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Original Research Article

THE ASSOCIATION BETWEEN GLYCEMIC CONTROL AND LIPID PROFILE IN TREATED TYPE 2 DIABETES MELLITUS

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Abstract: ***Background:*** Dyslipidemia is a well-established complication of diabetes mellitus; however, its specific correlation with the degree of glycemic control requires further elucidation. ***Aim:*** To study the levels of various lipid parameters and atherogenic indices in patients with type 2 diabetes mellitus stratified by different levels of glycemic control. ***Material and Methods***: In this cross-sectional study, 36 diagnosed patients with type 2 diabetes mellitus on standard treatment (including lipid-lowering drugs) were divided into two groups based on glycated hemoglobin (HbA1c) levels: Group I (HbA1c ≤ 8%, n=17) and Group II (HbA1c > 8%, n=19). Their fasting lipid profiles were analyzed and compared. ***Result***: A significant positive correlation was observed between HbA1c and serum triglycerides (TG) (r=0.323, p=0.05) in the total cohort. No statistically significant correlations were found between HbA1c and total cholesterol (r=0.262, p=0.12), LDL-C (r=0.061, p=0.72), or HDL-C (r=0.080, p=0.64). ***Conclusion***: Poor glycemic control in type 2 diabetes mellitus is specifically associated with hypertriglyceridemia, while other conventional lipid parameters appear unaffected in this treated cohort. This suggests that optimizing glycemic control may help mitigate cardiovascular risk by directly influencing triglyceride levels.

Keywords: Type 2 Diabetes Mellitus, Dyslipidemia, Glycemic Control, HbA1c, Hypertriglyceridemia

INTRODUCTION

Diabetes mellitus is a prevalent global metabolic disorder, characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. This dysregulation leads to disturbances in carbohydrate, fat, and protein metabolism [1,2]. Chronic hyperglycemia is associated with long-term organ damage and dysfunction, particularly affecting the eyes, kidneys, nerves, heart, and blood vessels [3]. Among various complications, dyslipidemia is a critical modifiable cardiovascular risk factor in individuals with type 2 diabetes [4,5]. The characteristic lipid abnormalities in this condition—elevated triglycerides (TG), low high-density lipoprotein cholesterol (HDL-C), and a preponderance of small, dense low-density lipoprotein (LDL) particles—are largely driven by increased free fatty acid flux secondary to insulin resistance, exacerbated by pro-inflammatory adipokines [6,7].

Glycated hemoglobin (HbA1c) serves as a key diagnostic and monitoring indicator for long-term glycemic control. It forms through a non-enzymatic glycation process, reflecting integrated blood glucose levels over the preceding 6–8 weeks, and is typically maintained below 5.8% in normoglycemic individuals [8]. Hypertriglyceridemia, defined as a fasting TG level of ≥150 mg/dL, is a significant component of diabetic dyslipidemia and an independent risk factor for pancreatitis and cardiovascular disease [9].

While the association between diabetes and dyslipidemia is well-known, the specific relationship between the degree of glycemic control and individual lipid parameters, especially in patients already on lipid-lowering therapy, remains a pertinent clinical question. This study aims to investigate this relationship by correlating HbA1c

levels with a detailed lipid profile and atherogenic indices in patients with type 2 diabetes.

MATERIAL AND METHODS

Study Population and Design

A cross-sectional study was conducted on 36 diagnosed patients with type 2 diabetes mellitus, all of whom were on standard anti-diabetic and lipid-lowering therapy. Participants were stratified into two groups based on their glycated hemoglobin (HbA1c) levels: Group I (HbA1c ≤ 8%) and Group II (HbA1c > 8%). A comparative analysis of their fasting lipid profiles was performed.

Biochemical Investigations

Venous blood samples were collected after a 10–12 hour overnight fast. Serum was separated and analyzed using an EM-360 fully automated analyzer.

1. Measurement of Glycated Hemoglobin (HbA1c)

HbA1c was quantified using a latex agglutination inhibition rate assay.

- Principle: Total hemoglobin and HbA1c concentrations were measured separately. After red blood cell lysis and protease hydrolysis, total hemoglobin was converted to alkaline haematin for photometric measurement. For HbA1c, an inhibition immunoassay was employed, where HbA1c in the sample competes with the agglutination reagent for antibody-binding sites.
- Calibration and Quality Control: Calibration was performed using the Randox Haemoglobin A1c Calibrator series (Levels 1–6, Lot no. 1600HA). Daily assay performance was verified with Randox Haemoglobin A1c control materials (Levels 1 & 2, Lot no. 14411).
- Sample Pretreatment: Whole blood (10 µL) was mixed with 400 µL of hemoglobin denaturant reagent, incubated at room temperature for 5 minutes, and subsequently analyzed.

2. Measurement of Fasting Lipid Profile

Serum total cholesterol, triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were measured. Very-low-density lipoprotein cholesterol (VLDL-C) was calculated using the formula: TG/5.

- Equipment and Quality Control: Analyses were performed using a Beckman Coulter multicalibrator (Lot no. 0117) with Randox quality control materials.
- Triglycerides: Measured by the GPO-PAP enzymatic colorimetric method.
- Total Cholesterol: Measured by the CHOD-PAP enzymatic colorimetric method.
- HDL-C: Determined by a direct homogeneous method using a detergent that selectively solubilizes HDL particles.

Statistical Analysis

Data were analyzed using SPSS version 20.0. Continuous data were expressed as mean ± standard deviation (SD). The student's t-test was used to compare parameters between the two HbA1c groups. Pearson's correlation coefficient was applied to assess the relationship between HbA1c and lipid parameters. A p-value of < 0.05 was considered statistically significant.

RESULTS

Comparison of Lipid Profiles Stratified by Glycemic Control The demographic and biochemical characteristics are summarized in Table 1. Group II (poorer glycemic control) exhibited non-significant trends toward higher mean levels of total cholesterol, triglycerides, VLDL-C, LDL-C, and all atherogenic indices compared to Group I. The difference in HbA1c between the groups was highly significant ($p < 0.01$).

Correlation of HbA1c with Lipid Parameters: Correlation analysis for the total cohort (Table 2) revealed a significant positive correlation between HbA1c and both serum triglycerides ($r = 0.323$, $p = 0.05$) and VLDL-C ($r = 0.323$, $p = 0.05$). No significant correlations were observed between HbA1c and total cholesterol, HDL-C, LDL-C, or any atherogenic index.

Table 1: Comparison of Lipid Parameters and Atherogenic Indices Between HbA1c Groups

Parameter	Total Cohort (N=36)	Group I (HbA1c ≤ 8%) (n=17)	Group II (HbA1c > 8%) (n=19)	p-value
HbA1c (%)	8.49 ± 3.0	5.62 ± 0.91	11.05 ± 1.80	**0.00**
Total Cholesterol (mg/dL)	176.16 ± 35.27	163.82 ± 36.44	187.21 ± 31.08	0.28
Triglycerides (TG) (mg/dL)	158.25 ± 100.05	132.47 ± 40.76	181.31 ± 129.60	0.21
HDL-C (mg/dL)	41.19 ± 9.4	39.82 ± 8.77	42.42 ± 10.04	0.45
VLDL-C (mg/dL)	31.65 ± 20.0	26.49 ± 8.15	36.26 ± 25.92	0.21
LDL-C (mg/dL)	103.32 ± 33.3	97.50 ± 28.59	108.52 ± 37.12	0.82
Atherogenic Indices				
LDL/HDL Ratio	2.60 ± 1.09	2.49 ± 0.74	2.70 ± 1.35	0.73
TG/HDL Ratio	4.17 ± 4.0	3.37 ± 0.99	4.89 ± 5.49	0.33
Total Cholesterol/HDL Ratio	4.44 ± 1.26	4.17 ± 0.81	4.68 ± 1.54	0.57

Table 2: Correlation Analysis Between HbA1c and Lipid Parameters (Total Cohort, n=36)

Parameter	Correlation Coefficient (r)	p-value
Total Cholesterol	0.262	0.12
Triglycerides (TG)	**0.323**	**0.05***
HDL-C	0.080	0.64
VLDL-C	**0.323**	**0.05***
LDL-C	0.061	0.72
LDL/HDL Ratio	-0.009	0.95
TG/HDL Ratio	0.281	0.09
Total Cholesterol/HDL	0.172	0.31

*r = Pearson's correlation coefficient. * Denotes statistical significance ($p < 0.05$). *

DISCUSSION

This study demonstrates a specific and significant correlation between poor long-term glycemic control (higher HbA1c) and elevated triglyceride levels in

patients with type 2 diabetes who are on concurrent lipid-lowering therapy. This finding persists despite the lack of significant differences in mean lipid values between the HbA1c-stratified groups, a result likely confounded by the uniform use of statins or other lipid-modifying drugs in the cohort. The pathophysiological link between hyperglycemia and hypertriglyceridemia is robust. Insulin resistance and relative insulin deficiency, the hallmarks of poor glycemic control, promote hepatic VLDL overproduction through several mechanisms: increased free fatty acid delivery from adipose tissue, enhanced de novo lipogenesis, and increased synthesis and stabilization of apolipoprotein B100 [10-12]. Concurrently, the catabolism of triglyceride-rich lipoproteins (TRLs) is impaired due to reduced activity of lipoprotein lipase (LPL) and decreased hepatic clearance of remnant particles [13,14]. This dual defect leads to accumulation of TRLs, manifesting as hypertriglyceridemia and elevated VLDL-C. Our finding that conventional parameters like LDL-C and HDL-C did not correlate with HbA1c is clinically insightful. It suggests that in patients on standard therapy, the dyslipidemia most tightly coupled to glycemic status is the triglyceride-VLDL axis. This residual dyslipidemia may contribute to residual cardiovascular risk even when LDL-C targets are met. The TG/HDL ratio, often considered a proxy for insulin resistance and atherogenic small dense LDL, showed a positive but non-significant trend ($p=0.09$), potentially underscoring this relationship. The clinical implication is twofold. First, achieving optimal glycemic control remains paramount, not only for microvascular outcomes but also for mitigating this specific aspect of atherogenic dyslipidemia. Second, in patients with poor glycemic control (HbA1c >8%) and persistent hypertriglyceridemia, the addition of triglyceride-specific therapies (e.g., fibrates, high-dose omega-3 fatty acids) to statin therapy may be warranted for comprehensive cardiovascular risk reduction [15,16].

Limitations of this study include its cross-sectional design, small sample size, and the confounding effect of lipid-lowering medications. The absence of a drug-naïve control group limits the ability to observe the full, unmodified relationship between glycemia and lipids.

CONCLUSION

In patients with type 2 diabetes on lipid-lowering therapy, the degree of glycemic control maintains a specific and significant positive correlation with serum triglyceride and VLDL-C levels. This association underscores hypertriglyceridemia as a persistent, glycemic-sensitive risk marker. Management of diabetic dyslipidemia should, therefore, involve a dual focus: stringent glycemic control to address triglyceride metabolism and appropriate pharmacotherapy to manage all components of cardiovascular risk.

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DISCLOSURE STATEMENT

The authors have no financial or personal relationships that could be perceived as influencing the work reported in this paper.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this paper.

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