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Association of HbA1c with lipid profile in patients with type 2 diabetes mellitus and cardiovascular disease

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Abstract

Cardiovascular diseases (CVDs) are major causes of increased mortality and disability across the globe. Effective management of diabetes mellitus (DM) in CVD patients requires the integration of monitoring of glycemic status (HbA1c) and lipid metabolism. This cross-sectional analytical study included 121 patients and 80 control subjects to analyze the association of HbA1c levels with lipid profiles. Serum biochemical analyses of the lipid profile [total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and triglycerides] were performed with an Agilent 8453 UV-Visible spectrophotometer (Agilent Technologies, USA), and HbA1c was measured on an A1C EZ 2.0 glycohemoglobin analyzer. Comparisons of characteristics between the groups were performed using chi square or Fisher's exact tests and unpaired Student's t tests, and the relationships between HbA1c and other variables were determined by correlation and linear regression analyses. The cohort exhibited evidence of poor glycemic control (mean HbA1c of 8.23%) and was associated with increasing age (mean 50.7 years) and weight (mean 70.61 kg). Low levels of HDL-C and a positive family history are associated with disease. Increased age and high body weight, systolic blood pressure (SBP), diastolic blood pressure (DBP) and fasting blood glucose levels were associated with high HbA1c levels (≥ 6.5 %), whereas HDL-C levels were inversely associated with HbA1c. Key correlations included significant positive associations of age, weight, SBP, and DBP with HbA1c (all $p < 0.05$). HDL-C was negatively correlated with HbA1c ($p = 0.04$). The association of HbA1c with dyslipidemia confirms interlinked nature of risk factors for CVD and glycemic control. Our findings support integrated clinical management that addresses blood pressure alongside glycemic and lipid targets to help mitigate long-term cardiovascular risk in diabetic patients.

Keywords

Diabetes mellitus; Glycated hemoglobin; Cardiovascular diseases; Cholesterol, LDL; Triglycerides; Dyslipidemias

1. Introduction

Cardiovascular disease (CVD) is an umbrella term comprising various cardiac and vascular conditions [1]. Diabetes mellitus (DM), a metabolic disorder, is a potential risk factor for CVDs as well as strongly connected with CVDs being the major determinant of health complications and diabetes-related mortality. Type 2 diabetes mellitus (T2DM) patients are predisposed to a greater risk of mortality from CVDs including heart failure, myocardial infarction (MI), and stroke. Cardiovascular risks in diabetic patients are linked with multiple factors, including hyperglycemia, dyslipidemia, insulin resistance,

inflammation, obesity, high blood pressure, along with that diabetes complications such as nephropathy and retinopathy [2,3].

Obesity has damaging effects on cardiac health through its effects on established risk determinants, including glucose intolerance, dyslipidemia, hypertension, obstructive sleep apnea and hypoventilation, systemic inflammation, and a prothrombotic state, as well as through potential mechanisms that remain incompletely understood [4]. Thus, obesity markedly predisposes individuals to a wide variety of cardiovascular complications, including coronary artery disease, cardiac arrest, and heart failure due to its persistent impact on cardiovascular system of a patient [5].

The formation of glycosylated hemoglobin (Hb) is a progressive, continuous, and irreversible non enzymatic process. HbA1c levels are mainly affected by long-term blood glucose levels over approximately 2–3 months rather than short-term fluctuations caused by factors such as insulin therapy or immediate oral glucose administration [6]. HbA1c is a reliable biomarker that reflects long-term glycemic control and helps predict risk factors associated with diabetes-related complications in individuals with T2DM, however, the prognostic value of HbA1c in patients with underlying CVD, especially coronary heart disease, remains unclear owing to conflicting as well as scarce evidence. Notwithstanding its clinical importance, there is a dearth of literature assessing the correlation of HbA1c levels with lipid profiles, especially among South Asian populations, with a focus on Pakistan.

There is a dire need to determine the relationship between HbA1c and lipid profile in the South Asian population, given the substantial burden of T2DM and CVD in the region. Compared to the West, South Asians usually experience earlier onset and rapid progression of diseases and are particularly prone to metabolic abnormalities, including obesity, resistance to insulin, and an atherogenic lipid pattern characterized by increased triglycerides (TG), decreased high-density lipoprotein cholesterol (HDL-C), and elevated low-density lipoprotein cholesterol (LDL-C). This amalgamation of metabolic disturbances contributes significantly to their increased vulnerability to cardiovascular complications. Furthermore, genetic susceptibility, carbohydrate-rich dietary patterns, and sedentary lifestyles increase risk, with premature cardiovascular mortality being disproportionately high in South Asian population. However, research is required to determine exact predictive value of HbA1c for dyslipidemia and CVD risk in this cohort.

Our hypothesis suggests that in patients with T2DM and established CVD, higher HbA1c levels are significantly associated with adverse lipid profile changes, characterized by elevated TC, LDL C, and triglyceride levels and reduced HDL C levels.

The aim of our research is to comprehensively investigate the relationship between glycemic control (as measured by HbA1c) and lipid profiles in a Pakistani population characterized by a high incidence of both diabetes and CVD.

2. Methods

2.1. Study setting, ethics, and approval

The study was conducted in hospitals/medical centers located in Punjab Province. Samples were collected after approval from the Ethical Review Committee (ERC) Ref. No. (NMDC-ERC/CER/13/25) & Institutional Research Advisory Committee (IRAC) Ref. No. (RAC/0017/25-APR), Niazi Medical & Dental College and Niazi Welfare Foundation Teaching Hospital, Sargodha, followed by the approval of Heads (Medical Superintendents) of the respective hospitals/medical centre (Tayyab Hospital Sargodha, Niazi Welfare Foundation Teaching Hospital Sargodha, Mubarak Hospital Sargodha, Shazia Zahid Medical Centre Lahore, New Mahar Medical Hospital Silanwali, Noshani Hospital Sandhiliwali, Mughees Haris Surgical Complex Joharabad).

2.2. Sample size

The sample size for the study was calculated using Cohen's guidelines (r = 0.2; α = 0.05; power = 80%). The minimum required sample size was approximately 194; a total of 201 participants (121 patients and 80 controls) were enrolled.

2.3. Sampling method

Eligible participants were recruited by using non-probability convenience sampling technique, and selection was based on predefined inclusion and exclusion criteria.

2.4. Study design

This is a cross-sectional analytical study, and we carried out it over a period of six months from April to September 2025.

2.5. Target population

Adult patients with a confirmed T2DM diagnosis and/or established CVD.

2.6. Selection criteria

We recruited participants aged ≥ 18 years who had a confirmed T2DM diagnosis for at least one year, established CVD (defined as MI or unstable angina) and provided written informed consent. Patients were excluded if they had type 1 diabetes mellitus (T1DM), were pregnant, or had acute infections or other severe systemic illnesses that could acutely influence blood glucose or lipid levels, as well as individuals with known or suspected secondary causes of dyslipidemia, such as hypothyroidism or nephrotic syndrome, or those with conditions affecting HbA1c measurement, including severe anemia or hemoglobinopathies.

2.7. Study subjects

This study included 80 unrelated, healthy control subjects and 121 sporadic diabetic CVD patients. The patients (67 males, 54 females) were diagnosed with either MI or unstable angina. The initial diagnosis of CVD was established using electrocardiographic evidence of cardiac abnormalities, followed by confirmation with coronary angiography.

DM was diagnosed using standardized biochemical criteria. According to American Diabetes Association (ADA), a diagnosis is established by an HbA1c level of ≥ 6.5%, a fasting plasma glucose concentration of ≥ 126 mg/dL, or a random plasma glucose concentration of ≥ 200 mg/dL in individuals who present with classic symptoms of hyperglycemia. These criteria offer a consistent and reproducible mechanism to identify individuals at increased risk of microvascular and macrovascular complications.

The control participants group, comprised of unrelated 45 males and 35 females, recruited from similar geographical area who belonged to same ethnic group as study group with no prior diagnosis of CVD or diabetes, as well as absence of clinical evidence of other atherosclerotic vascular disorders with normal electrocardiography findings. We defined arterial hypertension as a systolic blood pressure (SBP) of ≥ 140 mmHg or a diastolic blood pressure (DBP) of ≥ 90 mmHg. Smoking status was assessed based on tobacco use history, with participants classified as smokers if they reported current or previous tobacco use and as nonsmokers if they had no history of tobacco exposure. Overweight and obesity were determined per the World Health Organization Western Pacific Region (WPRO) criteria for Asian populations, based on body mass index (BMI) cut-off values for overweight (≥ 23.0 kg/m^2) and obesity (≥ 25.0 kg/m^2) [7,8].

2.8. Data collection

Participants provided information for completion of the questionnaire. Personal information (name, sex, age), family history, socio-behavioral risk factor data (smoking status), physical measurements (weight, height, blood pressure), and medical history, including disease profile and clinical symptoms, were collected. The questionnaires were carefully completed according to the information and data provided by patients and their consulting physicians. BMI was calculated by the standard formula. The clinical histories of the control subjects were also obtained through an interview.

2.9. Sample collection

After an overnight fast of at least 8 hours, peripheral venous blood samples were collected from patients and controls using sterilized syringes. For biochemical analysis, 2.5 mL blood was collected into sterile plain serum tubes and allowed to clot before centrifugation at 4000 rpm for 5 minutes to separate the serum. The resulting serum supernatant was carefully transferred into microcentrifuge tubes in 0.5 mL aliquots and preserved at −20°C for further analysis.

2.10. Biochemical analysis

Biochemical parameters were analyzed employing an Agilent 8453 UV-Visible spectrophotometer (USA). The levels of serum TC, TG, and glucose were determined through enzymatic colorimetric analysis using commercially available gCHEM SERIES reagents (gCHEM, Italy). HDL-C levels were determined using enzymatic colorimetric assays with commercially available kits (Erba Lachema, Brno, Czech Republic). LDL-C concentrations were estimated using the Friedewald equation based on the measured values of TC, TG, and HDL-C; because LDL-C was derived using this equation, all samples were fasting and cases with triglycerides > 400 mg/dL were excluded from LDL-C estimation. HbA1c was measured on an A1C EZ 2.0 glycohemoglobin analyzer. The glucose value used in all analyses was fasting plasma glucose.

All biochemical analyses were performed under strict quality control measures, and all instruments were calibrated daily with help of standard references, while assay precision was determined by intra- and inter-assay coefficients. Reproducibility was confirmed by a duplicate analysis of randomly selected samples, ensuring reliability and consistency of results.

2.11. Statistical analysis

Frequencies (n) and percentages (%) were calculated for the baseline variable, whereas means ± standard deviations (SD) were calculated for biochemical and clinical parameters. Pearson chi-square test/Fisher's exact test was used to determine associations between categorical variables and to compare their distribution across patient and control groups. Continuous variables, including age, blood pressure, BMI, and lipid profile, were compared between the study groups using an unpaired Student’s t-test. Correlation as well as linear regression were performed to determine the relationship between HbA1c levels and relevant clinical variables. Statistical analyses using SPSS, version 26.0, were performed. Statistical significance was set at $p \leq 0.05$.

Levene's test was rendered to determine homogeneity of variance prior to conducting parametric tests. Regression diagnostics included the assessment of residual plots, and residuals’ normality. Residuals were approximately normally distributed, indicating the validity of the regression model.

3. Results

3.1. Characteristics of patients and control subjects

The study cohort consisted of 80 control subjects and 121 patients (Table 1). The proportion of participants aged > 45 years was significantly higher among the patient group compared to the control group ($p = 0.010$). As presented in Table 1, patient group included 54 (45%) females and 67 (55%) males, whereas control group consisted of 35 (44%) females and 45 (56%) males. Compared to control group, common risk factors such as high blood pressure and family history were observed at significantly higher frequencies among patients ($p < 0.0001$).

Table 1. Baseline profile of study participants by group.

Characteristics		Study Groups		*p* Values
		Controls n = 80	Patients n = 121	
		n (%)	n (%)	
Age	≤ 45 years	48 (60)	51 (42)	0.010 ***
	> 45 years	32 (40)	70 (58)	
Gender	Female	35 (44)	54 (45)	0.902
	Male	45 (56)	67 (55)	
Body mass index (BMI)	Normal	35 (44)	45 (37)	0.605
	Overweight/obese	45 (56)	76 (63)	
Hypertension	Normal	50 (62.5)	42 (35)	< 0.0001 ***
	Yes	30 (37.5)	79 (65)	
Smoking status	Non-smokers	53 (66)	76 (63)	0.619
	Smokers	27 (34)	45 (37)	
Family history	No family history	80 (100)	50 (41)	< 0.0001 ***
	Family history	0 (0)	71 (59)	

* The table presents a column-wise comparison of each characteristic between control participants and patient groups. ** Data are expressed as absolute numbers and corresponding percentages. *** Significant at $p \leq 0.05$.

3.2. Clinical and biochemical characteristics

Clinical and biochemical variables such as age, weight, blood pressure (BP), anthropometric measures (BMI), glycemic indices (blood glucose and HbA1c), and lipid parameters were compared between patients and controls (Table 2). The patients were significantly older (50.7 ± 12.7 years) than the control subjects were (42.9 ± 13.1 years), showing significant difference between the groups ($p < 0.001$). Patients had a significantly greater mean weight (70.61 ± 8.59 kg) than control subjects did (65.75 ± 12.86 kg; $p = 0.002$). The two groups did not differ significantly with respect to BMI ($p = 0.59$), possibly because of variability in height. The SBP was greater among patients (129.21 ± 10.8 mm Hg) than among control subjects (124.06 ± 10.73 mm Hg; $p = 0.001$). Table 2 further delineates that DBP was significantly greater in patients (89.01 ± 12.35 mm Hg) than in control subjects (82.44 ± 10.67 mm Hg; $p < 0.0001$).

Analysis showed a significant difference between study groups regarding the average blood glucose and HbA1c levels (228.1 ± 50.5 mg/dL; 8.23 ± 1.75 %) and the control group (114.49 ± 17.3 mg/dL; 5.06 ± 0.68 %) ($p < 0.0001$). The mean concentrations of TC and LDL-C were higher in patients than in control subjects, whereas triglyceride and VLDL-C levels were somewhat higher in the control group relative to patients, though these differences were not statistically significant. In contrast, the mean HDL-C levels were significantly greater among the controls than among the patients ($p = 0.008$).

Table 2. Differences in clinical and biochemical parameters between study groups.

Parameters	Study Groups		95% Confidence Intervals (CI)	*p* Values
	Controls (n = 80)	Patients (n = 121)		
	Mean ± SD	Mean ± SD		
Age (years)	42.94 ± 13.10	50.74 ± 12.70	4.14–11.47	< 0.0001 **
Weight (kg)	65.75 ± 12.86	70.61 ± 8.59	1.87–7.84	0.002 **
Body mass index (BMI) (kg/m^2)	24.32 ± 5.30	24.01 ± 2.96	-1.46–0.83	0.590
Systolic blood pressure (SBP) (mm Hg)	124.06 ± 10.73	129.21 ± 10.80	2.09–8.21	0.001 **
Diastolic blood pressure (DBP) (mm Hg)	82.44 ± 10.67	89.01 ± 12.35	3.23–9.90	< 0.0001 **
Fasting blood glucose (mg/dL)	114.49 ± 17.31	228.11 ± 50.51	102.05–125.18	< 0.0001 **
HbA1c	5.06 ± 0.68	8.23 ± 1.75	2.76–3.57	< 0.0001 **
Cholesterol (mg/dL)	187.10 ± 40.26	193.17 ± 45.80	-6.34–18.48	0.330
Triglycerides (mg/dL)	197.30 ± 114.93	184.44 ± 70.75	-38.69–12.96	0.320
Low-density lipoprotein cholesterol (LDL-C) (mg/dL)	104.86 ± 31.77	109.96 ± 36.48	-4.76–14.95	0.300
High-density lipoprotein cholesterol (HDL-C) (mg/dL)	47.39 ± 14.92	42.70 ± 10.03	-8.15–-1.21	0.008 **
Very Low-density lipoprotein cholesterol (VLDL-C) (mg/dL)	38.68 ± 20.81	37.15 ± 15.04	-6.54–3.48	0.540

* The table presents a column-wise comparison of each characteristic using unpaired Student's t test between study groups. ** Significant at $p \leq 0.05$.

3.3. Differences in clinical and biochemical characteristics by HbA1c status

Table 3 summarizes the characteristics of our study population in terms of comparisons between subjects with normal HbA1c (< 6.5%; n = 92, comprising all 80 controls and 12 patients) and those with high HbA1c (≥ 6.5%; n = 109, all patients) using t tests. Subjects with high HbA1c levels were significantly older (51.14 ± 12.88 years) than those with normal HbA1c levels were (43.49 ± 12.92 years). Analysis revealed significant variation in this parameter ($p < 0.0001$).

High-HbA1c group had a greater average body weight (70.71 ± 8.84 kg) than those in the normal-HbA1c group (66.27 ± 12.25 kg) did ($p = 0.003$). Similarly, SBP was observed to be greater in the high HbA1c group (129.40 ± 10.68 mm Hg) than in the normal HbA1c group (124.51 ± 10.92 mm Hg) ($p = 0.002$). Similarly, DBP was markedly elevated in the high-HbA1c group (89.18 ± 12.29 mm Hg) compared with that in the normal-HbA1c group (83.02 ± 11.07 mm Hg), indicating significant association ($p < 0.0001$). The mean blood glucose level was markedly greater in the high-HbA1c group (234.64 ± 45.39 mg/dL) than in the normal-HbA1c group (121.57 ± 31.65 mg/dL), underscoring the strong link between increased HbA1c and hyperglycemia ($p < 0.0001$). Furthermore, patients in the high-HbA1c group had lower HDL-C levels (42.95 ± 9.98 mg/dL) than those in the normal-HbA1c group did (46.48 ± 14.58 mg/dL; $p = 0.04$). However, comparison between the two groups showed no significant differences in BMI or lipid profile.

Table 3. Comparison of clinical and biochemical profiles according to HbA1c status.

Parameters	Subjects with Normal HbA1c (n = 92)	Subjects with High HbA1c (n = 109)	95% Confidence Intervals (CI)	*p* Values
	Mean ± SD	Mean ± SD		
Age (Years)	43.49 ± 12.92	51.14 ± 12.88	4.04–11.25	< 0.0001 **
Weight (kg)	66.27 ± 12.25	70.71 ± 8.84	1.49–7.37	0.003 **
Body mass index (BMI) (kg/m^2)	24.13 ± 5.03	24.13 ± 3.00	-1.14–1.12	0.990
Systolic blood pressure (SBP) (mm Hg)	124.51 ± 10.92	129.40 ± 10.68	1.87–7.90	0.002 **
Diastolic blood pressure (DBP) (mm Hg)	83.02 ± 11.07	89.18 ± 12.29	2.87–9.45	< 0.0001 **

Parameters	Subjects with Normal HbA1c (n = 92)	Subjects with High HbA1c (n = 109)	95% Confidence Intervals (CI)	*p* Values
	Mean ± SD	Mean ± SD		
Fasting blood glucose (mg/dL)	121.57 ± 31.65	234.64 ± 45.39	101.99–124.16	< 0.0001 **
Cholesterol (mg/dL)	185.76 ± 39.18	194.97 ± 46.91	-2.94–21.37	0.130
Triglycerides (mg/dL)	192.30 ± 109.79	187.24 ± 71.61	-30.49–20.37	0.700
Low-density lipoprotein cholesterol (LDL-C) (mg/dL)	105.75 ± 33.84	109.77 ± 35.45	-5.67–13.71	0.410
High-density lipoprotein cholesterol (HDL-C) (mg/dL)	46.48 ± 14.58	42.95 ± 9.98	-6.96–-0.10	0.040 **
Very Low-density lipoprotein cholesterol (VLDL-C) (mg/dL)	38.48 ± 21.93	37.46 ± 14.35	-6.10–4.06	0.690

* The table shows a column-wise comparison of each characteristic using unpaired Student's t test among the study population based on HbA1c status. ** Significant at $p \leq 0.05$.

3.4. Pearson correlation analysis of HbA1c with other parameters

Pearson correlation analysis results are summarized in Table 4, highlighting relationships between HbA1c and independent parameters. Elevated HbA1c levels were strongly and positively linked to blood glucose levels ($p < 0.0001$). Weak but statistically significant positive associations were identified between HbA1c levels and age, body weight, SBP and DBP (all, $p < 0.05$). Conversely, HbA1c was weakly, negatively, and significantly related to HDL-C ($p = 0.04$). No significant relationship was recorded between HbA1c levels and other evaluated parameters, including BMI, and cholesterol levels ($p > 0.05$).

Table 4. Correlation analysis of HbA1c and related parameters.

Variables	Correlation Coefficient (r)	*p* Values
Age (years)	0.19	0.005 *
Weight (kg)	0.23	0.001 *
Body mass index (BMI) (kg/m2)	0.07	0.270
Systolic blood pressure SBP (mm Hg)	0.19	0.005 *
Diastolic blood pressure (DBP) (mm Hg)	0.17	0.010 *
Fasting blood glucose (mg/dL)	0.75	< 0.0001 *
Cholesterol (mg/dL)	0.10	0.120
Triglycerides (mg/dL)	-0.01	0.850
Low-density lipoprotein cholesterol (LDL-C) (mg/dL)	0.05	0.420
High-density lipoprotein cholesterol (HDL-C) (mg/dL)	-0.14	0.040 *
Very low-density lipoprotein cholesterol (VLDL-C) (mg/dL)	-0.01	0.850

* Significant at $p \leq 0.05$.

3.5. Linear regression analysis of HbA1c with other parameters

Regression analysis results for HbA1c levels and age, weight, BMI, SBP, DBP, and blood glucose and lipid parameters are presented in Table 5. Regression model accounted for 56% of the variability in HbA1c levels ($R^2 = 0.56$), and the model assumptions were adequately met. The residuals were normally distributed. Blood glucose and HDL-C were the only parameters that were significantly related to HbA1c. After controlling for other variables, the regression coefficient (b = 0.023) revealed that a 1 mg/dL increase in blood glucose corresponded to a 0.023-unit increase in HbA1c. Among the evaluated parameters, blood glucose exhibited the strongest association with HbA1c ($p < 0.0001$). Table 5 further corroborates that HDL-C is inversely associated with HbA1c. The regression coefficient (b = −0.027) indicates that each 1 mg/dL increase in HDL-C is associated with a 0.027-unit reduction in HbA1c after controlling for other variables ($p = 0.030$).

Table 5. Regression analysis of factors associated with HbA1c levels.

Parameters	Unstandardized Coefficient (b)	Standardized Coefficient (β)	*p* Values
Age (Years)	-0.000	-0.050	0.300
Weight (kg)	0.010	0.090	0.110
Body mass index (BMI) (kg/m^2)	0.010	0.020	0.590
Systolic blood pressure (SBP) (mm Hg)	-0.000	-0.000	0.960
Diastolic blood pressure (DBP) (mm Hg)	0.000	0.040	0.500
Fasting blood glucose (mg/dL)	0.023	0.740	< 0.0001 **
Cholesterol (mg/dL)	0.000	0.100	0.160
Triglycerides (mg/dL)	-0.030	-1.430	0.800
Low-density lipoprotein cholesterol (LDL-C) (mg/dL)	0.000	0.040	0.530
High-density lipoprotein cholesterol (HDL-C) (mg/dL)	-0.027	-0.150	0.030 **
Very Low-density lipoprotein cholesterol (VLDL-C) (mg/dL)	0.160	1.420	0.800

* Dependent variable: HbA1c. ** Significant at $p \leq 0.05$.

4. Discussion

Our study shows HbA1c levels and key cardiovascular risk factors relationship among patients with diabetes. Baseline parameters like age > 45 years, hypertension and positive family history were significantly linked to disease ($p < 0.05$). Consistent results have been documented in previous studies [9,10,11]. Our results show that among the major risk factors, including weight, blood pressure were higher among patient groups than in control group. Previous studies have shown excess body weight and visceral obesity to be contributing risk factors in development of hypertension and cardiovascular disease [12]. Biochemical analysis revealed that patients had numerically higher cholesterol and LDL-C levels than healthy control subjects did, although this difference was not statistically significant. Comparable outcomes have been documented in prior studies, likewise, indicating no significant correlations between these variables [10]. These results are not in agreement with those of multiple prior studies, which reported significant correlations between HbA1c and both TC and LDL-C levels [13]. However, no significant associations were detected for TC, LDL-C, TG, or VLDL-C. Our results indicated that patients have lower HDL-C levels than control subjects do. These findings reveal a positive effect of HDL-C on health. A previous population survey similarly documented a high burden of lipid abnormalities in a comparable adult population [14].

We divided our study population on the basis of the cutoff value for HbA1c according to the ADA criteria. Study participants were categorized into a group of subjects with normal HbA1c levels (< 6.5) and individuals with higher levels of HbA1c (≥ 6.5 %). High HbA1c levels were associated with increasing age, blood pressure, body weight, and blood glucose levels, whereas HDL-C had a negative correlation with HbA1c, as per the findings of current study. Similar nature associations between glycemic status and cardiovascular risk factors have been reported by previous studies [13,15].

Pearson correlation analysis indicated a strong positive association of HbA1c with age, weight, SBP, DBP and glucose levels. Findings of current study align with previously conducted studies reporting significant and positive correlations between HbA1c levels, increased SBP, and family history [11]. Our results are in agreement with those of a number of studies that documented a significant inverse relationship between HbA1c levels and HDL-C levels [16,17].

The observed positive association between SBP, HbA1c, and fasting glucose levels is consistent with previous studies, highlighting relationship between impaired glycemic control and cardiovascular risk factors [9,18]. These findings highlight importance of

maintaining optimal blood pressure, as persistent hyperglycemia has been linked to vascular alterations such as arterial stiffness and impaired endothelial function, potentially contributing to increased SBP. Effective control of glycemia and blood pressure is essential for reducing cardiovascular risk among patients with diabetes.

A negative association for HbA1c and HDL-C levels was observed, which is in agreement with findings of Madhuri and Shet, revealing a negative correlation between these variables [19]. LDL-C was not significantly correlated with HbA1c, as reported by present study, which differs from findings of Vergès et al., who observed either no association or a positive relationship between these parameters [20]. These differences may reflect variations in metabolic profiles, genetic predispositions, lifestyle patterns, and clinical factors across populations, which can affect lipid parameters beyond glycemic status.

Insulin resistance reduces hepatic LDL receptor expression, resulting in impaired LDL clearance from circulation. Furthermore, increased influx of free fatty acids into liver promotes production of VLDL, resulting in hypercholesterolemia [20].

A study from Pakistan by Shahid et al. assessed HbA1c levels and fasting lipid profiles relationship among local diabetic population and showed that TC, and other parameters of lipid profile were positively correlated with HbA1c levels; however, LDL-C levels were negatively correlated [21]. Similarly, an Arabian study assessed HbA1c levels and lipid profiles relationship and revealed significant linkage between the levels of both TC and triglyceride levels and between the levels of these two variables in T2DM patients [22]. A recent Indonesian study similarly examined the relationship between HbA1c and the lipid profile in patients with T2DM [23].

Absence of a significant relationship between TC and glycemic indices in current study contrasts with findings from other scientific studies [24]. While previous studies have linked hypercholesterolemia with poor glycemic control, observations made by current study highlighted metabolic alterations in diabetes are heterogeneous, with individual lipid fractions revealing different associations with glycemic indices.

The variation in findings of current study and other earlier studies may highlight differences in underlying population characteristics. South Asian populations, including nondiabetic individuals, are reported to have a high baseline incidence of dyslipidemia, influencing HbA1c and lipid profile relationship. A key strength is the use of complete biochemical profiles of patients, which enabled comprehensive assessment through comparative, correlation, and regression analyses. However, the use of a cross-sectional study design, sample size, and lack of information on dietary habits, lifestyle patterns, duration of diabetes, and regular physical activity may be limitations to this study. Because the correlation and regression analyses were performed on the combined sample of patients and controls, the observed associations partly reflect between-group (case–control) differences and may not represent within-patient relationships. Furthermore, unmeasured confounding factors may have affected the observed associations; therefore, comprehensive clinical management strategies combining strict glycemic control with lipid-lowering agents are important for complications risk reduction.

5. Conclusions

The study identified a significant association between diabetes and several risk factors, including older age, hypertension, and family history. High levels of HbA1c were positively associated with age, blood pressure, body weight, and, most strongly, blood glucose levels, reflecting complex metabolic alterations associated with diabetes. The observed negative relationship between HbA1c and HDL-C highlights that reduced levels of protective lipoprotein may be associated with worsening glycemic status in diabetic patients. Although patients showed numerically higher TC and LDL-C than controls, these

differences were not statistically significant and were not associated with HbA1c levels, highlighting possible metabolic differences within the South Asian population.

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Data availability: The data supporting this study's findings are available from the corresponding author, Gul Zareen Asifa, upon reasonable request.

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