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ORIGINAL RESEARCH

Glycemic Control and Cardiometabolic Risk in Black Zimbabweans with Type 2 Diabetes Mellitus

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Purpose: Type 2 diabetes mellitus (T2DM) frequently presents with modified cardiometabolic risk profiles, indicative of an elevated susceptibility to cardiovascular disease (CVD). Cardiometabolic risk factors such as obesity, hyperglycemia, hypertension, insulin resistance and dyslipidemia are known contributors to increased CVD hazard in individuals with T2DM. This study evaluated the glycemic control-based cardiometabolic risk profiles of black Zimbabweans with T2DM.

Patients and Methods: A cross-sectional study of 116 T2DM patients recruited from diabetic clinics at Parirenyatwa and Sally Mugabe Hospitals, Harare, Zimbabwe, was conducted. Blood samples were collected for glycated hemoglobin (HbA1c) and lipid profile assessment. The Framingham risk scores (FRS) based on body mass index (BMI) and lipid profile were used to determine CVD risk. Parametric variables were analyzed using one-way analysis of variance (ANOVA) with post hoc Bonferroni correction, while non-parametric variables were compared using the Kruskal–Wallis test with post hoc Dunn test for multiple comparisons.

Results: The overall frequency of dyslipidemia was 83.6% (n=97) and hypoalphalipoproteinemia was the most prevalent dyslipidemia (79.3%). Median HDLC levels were significantly lower in participants with poor glycemic control (1.12 mmol/L) compared to those with good glycemic control group (1.37 mmol/L) (p=0.011). Despite lack of significant variations in Framingham Risk Scores, there was a trend towards lower FRS-BMI in the good control group (29.8%) compared to the inadequate control (35.4%) and poor control (32.7%) groups (p=0.078).

Conclusion: Duration since DM diagnosis was observed to be an important risk factor for poor glycemic control being significantly shorter in those with good glycemic control compared to those with inadequate and poor control. Overall, there was no significant difference in HbA1c status by age but individuals with poor glycemic control were significantly older than those with good control. The most prevalent dyslipidemia among the study participants was hypoalphalipoproteinemia which is reportedly associated with genetic predisposition, warranting further investigations.

Keywords: glycated hemoglobin, dyslipidemia, hypoalphalipoproteinemia, Framingham risk score

Introduction

Diabetes mellitus (DM) is an increasingly important public health problem globally.[1] Type 2 diabetes mellitus (T2DM) is a cardiometabolic disorder characterized by hyperglycemia consequent to relative insulin deficiency and/or insulin resistance.[2] Globally, 537 million adults were living with DM in 2021 and the prevalence is projected to increase to 643 million by 2030 and to 783 million people by 2045.[3] Approximately 75% of all diabetics are resident in low- and middle-income countries of whom approximately 24 million adults living with DM are in the African region, and of these, about 54% are undiagnosed DM patients.[3] In 2015, about 850000 Zimbabweans were estimated as living with DM.[4]

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Approximately 5% of global deaths annually are attributed to DM and furthermore, T2DM is a well-known risk factor for the pathogenesis of cardiovascular diseases (CVDs).[5] Diabetes mellitus is associated with various metabolic complications which include CVD, hypertension, stroke, nephropathy, retinopathy, neuropathy, lower limb amputations among others.[6] The mainstay of T2DM treatment is optimum glycemic control achieved through dietary control of calorific intake, oral hypoglycemic agents and/or exogenous insulin injections.[7] Glycated hemoglobin (HbA1c) formed by the irreversible ketamine reaction between glucose and the N-terminal valine chain of hemoglobin A is the most widely used biomarker for the assessment of long-term glycemic status.[8] The HbA1c value reflects the weighted mean plasma glucose concentration during the preceding 2–3 months but is relatively insensitive to short-term lifestyle changes.[9] HbA1c has also been reported to be an independent risk factor for the development of coronary heart disease (CHD), stroke and other chronic metabolic complications commonly associated with poorly controlled DM.[10] The American Diabetes Association (ADA) designated an HbA1c level <7% as the optimum goal of blood glucose control in T2DM.[11]

Elevated HbA1c has been reported to be an independent risk factor for CVD in both diabetics and non-diabetic individuals with a reported 18% increased risk of CVD for each 1% rise in absolute HbA1c levels in diabetics.[12] This association is probably mediated via modulation of co-existent CVD risk factors such as dyslipidemia and hypertension.[12] The association between HbA1c and microvascular complications in diabetic individuals has been consistently reported, although the correlation between macrovascular complications and HbA1c still remains unclear.[13] Against this background, it is plausible that HbA1c could potentially be utilized as a biomarker for predicting dyslipidemia as well as CVD in T2DM patients. The correlation between HbA1C and lipid profiles, if proved to be consistent, could permit routine use of HbA1c as a dual biomarker for glycemic status and dyslipidemia in T2DM patients, thus reducing the cost of monitoring diabetics in settings with limited resources.

In T2DM, insulin secretion and/or function abnormalities and low adiponectin levels result in decreased lipoprotein lipase activity that consequently causes elevated serum low-density lipoprotein cholesterol (LDLC), high levels of serum triglycerides (TG) and low levels of high-density lipoprotein cholesterol (HDLC).[14] This dyslipidemia in T2DM patients is therefore largely attributed to both increased production and delayed catabolism of very-low-density lipoprotein (VLDL) secondary to insulin abnormalities.[2] Insulin normally inhibits the activation of hormone-sensitive lipase in adipose tissue and decreases the circulating non-esterified free fatty acids from adipocytes thus, defects in insulin secretion or function predisposes to dyslipidemias.[15] Furthermore, insulin normally stimulates the expression of LDL receptors, thus facilitating clearance of cholesterol bearing lipoproteins by peripheral tissues.[16] In addition, qualitative defects in LDLC have also been reported to occur in T2DM. These include occurrence of smaller and denser more atherogenic LDLC particles and LDLC particles modified by glycation or oxidation, both of which enhance atherogenesis.[17]

Deterioration of glycemic control in T2DM patients exacerbates lipid and lipoprotein abnormalities.[18] Dyslipidemia has been reported in 2 to 20% of T2DM patients.[19] However, reports on lipid studies among patients with DM in sub-Saharan Africa are scanty.[20] A major goal of CVD risk evaluation is to identify individuals that would benefit from CVD risk mitigation and lipid modification therapies.[21] The limited number of reports on lipid variables among individuals with T2DM in sub–Saharan Africa could be an indication that lipid profile evaluation might not be readily accessible in routine practice or perhaps that most patients in this setting may not be prone to dyslipidemias.[22] The combination of dyslipidemia, hypertension and hyperglycemia occurring in T2DM thus, creates a fertile atherogenic environment that accelerates atherosclerosis progression.[23] Optimal control of both conditions would therefore reduce CVD risk.[23,24] Diabetic dyslipidemia, if recognized early, would necessitate the institution of aggressive CVD preventive strategies in T2DM patients.[25]

Given the high prevalence of CVDs, it is crucial to identify individuals at high risk early on.[1] One of the primary methods for predicting the 10-year risk of CVD is by using the Framingham Risk Score (FRS).[2] The FRS developed by D'Agostino et al, (2008) is calculated using either a laboratory-based or a non-laboratory-based algorithm.[26] The laboratory-based model is based on age, sex, systolic blood pressure (SBP) and treatment status, current smoking, diabetes mellitus, cholesterol, and HDLC.[2] Nevertheless, in developing countries, laboratory markers may not always be available at primary healthcare centres or laboratory tests may be unaffordable to most and in such instances, the non-laboratory-based

algorithm can be used.[2] The non-laboratory-based model is based on age, sex, SBP, treatment status, current smoking, diabetes mellitus and body mass index (BMI).[3] The FRS is the most widely utilized and validated tool for evaluating the 10-year risk of CVD in individuals with no prior history of the disease. In a study by Jahangiry et al (2017) on FRS estimation in people with metabolic syndrome, it was shown that 16.3% of people were at moderate risk, and 6.3% were at high risk of developing CVDs over the next 10 years.[27] Notably, systolic and diastolic pressures have been useful predictors and are incorporated in the FRS. Other parameters, including mean arterial pressure (MAP) and pulse pressure (PP), which are not part of the FRS, have also demonstrated potential use for CVD risk prediction.[28]

Although, there is compelling evidence from elsewhere that dyslipidemia is highly prevalent among T2DM patients, there is a paucity of published studies that explore the nature of the association between T2DM, glycemic control and diabetic dyslipidemia co-morbidity in sub–Saharan Africa where phenotypic and genotypic differences might influence variations in findings. The present study explored the association between glycemic control and lipid profile parameters among Zimbabwean T2DM patients.

Materials and Methods

Study Site and Participants

In this analytical cross-sectional study, we enrolled 116 consecutive T2DM patients above 30 years of age, presenting at the weekly outpatient diabetic clinics at Sally Mugabe and Parirenyatwa Central Hospitals, Harare, Zimbabwe. Enrolment was done during the period January to June 2018. These hospitals serve as referral centers for the northern half of Zimbabwe, with the majority of patients originating from the Harare metropolis through a hierarchical referral system.

Ethical Considerations

Ethical approval was obtained from the Joint Research Ethics Committee of the University of Zimbabwe College of Health Sciences and Parirenyatwa Group of Hospitals (JREC/30/18) and Sally Mugabe Hospital Ethics Committee (HCHEC 200318/33). In accordance with the Declaration of Helsinki, all study participants provided written informed consent and were allowed to opt out of the study at any time. Both sociodemographic and laboratory data were anonymized by assigning unique study identification numbers and further kept under lock and key with access restricted to authorized personnel only.

Data Collection

A questionnaire eliciting clinico-demographic information was administered to eligible consenting patients, and additional clinical details were abstracted from the patients' medical records at the clinic. The collected data included anthropometry, blood pressure, smoking status, current medication and family medical history, essential for CVD risk profiling. Participants had their weights measured using a calibrated scale and height was determined using a right-angled stadiometer. These measurements were used to calculate the BMI by dividing the weight in kilograms (kg) by their height in meters (m) squared.

Blood pressure was measured using an upper arm blood pressure monitor (Philips Suresigns VSi, Markham, Canada) whilst the participant was seated in a relaxed position. Two readings were taken, and the average was reported as the blood pressure. Systolic and diastolic pressure readings were used to calculate mean arterial pressure (MAP) in mmHg, as the diastolic pressure plus one-third of the difference between the systolic pressure and the diastolic pressure. Subsequently, two non-fasting 5 mL whole blood samples were collected by venepuncture into ethylene diamine tetraacetic acid (EDTA) and plain tubes for HbA1c and lipid profile assays, respectively. Patients with incomplete clinical records and those suffering from self-reported kidney disease, type 1 diabetes mellitus, liver disease, cardiac disease, thyroid disease, familial dyslipidemias, taking statins, hemolytic disease or hemoglobinopathies were excluded.

Laboratory Methods

All laboratory assays were carried out using the Dimension Siemens Xpand Plus auto analyzer (Siemens Healthcare Diagnostics, Newark, USA) in the clinical biochemistry laboratory at Sally Mugabe Hospital,

Harare, Zimbabwe, using the principles of good laboratory practice. An immunoturbidimetric method was used to measure HbA1c with the result expressed as a percentage of total hemoglobin. Total hemoglobin results were used to assess the anemia status as a further exclusion criteria for patients with low hemoglobin (<12g/dl). Total cholesterol was measured using routine colorimetric methods, and both HDLC and LDLC were measured using routine direct methods. The reagents for all analyses were supplied by the auto analyzer manufacturer.

Cardiometabolic Risk Values

The 10-year CVD risk for participants was calculated using two 2018 version FRS calculators from the Framingham heart study website (https://www.framinghamheartstudy.org/fhs-risk-functions/cardiovascular-disease-10-year-risk/), which incorporates the BMI and lipid profile results. Cardiometabolic risk was evaluated using lipid profiles and Castelli's Risk Index (CRI-I), in line with NCEP ATP III guidelines,[29] and glycemic control was categorized based on %HbA1c as per ADA standards.[30] Hypertension was classified according to ADA guidelines as SBP ≥140 mmHg, DBP ≥90 mmHg, or if the participant was taking anti-hypertensive drugs,[31] and BMI was stratified into four WHO categories.[32]

Data Analysis

Categorical variables were summarized by counts and proportions, while parametric numerical data were expressed as mean ± standard deviation (SD). Non-normally distributed numerical data were summarized as median (interquartile range) (IQR). The *Z*-test for proportions, Fisher's exact and chi-square tests were used to compare proportions for categorical variables. Parametric variables were compared using the one-way analysis of variance (ANOVA) with the post hoc Bonferroni correction, and non-parametric variables were compared using the Kruskal Wallis test with the post hoc Dunn test for multiple comparisons. Statistical significance level (alpha) was set at 0.05.

Results

Clinicodemographic Characteristics

Table 1 presents clinicodemographic characteristics of 116 participants with T2DM, stratified by HbA1c status. The study participants were predominantly female 68.1% (n=79) and the majority; 59.5% (n=69) had inadequate and poor glycemic control. All the clinicodemographic variables explored were comparable across the three glycemic control groups (p>0.05). In terms of age, participants with inadequate glycemic control were the oldest; median (IQR), 65(55–70) years. In terms of sex, among males, the greatest proportion, 40.5% (n=15) were in the poor glycemic control group, whereas the majority of

Table 1 Clinicodemographic Characteristics of Participants by HbA1c Status

Variable		HbA1c Control Status n(%)			p-value
		Good (< 7%) n=47	Inadequate (7–8%) n=27	Poor (> 8%) n=42	
Age (years)	median (IQR)	59(48–69)	65(55–70)	59(49–68)	0.141
Sex	Male	14(37.9)	8(21.6)	15(40.5)	0.845
	Female	33(41.8)	19(24.0)	27(34.2)	
Age category (years)	34–44	6(42.9)	1(7.1)	7(50.0)	0.104
	45–54	15(55.6)	5(18.5)	7(25.9)	
	55–64	11(39.3)	4(14.3)	13(46.4)	
	65+	15(31.9)	17(36.2)	15(31.9)	
BMI	mean ± sd	26.6±4.2	26.7±3.8	26.8±3.9	0.834

(*Continued*)

Table 1 (Continued).

Variable		HbA1c Control Status n(%)			p-value
		Good (< 7%) n=47	Inadequate (7–8%) n=27	Poor (> 8%) n=42	
BMI category	Normal	18(43.9)	9(22.0)	14(34.1)	0.938
	Overweight	18(36.0)	13(26.0)	19(38.0)	
	Obese	11(44.0)	5(20.0)	9(36.0)	
SBP mmHg	median (IQR)	143(127–158)	144(129–157)	139(126–160)	0.402
DBP mmHg	mean ± sd	81.5±13.4	81.1±15.2	78.7±10.9	0.585
MAP	mean ± sd	102.0±14.3	102.6±13.4	100.5±13.0	0.797
Hypertension status	Yes	16(39.0)	8(19.5)	17(41.5)	0.66
	No	31(41.3)	19(25.3)	25(33.4)	
DM Duration (months)	median (IQR)	36(12–60)	84(24–120)	60(16–144)	0.007**
Smoking status	Yes	0	2(100.0)	0	0.053
	No	47(41.3)	25(21.9)	42(36.8)	
Alcohol consumption	Yes	2(100)	0	0	0.344
	No	45(39.5)	27(23.7)	42(36.8)	
Antihypertensive medication	Yes	17(54.8)	4(12.9)	10(32.3)	0.131
	No	30(35.3)	23(27.0)	32(37.7)	

Note: **p-value < 0.01.
Abbreviations: BMI, Body Mass Index; DBP, Diastolic Blood Pressure; SBP, Systolic Blood Pressure, MAP, Mean arterial pressure.

females 41.8% (n=33) were in the good glycemic control group. Overall, 64.7% (n=75) of the participants were overweight or obese, but there was no significant difference in mean BMI (p=0.834) or proportions of normal weight, overweight or obese individuals by glycemic control status (p=0.938), although the highest prevalence of obesity was observed among participants with good glycemic control; 44% (n=11). There was no significant difference in median SBP (p=0.402), mean DBP (p=0.147) or hypertension status (p=0.660) by the glycemic control groups, although patients with poor glycemic control had the highest prevalence of hypertension; 41.5% (n=17). Mean arterial pressure by HbA1c control status revealed no significant differences among the glycemic control groups (p=0.797). Individuals with good glycemic control had significantly the shortest median duration since DM diagnosis; 36(12–60) months compared to those with inadequate control; 84(24–120) months and those with poor glycemic control 60(16–144) months (p=0.007).

Dyslipidemia Distribution

The overall prevalence of dyslipidemia among the study participants was 83.6% (n=97). Low HDL-cholesterol had the highest frequency of dyslipidemia cases (79.3%), while hypercholesterolemia showed the lowest frequency (7.8%). The frequencies of elevated LDL-cholesterol and elevated non-HDLC were almost similar (Figure 1).

Cardiovascular Risk Factors

Participants with poor glycemic control (>8.0%) had a significantly higher proportion of individuals in the below average HDLC CVD Risk category (29.2%, p = 0.043) compared to the other groups. Furthermore, a significantly higher proportion of individuals with poor glycemic control 31(38.8) were classified as being at higher risk of CVD compared to the other glycemic control categories based on Framingham Risk Score-BMI (p=0.049). In contrast, the good glycemic control group had a higher prevalence of dyslipidemia (57.9%) compared to those with intermediate

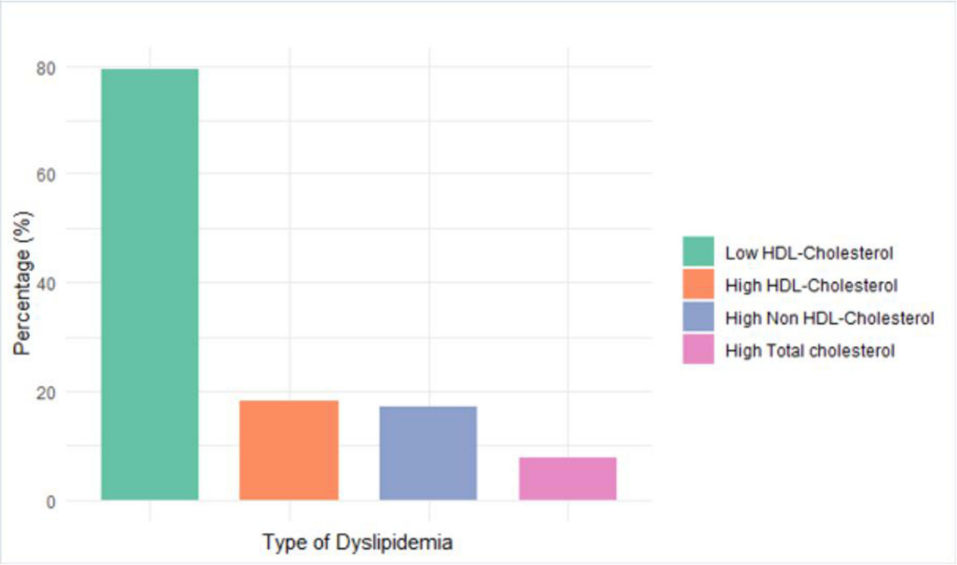

Figure 1 Distribution of Dyslipidemia Categories in Diabetes Patients.

control (10.5%) and poor control (31.6%) although the difference was not statistically significant (p=0.187). The analysis did not show significant differences in other lipid categories or Framingham Risk Score-Lipids among glycemic control groups (Table 2).

Table 2 Cardiovascular Risk Factor Categories by HbA1c Categories

Variable		HbA1c Status n(%)			p-value
		Good Control (< 7%) n=47	Inadequate Control (7–8%) n=27	Poor Control (> 8%) n=42	
Total Cholesterol CVD Risk Categories	Desirable	43(40.2)	25(23.3)	39(36.5)	0.883
	Borderline	3(42.9)	1(14.2)	3(42.9)	
	High	1(50.0)	1(50.0)	0	
LDLC Risk CVD Categories	Optimal	39(41.1)	21(22.1)	35(36.8)	0.901
	Borderline	4(30.8)	4(30.8)	5(38.4)	
	High	3(50.0)	1(16.7)	−33.3	
	Very High	1(50.0)	1(50.0)	0	
HDLC CVD Risk categories	Below Average	14(58.3)	1(2.5)	7(29.2)	0.043*
	Average Risk	15(51.7)	6(20.7)	8(27.6)	
	High Risk	18(28.6)	18(28.6)	27(42.8)	
NHDLC CVD Risk Category	Optimal	41(42.7)	22(22.9)	33(34.4)	0.507
	Near Optimal	4(26.7)	4(26.7)	7(46.6)	
	Borderline	2(50.0)	0	2(50.0)	
	High	0	1(100.0)	0	

(*Continued*)

Table 2 (Continued).

Variable		HbA1c Status n(%)			p-value
		Good Control (< 7%) n=47	Inadequate Control (7–8%) n=27	Poor Control (> 8%) n=42	
TC: HDLC Ratio CVD Risk Category	Optimum	33(45.2)	16(21.9)	24(32.9)	0.592
	Average Risk	13(35.1)	9(24.3)	15(40.5)	
	Above Average	1(16.7)	2(33.3)	3(50.0)	
Dyslipidemia	Yes	11(57.9)	2(10.5)	6(31.6)	0.187
	No	36(37.1)	25(25.8)	36(37.1)	
Framingham Risk Score-BMI	Low Risk	7(50.0)	1(7.1)	6(42.9)	0.049*
	Intermediate Risk	14(63.6)	3(13.6)	5(22.7)	
	High Risk	26(32.4)	23(28.8)	31(38.8)	
Framingham Risk Score-Lipids	Low Risk	13(52.0)	3(12.0)	9(36.0)	0.517
	Intermediate Risk	13(40.6)	8(25.0)	11(34.4)	
	High Risk	20(34.5)	16(27.6)	22(37.9)	

Note: *p-value < 0.05.
Abbreviations: CVD, cardiovascular disease; TC, Total Cholesterol; LDLC, Low Density Lipoprotein Cholesterol; HDLC, High Density Lipoprotein Cholesterol; NHDLC, Non-High Density Lipoprotein Cholesterol; FRS_BMI, Framingham Risk Score incorporating Body Mass Index; FRS-Lipids, Framingham Risk Score incorporating lipid profiles.

Lipid Profiles and Framingham Risk Scores by Glycemic Control Groups

HDLC levels were significantly lower in patients in the poor glycemic control group (1.12 mmol/L) compared to the good glycemic control group (1.37 mmol/L) (p=0.011). Although there were no significant differences in TC, LDLC, NHDLC, or TC/HDLC ratio among the glycemic control groups, there was a trend towards lower TC/HDLC ratio in the good control group (2.8) compared to the inadequate control (3.3) and poor control (3.3) groups (p=0.061). Framingham Risk Scores based on BMI and those based on lipids showed no significant differences, although there was a trend towards lower FRS-BMI in the good control group (29.8%) compared to the inadequate control (35.4%) and poor control (32.7%) groups (p=0.078) (Table 3).

Table 3 Lipid Profiles and Framingham Risk Scores by HbA1c Category

Variable	HbA1c Status			p-value
	Good Control (<7.0%) n=47	Inadequate Control (<7.0–8.0%) n=27	Poor Control (>8.0%) n=42	
TC mmol/L	3.84±0.85	3.75±0.95	3.84±0.83	0.719
LDLC mmol/L	2.67±1.0	2.69±0.9	2.63±0.8	0.366
HDLC mmol/L	1.37(1.08–1.64)	1.13(0.93–1.38)	1.12(0.99–1.33)	0.011*
NHDLC mmol/L	2.44±0.84	2.57±0.87	2.64±0.79	0.649
TC/HDLC	2.8(2.3–3.4)	3.3(2.7–3.7)	3.3(2.7–3.9)	0.061
FRS-BMI	29.8(14.3–40.6)	35.4(27.4–51.1)	32.7(16.9–47.6	0.078
FRS-Lipids	18.1(9.7–25.6)	21.5(14.4–39.2)	20.6(11.5–33.8)	0.078

Note: *p-value < 0.05.
Abbreviations: TC, Total Cholesterol; LDLC, Low Density Lipoprotein Cholesterol; HDLC, High Density Lipoprotein Cholesterol; NHDLC, Non-High Density Lipoprotein Cholesterol; FRS-BMI, Framingham Risk Score incorporating Body Mass Index; FRS-Lipids, Framingham Risk Score incorporating lipid profiles.

Discussion

The present study evaluated lipid profile parameters and the 10-year cardiovascular disease risk profiles of T2DM patients stratified by glycemic control. Our findings revealed significant differences in glycemic control by duration since DM diagnosis. Overall, the prevalence of dyslipidemia was 83.6% with hypoalphalipoproteinemia (79.3%) being the most prevalent dyslipidemia among the T2DM patients whilst the proportions of hyperbetalipoproteinemia and elevated non-HDLC levels were almost equivalent. Furthermore, a significantly higher proportion of T2DM patients with poor glycemic control had a higher 10-year risk of CVD based on the Framingham risk calculators and significantly lower serum HDLC levels. There were no significant differences in the levels of the other measured parameters.

In this study, there was a significant variation in the median duration since DM diagnosis across the different HbA1c statuses. Individuals with good glycemic control had a significantly shorter duration since DM diagnosis compared to those with inadequate and poor control. The duration since DM diagnosis is often associated with disease progression and the development of complications. This finding aligns with previous research that similarly reported an association between poor glycemic control and longer duration since DM diagnosis.[31,32] This finding could be explained by the chronic and progressive nature of diabetes mellitus. A longer duration with the condition may eventually make it difficult for a patient to maintain good glycemic control since long-term T2DM is associated with impaired insulin secretion due to deteriorating beta cell dysfunction.[33–35] However, in contradiction to our findings, an earlier study by Nichols et al (2000) reported that the duration since diabetes diagnosis was not significantly associated with glycemic control.[36]

The study also found a high prevalence of dyslipidemia among the study participants (83.6%) aligning with previous findings in individuals with T2DM. The most commonly observed dyslipidemia according to previous studies in T2DM is hypertriglyceridemia.[29] However, in the present study, triglycerides were not measured because participants were not fasting. Mean total cholesterol levels were unexpectedly within the desirable range while the overall prevalence of hypercholesterolemia was also surprisingly low (7.8%).[30] The possible reason for this finding could have been the exclusion criteria which among other issues excluded potential participants with underlying conditions such as chronic kidney disease or liver disease, which are known to contribute to T2DM associated hypercholesterolemia.[30,31] The relatively low prevalence of hypobetalipoproteinemia observed in the current study also aligns with the fact that LDLC is the major cholesterol bearing lipoprotein, thus low prevalence of hypercholesterolemia is physiologically associated with low prevalence of hypobetalipoproteinemia.

Additionally, the observed high prevalence of low HDL-C aligns with expectations, given the association of poorly controlled T2DM with hypoalphalipoproteinemia.[32] This highlights the importance of monitoring lipid profiles comprehensively in T2DM management, considering their significant implications for cardiovascular health. There are several factors that can contribute to hypoalphalipoproteinemia in individuals with type 2 diabetes mellitus. T2DM is associated with an increase in triglyceride (TG) rich lipoproteins in plasma. Consequently, there will be increased exchange of TG for cholesterol in a process mediated by cholesterol ester transfer protein (CETP) which essentially depletes HDLC of cholesterol esters resulting in the formation of small dense TG-rich HDLC. Furthermore, the activity of hepatic lipase is upregulated by hyperglycemia and in insulin resistance and this results in the rapid metabolism of the TG-rich HDLC leaving a lipid depleted lipoprotein that undergoes accelerated clearance.[37]

Based on the FRS-BMI, the majority of the participants (68.9%) were classified as being at high risk of CVD within the coming 10 years and the same pattern was observed across the glycemic control categories. No significant differences were observed by glycemic control among the other parameters. Based on the FRS calculations based on lipid profile, significantly higher scores were observed for those with inadequate glycemic control compared to those with good control. However, there were no significant differences in the mean and median concentrations of other serum lipid profile parameters. Although, the FRS has received class 1 recommendation from the ACC and AHS, it is possible that racial differences could modulate the FRS risk scoring since the risk score calculator was validated for Caucasians. There is an increasingly strong call for incorporation of racial variation in the FRS calculator.[38]

Strengths and Limitations

A major strength of the current study is the ethnic homogeneity of the study population that eliminated effects of ethnic differences. The generalizability of the findings may, however, be limited by the non-random participant enrolment approach used. In addition, the cross-sectional nature of the study design did not allow for follow-up to ascertain the possibility of incident CVD events.

Conclusion

Duration since DM diagnosis was observed to be associated with poor glycemic control being significantly shorter in those with good glycemic control compared to those with inadequate and poor control. Overall, there was no significant difference in HbA1c status by age; however, individuals with inadequate glycemic control were significantly older than those with good control. The most prevalent dyslipidemia among the study participants was hypoalphalipoproteinemia. These findings emphasize the importance of regular monitoring of lipid profiles in patients with T2DM and implementing appropriate interventions to manage dyslipidemia and reduce cardiovascular risk. Additional research is required to explore the underlying mechanisms contributing to the high prevalence of dyslipidemia in T2DM in this population and to identify specific targets for therapeutic interventions.

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Disclosure

The authors declare no conflicts of interest in this work.

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