Original Article

Assessment of Dyslipidaemia and Cardiovascular Disease Risk Factors among Type 2 Diabetes Mellitus Patients Attending Kabutare District Hospital, Huye, Rwanda

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Cite as: Mapira TH , Musangwa J, Mugenga A, Umurerwa J, Ufitikirezi D, Musarurwa C. Assessment of Dyslipidaemia and Cardiovascular Disease Risk Factors among Type 2 Diabetes Mellitus Patients Attending Kabutare District Hospital, Huye, Rwanda. Rwanda J Med Health Sci. 2024;7(3): 471-484. https://dx.doi.org/10.4314/rjmhs.v7i3.8

Abstract

Background

Dyslipidaemia is a major contributing factor to the development of cardiovascular disease (CVD) in type 2 diabetic mellitus (T2DM) patients. People with T2DM are at a significantly high risk of developing dyslipidaemia which in turn is a risk factor for CVD. The objective of this study was to assess the risk factors for CVD in T2DM patients.

Methodology

A descriptive cross-sectional study was conducted on 100 T2DM patients consecutively presenting at Kabutare District Hospital, Huye District southern Rwanda. Excluded were patients on lipid lowering drugs and those with chronic renal and liver diseases.

Results

The overall prevalence of dyslipidaemia was 79% with hypoalphalipoproteinaemia (47%) being the most common and hypercholesterolaemia (29%) the least frequent. When stratified according to glycaemic control, median diastolic blood pressure was significantly higher (p=0.045) in participants whose median fasting plasma glucose (FPG) was ≥5.6 mmol/L. Median fasting triglycerides (p=0.006) and non-HDL-C (p=0.019) concentrations were significantly lower in euglycaemic participants compared to participants with median FPG ≥5.6 mmol/L. Dysglycaemia was significantly associated with dyslipidaemia status (p=0.001).

Conclusion

Effective management and monitoring of dyslipidaemia, particularly among those with poor glycaemic control, is crucial in mitigating CVD risks in this population.

Rwanda J Med Health Sci 2024;7(3):471-484

Keywords: dyslipidaemia, type 2 diabetes mellitus, cardiovascular disease, Hypoalphalipoproteinaemia and hyperbetalipoproteinaemia

Introduction

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality worldwide, and individuals with type 2 diabetes mellitus (T2DM) are at a significantly higher risk of developing CVD compared to the general population.[1] Dyslipidaemia, characterized by abnormal levels of lipids in the blood, is one of the major risk factors for CVD in T2DM patients.[2] The assessment of dyslipidaemia and other risk factors for CVD among T2DM patients is crucial for effective management and prevention of cardiovascular complications. Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and impaired insulin secretion and is associated with various complications, including CVD, which is the leading cause of death among T2DM patients.[3] The risk of CVD in T2DM patients is two to four times higher compared to individuals without diabetes.[4] Dyaslipidaemia, a common comorbidity in T2DM, is characterized by elevated levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), as well as decreased levels of high-density lipoprotein cholesterol (HDL-C). These lipid abnormalities contribute to the development of atherosclerosis and subsequent CVD events.[5]

Dyslipidaemia in type 2 diabetes mellitus (DM) can arise from a combination of genetic and metabolic abnormalities and lifestyle factors. Some of the key causes include insulin resistance, which is the hallmark of T2DM. Insulin resistance leads to decreased insulin-mediated suppression of lipolysis in adipose tissue. This results in increased free fatty acid release into the bloodstream, contributing to elevated triglyceride levels and reduced HDL cholesterol levels.[3] Obesity-related adipose tissue dysfunction and genetic factors further exacerbate this risk.[6] Polymorphisms in genes involved in lipid metabolisms such as those encoding for lipoprotein lipase, apoproteins and cholesterol transporters can also influence lipid levels thus dyslipidaemia risk.[7]

Other causes of dyslipidaemia in T2DM include dietary intake of saturated fats, physical inactivity and medications that may influence lipid metabolism.[8] Despite the known relationship between dyslipidaemia and CVD in T2DM, there is limited data on the specific prevalence and patterns of dyslipidaemia in different populations, especially in sub-Saharan Africa. The prevalence of dyslipidemia in T2DM patients varies widely across different populations. For example, a facility-based cross-sectional study in Ethiopia in 2020 reported an overall prevalence of dyslipidaemia of 81.5%.[9] In North-western Nigeria, the prevalence of dyslipidaemia in T2DM was 69.3%,[10] in South Africa was 86.7%,[11] and in Tanzania it was 45.1%.[12]

In Rwanda, a 2018 study conducted on diabetic patients at Gisenyi District Hospital reported a dyslipidaemia prevalence of 15.78% overall and 22.58% in women, which are much lower than findings from neighbouring countries [13] These findings, which are lower than those reported in other sub-Saharan and East African countries, suggest that dyslipidaemia may be under-reported or under-diagnosed in Rwanda, particularly against the backdrop of the country's growing diabetes burden. This study addresses this gap by providing updated findings on the prevalence of dyslipidaemia and other CVD risk factors among T2DM patients in Rwanda, specifically at Kabutare District Hospital. By investigating the specific lipid abnormalities and their correlation with cardiovascular outcomes, this research aimed to inform more effective interventions and public health strategies to reduce CVD morbidity and mortality in Rwanda's diabetic population.

Methods

Study Setting and Study Population

The study was conducted at Kabutare District Hospital a public institution with a diabetic clinic and is located in Huye district, southern province of Rwanda.

The study population is both male and female T2DM patients aged 20–80 years, from both rural and urban settings. The hospital is accessible to a wide range of patients including those from under-served communities thus ensuring a diverse sample population. Such diversity also helps provide insights into how different factors influence health outcomes.

Study Design and Participant Recruitment

A descriptive cross-sectional study was conducted between December 2021 and February 2022 at Kabutare District Hospital, enrolling 100 adult Type 2 Diabetes Mellitus (T2DM) patients. Participants were selected consecutively until the desired sample size was achieved. Both male and female patients, aged 20-80 years, from both rural and urban areas, were included in the study. The inclusion criteria required that T2DM patients were on either dietary control or receiving a combination of dietary control and oral hypoglycaemic agents. The exclusion criteria were for ppatients with T2DM being on anti-lipid drugs and anti-hypertensive drugs, and having chronic renal and liver diseases.

Sample Size

A priori sample size calculation was conducted using Fisher's formula described by Daniel & Cross,[14] to determine the required number of participants. Assuming a small effect size (d = 0.05) and a proportion of 0.1, we calculated that a minimum of 138 participants would be needed to achieve sufficient statistical power at an alpha level of 0.05. Based on this calculation, we aimed to recruit a total of 151 participants to account for potential attrition and ensure robust estimates but due to time constraint, 100 participants were recruited.

Data Collection Methods and Procedures

The study aimed to assess fasting lipid profiles and FPG levels in T2DM patients. Data collection was done using a pre-tested, structured questionnaire to gather information on participants' demographic characteristics and potential hyperlipidaemia risk factors, such as obesity, lipid-lowering drug use, cigarette smoking, alcohol consumption, dietary habits, and physical activity levels.

We ensured the validity and reliability of the questionnaire by conducting a pilot study to test the clarity and comprehension of the questions. Based on the feedback, we made necessary adjustments to improve the questionnaire's validity and reliability.

Physical measurements included

Body Mass Index (BMI): Participants' height and weight were measured using a stadiometer and a scale, respectively, and BMI was calculated using the formula weight (kg)/height (m^2).

Blood Pressure: Measured using a digital automatic blood pressure machine after participants had rested for five minutes.

Laboratory testing

Fasting venous blood was collected in fluoride oxalate tubes for plasma glucose analysis and into plain red top tubes serum for lipid analysis (TG, TC, HDL-C and LDL-C). For glucose analysis, plasma was immediately separated from whole blood and analysed within one hour of collection. Blood samples were collected using the standard venipuncture procedure. For lipid analysis, blood was allowed to clot and serum was harvested after centrifugation at 3000 rpm for 5 minutes. Glucose and lipids were analysed using the Humalyzer 2000 UV/VS spectrophotometer (Human Diagnostics, Wiesbaden, Germany) using reagents supplied by the instrument manufacturer. Low-density lipoprotein cholesterol (LDL-C), was calculated using the Friedewald equation.[15] In all cases, principles of good clinical laboratory practice were followed.

Briefly, plasma glucose was measured using the glucose oxidase method in which glucose was oxidised by glucose oxidase to yield gluconic acid and hydrogen peroxide. The hydrogen peroxide generated subsequently reacts with a chromogenic substrate to produce a coloured product whose intensity was measured spectrophotometrically.[16]

Triglycerides, total cholesterol and cholesterol fractions were measured using enzymatic methods. Triglycerides in the sample were hydrolysed to glycerol and fatty acids by lipase. The glycerol was phosphorylated by glycerol kinase to form glycerol-3-phosphate which was oxidised to form hydrogen peroxide which reacted with a chromogenic substrate to for a coloured compound whose intensity was measured spectrophotometrically.[17] High density lipoprotein cholesterol was measured using a homogenous method in which other lipoproteins were blocked from the reaction before determination of the cholesterol content.[18] For total cholesterol and HDL-C determination, cholesterol esterase hydrolyses cholesterol esters in the sample to free cholesterol and fatty acids. In a subsequent reaction, cholesterol oxidase catalyses the oxidation of cholesterol to form cholest-4-en-3-one and hydrogen peroxide. Peroxidase then catalyses a reaction between hydrogen peroxide and a chromogenic substrate to yield a coloured product whose intensity is measured spectrophotometrically.[19]

The American Diabetic Association criteria was used to define impaired fasting glucose as: 5.6–6.9 mmol/L; and diabetes mellitus as FPG:>7.0 mmol/L. Plasma glucose levels <5.6 mmol/L were therefore deemed euglycaemic.[20] In the current study glycaemic control was defined as FPG ≥5.6 mmol/L. Dyslipidaemia was defined as the presence of at least one or more lipid profile abnormalities from the following according to ATP III guidelines in (mmol/L); TC levels: <5.2 mmol/L were deemed desirable and TC concentrations ≥5.2mmol/L were deemed hypercholesterolaemic; TG levels: < 1.7 mmol/L were deemed desirable whilst levels≥1.7 mmol/L were deemed hypertriglyceridaemic; LDL-C levels: <2.58 mmol were deemed optimal whilst levels ≥2.58 mmol/L were deemed as hyperbetalipoproteinaemia and HDL-C levels: <1.03 mmol/L for males and <1.29 mmol/L for females were deemed hypoalphalipoproteinameic and those equal to or less than these cut-off points were deemed desirable. Hypertension was defined as systolic blood pressure (SBP) >130 mmHg and/or diastolic blood pressure (DBP) >80mmHg whilst for the body mass index (BMI), participants with BMI <18.5 were classified as underweight, 18.5–24.9 as normal and those with BMI≥ 25.0 were classified as overweight.[21]

Data analysis

Normally distributed numerical data were summarised as a mean ±standard deviation (SD) while non-normal data were summarised as median (interquartile range) (IQR). Categorical data was summarised as count (n) and proportion (%). For normally distributed data, comparisons between two groups were done using the student's t-test whilst comparisons of two or more groups were done using the ANOVA test with Bonferroni post hoc analysis. Two group comparisons for non-parametric data were achieved using the Wilcoxon Rank-Sum Test whilst the Kruskal Wallis Test with the Dunn post hoc analysis was used for the comparison of more than two groups. The two-sample Z test of proportions was used to compare the proportions of two groups whilst multiple group proportions were achieved using Fisher's exact test. In all cases of statistical comparisons, the level of significance was set at 0.05.

Ethical considerations

Ethical clearance with reference number CMHS/IRB/357/2021 was granted by the University of Rwanda College of Medicine and Health Sciences and permission to conduct the study was granted by the Kabutare District Hospital authorities. Participation in the study was on voluntary basis. Consenting patients gave written informed consent and had the right to withdraw from the study at any stage during the study period. Participant confidentiality was maintained by using unique study identification numbers and data was stored in password-protected databases accessed only by authorized study personnel. Participants with abnormal laboratory findings were referred for further treatment and management.

Results

Table 1. Sociodemographic and clinical characteristics of study participants by gender (N=100)

Variables	All Participants N=100	Females n=53	Males n=47	P Value
Age ± SD (years)	50.5±15.1	55.1±16.1	45.4±12.0	**0.001**
SBP mmHg	126(117–138)	134(119–139)	123(115–138)	0.074
DBP mmHg	78.5(72–86)	76(72–86)	89(72–85)	0.978
BMI	20.6(18.7–23.2)	20.6(18.8–22.8)	20.5(18.6–23.2)	0.975
Compliance with Recommended Diet n (%)				
No	23(23)	13(24.5)	10(21.3)	0.813
Yes	77(77)	40(75.5)	37(78.7)	
Alcohol Consumption n (%)				
No	89(89)	48(90.6)	41(87.2)	0.751
Yes	11(11)	5(9.4)	6(12.8)	
Comorbidities n (%)				
No	61(61)	27(50.9)	34(72.3)	**0.040**
Yes	39(39)	26(49.1)	13(27.7)	
Regular Physical Exercises n (%)				
No	63(63)	35(66.0)	28(59.6)	0.539
Yes	37(37)	18(34.0)	19(40.4)	
Concurrent Non-Diabetic Drugs n (%)				
No	61(61)	27(50.9)	34(72.3)	**0.040**
Yes	39(39)	26(49.1)	13(27.7)	

Key: SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; BMI=Body Mass Index; SD=Standard Deviation.

One hundred (100) T2DM participants comprising 53 (53%) females and 47 (47%) males were enrolled into the study. The ages of the participants ranged from 20 to 80 years. The sociodemographic and clinical characteristics of the participants by gender are presented in Table 1.

There was significant statistical difference in the mean age (p= 0.001) by gender with female participants being older at 55.1±16.1 years than their male counterparts at 45.4±12.0 years. A statistically significant difference by gender was also observed in the proportion of participants with comorbid conditions (p=0.04), with more females at 26 (49.1%) self-reporting comorbidities than males at 13 (27.7%).

Similarly, significantly more female participants at 26(49.1%) reported using concurrent non-diabetic drugs compared to males at 13 (27.7%) (p=0.04).

There were no statistically significant differences observed by gender in blood pressure, alcohol consumption, engaging in regular physical exercises, BMI and compliance with dietary recommendations (p>0.05). Furthermore, none of the participants smoked cigarettes and none were on lipid-lowering drugs. All participants were on anti-diabetic drugs.

The prevalence of putative risk factors for CVD was evaluated for the study population and further compared by gender. The results are presented in Table 2.

Table 2. Comparison of cardiovascular disease risk categories by gender (N=100)

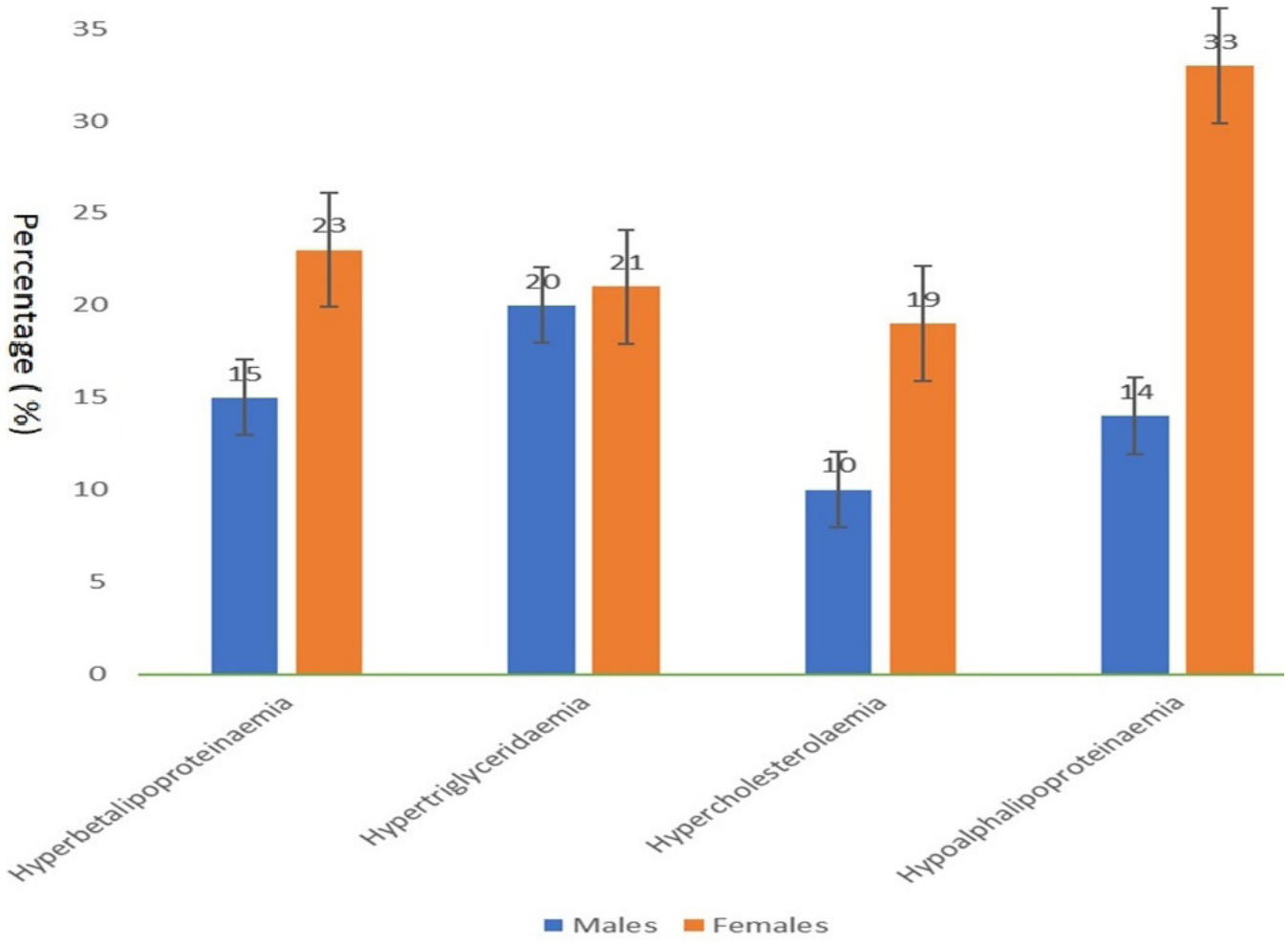
Variables	Frequency n (%)	Females n=53	Males n=47	P Value
Dyslipidaemia Status n (%)				
Dyslipidaemic	79(79)	45(84.9)	34(72.3)	0.124
Non-Dyslipidaemic	21(21)	8(15.1)	13(27.7)	
BP Category				
>130/80mmHg	52(52)	26(49.1)	27(50.9)	0.844
≤130/80 mmHg	48(48)	22(46.8)	25(53.2)	
BMI Category				
Underweight:<18.5	23(23)	13(24.5)	10(21.2)	0.328
Normal weight:18.5–25	66(66)	32(60.4)	34(72.3)	
Overweight:>25	11(11)	8(15.1)	3(6.4)	
TC Category				
Desirable <5.2mmol/L	71(71)	34(64.2)	37(78.7)	0.109
High≥5.2mmol/L	29(29)	19(35.8)	10(21.3)	
LDL-C Category				
Optimal: <2.59mmol/L	62(62)	30(56.6)	32(68.1)	0.238
High≥:2.59mmol/L	38(38)	23(43.4)	15(31.9)	
HDL-C Category				
Low: Males<1.03 Females<1.29mmol/L	47(47)	33(62.3)	14(29.8)	**0.001**
Normal: Males ≥1.03; Females ≥1.29mmol/L	53(53)	20(37.7)	33(70.2)	
TG Category				
Normal:<1.7mmol/L	59(59)	32(60.3)	27(57.5)	0.766
High ≥1.7 mmol/L	41(41)	21(39.7)	11(23.4)	
NHDL Category				
<3.37mmol/	60(60)	29(54.7)	31(66.0)	0.252
≥3.37 mmol/L	40(40)	24(45.30	16(34.0)	
FPG Category				
FPG<5.6mmol/L	20(20)	10(18.9)	10(21.3)	0.764
FPG ≥5.6mmol/L	80(80)	43(81.1)	37(78.7)	

Key: BP: Blood pressure; BMI:Body mass index; HDL-C: High density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; NHDL: Non-high density lipoprotein cholesterol; TC:Total cholesterol; TG:Triglycerides.

The overall prevalence of dyslipidaemia was 79% (n=79). Dyslipidaemia was more prevalent among female participants at 84.9% (n=45) compared to males at 72.3% (n=34) but the difference was not statistically significant (p=0.124) A statistically significant difference by gender was observed in the proportions of individuals with low HDL-C, with significantly more women (62.3%) compared to men (29.8%) having hypoalphalipoproteinaemia.

No statistically significant differences by gender were observed in any of the other putative risk factors for CVD as presented in Table 3. These included FPG, blood pressure, total cholesterol, triglycerides, and BMI, LDL-C, and NHDL categories.

The prevalence of the individual components that defined dyslipidaemia were determined for the overall study population and the results are presented in Figure 1.



Note: The error bars represent the 95% CI for each percentage value.
Figure 1. Prevalence of dyslipidaemia by individual lipid profile parameters

Table 3. Comparison of lipid profile parameters, age, and BMI and blood pressure measurements by plasma glucose status (N=100)

Variables*	Overall	FPG<5.6mmol/L n=20	FPG ≥5.6mmol/L n=80	P Value
SBP mmHg	126(117–138)	120(114.5–137.5)	128(118–138)	0.386
DBP mmHg	78.5(72–86)	73.5(65–80.5)	79.5(72-86)	**0.045**
BMI	20.6(18.7–23.2)	21.5(17.5–23.2)	20.5(18.8–23.2)	0.611
TC mmol/L	4.13(3.5–5.4)	3.79(3.42–4.23)	4.42(3.45–5.48)	0.054
HDL-C mmol/L	1.19(1.00–1.54)	1.42(1.05–1.57)	1.16(1.00–1.51)	0.330
LDL-C mmol/L	2.11(1.60 –3.24)	1.79(1.45–2.29)	2.30(1.64–3.32)	0.088
TG mmol/L	1.46(1.12–2.37)	1.19(0.95–1.37)	1.64(1.15–2.46)	**0.006**
NHDL-C mmol/L	2.87(2.23–4.16)	2.36(1.99–2.89)	3.15(2.32–4.30)	**0.019**
Age years	50.5±15.1	50.5±14.7	50.5±15.2	0.982

Key: *All median IQR except for age mean ±SD; BMI: Body mass index; HDLC: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; TC: Total cholesterol; TG triglycerides; NHDL-C: Non-High density lipoprotein cholesterol; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; SD: Standard Deviation.

With regard to the individual lipid profile parameters hypoalphalipoproteinaemia had the highest prevalence at 47% distributed as 14% in males 33% in females. On the other hand, hypercholesterolaemia had the lowest prevalence at 29% being 10% in males and 19% in females. The prevalence of hypertriglyceridaemia and hyperbetalipoproteinaemia was 41% and 38% respectively with higher proportions observed in females compared to males.

Lipid profile parameters, BMI, blood pressure measurements and age were summarised and compared by FPG categories as presented in Table 3.

A statistically significant difference was observed by glycaemic control in the median DBP with median DBP being significantly higher at 79.5 (72–86) in participants with median FPG ≥5.6 mmol/L compared to 73.5 (65–80.5) in euglycaemic individuals with median FPG<5.6 mmol/L (p=0.045). Furthermore, median fasting serum TG were significantly lower at 1.19 (0.95–1.37) mmol/L in euglycaemic participants than in participants with median FPG ≥5.6 mmol/L at 1.64 (1.15–2.46) mmol/L, (p=0.006). Non-high-density lipoprotein cholesterol (NHDL-C) concentrations were significantly lower at 2.36 (1.99–2.89) mmol/L in euglycaemic participants compared with 3.15 (2.32–4.30) mmol/L in participants with median FPG ≥5.6 mmol/L, (p=0.019). There were no statistically significant differences in mean age, median BMI, SBP, TC, HDL-C and LDL-C between participants with median FPG<5.6 mmol/L and those with FPG ≥5.6 mmol/L.

Putative risk for dyslipidaemia were compared between dyslipidaemic and normolipidaemic participants and the results are presented in Table 4.

The prevalence of dyslipidaemia only varied by glycaemic control status (p=0.001), with fasting normogycaemic participants (FPG <5.6 mmol/L) having a lower prevalence of dyslipidaemia (12.7%) compared to those with poor glycaemic control (FPG ≥5.6mmol/L) who had a dyslipidaemia frequency of 87.3%. There were no significant differences in the proportions of dyslipidaemia for the other parameters; p>0.05 in all cases.

Table 4. Risk factors for dyslipidaemia (N=100)

Variable	Dyslipidaemia Status		P -Value
	Dyslipidaemic	Normolipidaemic	
	n(%)	n(%)	
Mean age (years)*	51.2±15.7	47.9±12.2	0.373
Glycaemic control			
Good (FPG <5.6 mmol/L)	10(12.7)	10(47.6)	**0.001**
Poor (FPG ≥5.6mmol/L)	69(87.3)	11(52.4)	
Sex			
Female	45(57.0)	8(38.1)	0.145
Male	34(43.0)	13(61.9)	
BMI Status			
Underweight	16(20.3)	7(33.3)	0.118
Normal Weight	52(65.8)	14(66.7)	
Over weight	11(13.9)	0	
Hypertension Status			
Normal BP	35(44.3)	13(61.9	0.219
Hypertensive	44(55.7)	8(38.1)	
Engaging in Regular Physical Exercise			
No	52(65.8)	11(52.4)	0.312
Yes	27(34.2)	10(47.6)	
Regular Alcohol Consumption			
No	70(88.6)	19(90.5)	1.000
Yes	9(11.4)	2(9.5)	
Follows Recommended Diabetic Diet			
No	20(25.3	3(14.3)	0.388
Yes	59(74.7)	18(85.7)	

Key: *Compared using the student's t-test. Proportions compared using the Fisher's exact Chi -square test

Discussion

Dyslipidemia is a recognised complication of T2DM but the prevalence of diabetic dyslipidaemia has been reported to vary in different populations. Such variation would call for tailored and targeted interventions to reduce cardiovascular morbidity and mortality. The present study reveals an alarmingly high prevalence of dyslipidaemia (79%) in the Rwandan T2DM patients highlighting a critical need for targeted lipid management strategies to reduce CVD risk. The most prevalent individual dyslipidaemia was hypoalphalipoproteinaemia, whilst the least prevalent was hypercholesterolaemia. There were no significant differences in the prevalence of dyslipidaemia by gender. Hypoalphalipoproteinaemia was significantly more prevalent in females compared to males. Female participants were also significantly older than their male counterparts and significantly more females suffered from and were on treatment for co-morbid conditions compared to males. No significant differences were observed for other putative risk factors for CVD by gender. On the other hand, a comparison of risk factors for CVD by glycaemic control revealed that DBP, TG and NHDLC levels were significantly higher in T2DM patients with poor glycaemic control compared to euglycaemic.

The findings from the present study confirm the high prevalence of dyslipidaemia in T2DM patients and add to the body of knowledge on the subject in this resource-limited setting where there is a paucity of published data on this subject. Our findings also confirm the geographical variation in the prevalence of dyslipidaemia among T2DM. In stark contrast with findings from the present study, a previous study conducted in Kigali, Rwanda at Muhima District Hospital, reported a dyslipidaemia prevalence of 20% among T2DM patients. The commonest dyslipidaemia components in that study were hypercholesterolemia (20%) and low HDL-C (29%).[13] This prevalence is much lower compared to that obtained in the current study. This difference with the current study may be attributed to the different socioeconomic classes among the study populations, the study methodology, and the participants' lifestyles. In addition, another possible cause of the observed difference could have been the different criteria used to define dyslipidaemia. However, findings from the present study are much more concordant with findings form other studies, regionally and elsewhere.[9-11]

A study conducted at two health centres in Kenya reported a prevalence of dyslipidaemia in T2DM patients of 86.1%, which was marginally higher than the prevalence observed in the present study. [22] In concordance with our study, elevated FPG levels were associated with dyslipidaemia. However, unlike the study from Kenya, we did not find any significant associations between dyslipidaemia and BMI or with regular physical exercise. A systematic review and meta-analysis on the prevalence of dyslipidaemia in T2DM patients in Nigeria reported a prevalence of 25-97.1% with hypertriglyceridaemia and hypoalphalipoproteinaemia reported as being the most common.[23] A single centre study from south west Ethiopia reported a dyslipidaemia prevalence of 68.1% and advancing age ≥30 years, physical inactivity, being obese, hypertension, and high blood glucose value were significantly associated with diabetic dyslipidaemia in this study. [24]From Southern Africa, the overall prevalence of dyslipidaemia was reported to be 89% in T2DM patients from Western Cape, South Africa. In that study the most common dyslipidaemias were low HDL-C (65%) and hypertriglyceridaemia; 64%.[25] In summary findings from all these studies strongly suggest that people with T2DM are at a significantly high risk of developing dyslipidaemia which in turn is a risk factor for CVD.

Poorly controlled T2DM as shown by elevated FPG levels arises from a combination of factors including insulin resistance and possible genetic predispositions. Insulin resistance leads to increased lipolysis in adipose tissue thus enhanced release of free fatty acids into circulation. These fatty acids are then esterified to glycerol to form triglycerides hence hypertriglyceridaemia.[26] On the other hand, hyperglycaemia can stimulate hepatic lipogenesis and inhibit lipid oxidation, thus further exacerbating dyslipidaemia. Additionally, disturbances in insulin signalling pathways can disrupt lipid metabolism leading to abnormalities in lipoprotein synthesis and metabolism.[27] Lifestyle factors such as sedentary behaviour and poor dietary choices such as high intake of saturated fats and refined carbohydrates have also been reported to contribute to T2DM dyslipidaemia as has been co-morbid conditions such as obesity and hypertension.[28]

In the present study, we report no statistically significant differences in dyslipidaemia proportions by BMI, dietary habits and hypertension status. However, only 11% of the participants were overweight whilst only 23% reported poor dietary habits. It is possible that any underlying differences might have been masked by possible inadequate power of our study. Furthermore, the lack of association could also be explained by the complex interactions of genetic, environmental and healthcare-related factors that could possibly confound and dampen possible associations. In addition, co-morbid conditions could also confound the interplay between dyslipidaemias and putative risk factors. In the present study, the participants were only asked about any co-morbid conditions that they were suffering from but were not asked to specify the nature of such conditions. Medical conditions such as kidney and liver disease and thyroid abnormalities tend to impact lipid metabolism. Furthermore, cultural norms and socioeconomic factors might also impact the prevalence of dyslipidaemia in such participants.

Low serum HDL-C levels are almost universally reported in T2DM. The pathogenesis of low HDL-C in type 2 diabetes mellitus involves multiple factors that contribute to the dysregulation of lipid metabolism. Overall, the pathogenesis of low HDL-C in T2DM involves a complex interplay of insulin resistance, abnormal lipid profile, obesity, inflammation, glycation, oxidative stress, and medication effects.[29] The hyperglycaemia in T2DM leads to enhanced glycation of lipoproteins, including HDL-C. Glycated HDL-C is less functional and has reduced ability to remove cholesterol from peripheral tissues, resulting in low HDL-C levels. In addition, T2DM is associated with chronic low-grade inflammation and inflammatory cytokines, such as tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which can inhibit the production of HDL-C and promote its catabolism, leading to decreased levels. Increased oxidative stress as occurs in T2DM can also impair the function of HDL-C. Oxidized HDLC loses its anti-inflammatory and antioxidant properties, leading to decreased levels of functional HDL-C.[29]

Although the difference was not significant, female participants had a higher prevalence of overall dyslipidaemia compared to males and a significantly higher prevalence of low HDL-C compared to males. The latter was a surprising finding since females are generally expected to have a better serum HDL-C profile compared to males in whom low HDL-C levels generally predispose them more to a greater CVD risk compared to females.[30] The possible explanations for the observed findings include the possible use of hormone-based oral contraceptives that have been reported by some to be associated with dyslipidaemia. In addition, the serum HDL-C levels in females also change with age becoming lower post-menopausal.[31]

The population in the current study included a spectrum of pre and post-menopausal women. In addition, differences in lifestyle factors such as dietary choices

and physical activity levels could also have caused the observed differences.[28]

Strengths and Limitations of the study

The main strength of the present study was the determination of a full lipid profile, which allowed for the assessment of the prevalence of each individual dyslipidaemia component. Furthermore, the cross-sectional design allowed for determination of a snapshot of the prevalence of dyslipidaemia in the study population and an exploration of possible risk factors thereof. The novelty of our study lies in geographical and population focus since it dealt with the previously understudied population.

However, the cross-sectional design has inherent limitations revolving around temporality and causation. Furthermore, the study design is also susceptible to recall and selection bias despite our efforts to minimise the effects of such biases. Another limitation of the present study was our inability to use glycated haemoglobin for the evaluation of glycaemic control. The FPG used in the present study is subject to diurnal variation and is therefore not the best indicator of glycaemic control. Use of FPG might have caused an overestimation of the frequency of poor glycaemic control. Regardless, a study that retrospectively analysed data on 2888 patients with type 2 diabetes mellitus and compared the utility of FPG and HbA1c to determine optimal glucose control, reported that FPG could be used as an effective proxy when HbA1c determination is not available.[30]

Conclusion

Findings from the present study underscore the significant burden of dyslipidaemia among patients with T2DM in Rwanda and the findings carry important implications for both clinical practice and public health interventions. The high prevalence of dyslipidaemia highlights the urgent need for comprehensive screening and management strategies tailored specifically to individuals with T2DM.

Given the well-established link between dyslipidaemia and increased CVD risk, early detection and effective management of lipid abnormalities are paramount to reducing the risk of cardiovascular complications in this vulnerable population.

Furthermore, the findings emphasize the importance of adopting a multifaceted approach to diabetes care that addresses not only glycaemic control but also the management of associated comorbidities. Integrating lifestyle modifications, pharmacotherapy, and regular monitoring into diabetes management protocols can help mitigate the adverse cardiovascular outcomes associated with dyslipidaemia in T2DM patients.

Future research directions could explore patient-level interventions, such as personalized medicine and lifestyle modifications, to improve outcomes in patients with T2DM and dyslipidaemia. Additionally, studies could investigate healthcare system-level factors, policy interventions, and technological innovations, such as predictive models and mobile health interventions, to improve diabetes and dyslipidaemia care in Rwanda.

Conflict of interest

None

Authors' contribution

JNM, AM, JU, DU, HTM all played a role in the conception, design, interpretation, and writing of the manuscript; CM contributed with conception, data analysis, writing and editing the manuscript.

Acknowledgements

We are extremely thankful to all participants for voluntary provision of information in the study. We also thank the diabetic clinic staff and laboratory department at KABUTARE District Hospital for their collaboration and guidance. We would also like to sincerely thank the Department and staff of Biomedical Laboratory Sciences, School of Health Sciences, College of Medicine and Health Sciences, University of Rwanda, for their unwavering support.

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