAS-REC A.10 (36) 20–21. Informed consent was obtained from all participants. Confidentiality of data was ensured by securing the electronic data file until it was used for analysis.

## 3. Results

### 3.1. Socio-demographic and lifestyle characteristics of study participants

A total of two hundred and ten (210) respondents were recruited into this study. The majority were women [114 (54.3%)], more than 50 years [118 (118%)] and 150 (71.4%) were married. Approximately half [105 (50%)] of the total study respondents had attained basic education at the time of this study. Self-reports on alcohol intake and current smoking status were 33 (15.7%) and 3 (1.4%), respectively. Dietary fat [155 (73.8%)] and salt intakes [183 (87.1%)] were predominantly moderate, while the majority [101 (48.1%)] avoided dietary sugar intake (Table 1).

**Table 1.** Demographic and lifestyle characteristics of study participants.

Parameters	Frequency	Percentage
Total	210	100
<b>Gender</b>		
Male	96	45.7
Female	114	54.3
<b>Age (years)</b>		
20–30	4	1.9
31–40	29	13.8
41–50	59	28.1
>50	118	56.2
<b>Educational Level</b>		
None	17	8.1
Basic	105	50
Secondary	49	23.3
Tertiary	39	18.6
<b>Marital Status</b>		
Single	60	28.6
Married	150	71.4
<b>Alcohol Intake</b>		
No	177	84.3
Yes	33	15.7
<b>Smoking Status</b>		
No	207	98.6
Yes	3	1.4
<b>Dietary Fat Intake</b>		
None	36	17.1
Moderate	155	1
High	19	9.1
<b>Dietary Sugar Intake</b>		
None	101	48.1
Moderate	99	47.1
High	10	4.8
<b>Dietary Salt Intake</b>		
None	8	3.8
Moderate	183	87.1
High	19	9.1
<b>Type of treatment</b>		
Oral only	70	33.3
Oral + Insulin	140	66.7

Data presented as frequency and the corresponding proportion in parenthesis.

**Table 2.** Anthropometric, haemodynamic and biochemical parameters of study participants stratified by gender.

Parameter	Total	Male	Female	p-value
Age	49.98 ± 7.90	48.73 ± 8.53	51.03 ± 7.21	<b>0.0365</b>
Weight (kg)	74.11 ± 14.87	75.13 ± 15.29	73.23 ± 14.52	0.3584
Height (cm)	163.70 ± 11.04	170.40 ± 12.26	158.20 ± 5.49	<b>&lt; 0.0001</b>
BMI (kg/m <sup>2</sup> )	27.70 ± 5.57	25.73 ± 4.88	29.36 ± 5.59	<b>&lt; 0.0001</b>
WC (cm)	91.91 ± 12.95	89.30 ± 12.77	94.23 ± 12.83	<b>0.0064</b>
HC (cm)	100.80 ± 13.88	95.88 ± 13.55	104.90 ± 12.86	<b>&lt; 0.0001</b>
WHtR	0.56 ± 0.09	0.53 ± 0.09	0.59 ± 0.08	<b>&lt; 0.0001</b>
WHR	0.92 ± 0.08	0.94 ± 0.11	0.89 ± 0.05	<b>0.0008</b>
SBP (mmHg)	125.10 ± 21.13	125.90 ± 21.81	124.40 ± 20.61	0.6013
DBP (mmHg)	81.98 ± 14.81	85.16 ± 16.57	79.26 ± 12.58	<b>0.0047</b>
FBG (mmol/l)	12.78 ± 5.89	13.14 ± 6.59	12.47 ± 5.22	0.4260
TC (mmol/l)	5.204 ± 1.29	4.68 ± 1.03	5.64 ± 1.32	<b>&lt; 0.0001</b>
TG (mmol/l)	1.62 ± 0.74	1.53 ± 0.65	1.69 ± 0.79	0.0967
HDL-C (mmol/l)	1.39 ± 0.41	1.39 ± 0.46	1.39 ± 0.37	0.9891
LDL-C (mmol/l)	3.05 ± 1.072	2.63 ± 0.99	3.41 ± 1.00	<b>&lt; 0.0001</b>
VLDL-C (mmol/l)	1.02 ± 3.7	0.71 ± 0.31	0.81 ± 0.52	0.0932
Coronary-Risk Ratio	5.42 ± 1.65	5.10 ± 1.54	5.70 ± 1.69	<b>0.0074</b>
Non-HDL-C (mmol/l)	3.81 ± 1.247	3.29 ± 1.04	4.25 ± 1.24	<b>&lt; 0.0001</b>

Data presented as means ± standard deviations. BMI-Body Mass Index, WC-Waist Circumference, HC-Hip Circumference, WHtR-Waist-to-Height Ratio, WHR-Waist-to-Hip Ratio, SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, FBG-Fasting Blood Glucose, TC-Total Cholesterol, TG-Triglycerides, HDL-C-High Density Lipoprotein Cholesterol, LDL-C-Low Density Lipoprotein Cholesterol, VLDL-C- Very Low Density Lipoprotein Cholesterol, Non-HDL-C-Non High Density Lipoprotein Cholesterol, p-value is significant at < 0.05. The bolded p-values indicate statistical significance.

### 3.2. Anthropometric, haemodynamic and biochemical characteristics of study participants stratified by gender

The average age of the study population was 49.98 ± 7.90 years, with the female participants recording a significantly higher mean age (51.03 ± 7.21 years) compared to their male counterparts (48.73 ± 8.53 years); (p = 0.0365). Overall, the anthropometric profiles (BMI, WC, HC and WHtR) were significantly higher among the female population compared to their male peers, with the exception of the average height which was significantly higher among males compared to females. The average DBP of the male participants was significantly higher (85.16 ± 16.57mmHg) in comparison to the female participants (79.26 ± 12.58 mmHg); p = 0.0047, whereas the average SBP was found to be statistically comparable between both genders (125.90 ± 21.81 vs 124.40 ± 20.61 mmHg; p

= 0.6013). Significantly higher TC, LDL-C, and coronal risk ratios, as well as non-HDL-C levels, were observed in a higher proportion of females compared to males (Table 2).

### 3.3. Pattern of dyslipidaemia, atherogenic index and elevated levels of non-HDL-C among study participants stratified by gender

As shown in Table 3 below, dyslipidaemia was found to be present in 114 (67.1%) of the total study population, with a significant proportion of females with dyslipidaemia [100 (87.7%)] compared to males [41 (42.7)] (p < 0.0001). Atherogenic dyslipidaemia, isolated dyslipidaemias, and mixed dyslipidaemias respectively, represented 22 (10.5%), 122 (58.09%), and 112 (53.33%) of the total population. High TC [66 (31.4%)] was the most common isolated dyslipidaemia, while the co-

**Table 3.** Pattern of dyslipidaemia, atherogenic index and elevated non-HDL-C among study participants stratified by gender.

Lipid abnormality	Total (210)	Male (96)	Female (114)	p-value
<b>Dyslipidaemia</b>				
High TC and/or high LDL-C, and/or High TG, and/or Low HDL-C	141 (67.1)	41 (42.7)	100 (87.7)	<b>&lt; 0.0001</b>
<b>Atherogenic Dyslipidaemia</b>				
High TG, High LDL-C, and Low HDL-C	22 (10.5)	6 (6.3)	16 (14.0)	0.1076
<b>Isolated Dyslipidaemias</b>				
High TC	66 (31.4)	22 (22.9)	44 (38.6)	<b>0.0221</b>
High TG	22 (10.5)	14 (14.6)	8 (7.0)	0.1194
High LDL-C	21 (10.0)	9 (9.4)	12 (10.5)	0.9632
Low HDL-C	13 (6.2)	7 (7.3)	6 (5.3)	0.7488
<b>Mixed Dyslipidaemias</b>				
High TG and High LDL-C	53 (25.2)	17 (17.7)	36 (31.6)	<b>0.0319</b>
High LDL-C and low HDL-C	37 (17.6)	7 (7.3)	30 (26.3)	<b>0.0006</b>
High TG and low LDL-C	22 (10.5)	14 (14.6)	8 (7.0)	0.1194
<b>Prevalence of Elevated non-HDL-C</b>				
Elevated non-HDL-C	135 (64.3)	45 (46.9)	90 (78.9)	<b>&lt; 0.0001</b>
Atherogenic Index	74 (35.2)	31 (32.3)	43 (37.7)	0.4995

Data presented as frequency and the corresponding proportion in parentheses. TC-Total Cholesterol, TG-Triglycerides, HDL-C-High Density Lipoprotein Cholesterol, LDL-C-Low Density Lipoprotein Cholesterol, Non-HDL-C-Non High Density Lipoprotein Cholesterol, p-value is significant at < 0.05. The bolded p-values indicate statistical significance.

occurrence of high TG and LDL-C [53 (25.2%)] was the predominant mixed dyslipidaemias, with gender differences significantly tilted toward women. The prevalence of elevated levels of non-HDL-C and atherogenic index was 135 (64.3%) and 74 (35.2%), respectively, with significant female participants [90 (78.9%)] who presented elevated levels of non-HDL-C compared to males [45 (46.9%)] ( $p < 0.0001$ ).

#### 3.4. Chi-square test analysis of factors associated with elevated levels of non-HDL-C in type patients with type 2 diabetes mellitus

The Chi-square test analysis shows that factors such as gender, BMI, and WC were significantly associated with elevated levels of non-HDL-C among the study participants. However, alcohol intake was less likely to be associated with elevated levels of non-HDL-C. However, age, duration of diabetes mellitus, and blood pressure (SBP and DBP), as well as FBG, hours of work and exercise, were not significantly associated with elevated levels of non-HDL-C (Table 4).

**Table 4.** A Chi-square test analysis of factors associated with elevated non-HDL-C in the studied participants.

Variables	Total	Elevated Non-HDL-C	Normal Non-HDL-C	p-value
<b>Total</b>	210 (100.0)	135 (64.3)	75 (35.7)	
<b>Age (years)</b>				0.3737
<50	80 (39.1)	48 (60.0)	32 (40.0)	
≥50	130 (61.9)	87 (66.9)	43 (33.1)	
<b>Gender</b>				<b>&lt; 0.0001</b>
Male	96 (45.7)	45 (46.9)	51 (53.1)	
Female	114 (54.3)	90 (78.9)	24 (21.1)	
<b>Duration of diabetes (years)</b>				0.9671
Short term (<5)	111 (52.9)	71 (63.9)	40 (36.1)	
Long term (≥5)	99 (47.1)	64 (64.6)	35 (35.4)	
<b>SP (mmHg)</b>				0.3565
≤140	183 (87.1)	115 (62.8)	68 (37.2)	
>140	27 (12.9)	20 (74.1)	7 (25.9)	
<b>DP(mmHg)</b>				1.0000
≤90	175 (83.3)	112 (64.0)	63 (36.0)	
>90	35 (16.7)	23 (65.7)	12 (34.3)	
<b>BMI (kg/m<sup>2</sup>)</b>				<b>0.0008</b>
Normal (18.5–24.9)	68 (32.4)	30 (44.1)	38 (55.9)	
Overweight (25.02–29.9)	69 (32.9)	51 (73.9)	18 (26.1)	
Grade 1 obesity (30.0–34.9)	50 (23.8)	37 (74.0)	13 (26.0)	
Grade 2 obesity (35.0–39.9)	20 (9.5)	14 (70.0)	6 (30.0)	
Morbid obesity (≥40.0)	3 (1.4)	3 (100.0)	0 (0.0)	
<b>WC (cm)</b>				<b>&lt; 0.0001</b>
Normal	78 (37.1)	33 (42.3)	45 (57.7)	
Truncal obesity	132 (62.9)	102 (77.3)	30 (22.7)	
<b>Glycaemic Control (mmol/l)</b>				0.8860
FBG ≤7.0	37 (17.6)	23 (62.2)	14 (37.8)	
FBG >7.0	173 (82.4)	112 (64.7)	61 (35.3)	
<b>No. of hours spent working (stress)</b>				0.0746
1–8 h s	139 (66.2)	83 (59.7)	56 (40.3)	
>8 h s	71 (33.8)	52 (73.2)	19 (26.8)	
<b>Exercise</b>				0.9630
No	55 (26.2)	36 (65.5)	19 (34.5)	
Yes	155 (73.8)	99 (63.9)	56 (36.1)	
<b>Alcohol Intake</b>				<b>0.008</b>
No	177 (84.3)	121 (68.4)	56 (31.6)	
Yes	33 (15.7)	14 (42.4)	19 (57.6)	

Data presented as frequency and the corresponding proportion in parentheses. BMI-Body Mass Index, WC-Waist Circumference, SBP-Systolic Blood Pressure, DBP-Diastolic Blood Pressure, FBG-Fasting Blood Glucose, p-value is significant at  $< 0.05$ . The bolded p-values indicate statistical significance.

#### 3.5. Binary and multivariate logistic regression analysis of risk factors associated with elevated non-HDL-C among patients with type 2 diabetes mellitus

The logistic regression analysis of factors associated with elevated non-HDL-C levels showed that the female gender (AOR: 4.07; CI: 2.20–7.51;  $p < 0.0001$ ), being overweight (AOR: 3.1; CI: 1.45–6.61;  $p = 0.0035$ ) and having grade 1 obesity (AOR: 2.8; CI: 1.20–6.49;  $p = 0.0168$ ) and truncal obesity (AOR: 3.09; CI: 1.54–6.19;  $p < 0.0001$ ) were significantly associated with elevated levels of non-HDL-C after adjustment for age and sex, while alcohol intake (COR: 0.34; CI: 0.16–0.73;  $p = 0.006$ ) was less likely to be associated with elevated levels of non-HDL-C (Table 5).

## 4. Discussion

Due to the ever increasing economic growth and changing lifestyles in developing countries, the prevalence of abnormal serum lipid profile is increasing, particularly in populations with chronic illness and less physical activity [26]. Over the years, several studies have demonstrated that lipid abnormalities are a major metabolic feature in patients with diabetes mellitus [27, 28, 29]. Moreover, individuals with diabetes mellitus have two to four-fold excess risk of coronary artery disease [30] due in part to the presence of dyslipidaemia compared to non-diabetics. However, in Ghana, comprehensive data on the pattern of dyslipidaemia and factors associated with dyslipidaemic variables among patients with type 2 diabetes mellitus remain scarce in the literature.

In the present study, we found a high rate of dyslipidaemia [114 (67.1%)] among the study respondents (Table 3) similar to those reported previously in Ho (3–42%) [18] and Kumasi (63%) [31]. The mechanism that underlies dyslipidaemia in diabetes mellitus is complex. A previous report suggests the role of insulin resistance, leading to increased rate of lipolysis in adipocytes and flow of free fatty acids into the liver resulting in the overproduction of lipoproteins rich in triglycerides [32, 33, 34]. Another proposed mechanism includes decreased activity of endothelial bound lipoprotein lipase which is believed to delay the clearance of lipoproteins leading to their build up in circulation [35]. Notwithstanding, the rate of dyslipidaemia recorded in this study also compares with the earlier reports of Bekele, et al. [26] (65.6%) in Ethiopia and Bello-Ovosi, et al. [22] (69.3%) in Nigeria.

Invariably, diabetic dyslipidaemia manifests in circulation with characteristic patterns that include the single, combined, and mixed phenotypes. In our study, the frequency of atherogenic dyslipidaemia, isolated dyslipidaemias and mixed dyslipidaemias was 10.5%, 58.09% and 53.33%, respectively (Table 3). The pattern of dyslipidaemia observed in this study is consistent with results from different parts of Ghana [36, 37, 38], in Africa [4] and Asia [34, 39]. The existence of atherogenic dyslipidaemia is linked to apolipoprotein B level, leading to a shift of the LDL pool toward small, dense, cholesterol-ester depleted LDL, believed to be more atherogenic and a higher risk for arteriosclerotic cardiovascular disease [40, 41]. Isolated hypercholesterolaemia and the mixed occurrence of high TG and high LDL-C levels were found in 31.4% and 25.2%, respectively, of the study respondents (Table 3). Mixed dyslipidemias mainly reflect the hepatic overproduction of VLDL particles, with increases in both TG and LDL-C levels [42]. Moreover, the isolated and mixed dyslipidaemias correlate with the initiation and progression of atherosclerosis as well as other clinical consequences such as myocardial infarction, stroke, peripheral vascular disease, and heart failure [43, 44, 45]. Of note also, is the finding of gender disparity in the rate of dyslipidaemia, with a preponderance towards the females (Table 3). This could be due to the relatively weightier or obese tendencies exhibited by the female respondents in this study (Table 2). There is strong and consistent link between adiposity and altered lipid metabolism [46, 47].

Non-HDL-C defines all cholesterol present in potentially atherogenic lipoprotein particles including LDL-C, lipoprotein a, VLDL-C, VLDL

**Table 5.** Binary and multivariate logistic regression analysis of risk factors associated with elevated levels of non-HDL-C among patients with type 2 diabetes mellitus.

Parameter	COR	95% CI	p-value	AOR	95% CI	p-value
<b>Gender</b>						
Male	1					
Female	4.25	2.32–7.77	< 0.0001	4.07*	2.20–7.51	<0.0001
<b>BMI (kg/m<sup>2</sup>)</b>						
Normal (18.5–24.9)	1					
Overweight (25.0–29.9)	3.59	1.75–7.37	<b>0.0005</b>	3.1	1.45–6.61	<b>0.0035</b>
Grade 1 Obesity (30.0–34.9)	3.61	1.63–7.97	<b>0.0014</b>	2.8	1.20–6.49	<b>0.0168</b>
Grade 2 Obesity (35.0–39.9)	2.95	1.01–8.61	0.0734	1.79	0.57–5.61	0.3211
<b>WC (cm)</b>						
Normal	1					
Truncal Obesity	4.64	2.53–8.50	< 0.0001	3.09	1.54–6.19	<b>0.0015</b>
<b>Alcohol Intake</b>						
No	1			-		
Yes	0.34	0.16–0.73	<b>0.006</b>	-		

Data presented as odd-ratios and the corresponding 95% confidence intervals. COR-Crude Odd Ratio, AOR-Adjusted Odd Ratio, CI-Confidence Intervals, BMI-Body Mass Index, WC-Waist Circumference. The (\*) denotes the odd ratio value after adjustment for age only. The p-value is significant at < 0.05. The bolded p-values indicate statistical significance.

remnant and intermediate-density lipoprotein (IDL-C). In recent times, its elevation in serum has attracted much attention as a better predictor of cardiovascular risk in comparison with other lipid parameters [14]. In this study, the prevalence of elevated levels of non-HDL-C was 64.3%, with a female preponderance (78.9%) ( $p < 0.0001$ ) (Table 3). The findings are not uncommon as similar observations of high rates of elevated non-HDL-C levels were reported in Thailand [48] and Nepal [34]. Furthermore, the multivariate logistic regression analysis revealed that women were four times more likely to develop elevated levels of non-HDL-C after adjustment for age (AOR: 4.07; CI: 2.20–7.51;  $p < 0.0001$ ). Likewise, being overweight (AOR: 3.1; CI: 1.45–6.61;  $p = 0.0035$ ), having grade 1 obesity (AOR: 2.8; CI: 1.20–6.49;  $p = 0.0168$ ), and truncal obesity (AOR: 3.09; CI: 1.54–6.19;  $p < 0.0001$ ) were three times each more at risk of developing elevated levels of non-HDL-C after adjustment for age and gender (Table 5). The reason for the association between femininity and elevated levels of non-HDL-C levels is not clear, but may be linked to the hormonal changes that predispose to unfavourable lipid profiles during menopause [49]. Overweight, grade 1 obesity, and truncal obesity represent different morbid levels of adiposity, and their associations with the risk of elevated levels of non-HDL-C offer opportunities for lifestyle modifications to reduce risk or prevent the development of cardiovascular disease. In contrast, however, alcohol intake was found to be protective against elevated levels of non-HDL-C (COR: 0.34; CI: 0.16–0.73;  $p = 0.006$ ) (Table 5). This finding suggests a probable role of alcohol intake for cardiovascular risk reduction, though relationships between the two variables are said to be complex and interconnected [50]. Thus, further studies are warranted to validate the current findings and establish levels of alcohol intake that would be beneficial for cardiovascular risk modification. In agreement with this finding, earlier works have found that cardiovascular risk factors were significantly more prevalent in respondents who did not consume alcohol compared to low-to moderate-drinkers [51, 52]. A plausible mechanism to explain the positive effects mediated by moderate alcohol intake on cardiovascular disease risk include increased insulin sensitivity and increased HDL-C levels [53], a lipoprotein particle believed to be involved in the reverse cholesterol transport mechanism.

However, this study is limited by the cross-sectional study design; hence, no causal effect can be described. The dietary intake assessments including alcohol intake, and smoking status were based on self-reports, hence, the potential of re-call bias could affect the interpretation of our findings. Some antidiabetic medications are known to affect the levels of lipid in circulation hence this may affect our results. Glycaemic control

was assessed based on plasma glucose levels instead of the more sensitive HbA1c due to low funds for this research. However, the strength of our paper lies in the assessment of non-traditional lipid profile levels (non-HDL-C) and associated factors which could help explain the residual risk of cardiovascular disease among patients with type 2 diabetes mellitus.

## 5. Conclusion

The overall prevalence of dyslipidemia and elevated levels of non-HDL-C were high among the study respondents. Hypercholesterolaemia and co-occurrence of high TG and high LDL-C levels were the commonest isolated dyslipidaemia and mixed dyslipidaemias, respectively. Female gender, overweight, grade 1 obesity, and truncal obesity as well as alcohol intake, were significant predictors of elevated levels of non-HDL-C.

## Declarations

### Author contribution statement

Sylvester Yao Lokpo, Roger Laryea, James Osei-Yeboah and William K.B.A. Owiredu: Conceived and designed the experiments; Performed the experiments; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Richard K.D. Ephraim, Esther Ngozi Adejumo, Samuel Ametepe, Michael Appiah and Peter Nogo: Performed the experiments; Analyzed and interpreted the data; Wrote the paper.

Patrick Affrim, Precious Kwablah Kwadzokpui and Ohene Kwaku Abeka: Contributed reagents, materials, analysis tools or data; Wrote the paper.

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### Data availability statement

Data will be made available on request.

### Declaration of interest's statement

The authors declare no conflict of interest.

### Additional information

No additional information is available for this paper.

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