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Postprandial Hypertriglyceridemia and its Association with Carotid Intima-Media Thickness in Patients with Type 2 Diabetes Mellitus: An Observational Study

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ABSTRACT:

Background: Dyslipidemia is a well-recognized metabolic disturbance in type 2 diabetes mellitus (T2DM), and fasting lipid measurement may underestimate atherogenic risk because most of the day is spent in a postprandial state. Postprandial hypertriglyceridemia (PPHTG) may contribute to subclinical atherosclerosis, but its relationship with carotid intima-media thickness (CIMT) is not routinely assessed in clinical practice.

Objective: To evaluate postprandial triglyceride levels and their association with CIMT, measured by high-resolution B-mode ultrasonography, in patients with T2DM.

Methods: In this observational study conducted over 18 months in the Department of General Medicine, 50 adults with T2DM (age 18–65 years) were enrolled after excluding type 1 diabetes, diabetes duration under one year, established cardiovascular or ischemic heart disease, and hypertension or hyperlipidemia. Fasting triglycerides (FTG) and 4-hour postprandial triglycerides (PPTG) were measured, and participants were stratified into three groups: Group A (FTG <150 mg/dL and PPTG <150 mg/dL, n=15), Group B (FTG <150 mg/dL and PPTG ≥150 mg/dL, n=15), and Group C (FTG ≥150 mg/dL and PPTG ≥150 mg/dL, n=20). CIMT was measured by high-resolution B-mode ultrasonography. Group comparisons used chi-square and ANOVA, and associations with CIMT were assessed by Pearson correlation; $p<0.05$ was considered

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significant.

Results: Mean CIMT increased progressively across groups (Group A 0.97±0.32 mm, Group B 1.68±0.59 mm, Group C 1.91±0.49 mm; p<0.001). PPTG correlated strongly with CIMT (r=0.621, p<0.001), as did duration of diabetes (r=0.681, p<0.001) and age (r=0.622, p<0.001); FTG showed a weaker but significant correlation (r=0.327, p=0.021). Notably, Group B — with normal fasting but elevated postprandial triglycerides — already showed significantly increased CIMT compared with Group A, indicating that isolated postprandial hypertriglyceridemia is associated with early vascular change. Fasting and postprandial blood glucose, HbA1c, blood pressure, LDL, HDL, and BMI showed no significant association with CIMT (all p>0.05).

Conclusion: Postprandial hypertriglyceridemia, even in the presence of normal fasting triglycerides, is significantly associated with increased CIMT in patients with T2DM and appears to be a more sensitive marker of subclinical atherosclerosis than conventional fasting lipid or glycemic parameters. Postprandial triglyceride assessment, alongside CIMT screening, may merit inclusion in the routine cardiovascular risk evaluation of patients with T2DM.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a highly prevalent metabolic disorder characterized by insulin resistance, relative insulin deficiency, and chronic hyperglycemia, and its global burden continues to rise, particularly in developing countries such as India, where urbanization, sedentary behaviour, and dietary transition have accelerated its incidence.[1] Cardiovascular disease remains the leading cause of morbidity and mortality among patients with T2DM, and dyslipidemia — typified by elevated triglycerides, low HDL-cholesterol, and a preponderance of small, dense LDL particles — is a major contributor to this risk.[2,4]

Lipid profiles are conventionally measured in the fasting state, yet individuals spend the majority of their waking hours in a postprandial condition. Postprandial hypertriglyceridemia — an exaggerated or prolonged rise in triglyceride-rich lipoproteins after a meal — arises in T2DM largely from impaired clearance of chylomicrons and VLDL secondary to insulin resistance and reduced lipoprotein lipase activity.[3,9,10] The resulting triglyceride-rich remnant particles are believed to penetrate the arterial intima directly, promoting endothelial dysfunction, oxidative stress, and inflammation — key early steps in atherogenesis.[5,6,11]

Carotid intima-media thickness (CIMT), measured by high-resolution B-mode ultrasonography, is a validated, non-invasive surrogate marker of subclinical atherosclerosis and an independent predictor of future cardiovascular events.[13-15] Patients with T2DM consistently demonstrate greater CIMT than non-diabetic counterparts, reflecting accelerated vascular ageing in this population.[12] While fasting lipid abnormalities and CIMT have been extensively studied, postprandial triglyceride excursions — which better reflect the body's real-world handling of dietary lipid load — remain comparatively under-investigated, and current clinical guidelines continue to emphasize fasting profiles alone.[7,8]

Given this gap, the present study was designed to evaluate postprandial triglyceride levels in patients with T2DM and to examine their association with CIMT, with the broader aim of clarifying whether postprandial lipid assessment adds clinically meaningful information to conventional cardiovascular risk stratification in this population.

Aim and Objectives

Aim: To assess the role of postprandial hypertriglyceridemia and CIMT in patients with T2DM.

- To investigate the role of postprandial triglyceride levels in atherosclerosis among patients with type 2 diabetes.
- To determine the association between postprandial triglyceride levels and carotid intima-media thickness, measured by high-resolution B-mode ultrasonography, in the study population.

MATERIALS AND METHODS

Study Design and Setting

This was an observational study conducted in the Department of General Medicine, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu, over a period of 18 months, following institutional ethics committee approval. Written informed consent was obtained from all participants after they were informed of the study objectives and protocol.

Participants

Fifty adult patients with T2DM, aged 18–65 years, of either sex, were enrolled.

Exclusion criteria: type 1 diabetes mellitus; diabetes duration of less than one year; clinical or electrocardiographic evidence of cardiovascular or ischemic heart disease; and pre-existing hypertension or hyperlipidemia.

Sample Size

Sample size was calculated on the basis of the expected correlation between postprandial hypertriglyceridemia and CIMT in the T2DM population (correlation coefficient r = 0.45), with 95% confidence ($Z1-\alpha = 1.96$) and 80% power ($Z1-\beta$), yielding a minimum requirement of 50 participants.

Study Procedure

Fasting blood sugar (FBS), postprandial blood sugar (PPBS), HbA1c, fasting lipid profile (total cholesterol, LDL, HDL, VLDL), and fasting triglycerides (FTG) were measured after an 8–12 hour fast. Postprandial triglycerides (PPTG) were measured 4 hours after a standard meal. CIMT was assessed bilaterally by high-resolution B-mode ultrasonography of the common carotid arteries, performed by a single trained operator.

Participants were subsequently stratified into three groups on the basis of FTG and PPTG values:

- Group A: FTG <150 mg/dL and PPTG <150 mg/dL (normal fasting and postprandial triglycerides), n=15
- Group B: FTG <150 mg/dL and PPTG ≥150 mg/dL (isolated postprandial hypertriglyceridemia), n=15
- Group C: FTG ≥150 mg/dL and PPTG ≥150 mg/dL (combined fasting and postprandial hypertriglyceridemia), n=20

Statistical Analysis

Data were analysed using IBM SPSS software (version 20/24). Categorical variables were expressed as frequencies and percentages and compared using the chi-square test; continuous variables were expressed as mean ± standard deviation and compared across the three groups using one-way ANOVA. Associations between clinical/biochemical parameters and CIMT were assessed using Pearson's correlation coefficient (r). A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Fifty patients with T2DM were included and distributed into Group A (n=15), Group B (n=15), and Group C (n=20) based on fasting and postprandial triglyceride levels.

Demographic and Anthropometric Characteristics

Mean age increased progressively from Group A (50.3±5.8 years) to Group B (53.3±7.8 years) to Group C (57.6±7.8 years) (p=0.017), indicating that older patients were more likely to have combined fasting and postprandial hypertriglyceridemia. Males predominated across all groups, with no significant gender difference between groups (p=0.062). Body weight (p=0.112) and BMI (p=0.132) did not differ significantly across groups, although BMI showed a non-significant upward trend in Group C (Table 1).

Table 1. Demographic and anthropometric comparison across study groups

Parameter	Group A	Group B	Group C	p-value
Age, years (mean±SD)	50.33±5.76	53.27±7.83	57.60±7.82	0.017*

Parameter	Group A	Group B	Group C	p-value
Male : Female, n	12:3	7:8	16:4	0.062
Weight, kg (mean±SD)	57.0±9.98	64.8±10.47	60.85±9.57	0.112
BMI, kg/m² (mean±SD)	26.93±2.84	26.40±2.80	29.05±3.61	0.132

Clinical and Glycemic Parameters

Duration of diabetes was significantly longer in Groups B and C compared with Group A (7.8±3.3 vs 13.3±5.1 and 12.8±3.6 years respectively; p<0.001). Systolic and diastolic blood pressure were comparable across groups (p=0.949 and p=0.086 respectively). FBS, PPBS, and HbA1c did not differ significantly between groups, indicating that glycemic control alone did not predict the pattern of triglyceride abnormality (Table 2).

Table 2. Clinical and glycemic parameters across study groups

Parameter	Group A	Group B	Group C	p-value
Duration of DM, years	7.8±3.26	13.33±5.05	12.8±3.55	<0.001*
SBP, mmHg	139.6±12.48	141.07±11.14	140.3±13.22	0.949
DBP, mmHg	89.6±9.44	82.53±10.40	85.8±5.85	0.086
FBS, mg/dL	167.4±47.24	195.07±49.13	187.15±38.80	0.241
PPBS, mg/dL	236.4±73.19	236.93±71.65	256.75±57.74	0.385
HbA1c, %	8.17±1.74	7.34±1.74	7.37±1.40	0.239

Lipid Profile and Triglyceride Parameters

Total cholesterol (p=0.02) and triglycerides (p<0.001) were significantly higher in Group C, while LDL (p=0.068) and HDL (p=0.686) did not differ significantly across groups. As expected by group definition, FTG was markedly higher in Group C (173.25±15.55 mg/dL) than in Groups A and B (p<0.001), while PPTG rose progressively from Group A (131.87±13.7 mg/dL) to Group B (175.8±13.5 mg/dL) to Group C (183.95±11.61 mg/dL) (p<0.001) — notably, Group B showed markedly elevated PPTG despite normal fasting triglycerides, confirming that isolated postprandial hypertriglyceridemia is identifiable independent of the fasting profile (Table 3).

Table 3. Lipid and triglyceride parameters across study groups

Parameter	Group A	Group B	Group C	p-value
Total cholesterol, mg/dL	219.68±58.39	210.91±34.17	254.60±44.52	0.02*
LDL, mg/dL	140.87±56.54	130.33±37.25	167.00±46.10	0.068
HDL, mg/dL	42.53±12.29	45.87±11.04	42.65±12.78	0.686
FTG, mg/dL	128.07±13.87	124.4±14.73	173.25±15.55	<0.001*
PPTG, mg/dL	131.87±13.7	175.8±13.5	183.95±11.61	<0.001*

Carotid Intima-Media Thickness

Mean CIMT increased progressively across the three groups: Group A 0.967±0.315 mm, Group B 1.68±0.585 mm, and Group C 1.905±0.49 mm (p<0.001). Importantly, Group B — with normal fasting but elevated postprandial triglycerides —

already showed a substantially higher CIMT than Group A, indicating that postprandial hypertriglyceridemia alone is associated with measurable early vascular change even before fasting triglycerides become abnormal (Table 4).

Table 4. Carotid intima-media thickness across study groups

Parameter	Group A	Group B	Group C	p-value
CIMT, mm	0.967±0.315	1.68±0.585	1.905±0.49	<0.001*

Correlation of Clinical and Biochemical Parameters with CIMT

On Pearson correlation analysis, age (r=0.622, p<0.001), duration of diabetes (r=0.681, p<0.001), FTG (r=0.327, p=0.021), and PPTG (r=0.621, p<0.001) each showed a statistically significant positive correlation with CIMT, with duration of diabetes and PPTG showing the strongest associations (Table 5). In contrast, FBS, PPBS, HbA1c, systolic and diastolic blood pressure, total cholesterol, LDL, HDL, and BMI showed no statistically significant correlation with CIMT (Table 6), indicating that conventional glycemic and fasting-lipid parameters were less informative than postprandial triglycerides in this cohort.

Table 5. Parameters showing significant correlation with CIMT

Variable	r value	p value	Correlation with CIMT
Age	0.622	<0.001	Significant positive
Duration of diabetes	0.681	<0.001	Significant positive (strongest)
FTG	0.327	0.021	Significant positive (moderate)
PPTG	0.621	<0.001	Significant positive (strong)

Table 6. Parameters showing no significant correlation with CIMT

Variable	r value	p value	Correlation with CIMT
FBS	0.267	0.061	Not significant
PPBS	−0.250	0.079	Not significant
SBP	−0.024	0.866	Not significant
DBP	−0.211	0.141	Not significant
Total cholesterol	0.238	0.096	Not significant
LDL	0.177	0.219	Not significant
HDL	0.127	0.381	Not significant
HbA1c	−0.056	0.698	Not significant
BMI	0.195	0.174	Not significant

DISCUSSION

This study evaluated postprandial hypertriglyceridemia and its association with CIMT in 50 patients with T2DM stratified according to fasting and postprandial triglyceride status. The principal finding was a progressive, statistically significant increase in CIMT from Group A to Group C, with PPTG and duration of diabetes emerging as the strongest correlates of CIMT — stronger than any conventional glycemic or fasting-lipid parameter assessed.

A particularly notable observation was that Group B, comprising patients with normal fasting but elevated postprandial triglycerides, already exhibited significantly higher CIMT than Group A. This suggests that isolated postprandial hypertriglyceridemia — which would be missed entirely by fasting lipid testing alone — is sufficient to initiate measurable vascular change. This observation is consistent with the work of Nordestgaard et al., who demonstrated that non-fasting triglycerides are a stronger predictor of cardiovascular events, including myocardial infarction and stroke, than fasting triglyceride measurements.[8] It is also consistent with mechanistic data from Ceriello et al., who showed that postprandial hyperglycemia and hypertriglyceridemia jointly promote endothelial dysfunction, oxidative stress, and low-grade inflammation — processes that plausibly translate into the accelerated intimal thickening observed in the present cohort.[11]

The finding that CIMT was significantly greater in patients with T2DM in the higher-triglyceride groups, and that CIMT correlated positively with disease duration, aligns with earlier work by Teno et al., who reported significantly greater CIMT in T2DM compared with non-diabetic controls and a positive association between CIMT and lipid abnormality.[12] Taken together, the present results reinforce the broader physiological argument that triglyceride-rich remnant lipoproteins generated in the postprandial state have direct atherogenic potential, independent of fasting lipid status.[9,10]

In contrast, conventional risk parameters — FBS, PPBS, HbA1c, blood pressure, LDL, HDL, and BMI — showed no significant association with CIMT in this cohort. This does not imply that glycemic control or adiposity are clinically unimportant; rather, it suggests that within a T2DM population where most patients already carry some degree of dysglycemia, postprandial triglyceride handling may distinguish vascular risk more sharply than glycemic indices that are already uniformly deranged. This is in keeping with a substantial body of literature indicating that HbA1c-based glycemic control does not fully capture cardiovascular risk in T2DM, and that lipid-related pathways — particularly postprandial ones — contribute additively to atherogenesis.[1,2,4]

From a clinical standpoint, these findings support incorporating postprandial triglyceride measurement into the cardiovascular risk assessment of patients with T2DM, rather than relying on fasting lipid profiles alone. Given that CIMT is an established, low-cost, non-invasive surrogate for subclinical atherosclerosis, pairing postprandial triglyceride testing with periodic CIMT screening — particularly in patients with long-standing diabetes — may allow earlier identification of patients at elevated cardiovascular risk who would otherwise appear reassuring on fasting lipid testing.[13-15]

LIMITATIONS

- The single-center design and modest sample size (n=50) limit the generalizability of these findings and reduce power to detect weaker associations.
- The cross-sectional, observational design precludes causal inference between postprandial hypertriglyceridemia and CIMT progression; longitudinal follow-up would be required to establish temporality.
- A single postprandial triglyceride measurement at 4 hours after a non-standardized meal may not fully capture the individual variability of postprandial lipid excursions that a standardized fat-tolerance test would provide.
- Potential confounders such as physical activity, dietary pattern, smoking status, and concurrent lipid-lowering or antidiabetic therapy were not systematically captured or adjusted for.

CONCLUSION

Postprandial hypertriglyceridemia — including when isolated, in the presence of normal fasting triglycerides — is significantly associated with increased carotid intima-media thickness in patients with type 2 diabetes mellitus, and shows a stronger correlation with CIMT than fasting triglycerides or conventional glycemic and lipid parameters. These findings suggest that postprandial triglyceride assessment may serve as a more sensitive early marker of subclinical atherosclerosis than fasting lipid profiling alone, and support consideration of routine postprandial triglyceride measurement, alongside CIMT screening, in the comprehensive cardiovascular risk evaluation of patients with T2DM. Larger, prospective, multi-center studies are warranted to confirm these findings and to define the incremental value of postprandial triglyceride testing in routine diabetes care.

DECLARATIONS

Ethical approval: Obtained from the Institutional Ethics Committee prior to data collection; written informed consent was obtained from all participants.

Conflict of interest: The authors declare no conflict of interest.

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Author contributions: Both authors contributed to study conception, data acquisition, analysis, and manuscript preparation, and approved the final version.

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