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Original Research Article

Evaluation of Glycated Hemoglobin and Lipid Profile in Type 2 Diabetes Mellitus

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Abstract:

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by impaired glycemic control and lipid abnormalities that collectively elevate cardiovascular risk. This study aimed to evaluate glycated hemoglobin (HbA1c) and lipid profile parameters in T2DM patients and assess their correlation with glycemic control.

Materials and Methods: A cross-sectional study was conducted at a tertiary care hospital in India, enrolling 120 participants — 80 confirmed T2DM patients and 40 healthy age- and sex-matched controls. Fasting venous blood samples were analyzed for HbA1c by High-Performance Liquid Chromatography (HPLC), while total cholesterol (TC), triglycerides (TG), and HDL-C were measured by enzymatic colorimetric methods. LDL-C and VLDL-C were derived using the Friedewald equation. Data were analyzed using SPSS version 26.0, with $p < 0.05$ considered statistically significant.

Results: HbA1c was significantly higher in T2DM patients than controls (9.1 ± 1.8% vs. 5.2 ± 0.4%, $p < 0.001$). Diabetic patients exhibited significantly elevated TC (5.82 ± 1.12 vs. 4.61 ± 0.83 mmol/L), TG (2.31 ± 0.87 vs. 1.24 ± 0.41 mmol/L), LDL-C (3.75 ± 0.94 vs. 2.67 ± 0.71 mmol/L), and VLDL-C (1.05 ± 0.40 vs. 0.56 ± 0.19 mmol/L), with significantly reduced HDL-C (1.02 ± 0.24 vs. 1.38 ± 0.29 mmol/L; all $p < 0.001$). HbA1c correlated positively with TC ($r = +0.48$), TG ($r = +0.52$), LDL-C ($r = +0.44$), and VLDL-C ($r = +0.52$), and negatively with HDL-C ($r = -0.41$; all $p < 0.001$).

Conclusion: Patients with T2DM exhibit markedly elevated HbA1c alongside a significantly atherogenic lipid profile, highlighting the critical need for routine simultaneous monitoring of glycemic control and lipid status to mitigate cardiovascular risk.

Keywords: Type 2 diabetes mellitus; Glycated hemoglobin; Lipid profile; Cardiovascular risk.

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Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder that has emerged as one of the most pressing public health challenges of the twenty-first century. Characterized by progressive insulin resistance and relative insulin deficiency, it leads to sustained hyperglycemia and a cascade of systemic complications affecting multiple organ systems [1]. A systematic analysis based on Global Burden of Disease Study 2021 data demonstrated a steep rise in the worldwide burden of diabetes from 1990 to 2021, with projections indicating a continued increase in prevalence through 2050 across all global regions [2]. Consistent with these trends, the International Diabetes Federation estimated that global diabetes prevalence stood at 10.5% in 2021 and is projected to reach 11.3% by 2030 and 12.2% by 2040, underscoring the scale of this ongoing epidemic [3]. The disease disproportionately affects individuals in low- and middle-income countries, where healthcare resources for adequate monitoring and management remain limited.

Effective long-term management of T2DM relies heavily on reliable biomarkers of glycemic control. Glycated hemoglobin (HbA1c), formed through the nonenzymatic binding of glucose to the N-terminal valine residue of the hemoglobin beta-chain, serves as a well-established indicator of average blood glucose concentrations over the preceding two to three months [4]. The World Health Organization and the American Diabetes Association have both

endorsed HbA1c as the standard biomarker for monitoring glycemic status and predicting diabetes-related complications. Elevated HbA1c has been shown not only to reflect chronic hyperglycemia but also to correlate with the long-term risk of microvascular and macrovascular complications, including nephropathy, retinopathy, neuropathy, coronary heart disease, and stroke [4].

Beyond impaired glucose metabolism, patients with T2DM frequently demonstrate significant disturbances in lipid homeostasis. Impaired glycemic control has been shown to upregulate de novo lipogenesis and promote hepatic triglyceride synthesis, resulting in a dyslipidemic state characterized by elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and increased levels of small, dense low-density lipoprotein cholesterol (LDL-C), all of which collectively contribute to accelerated atherosclerosis [5]. This atherogenic lipid profile substantially amplifies cardiovascular risk, which remains the leading cause of morbidity and mortality among individuals with T2DM.

Given the interrelated pathophysiology of hyperglycemia and dyslipidemia, understanding the relationship between HbA1c and lipid profile parameters is of considerable clinical importance. Several studies have investigated this association with varying findings. A retrospective cross-sectional study conducted among T2DM patients in Saudi Arabia found a significant positive correlation between HbA1c levels and both total cholesterol and triglycerides, highlighting the link between glycemic control and the atherogenic lipid milieu [6]. Similarly, a more recent study among newly diagnosed T2DM patients at a tertiary hospital reported significant associations between elevated HbA1c and higher triglyceride levels as well as lower HDL-C concentrations, further supporting the utility of HbA1c as a potential indicator of dyslipidemia risk [7].

Despite this growing body of evidence, findings across different populations and geographic settings remain inconsistent. The present study was therefore undertaken to evaluate HbA1c and lipid profile parameters in patients with type 2 diabetes mellitus attending a tertiary care hospital in India, and to assess the correlation between glycemic control and lipid abnormalities, with the aim of informing more integrated clinical management approaches for this patient population.

Material and Methods

Study Design and Setting: A cross-sectional study was conducted at an Indian tertiary level teaching hospital. The study aimed to evaluate HbA1c and lipid profile parameters in patients diagnosed with T2DM in comparison to healthy non-diabetic individuals. Written informed consent was obtained from each participant. The study was conducted in accordance with the Declaration of Helsinki guidelines for research involving human subjects.

Study Participants: A total of 120 participants were recruited for this study and divided into two groups. The first group comprised 80 patients previously diagnosed with type 2 diabetes mellitus, while the second group consisted of 40 apparently healthy individuals who served as controls. Participants were matched for age and sex across both groups.

Inclusion criteria for the diabetic group included a confirmed diagnosis of type 2 diabetes mellitus based on the American Diabetes Association (ADA) diagnostic criteria, age between 30 and 65 years, and willingness to participate in the study. Patients who were pregnant, had type 1 diabetes mellitus, were diagnosed with renal or hepatic disease, had thyroid disorders, or were on lipid-lowering medications such as statins were excluded from the study. Control participants were included if they had no history of diabetes, cardiovascular disease, or any chronic illness, and were excluded if they had any acute or chronic condition that could influence the measured parameters.

Blood Sample Collection: Following a minimum fasting period of 10 to 12 hours, approximately 7 mL of venous blood was collected from each participant by venipuncture of the antecubital vein under aseptic conditions. Blood was distributed into two separate collection tubes. Three milliliters were transferred into a dipotassium ethylenediaminetetraacetic acid (K_2-EDTA) vacutainer tube for HbA1c analysis, and the remaining 4 mL were dispensed into a plain vacutainer tube and allowed to clot at room temperature for 15 to 20 minutes, followed by centrifugation at 3000 rpm for 10 minutes to obtain serum, which was used for lipid profile analysis. All samples were processed within two hours of collection to ensure analytical accuracy and minimize pre-analytical errors.

Laboratory Measurements: HbA1c was measured from whole EDTA blood using High-Performance Liquid Chromatography (HPLC) on the Bio-Rad D-10 Hemoglobin Testing System (Bio-Rad Laboratories, Hercules, CA, USA). The assay was performed according to the manufacturer's instructions, and results were expressed as a percentage (%) of total hemoglobin. The analyzer was calibrated daily using certified reference materials, and internal quality controls were run with each batch of samples.

Serum lipid profile parameters, including total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C), were measured using enzymatic colorimetric methods on the Mindray BS-200 fully automated biochemistry analyzer (Mindray Medical International Limited,

Shenzhen, China), utilizing commercially available reagent kits (Spinreact, S.A., Girona, Spain). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation as follows:

LDL-C (mmol/L) = TC − HDL-C − (TG/2.2)

Very low-density lipoprotein cholesterol (VLDL-C) was estimated by dividing the triglyceride value by 2.2, as previously described. All assays were performed in duplicate, and the mean value was recorded. The laboratory maintained strict quality control measures in accordance with standard operating procedures.

Statistical Analysis: Data were analyzed using the Statistical Package for Social Sciences (SPSS) software, version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD). Differences between the diabetic and control groups were assessed using the independent samples Student's t-test for normally distributed variables. Pearson's correlation coefficient was applied to examine the relationship between HbA1c and lipid profile parameters. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 120 participants were enrolled in the present study, comprising 80 patients diagnosed with type 2 diabetes mellitus and 40 healthy control subjects. As presented in Table 1, the mean age of the diabetic group was 52.4 ± 8.3 years, while that of the control group was 50.9 ± 7.6 years, with no statistically significant difference observed between the two groups ($p = 0.312$). Sex distribution was comparable across both groups, with a male-to-female ratio of 44:36 in the diabetic group and 22:18 in the control group ($p = 0.967$), indicating adequate matching between the groups. However, the mean body mass index (BMI) was significantly higher in diabetic patients (29.8 ± 3.7 kg/m²) compared to controls (26.3 ± 2.9 kg/m²), with a statistically significant difference ($p < 0.001$), as shown in Table 1. The mean duration of diabetes among T2DM patients was 7.2 ± 4.1 years.

Table 2 illustrates the comparison of HbA1c and lipid profile parameters between the two study groups. The mean HbA1c level in the T2DM group was significantly elevated at 9.1 ± 1.8%, compared to 5.2 ± 0.4% in the control group, reflecting poor long-term glycemic control among diabetic patients ($p < 0.001$). This marked difference confirms the validity of HbA1c as a reliable indicator for distinguishing between diabetic and non-diabetic individuals.

As demonstrated in Table 2, statistically significant differences were observed in all measured lipid profile parameters between the T2DM and control groups. The mean total cholesterol (TC) level was notably higher in diabetic patients (5.82 ± 1.12 mmol/L) than in controls (4.61 ± 0.83 mmol/L), with a p-value of less than 0.001. Similarly, serum triglyceride (TG) concentrations were considerably elevated in the T2DM group (2.31 ± 0.87 mmol/L) relative to the control group (1.24 ± 0.41 mmol/L), again reaching statistical significance ($p < 0.001$).

With regard to LDL cholesterol, the T2DM group exhibited a mean LDL-C level of 3.75 ± 0.94 mmol/L, which was significantly greater than the 2.67 ± 0.71 mmol/L recorded among controls ($p < 0.001$). Likewise, VLDL cholesterol levels were markedly higher in diabetic patients (1.05 ± 0.40 mmol/L) compared to healthy subjects (0.56 ± 0.19 mmol/L), with $p < 0.001$ (Table 2, Figure 1).

In contrast, HDL cholesterol levels followed an opposing trend. The mean HDL-C concentration was significantly lower in the T2DM group (1.02 ± 0.24 mmol/L) than in the control group (1.38 ± 0.29 mmol/L), indicating a reduction in the cardioprotective lipoprotein fraction among diabetic patients ($p < 0.001$), as shown in Table 2. Collectively, these findings suggest a distinctly atherogenic lipid profile in patients with type 2 diabetes mellitus.

The Pearson correlation analysis performed within the T2DM group revealed significant associations between HbA1c and all measured lipid parameters, as summarized in Table 3. HbA1c demonstrated a significant positive correlation with total cholesterol ($r = +0.48$, $p < 0.001$), triglycerides ($r = +0.52$, $p < 0.001$), LDL cholesterol ($r = +0.44$, $p < 0.001$), and VLDL cholesterol ($r = +0.52$, $p < 0.001$), indicating that worsening glycemic control was associated with a progressive rise in atherogenic lipid fractions. Conversely, a significant negative correlation was identified between HbA1c and HDL cholesterol ($r = −0.41$, $p < 0.001$), suggesting that poorer glycemic control corresponds to lower levels of the protective lipoprotein fraction (Table 3). These correlations collectively underscore the close relationship between chronic hyperglycemia and dyslipidemia in type 2 diabetes mellitus.

Table 1: Demographic and Baseline Characteristics of Study Participants

Parameter	T2DM Group (n=80)	Control Group (n=40)	P-value
Age (years), Mean ± SD	52.4 ± 8.3	50.9 ± 7.6	0.312
Sex (Male/Female)	44 / 36	22 / 18	0.967
BMI (kg/m²), Mean ± SD	29.8 ± 3.7	26.3 ± 2.9	<0.001
Duration of Diabetes (years)	7.2 ± 4.1	—	—

Table 2: Comparison of HbA1c and Lipid Profile Parameters Between T2DM and Control Groups

Parameter	T2DM Group (n=80) Mean ± SD	Control Group (n=40) Mean ± SD	P-value
HbA1c (%)	9.1 ± 1.8	5.2 ± 0.4	<0.001
Total Cholesterol, TC (mmol/L)	5.82 ± 1.12	4.61 ± 0.83	<0.001
Triglycerides, TG (mmol/L)	2.31 ± 0.87	1.24 ± 0.41	<0.001
HDL Cholesterol, HDL-C (mmol/L)	1.02 ± 0.24	1.38 ± 0.29	<0.001
LDL Cholesterol, LDL-C (mmol/L)	3.75 ± 0.94	2.67 ± 0.71	<0.001
VLDL Cholesterol, VLDL-C (mmol/L)	1.05 ± 0.40	0.56 ± 0.19	<0.001

Table 3: Pearson Correlation between HbA1c and Lipid Profile Parameters in the T2DM Group (n=80)

Lipid Parameter	Pearson's r	P-value
Total Cholesterol (TC)	+0.48	<0.001
Triglycerides (TG)	+0.52	<0.001
HDL Cholesterol (HDL-C)	−0.41	<0.001
LDL Cholesterol (LDL-C)	+0.44	<0.001
VLDL Cholesterol (VLDL-C)	+0.52	<0.001

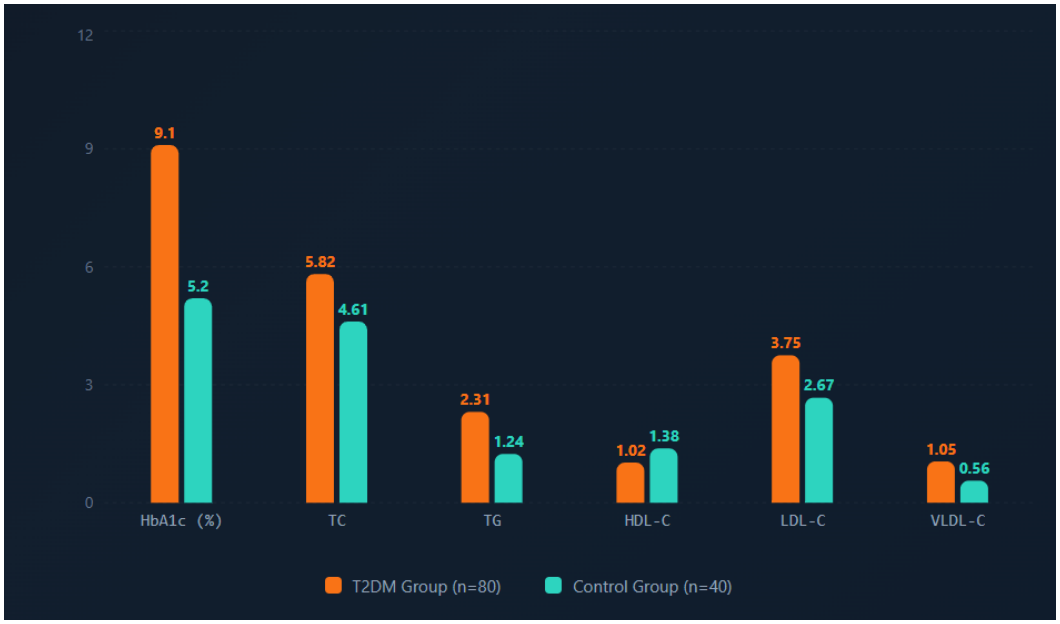

Figure 1: Comparison of Biochemical Parameters: T2DM vs Controls

Discussion

The present study evaluated HbA1c and lipid profile parameters in patients with type 2 diabetes mellitus and compared them to healthy controls, while also investigating the relationship between glycemic control and lipid disturbances. The findings demonstrate significant biochemical differences between the two groups across all measured parameters, a pattern that is broadly consistent with what has been reported in the existing literature.

The mean HbA1c level recorded in the T2DM group in this study was 9.1 ± 1.8%, indicating poor long-term glycemic control based on the internationally accepted threshold of 7.0% recommended by the American Diabetes Association. This level of suboptimal control is not unexpected. A cross-sectional study by Yahaya et al. conducted among T2DM patients in Uganda reported that approximately seven out of every ten patients exhibited inadequate glycemic control when HbA1c ≥7% was used as the cut-off, with lack of adherence to regular follow-up identified as a key contributing factor [8]. The magnitude of poor glycemic control observed in our cohort likely reflects similar systemic barriers, including irregular monitoring and inconsistent treatment adherence, which remain prevalent challenges in resource-limited healthcare settings such as India.

In the present study, the mean BMI of the T2DM group was significantly higher than that of the control group (29.8 ± 3.7 vs. 26.3 ± 2.9 kg/m²), suggesting a strong link between excess body weight and the presence of type 2 diabetes. This observation is well supported in the literature. Deng et al. demonstrated in a recent cross-sectional study among 200 T2DM patients that a significant positive correlation exists between BMI and HbA1c levels (r

= 0.45, $p < 0.001$), with obese patients exhibiting substantially higher HbA1c values compared to those with normal BMI, underscoring the clinical importance of weight management as part of comprehensive diabetes care [9].

Patients with T2DM in the current study exhibited a distinctly atherogenic lipid profile, including significantly elevated TC, TG, LDL-C, and VLDL-C, alongside markedly reduced HDL-C, compared to healthy controls. This pattern aligns with the well-characterized dyslipidemia of T2DM, which Strikić et al. described in their narrative review as being primarily defined by elevated triglycerides, reduced HDL cholesterol, and the predominance of small, dense LDL particles — a combination that collectively promotes atherosclerosis and substantially amplifies cardiovascular risk [10]. The review further emphasized that cardiovascular disease remains the leading cause of morbidity and mortality among individuals with T2DM, making the identification and management of dyslipidemia a critical clinical priority [10].

From a pathophysiological standpoint, the observed lipid disturbances are mechanistically rooted in insulin resistance. Adiels et al. demonstrated that the fundamental defect in metabolic syndrome-associated dyslipidemia is the overproduction of large VLDL1 particles by the liver, driven by increased free fatty acid flux under conditions of insulin resistance, which subsequently initiates a cascade of interrelated lipoprotein changes resulting in elevated remnant particles, smaller and denser LDL, and lower HDL-C concentrations [11]. This mechanistic framework explains the comprehensive pattern of dyslipidemia observed in our diabetic cohort.

One of the central findings of the present study was the significant positive correlation between HbA1c and all atherogenic lipid fractions, alongside a significant negative correlation with HDL-C. These associations highlight the biochemical interplay between chronic hyperglycemia and lipid dysregulation. Consistent with our findings, Joseph et al. analyzed the relationship between HbA1c and lipid profile across a large hospital-based population and reported a significant positive correlation between HbA1c and TC, LDL-C, and VLDL-C, while demonstrating a significant negative correlation between HbA1c and HDL-C, concluding that worsening glycemic control is associated with a progressively higher risk of cardiovascular complications [12].

The inverse relationship between HbA1c and HDL-C observed in our study reflects the well-documented suppression of reverse cholesterol transport under conditions of chronic hyperglycemia. Elevated glucose promotes glycation of HDL apolipoproteins, impairing HDL functionality and accelerating its catabolism. A large nationwide cross-sectional study conducted among 30,195 Thai adults with T2DM further confirmed that higher HDL-C levels were significantly associated with better glycemic control, with a consistent negative linear relationship demonstrated in male participants (adjusted $\beta = -0.076$, $p < 0.001$), reinforcing the protective role of adequate HDL-C in glycemic regulation [13].

The positive correlation observed between HbA1c and TG in the present study is mechanistically supported by the effect of insulin deficiency on lipoprotein lipase activity. In states of poor glycemic control, reduced lipoprotein lipase activity impairs TG clearance while simultaneously stimulating hepatic VLDL overproduction, resulting in hypertriglyceridemia. The positive correlation between HbA1c and LDL-C is similarly explained by the reduction in LDL receptor expression under hyperglycemic conditions, which diminishes LDL clearance from circulation and raises its plasma concentration [14]. A cross-sectional study evaluating HbA1c and lipid parameters among T2DM patients in a tertiary care hospital in India reported a significant positive correlation of TC, LDL-C, VLDL-C, and TG with both HbA1c and duration of diabetes ($p < 0.05$), a finding that closely mirrors the correlations observed in the present study [14].

The combined presence of elevated HbA1c and an atherogenic lipid profile carries significant prognostic implications for cardiovascular outcomes in patients with T2DM. Pei et al., in an analysis of data from the ACCORD study, examined the relationship between HbA1c variability and major cardiovascular adverse outcomes in high-risk T2DM patients, and demonstrated that glycemic control status significantly influenced the risk of cardiovascular events and all-cause mortality, with optimal glycemic management being associated with a meaningful reduction in these risks [15]. These findings reinforce the argument that simultaneous monitoring and control of both HbA1c and lipid parameters represent an indispensable dual strategy in reducing the burden of cardiovascular disease in this patient population.

Several limitations of the present study warrant acknowledgment. The cross-sectional design precludes the establishment of causal relationships between glycemic control and lipid abnormalities. The study was conducted at a single tertiary care center in India, which may limit the generalizability of the findings to the broader population. Additionally, confounding variables such as physical activity levels, dietary habits, and medication adherence were not systematically controlled for, and these factors may have influenced both HbA1c and lipid values. Future longitudinal, multicenter studies with larger sample

sizes and comprehensive covariate adjustment are warranted to better characterize the causal dynamics between glycemic control and dyslipidemia in Indian T2DM patients.

Conclusion

The findings of the present study demonstrate that type 2 diabetes mellitus is associated with significant alterations in both glycemic and lipid metabolism. Patients with T2DM exhibited markedly elevated HbA1c levels, reflecting poor long-term glycemic control, alongside a distinctly atherogenic lipid profile characterized by raised total cholesterol, triglycerides, LDL-C, and VLDL-C, with a concomitant reduction in the cardioprotective HDL-C fraction. Furthermore, the significant positive correlations observed between HbA1c and atherogenic lipid parameters, along with its negative correlation with HDL-C, highlight the intricate interplay between chronic hyperglycemia and dyslipidemia, both of which synergistically contribute to the heightened cardiovascular risk seen in this patient population. These results reinforce the clinical importance of routine and simultaneous monitoring of HbA1c and lipid profile in the management of T2DM, as such an approach may facilitate early identification of cardiovascular risk factors and guide more comprehensive therapeutic interventions aimed at reducing the burden of diabetes-related complications.

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