ORIGINAL ARTICLE

Comparative Analysis of Dyslipidemia and Atherogenic Indices among Controlled and Uncontrolled Diabetes Mellitus Patients at a Tertiary Care Center

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ABSTRACT

Objective: To compare the prevalence, patterns, and atherogenic indices associated with dyslipidemia among patients with controlled and uncontrolled diabetes mellitus.

Study Design: Cross-sectional observational study.

Place and Duration of Study: The study was conducted at the Punjab Institute of Cardiology, Lahore, from October 2 to December 29, 2025.

Materials and Methods: Fasting blood samples of 503 eligible patients were analyzed for their lipid profiles and HbA1c levels. Participants were grouped into controlled and uncontrolled diabetes groups based on ADA criteria, while dyslipidemia classification was made using NCEP ATP III cutoffs. Clinical characteristics, lipid profile parameters, and atherogenic indices were compared between the two groups using independent t-test for parametric variables while Mann-Whitney U test was employed for non-parametric variables. Logistic regression analysis was used to assess the association of specific dyslipidemia phenotypes with glycemic control status.

Results: The overall prevalence of dyslipidemia in the study population was 87.3%, which was significantly higher in the uncontrolled group (89.5% vs. 82.8%, χ^2=4.510, *p=0.034*). Patients with uncontrolled diabetes showed significantly higher median values of HbA1c, triglycerides, duration of diabetes, Castelli Risk Index I and II, atherogenic coefficient, and cardiometabolic index, while HDL-C levels were lower. Uncontrolled diabetes was a strong, independent predictor of the combined dyslipidemia phenotype (OR: 3.43, 95% CI: 1.61–7.31, *p=0.001*), while LDL-C did not differ significantly between groups.

Conclusion: Uncontrolled diabetes mellitus is strongly associated with a more atherogenic lipid profile. Atherogenic indices should be incorporated into routine lipid profile along with a detailed assessment of dyslipidemia patterns, especially in the diabetic population.

Key Words: *Diabetes Mellitus, Dyslipidemias, Atherosclerosis, Lipoproteins, Cardiometabolic Risk Factors.*

Introduction

Dyslipidemia is defined as an abnormal concentration of lipids and lipoproteins in the bloodstream.[1] These lipids primarily include triglycerides and cholesterol, which are absorbed from the intestine and transported in circulation through lipoproteins such as chylomicrons, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL) for tissue utilization, storage, and excretion.[1] Any imbalance or defect altering the lipid metabolism pathway may result in a disturbed lipid profile or dyslipidemia.[2]

Dyslipidemia is a major risk factor for cardiovascular disease, which is responsible for an estimated 19.8 million deaths globally.[3] Elevated levels of these lipids, particularly high LDL and low HDL can result in the deposition of fats in the vessels, resulting in narrowing of the vessel-lumen and compromised tissue perfusion.[4] The formation of atherosclerotic plaques due to fat deposition can result in ischemia and stroke, both of which may be fatal.[4]

Dyslipidemia can develop due to hereditary/genetic defects (primary dyslipidemia) or due to poor lifestyle choices and underlying medical conditions (secondary dyslipidemia).[5] Of the many disorders that can disturb lipid metabolism, diabetes mellitus is of prime importance due to its high prevalence and strong association with dyslipidemia and cardiovascular disease.[6] Insulin resistance increases adipose tissue lipolysis, leading to enhanced free

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Received: February 25, 2026 ; Revised: June 05, 2026
Accepted: June 15, 2026
https://doi.org/10.57234/jiimc.september26.2939

fatty acid (FFA) delivery to the liver and increased hepatic VLDL synthesis, which contributes to dyslipidemia.[7] Furthermore, VLDL produced transfers triglycerides to HDL and LDL forming small dense LDL, which is readily oxidized and can deposit in arteries causing atherosclerosis.[7,8] All of these changes can result in a characteristic lipid profile pattern known as diabetic dyslipidemia.

Pakistan bears one of the highest burdens of diabetes mellitus globally, with an estimated 34.5 million adults suffering from the condition.[9] Pakistan also has above global average mortality rates from CVD and other heart-related illnesses.[10] Studies conducted in different health-care centers in the country have consistently reported a high frequency of dyslipidemia among individuals with diabetes mellitus.[11,12] The high prevalence of dyslipidemia among the Pakistani diabetic population warrants further investigation into patterns of lipid abnormalities and their association with glycemic control.

Despite the high frequency of dyslipidemia among Pakistani adults in general and among diabetic individuals, much of the existing literature is either dated or does not compare lipid abnormalities between controlled and uncontrolled diabetes, particularly in the population of Lahore. This study aims to address these gaps by comparing the prevalence and patterns of dyslipidemia along with associated cardiovascular risk between individuals with controlled and uncontrolled diabetes mellitus.

Materials and Methods

This cross-sectional observational study was conducted at the Department of Pathology, Punjab Institute of Cardiology, Lahore, Pakistan, from October 2 to December 29, 2025. Ethical approval for the study was obtained from the institutional ethical review committee (Ref. No: RTPGME-Research-358; August 18, 2025).

The sample size was calculated using the Kirkwood's formula for comparing two independent continuous means $n = 2\sigma^2 (Z\alpha + Z\beta)^2 / d^2$. Sample size estimation was based on previously reported HDL-C values among controlled and uncontrolled type 2 diabetes mellitus patients reported by Rashid and Haider (1.05 ± 0.27 mmol/L vs. 0.91 ± 0.23 mmol/L).[13] Assuming a two-sided significance level of 5% ($Z\alpha$ = 1.96) and 80% power ($Z\beta$ = 0.84), the minimum required sample size was calculated to be 51 participants per group, yielding a total minimum sample size of 102 participants.

Consecutive sampling was employed. Patients with diagnosed cases of diabetes mellitus who visited the diagnostic laboratory for routine testing and fulfilled the study eligibility criteria were recruited. The inclusion criteria consisted of individuals with an established diagnosis of type 2 diabetes mellitus (T2DM) who were on an overnight fast and were not taking any lipid-lowering drugs. Patients with chronic liver disease, chronic kidney disease, thyroid disorders, known familial dyslipidemias, pregnancy, nephrotic syndrome, corticosteroid use, alcohol abuse, or other identifiable causes of secondary dyslipidemia were excluded from the study.

After obtaining written informed consent, participants were interviewed using a structured questionnaire to collect demographic and clinical information, including age, gender, duration of diabetes, comorbidities, antidiabetic medication or insulin use, physical activity, smoking status, and family history of diabetes mellitus or cardiovascular disease. Height and weight were measured using standard procedures, and body mass index (BMI) was calculated as weight (kg)/height (m^2).

Blood samples for HbA1c estimation were collected in EDTA vacutainers, while samples for lipid profile analysis were collected in lithium heparin tubes. Samples were transported to the laboratory within one hour of collection at room temperature (25°C). Blood samples in heparin vacutainers were centrifuged at 3000 rpm for 10 minutes prior to analysis. Lipid parameters were analyzed using the Atellica CH Analyzer, while HbA1c levels were measured using the Bio-Rad D-100 Hemoglobin Testing System. Patient lab results were later retrieved using the Hospital Information and Management System (HIMS).

Based on HbA1c levels, patients were grouped into controlled and uncontrolled diabetes according to the guidelines set by the American Diabetes Association (i.e., HbA1c < 7 = controlled diabetes, HbA1c ≥ 7 = uncontrolled diabetes .[14] Dyslipidemia was defined according to NCEP ATP III cut-off values (Triglycerides: ≥150 mg/dL, HDL-C: <40 mg/dL in men, <50 mg/dL in women).[15] Atherogenic dyslipidemia was defined as the presence of

https://doi.org/10.57234/jiimc.september26.2939

hypertriglyceridemia and low HDL-C. Combined dyslipidemia was defined as the combined presence of increased triglycerides, total cholesterol, LDL-C, and low HDL-C levels.

Laboratory results along with patient demographics were entered onto excel spreadsheets, which were later exported to SPSS for analysis. Clinical characteristics, lipid profile parameters and atherogenic indices including non-HDL cholesterol, remnant cholesterol, the atherogenic index of plasma (AIP), Castelli risk index I, Castelli risk index II, the atherogenic coefficient (AC), and the cardiometabolic index (CMI) were compared between the two groups using independent t-test for parametric variables and Mann-Whitney U test for non-parametric variables. Chi-square was used to assess the association of dyslipidemia in relation to glycemic control. Dyslipidemia was stratified into specific phenotypes (e.g., isolated High TG, Combined Dyslipidemia, etc.), and multivariable binary logistic regression analysis was employed to assess the association of uncontrolled diabetes with specific dyslipidemia phenotypes after adjusting for confounding variables.

Results

A total of 503 eligible patients with type 2 diabetes mellitus were included in the study, comprising 47.3% males and 52.7% females. Approximately two-thirds of the study population (66.4%) had poor glycemic control (HbA1c ≥7%), while 33.6% had controlled diabetes (HbA1c <7%). Overall, 87.3% of participants demonstrated some form of dyslipidemia.

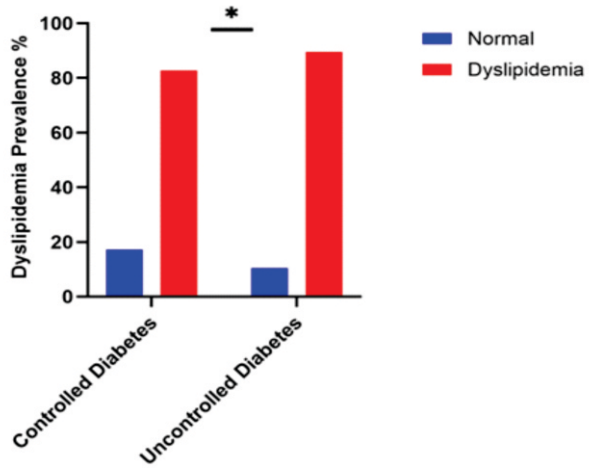

Figure 1: Comparison of Dyslipidemia Prevalence Between Controlled and Uncontrolled Diabetes.

Comparison of dyslipidemia frequency in controlled and uncontrolled diabetes is shown in Figure 1. Individuals with poor glycemic control had a higher prevalence of dyslipidemia (89.5 %) compared to those with adequate glycemic control (82.8%). Uncontrolled diabetes was found to be significantly associated with dyslipidemia $\chi^2 = 4.510$, $p = 0.034$.

Table I: Comparison of lipid parameters, atherogenic indices, and clinical characteristics between controlled and uncontrolled type 2 diabetes mellitus (n = 503)

Parameter	*Controlled Diabetes (n=169)*	*Uncontrolled Diabetes (n=334)*	*p-value*
Parametric Variables (Mean ±SD)			
Total Cholesterol (mg/dl)	178.81 ± 43.20	185.09 ± 39.14	0.101
LDL-Chol (mg/dl)	108.61 ± 35.63	112.58 ± 32.64	0.212
Non-HDL Chol (mg/dL)	135.44 ± 41.45	144.63 ± 37.86	**0.013**
AIP (Atherogenic index of Plasma)	0.177 ± 0.26	0.297 ± 0.26	**<0.001**
Age (Years)	53.89 ± 11.46	50.96 ± 10.87	0.005
Non-Parametric Variables (Median [IQR])			
HbA1c	6.50 (0.60)	8.75 (2.70)	**<0.001**
Triglyceride (mg/dl)	145.00 (92.00)	170.50 (111.00)	**<0.001**
HDL-Chol (mg/dl)	43.00 (12.00)	39.00 (11.00)	**<0.001**
BMI	29.39 (6.64)	29.70 (6.45)	0.784
Duration	3.00 (7.68)	6.00 (8.00)	**<0.001**
Non-Parametric Variables (Median [IQR])			
Castelli Risk Index I	4.12 (1.51)	4.74 (1.65)	**<0.001**
Castelli Risk Index II	2.57 (1.08)	2.81 (1.15)	**<0.001**
Remnant Cholesterol	26.00 (20.50)	29.00 (22.25)	0.180
Atherogenic Coefficient	3.12 (1.51)	3.74 (1.65)	**<0.001**
Cardiometabolic Index	100.78 (85.86)	131.54 (109.46)	**<0.001**

Data are presented as mean ± standard deviation (SD) for parametric variables and median (interquartile range [IQR]) for non-parametric variables. Parametric variables were analyzed using the independent t-test, while non-parametric variables were analyzed using the Mann-Whitney U test. Statistical significance was set at p <0.05.

Abbreviations: T2DM = Type 2 Diabetes Mellitus; LDL-Chol = Low-Density Lipoprotein Cholesterol; HDL-Chol = High-Density Lipoprotein Cholesterol; HbA1c = Glycated Hemoglobin; BMI = Body Mass Index; AIP = Atherogenic Index of Plasma; Castelli Risk Index I = Total Cholesterol/HDL-Cholesterol ratio; Castelli Risk Index II = LDL-Cholesterol/HDL-

Cholesterol ratio.

Phenotypic stratification demonstrated atherogenic dyslipidemia (High TG + Low HDL-C) to be the most frequent pattern in both controlled (23.1%) and uncontrolled diabetes mellitus (26.3%). Isolated low HDL levels were more prevalent in patients with adequate glycemic control (21.9%) compared to poor glycemic control (17.4%). High frequency of combined dyslipidemia was observed in individuals with uncontrolled diabetes (14.7%) vs. controlled diabetes (5.3%). The distribution of different patterns of dyslipidemia in relation to glycemic control is shown in Figure 2.

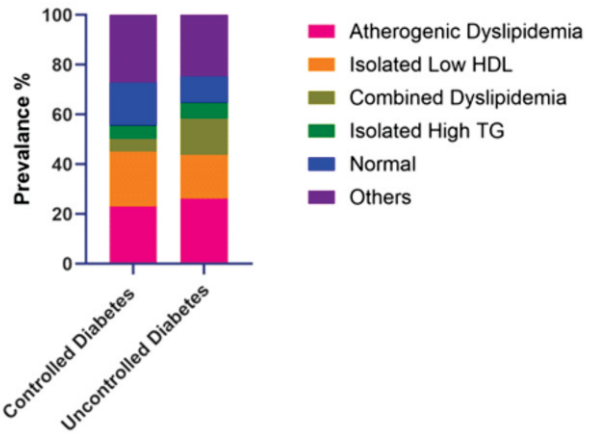

Figure 2: Distribution Of Dyslipidemia Patterns in Controlled and Uncontrolled Diabetes

The results of the logistic regression analysis are presented in Table II. Female gender was found to be associated with higher odds of having isolated low HDL (OR=1.92, p = 0.009), whereas uncontrolled diabetes was strongly associated with increased odds of combined dyslipidemia (OR=3.43, p = 0.001). Longer duration of diabetes slightly reduced the odds of isolated low HDL (OR=0.96, p = 0.036).

Table II: Multivariable Binary Logistic Regression Analysis Showing Independent Predictors of Dyslipidemia Phenotypes Among Patients with Diabetes Mellitus (n = 503)

Predictor	*Atherogenic Dyslipidemia OR (95% CI)*	*Isolated Low HDL OR (95% CI)*	*Isolated High TG OR (95% CI)*	*Combined Dyslipidmia OR (95% CI)*
Age (years)	0.98 (0.96–1.00)	1.04 (1.01–1.06) *	0.98 (0.95–1.01)	0.98 (0.96–1.01)
Female gender	1.06 (0.70–1.61)	1.92 (1.18–3.13) *	0.31 (0.14–0.71)	1.48 (0.83–2.62)
Duration of DM (years)	1.01 (0.98–1.04)	0.96 (0.92–0.99) *	—	0.94 (0.89–0.99)
Uncontrolled diabetes	1.10 (0.70–1.71)	0.91 (0.56–1.47)	1.03 (0.45–2.33)	3.43 (1.61–7.31) *
Exercise	1.20 (0.79–1.82)	1.26 (0.79–2.02)	—	—
BMI (kg/m²)	1.02 (0.99–1.04)	0.98 (0.94–1.02)	—	0.996 (0.98–1.02)

- Bolded ORs are statistically significant (p < 0.05).
- "—" indicates the predictor was not included in that model due to low events.
- *Statistically significant at p <0.05 in multivariable binary logistic regression analysis.

Discussion

This study demonstrates a high prevalence of dyslipidemia in patients with T2DM, specifically highlighting the profound impact of glycemic control. The principal finding is that uncontrolled diabetes is strongly associated with a more atherogenic dyslipidemic profile, as reflected by elevated composite risk indices and high-risk dyslipidemia phenotypes like combined dyslipidemia. These may provide additional insight into atherogenic risk profiles beyond isolated LDL-C levels.

The major abnormalities in the lipid profile of both controlled and uncontrolled diabetes were elevated triglycerides and low HDL-C. The observed pattern of lipid abnormalities, including hypertriglyceridemia and low HDL-C, is consistent with diabetic dyslipidemia. Although LDL-C differences were not significant, diabetic dyslipidemia is often associated with qualitative LDL abnormalities, particularly small dense LDL particles, which may contribute to residual atherogenic risk.[16] The high prevalence of dyslipidemia (87.3%) in our study population aligns with previous studies that have consistently shown > 90% prevalence of dyslipidemia among Pakistani diabetic population.[11,17] This is in agreement with the

https://doi.org/10.57234/jiimc.september26.2939

pathophysiological models where insulin resistance in diabetes drives hepatic VLDL overproduction and impaired lipolysis.[18]

In our study, all of the evaluated atherogenic indices were elevated in individuals with poor glycemic control. This aligns with recent clinical evidence that AIP and TyG index are significantly higher in the uncontrolled diabetic group, suggesting an association with an adverse atherogenic risk profile.[19] Elevated atherogenic ratios (TG/HDL-C, LDL-C/HDL-C, AIP) have also been observed in uncontrolled diabetes mellitus, which highlights their role as surrogate markers of lipid metabolism abnormalities that accompany worsening glycemic control.[20] High levels of Non-HDL-C in uncontrolled diabetes represent the total burden of atherogenic lipoproteins, while remnant cholesterol, carried by triglyceride-rich lipoproteins, is increasingly recognized as an independent risk factor for atherosclerosis.[21] These findings highlight the importance of non-LDL-C atherogenic fractions, which may represent additional markers of dyslipidemia burden that are often overlooked when deciding the treatment regimen and management of dyslipidemias in diabetic individuals, as these lipids may carry residual cardiovascular risk.[22] Nonetheless, in the absence of direct cardiovascular outcome assessment, these findings should be interpreted as indicators of an unfavourable lipid risk profile rather than predictors of cardiovascular events.

A key finding of our study is the association of uncontrolled diabetes with severe dyslipidemia phenotypes like combined dyslipidmia (OR=3.43). A study conducted in Riyadh also found significantly higher triglycerides, cholesterol, and lower HDL levels in admitted patients with poor glycemic control, while the differences in LDL-C were not statistically significant.[23] These findings indicate that poor glycemic control increases the likelihood of developing these highly atherogenic phenotypes characterized by elevated triglycerides, low HDL, and increased cholesterol levels.

Our regression analysis also revealed that female gender was associated with higher odds of developing isolated low HDL and decreased odds of having a normal lipid profile. This is supported by global epidemiological data that shows a higher prevalence of low HDL levels in women compared to men.[24] This increased prevalence of specific dyslipidemia phenotypes might be due to sex related differences in lipid metabolism, fat distribution, hormonal differences, and socioeconomic determinants.

Our study found no association of BMI with dyslipidemia. Prior research has also shown a limited ability of BMI to correlate with the severity of CVD and other metabolic disorders, including T2DM.[25] This indicates that insulin resistance may have a more direct effect on deranged lipid metabolism than adiposity alone. However, these associations may be influenced by potential confounders such as menopause, obesity-related heterogeneity, socioeconomic factors, and treatment adherence, which were not fully controlled in the present analysis.

The findings of this study highlight that achieving glycemic control (e.g., HbA1c < 7%) is associated with a more favourable lipid profile and reduced burden of atherogenic dyslipidemia. The results of this study also support the perspective that standard lipid panels and isolated LDL-C targets may not fully capture the complex lipid derangements present in uncontrolled diabetes. Incorporating calculated atherogenic indices into routine lab reporting could provide clinicians with a more comprehensive overview of a patient's lipid status.

Future studies should focus on prospective longitudinal designs to determine whether these atherogenic indices and dyslipidemia phenotypes translate into actual cardiovascular outcomes. Further research is also needed to evaluate optimal lipid-lowering strategies in different dyslipidemia phenotypes among diabetic patients, particularly in relation to glycemic control status.

Limitations

The single center nature of this study limits the generalizability of its findings to the general population. Also, the information regarding specific diabetic drugs (e.g., SGLT2 inhibitors, GLP-1 RAs, which can affect lipid levels), dietary habits, and level of physical activity was limited, which are potential confounding factors.

Conclusion

In conclusion, poor glycemic control is associated with more severe forms of dyslipidemia. Patients

with uncontrolled diabetes demonstrate a more atherogenic lipid profile and worse cardiometabolic indices. These findings suggest that dyslipidemia in diabetes is more pronounced in uncontrolled glycemic states and may warrant more comprehensive lipid assessment, including evaluation of specific dyslipidemia patterns and calculated atherogenic indices, in addition to standard lipid parameters. Improved glycemic control may help reduce the severity of dyslipidemia and associated cardiometabolic risk.

Acknowledgements The authors are thankful to the staff of the diagnostic lab, Punjab Institute of Cardiology for assisting in the data collection process. The authors also acknowledge the contributions of M. Abdul Khaliq and Amna Bibi, laboratory trainees, for their assistance in data collection and patient sampling.

Disclaimer The research was conducted independently and the views expressed in this study, regarding the interpretation and conclusions of results do not represent the official position or policy of Punjab Institute of Cardiology.

Conflict of Interest All the authors declare that there is no conflict of interest that could have influenced the work.

Funding Disclosure This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. Laboratory tests were conducted as part of routine patient care, and the associated costs were covered by the patients. No additional tests were carried out specifically for the study.

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CONFLICT OF INTEREST

Authors declared no conflicts of Interest.

GRANT SUPPORT AND FINANCIAL DISCLOSURE

Authors have declared no specific grant for this research from any funding agency in public, commercial or nonprofit sector.

DATA SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon request.