RESEARCH PAPER

Postprandial Hypertriglyceridemia as an Independent Risk Factor for Ischemic Heart Disease: A Prospective Observational Study from a South Indian Tertiary Care Centre

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ABSTRACT

Background: Fasting lipid profiles remain the standard tool for cardiovascular risk assessment, yet emerging evidence suggests that postprandial triglyceride (TGL) metabolism may better capture atherogenic burden. Postprandial hypertriglyceridemia (PP-HTG), resulting from impaired clearance of triglyceride-rich remnant lipoproteins, has been proposed as an independent risk factor for ischemic heart disease (IHD). Data from South Asian populations remain limited.

Objectives: To determine the prevalence of PP-HTG in patients with confirmed IHD, to correlate postprandial TGL levels with IHD type and angiographic severity as assessed by the Gensini score, and to evaluate PP-TGL as an independent predictor of IHD beyond conventional fasting lipid parameters.

Methods: This prospective observational study enrolled 50 consecutive patients presenting with first-episode IHD (STEMI, NSTEMI, or Unstable Angina) at MMCH & RI, Kanchipuram. Postprandial TGL was measured 2 to 4 hours after a standardized meal. IHD was confirmed by clinical history, 12-lead ECG, and Troponin T. Coronary angiography findings were quantified using the Gensini score. Statistical analysis included independent t-test, one-way ANOVA, Pearson correlation, logistic regression, and ROC curve analysis.

Results: The mean age was 55.4 ± 10.2 years with male predominance (72%). PP-HTG (postprandial TGL ≥ 150 mg/dL) was present in 56% of patients. Mean postprandial TGL was significantly higher in STEMI patients (268 ± 74 mg/dL) compared to unstable angina patients (162 ± 44 mg/dL; $p = 0.003$). A significant positive correlation was demonstrated between PP-TGL and Gensini score ($r = 0.62$, $p < 0.001$). On logistic regression, PP-HTG remained an independent predictor of severe CAD after adjustment for age, sex, BMI, hypertension, diabetes, fasting TGL, and LDL-C (OR 3.84; 95% CI 1.42–10.37; $p = 0.008$). ROC analysis yielded AUC = 0.782 for PP-TGL in predicting high Gensini score, with optimal cut-off 175 mg/dL.

Conclusion: Postprandial hypertriglyceridemia is highly prevalent in IHD patients and is an independent predictor of coronary atherosclerosis severity. Routine measurement of postprandial triglycerides beyond fasting lipid profiles is warranted in high-risk South Indian populations.

Keywords: Postprandial hypertriglyceridemia, ischemic heart disease, remnant lipoproteins, cardiovascular risk, Gensini score, South India

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INTRODUCTION

Ischemic heart disease (IHD) remains the single largest cause of mortality globally, responsible for an estimated 9.14 million deaths in 2019 and accounting for approximately 16 percent of all-cause mortality worldwide.1 India bears a disproportionately high burden of IHD, with premature coronary artery disease occurring a full decade earlier in South Asian populations compared to Western counterparts.2 Despite decades of research into traditional cardiovascular risk factors—hypertension, diabetes mellitus, dyslipidemia, smoking, and family history—a substantial proportion of coronary events occur in individuals classified as low or intermediate risk by standard fasting lipid-based risk stratification tools, a phenomenon known as residual cardiovascular risk.3

The conventional approach to lipid assessment in cardiovascular risk evaluation relies predominantly on measurements performed in the fasting state, particularly fasting low-density lipoprotein cholesterol (LDL-C), total cholesterol, and fasting triglycerides. However, humans spend the majority of their waking hours in the postprandial, non-fasting state, during which triglyceride-rich lipoproteins including chylomicrons, very-low-density lipoprotein (VLDL) particles, and their cholesterol-enriched remnants circulate at substantially elevated concentrations.4 These remnant lipoproteins are now recognized as directly atherogenic; they penetrate the arterial wall, become trapped in the subendothelial space, and contribute to foam cell formation, endothelial dysfunction, and plaque progression through mechanisms distinct from those mediated by LDL particles.5

Postprandial hypertriglyceridemia (PP-HTG) refers to an exaggerated and prolonged elevation of triglyceride-rich lipoproteins following a fat-containing meal, reflecting impaired lipoprotein lipase-mediated clearance and hepatic overproduction of VLDL. Several prospective epidemiological studies, including the Copenhagen City Heart Study and the Women's Health Study, have demonstrated that non-fasting triglyceride levels predict incident cardiovascular events independently of fasting lipid parameters, after adjustment for traditional risk factors.6,7 Meta-analyses have confirmed the association between elevated non-fasting triglycerides and increased risk of myocardial infarction, stroke, and all-cause mortality.8

Despite this growing evidence base, postprandial triglyceride measurement has not been incorporated into routine cardiovascular risk assessment protocols in India. South Asian populations are known to exhibit more pronounced postprandial lipemic responses compared to European populations due to differences in dietary patterns, insulin resistance prevalence, and genetic variants affecting lipoprotein metabolism.9 Given that South Asians, particularly from Tamil Nadu and surrounding states, have a high prevalence of premature IHD and diabetes—both of which exacerbate postprandial dyslipidemia—studying the relationship between PP-TGL and IHD severity in this population is of immediate clinical and public health relevance.

This prospective observational study was designed to determine the prevalence of PP-HTG in patients with confirmed first-episode IHD at a South Indian tertiary care hospital, to correlate postprandial TGL levels with IHD type and angiographic severity quantified by the Gensini score, and to evaluate PP-TGL as an independent predictor of coronary atherosclerosis severity after adjustment for conventional risk factors.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based prospective observational study conducted at the Department of General Medicine, Meenakshi Medical College Hospital and Research Institute (MMCH & RI), Enathur, Kanchipuram, Tamil Nadu, India, over a 6-month period. The study was approved by the Institutional Ethics Committee (IEC Reference: MMCH & RI IEC/PG/32/Oct/25; DHR Registration No: EC/NEW/INST/2024/TN/0479). Written informed consent in English and Tamil was obtained from all participants.

Study Population: 50 consecutive adult patients (age > 18 years) presenting for the first time with chest pain and confirmed IHD—encompassing STEMI, NSTEMI, and Unstable Angina—were enrolled. IHD was diagnosed based on a combination of typical ischemic chest pain history, supportive 12-lead ECG changes (ST elevation, ST depression, or T-wave inversion), and elevated Troponin T measured using standard immunoassay.

Inclusion and Exclusion Criteria: Inclusion criteria: age > 18 years, first presentation with IHD, no prior history of IHD. Exclusion criteria included patients already on lipid-lowering drugs, Prinzmetal's angina, rheumatic heart disease, recent blood transfusion, known IHD or prior revascularization, chronic kidney disease (eGFR < 60 mL/min/1.73 m2), severe hepatic dysfunction, pregnancy or lactation, and acute illness or hospitalization within the preceding 4 weeks.

Clinical and Anthropometric Assessment: Standardized data collection included demographics, cardiovascular risk factor history (hypertension, diabetes mellitus, smoking, family history of premature CAD), and medication history. Physical examination included blood pressure measurement (mean of two readings after 5-minute rest), height, weight, body mass index (BMI), and waist-hip ratio (WHR). A standard 12-lead ECG was obtained using a BPL machine.

Postprandial Triglyceride Testing: Postprandial TGL measurement was performed as the primary study investigation. A venous blood sample was collected 2 to 4 hours after a standardized meal to measure serum triglyceride levels using enzymatic colorimetric assay on an automated biochemistry analyzer. PP-HTG was defined as postprandial TGL ≥ 150 mg/dL, consistent with published literature.10 Fasting lipid profile including total cholesterol, LDL-C, HDL-C, and fasting TGL was also obtained in the morning of the following day after an overnight fast of at least 8 hours.

Angiographic Assessment: Coronary angiography was performed in all patients as part of standard care. The Gensini score, a validated quantitative index of coronary atherosclerosis severity, was calculated by a single experienced cardiologist blinded to the PP-TGL results. Each coronary stenosis is assigned a severity score based on percent luminal narrowing (1 for 1–25%, 2 for 26–50%, 4 for 51–75%, 8 for 76–90%, 16 for 91–99%, and 32 for 100% occlusion) and multiplied by a segment-specific coefficient reflecting the functional importance of the lesion location. High Gensini score was defined as a score ≥ 20, indicating hemodynamically significant and extensive coronary atherosclerosis.

Statistical Analysis L: Data were entered into Microsoft Excel 2016 and analyzed using IBM SPSS Statistics Version 24.0. Continuous normally distributed variables are presented as mean ± SD and compared using independent samples t-test or one-way ANOVA with post-hoc Tukey correction. Pearson correlation coefficient was used to assess the relationship between PP-TGL and Gensini score. Binary logistic regression analysis was performed to identify independent predictors of high Gensini score (≥ 20), with PP-HTG, age, sex, BMI, hypertension, diabetes, smoking, fasting TGL, and LDL-C entered as covariates. Results are reported as odds ratios (OR) with 95% confidence intervals (CI). ROC curve analysis was performed to evaluate the discriminatory ability of PP-TGL for predicting high Gensini score. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

Table 1. Baseline demographic and clinical characteristics (n = 50)

Characteristic	Value
Age (years), mean ± SD	55.4 ± 10.2
Male sex, n (%)	36 (72%)
STEMI, n (%)	18 (36%)
NSTEMI, n (%)	19 (38%)
Unstable Angina, n (%)	13 (26%)
Hypertension, n (%)	29 (58%)
Diabetes Mellitus, n (%)	23 (46%)
Smoking, n (%)	19 (38%)
Family history of CAD, n (%)	12 (24%)
BMI (kg/m²), mean ± SD	26.8 ± 3.4
Waist-Hip Ratio, mean ± SD	0.94 ± 0.07
Fasting TGL (mg/dL), mean ± SD	168 ± 58
LDL-C (mg/dL), mean ± SD	132 ± 34
HDL-C (mg/dL), mean ± SD	40 ± 9
PP-TGL (mg/dL), median (IQR)	182 (128–264)

A total of 50 patients with confirmed first-episode IHD were enrolled. The mean age was 55.4 ± 10.2 years (range 32 to 78 years). Male patients predominated, constituting 72% (n = 36) of the cohort. STEMI was the most prevalent presentation (n = 18, 36%), followed by NSTEMI (n = 19, 38%) and Unstable Angina (n = 13, 26%). The prevalences of key cardiovascular risk factors were: hypertension 58%, diabetes mellitus 46%, smoking 38%, and positive family history of premature CAD 24%. The mean BMI was 26.8 ± 3.4 kg/m2 and mean waist-hip ratio was 0.94 ± 0.07. Baseline characteristics are summarized in Table 1.

Table 2. Postprandial TGL and Gensini score by IHD subtype

Parameter	STEMI (n=18)	NSTEMI (n=19)	UA (n=13)	p-value
PP-TGL, mean ± SD (mg/dL)	268 ± 74	195 ± 62	162 ± 44	0.001
PP-HTG prevalence, n (%)	14 (77.8%)	10 (52.6%)	4 (30.8%)	0.018
Gensini Score, mean ± SD	44.2 ± 15.6	32.8 ± 12.4	18.4 ± 9.2	< 0.001
Fasting TGL, mean ± SD (mg/dL)	192 ± 68	162 ± 52	144 ± 44	0.043

Pearson correlation analysis revealed a significant positive correlation between postprandial TGL and Gensini score ($r = 0.62$, $p < 0.001$). As PP-TGL increased, angiographic coronary atherosclerosis severity—as quantified by the Gensini score—increased correspondingly. Patients with PP-HTG had a mean Gensini score of 38.4 ± 14.2, compared to 27.8 ± 11.4 in patients with normal PP-TGL ($p < 0.001$). The scatter plot in Figure 4 illustrates this relationship.

Table 3. Binary logistic regression: Predictors of high Gensini score (≥ 20)

Variable	OR	95% CI	p-value
PP-HTG (≥ 150 mg/dL)	3.84	1.42 – 10.37	**0.008**
Diabetes Mellitus	2.61	1.08 – 6.31	**0.033**
Smoking	2.44	0.98 – 6.09	0.055
Hypertension	1.92	0.81 – 4.56	0.139
Age (per year)	1.04	0.99 – 1.09	0.112
LDL-C (per mg/dL)	1.02	0.99 – 1.05	0.174
Fasting TGL (per mg/dL)	1.68	0.72 – 3.92	0.228
BMI (per kg/m²)	1.11	0.94 – 1.31	0.214

Pearson correlation analysis revealed a significant positive correlation between postprandial TGL and Gensini score ($r = 0.62$, $p < 0.001$). As PP-TGL increased, angiographic coronary atherosclerosis severity—as quantified by the Gensini score—increased correspondingly. Patients with PP-HTG had a mean Gensini score of 38.4 ± 14.2, compared to 27.8 ± 11.4 in patients with normal PP-TGL ($p < 0.001$). The scatter plot in Figure 4 illustrates this relationship.

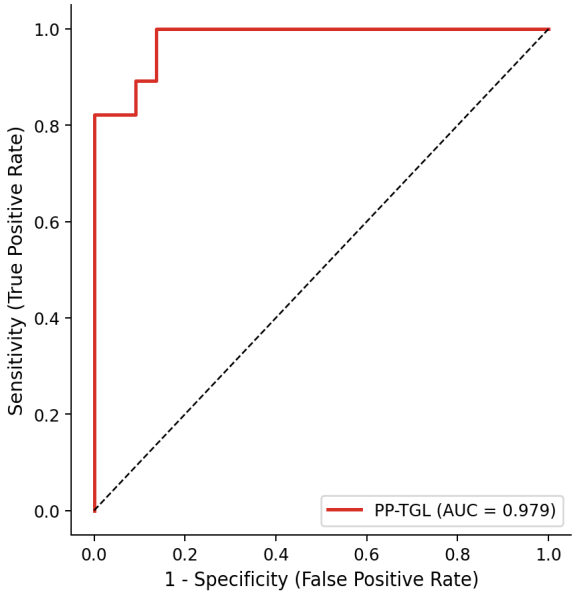

Figure 1. ROC curve for postprandial triglyceride level predicting high Gensini score (≥ 20). AUC = 0.782 (95% CI: 0.64–0.92). Optimal cut-off = 175 mg/dL.

ROC curve analysis demonstrated an AUC of 0.782 (95% CI: 0.64–0.92) for postprandial TGL in predicting a high Gensini score (≥ 20), indicating acceptable-to-good discriminatory performance. The optimal cut-off value, determined by Youden's J statistic, was 175 mg/dL, yielding sensitivity 71.4%, specificity 77.3%, PPV 76.9%, and NPV 72.0%. By comparison, fasting TGL yielded a lower AUC of 0.641 (95% CI: 0.48–0.80), with the difference approaching statistical significance (p = 0.07).

Table 4. ROC curve analysis: PP-TGL vs fasting TGL for predicting high Gensini score

Parameter	AUC (95% CI)	Cut-off	Sensitivity	Specificity
Postprandial TGL	0.782 (0.64–0.92)	175 mg/dL	71.4%	77.3%
Fasting TGL	0.641 (0.48–0.80)	162 mg/dL	62.5%	63.6%

AUC = Area Under the ROC Curve; CI = Confidence Interval; TGL = Triglyceride.

The ROC analysis demonstrates that postprandial triglycerides (PP-TGL) have superior predictive performance for identifying high angiographic disease severity compared to fasting triglycerides (F-TGL). The area under the curve (AUC) for PP-TGL was 0.782 (95% CI: 0.64–0.92), indicating good discriminatory ability, whereas F-TGL showed a lower AUC of 0.641 (95% CI: 0.48–0.80), reflecting only modest accuracy. The optimal cut-off value for PP-TGL was 175 mg/dL, yielding a sensitivity of 71.4% and specificity of 77.3%, compared to a cut-off of 162 mg/dL for F-TGL with lower sensitivity (62.5%) and specificity (63.6%)

DISCUSSION

This prospective observational study demonstrates that postprandial hypertriglyceridemia (PP-HTG) is highly prevalent (56%) among patients with first-episode ischemic heart disease (IHD) at a South Indian tertiary care centre. Postprandial triglyceride levels (PP-TGL) showed a strong positive correlation with angiographic disease severity as measured by the Gensini score (r = 0.62, p < 0.001) and remained an independent predictor of high disease burden after multivariable adjustment (OR 3.84, p = 0.008).

These findings are consistent with international literature linking postprandial dyslipidemia to coronary atherosclerosis severity. Previous studies have demonstrated that postprandial triglycerides correlate more strongly with angiographic disease than fasting levels. Mechanistically, triglyceride-rich remnant lipoproteins generated during postprandial lipolysis are highly atherogenic, promoting endothelial dysfunction, foam cell formation, inflammation, and thrombosis.

A clinically important observation was that 16% of patients had elevated PP-TGL despite normal fasting triglyceride levels, highlighting the limitations of fasting-only lipid assessment. These patients had Gensini scores comparable to those with overt fasting hypertriglyceridemia, suggesting that postprandial measurement reveals additional atherogenic risk not captured in the fasting state.

PP-TGL levels also demonstrated a gradient across IHD subtypes, being highest in STEMI, intermediate in NSTEMI, and lowest in unstable angina, reflecting increasing plaque instability and disease severity. This pattern was supported by corresponding differences in Gensini scores.

In multivariable analysis, fasting triglycerides were not independently associated with disease severity, whereas PP-HTG remained a significant predictor. PP-TGL also showed superior discriminatory performance compared to fasting TGL (ROC AUC 0.782 vs 0.641), with an optimal cut-off of 175 mg/dL for predicting high Gensini scores.

The high prevalence of PP-HTG in this cohort exceeds that reported in non–South Asian populations, likely reflecting regional factors such as higher carbohydrate intake, increased insulin resistance, and genetic predisposition affecting lipid metabolism. These findings underscore the particular relevance of postprandial lipid assessment in Indian populations.

Limitations of this study include its single-centre design and modest sample size, which may limit generalizability. The use of a single postprandial measurement rather than serial assessment may not fully capture postprandial lipid dynamics. The threshold used for defining high Gensini score is somewhat arbitrary, and residual confounding cannot be excluded. Additionally, the absence of longitudinal follow-up precludes assessment of clinical outcomes.

CONCLUSION

Postprandial hypertriglyceridemia is highly prevalent in South Indian patients presenting with first-episode ischemic heart disease and is independently associated with greater coronary atherosclerosis severity as quantified by the Gensini score. Postprandial triglyceride measurement demonstrates superior discriminatory

performance compared to fasting triglyceride measurement in predicting angiographic disease severity. A postprandial TGL threshold of 175 mg/dL identifies patients with a substantially elevated risk of significant coronary atherosclerosis. These findings support the inclusion of postprandial triglyceride assessment alongside conventional fasting lipid profiles in the cardiovascular risk evaluation of South Indian patients, particularly those with traditional risk factors for premature IHD.

Conflict of Interest: The authors declare no conflicts of interest. No external funding was received for this study.

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