Biomedical and Biopharmaceutical Research
Abbreviation: Biomed. Biopharm. Res.
Volume: 22: Issue: 02 |Year: 2025
Page Number: 325-330

STUDY OF POST PRANDIAL HYPERTRIGLYCERIDEMIA AS AN INDEPENDENT RISK FACTOR FOR ISCHEMIC HEART DISEASE

DR. RAGHAVENDRA.F.N[1], DR. ARPITHA.S.V[2], DR. SADANANDA ADIGA.M.N[3]
[1]Professor & Unit Chief, Department of General Medicine, Ballari Medical College Research Centre(BMCRC), Ballari
[2]Junior Resident/POST GRADUATE STUDENT, BMCRC, Ballari
[3]Associate Professor, Department of Biochemistry, BMCRC, Ballari

Corresponding Author

DR. RAGHAVENDRA.F.N
Professor & Unit Chief, Department of General Medicine, Ballari Medical College Research Centre(BMCRC), Ballari

Received: 03-06-2025

Accepted: 10-07-2025

ABSTRACT

Background: While fasting lipid profiles are routinely used to assess cardiovascular risk, postprandial hypertriglyceridemia (PPHTG) is increasingly recognized as a potential independent risk factor for ischemic heart disease (IHD). This study aimed to evaluate the association between PPHTG and IHD in patients with normal fasting triglyceride and cholesterol levels.

Objectives: To determine the prevalence of postprandial hypertriglyceridemia among patients with unstable angina and acute myocardial infarction (STEMI/NSTEMI) and to assess its correlation with anthropometric and metabolic risk factors.

Methods: This hospital-based observational study included 100 patients with IHD (49 unstable angina, 51 STEMI/NSTEMI) who had normal fasting triglyceride (<150 mg/dL) and cholesterol (<180 mg/dL) levels. Postprandial lipid profiles were assessed 4 hours after a standardized meal. Clinical, anthropometric, and biochemical data were collected and analyzed. Logistic regression was used to evaluate PPHTG as an independent risk factor for IHD.

Results: Postprandial hypertriglyceridemia was significantly more prevalent in STEMI/NSTEMI patients (74.5%) compared to unstable angina (53.1%) ($p = 0.013$). Mean postprandial triglyceride levels were significantly higher in STEMI/NSTEMI (267.2 ± 38.6 mg/dL) than in unstable angina (197.8 ± 32.4 mg/dL, $p = 0.001$). Logistic regression identified PPHTG as an independent risk factor for IHD (OR = 2.78; $p = 0.013$). Significant correlations were observed between PPHTG and both the waist-hip ratio and diabetes mellitus.

Conclusion: Postprandial hypertriglyceridemia is independently associated with ischemic heart disease and may serve as a valuable tool for detecting hidden cardiovascular risk, especially in patients with central obesity or diabetes, even when fasting lipid levels are normal. Routine assessment of postprandial lipids could enhance cardiovascular risk stratification and preventive strategies.

Keywords: Postprandial hypertriglyceridemia, Ischemic heart disease, STEMI, NSTEMI, Unstable angina, Lipid profile, Waist-hip ratio, Diabetes mellitus.

INTRODUCTION

Ischemic heart disease (IHD) remains a leading cause of morbidity and mortality worldwide, especially in developing countries where lifestyle-related risk factors are increasingly prevalent [1]. While traditional risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidemia are well-established contributors to atherosclerosis and coronary artery disease (CAD) [2], recent research suggests that postprandial lipid metabolism abnormalities may also play a crucial role in cardiovascular risk prediction [3].

Postprandial hypertriglyceridemia, defined as an exaggerated rise in serum triglyceride levels following a meal, is emerging as an important but under-recognized cardiovascular risk factor [4]. Unlike fasting triglyceride levels, which reflect baseline metabolic activity, postprandial measurements offer a dynamic view of lipid handling and endothelial function under physiological stress [5]. Elevated postprandial triglyceride levels have been linked to increased atherogenic remnant lipoproteins, oxidative stress, and endothelial dysfunction—all of which contribute to atherosclerotic plaque formation and instability [6].

Several population-based studies have demonstrated that non-fasting triglyceride levels are better predictors of cardiovascular events compared to fasting levels [7]. Moreover, patients with normal fasting lipid profiles may still

exhibit significant postprandial dyslipidemia, potentially masking their true cardiovascular risk [8]. This has important clinical implications, particularly for risk stratification and preventive cardiology.

Despite growing evidence, the role of postprandial hypertriglyceridemia as an independent risk factor for IHD in routine clinical practice remains underexplored, especially in South Asian populations who are known to have a higher predisposition to metabolic syndrome and cardiovascular disease [9]. This study aims to evaluate the prevalence and impact of postprandial hypertriglyceridemia among patients diagnosed with unstable angina or acute myocardial infarction (STEMI/NSTEMI), while controlling for traditional risk factors.

By comparing fasting and postprandial lipid profiles, and analyzing correlations with anthropometric, clinical, and metabolic parameters, the study seeks to determine whether postprandial hypertriglyceridemia independently contributes to IHD. The findings may offer novel insights for refining cardiovascular risk assessment and guide targeted interventions for patients with postprandial lipid abnormalities.

MATERIALS AND METHODS

Study Design

This observational study was conducted to investigate the relationship between postprandial hypertriglyceridemia and ischemic heart disease (IHD), encompassing unstable angina, ST-elevation myocardial infarction (STEMI), and non-ST-elevation myocardial infarction (NSTEMI). The study aimed to assess both primary and secondary outcomes related to lipid metabolism and cardiovascular risk in a hospital-based population.

Study Setting and Population

The study was conducted in the Department of General Medicine at a tertiary care hospital. Data were collected from patients diagnosed with unstable angina or acute myocardial infarction (STEMI/NSTEMI), based on clinical and electrocardiographic criteria. Eligible patients were consecutively enrolled until the target sample size was reached.

Sample Size

The sample size was calculated to be **89** based on an expected prevalence of postprandial hypertriglyceridemia in 64% of IHD patients, with a 95% confidence level and 10% absolute precision.

Inclusion Criteria

- Patients with unstable angina, defined by anginal chest pain or equivalent symptoms, ST-segment depression in at least two contiguous ECG leads, and normal Troponin T levels.
- Patients newly diagnosed with STEMI or NSTEMI as per standard clinical and ECG criteria.
- Fasting serum triglyceride level <150 mg/dL.
- Fasting serum cholesterol level <180 mg/dL.

Exclusion Criteria

- Use of lipid-lowering medications (e.g., statins, fibrates).
- Suspected cases of Prinzmetal angina.
- History of rheumatic heart disease.
- Use of oral contraceptive pills or hormone replacement therapy.
- Abnormal liver function tests (elevated AST/ALT).
- Abnormal renal function (elevated serum creatinine or reduced GFR).

Data Collection Procedure

After enrollment, the following data were collected:

- **Demographic details**: Age, sex, etc.
- **Medical history**: Comorbidities like diabetes mellitus, hypertension, smoking status.
- **Physical examination**: Vital signs, BMI, waist and hip circumference, waist-hip ratio.
- **Cardiovascular examination**: Heart sounds, murmurs, peripheral pulses.
- **ECG**: Confirmed diagnosis of unstable angina, STEMI or NSTEMI.
- **Laboratory tests**:
    - Complete blood count
    - Fasting and postprandial blood sugar
    - Postprandial lipid profile (drawn at 4 hours post-meal)
- **2D Echocardiography**: Assessment of cardiac structure and function, including regional wall motion abnormalities.

All laboratory tests were conducted using automated analyzers. No invasive or interventional procedures were performed as part of the study.

Outcome Measures

- **Primary Outcome**: Prevalence of elevated postprandial triglyceride levels in IHD patients with normal fasting lipid levels.

- **Secondary Outcomes**: Correlation between postprandial hypertriglyceridemia and:
    - High waist-hip ratio
    - Presence of diabetes mellitus

STATISTICAL ANALYSIS

All collected data were analyzed using appropriate statistical methods. Descriptive statistics, including means, standard deviations, and percentages, were used to summarize baseline characteristics of the study population. Comparative analyses were conducted using the independent t-test for continuous variables and the chi-square test for categorical variables to evaluate differences in postprandial triglyceride levels and other clinical parameters between patients with unstable angina and those with STEMI/NSTEMI. To examine associations between postprandial hypertriglyceridemia and other risk factors such as waist-hip ratio and diabetes mellitus, correlation analyses were performed using Pearson's or Spearman's correlation coefficients, as appropriate. A p-value of less than 0.05 was considered statistically significant for all analyses.

Ethical Considerations

Before initiation, ethical clearance was obtained from the Institutional Ethical Committee. Informed consent was obtained from all participants after they were informed of the purpose, procedures, and implications of the study.

RESULTS AND OBSERVATIONS

Table 1: Age Distribution (Mean ± SD and Categorical) by IHD Diagnosis

IHD Diagnosis	n	Mean Age (years)	SD	Age <50 (n, %)	Age 50–59 (n, %)	Age 60–69 (n, %)	Age ≥70 (n, %)
Unstable Angina	49	62.4	7.2	4 (8.2%)	14 (28.6%)	22 (44.9%)	9 (18.4%)
STEMI/NSTEMI	51	53.8	6.9	19 (37.3%)	21 (41.2%)	9 (17.6%)	2 (3.9%)
Total	100	58.0	8.3	23 (23.0%)	35 (35.0%)	31 (31.0%)	11 (11.0%)

Table 2: Sex Distribution by IHD Diagnosis

IHD Diagnosis	Male (n, %)	Female (n, %)	Total (n)
Unstable Angina	30 (61.2%)	19 (38.8%)	49
STEMI/NSTEMI	35 (68.6%)	16 (31.4%)	51
Total	65 (65.0%)	35 (35.0%)	100

Table 3: Baseline Characteristics of Study Population

Characteristic	Unstable Angina (n=49)	STEMI/NSTEMI (n=51)	P-value
Diabetes Mellitus (n, %)	18 (36.7%)	20 (39.2%)	0.795
Hypertension (n, %)	31 (63.3%)	29 (56.9%)	0.514
Smoking (n, %)	19 (38.8%)	22 (43.1%)	0.653
BMI (Mean ± SD)	27.5 ± 1.5	28.9 ± 1.7	0.002

Table 4: Postprandial Triglyceride Levels (Mean ± SD) by IHD Diagnosis

IHD Diagnosis	N	Mean (mg/dL)	SD	P-value
Unstable Angina	49	197.8	32.4	0.001
STEMI/NSTEMI	51	267.2	38.6	

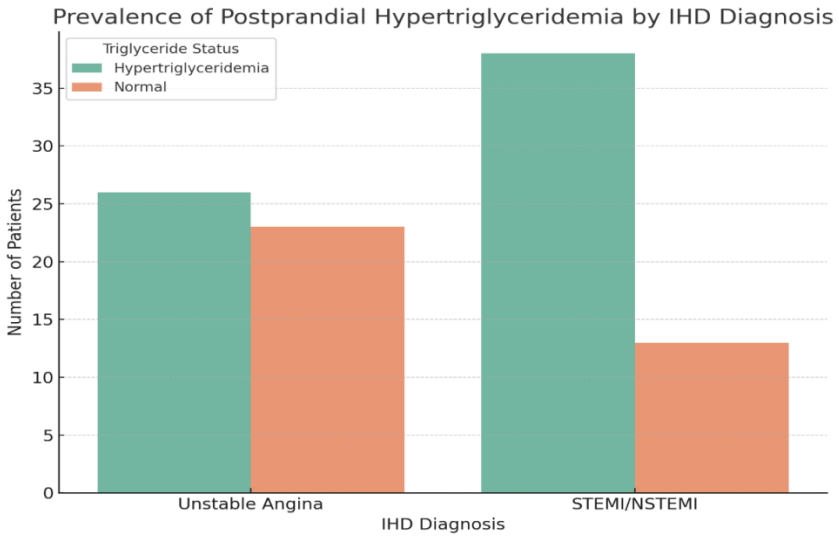

Figure 1; showing the prevalence of postprandial hypertriglyceridemia by IHD diagnosis (Unstable Angina vs STEMI/NSTEMI)

Table 5: Comparison of Lipid Profiles (Fasting and Postprandial) by IHD Diagnosis

Parameter	**Unstable Angina (Mean ± SD)**	**STEMI/NSTEMI (Mean ± SD)**	**P-value**
Fasting TG (mg/dL)	131.2 ± 7.8	141.8 ± 6.5	0.001
Fasting Cholesterol	159.6 ± 5.4	169.2 ± 4.8	0.001
Postprandial TG (mg/dL)	197.8 ± 32.4	267.2 ± 38.6	0.001
Postprandial HDL	41.6 ± 2.1	36.8 ± 1.9	0.001

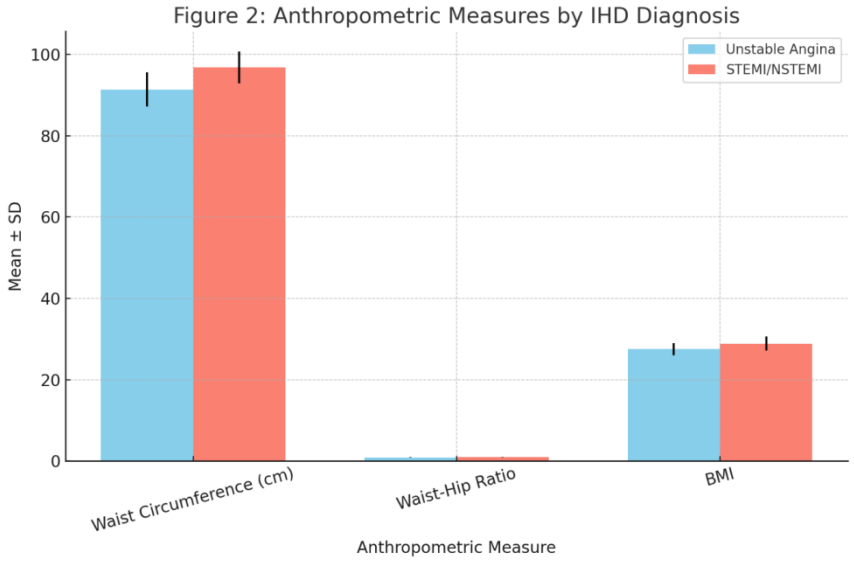

Table 6: Prevalence of Diabetes Mellitus by IHD Diagnosis

IHD Diagnosis	**Diabetes (n, %)**	**No Diabetes (n, %)**	**Total (n)**	**P-value**
Unstable Angina	18 (36.7%)	31 (63.3%)	49	0.795
STEMI/NSTEMI	20 (39.2%)	31 (60.8%)	51	

Figure 3: Correlation Between Postprandial Triglycerides and Waist-Hip Ratio

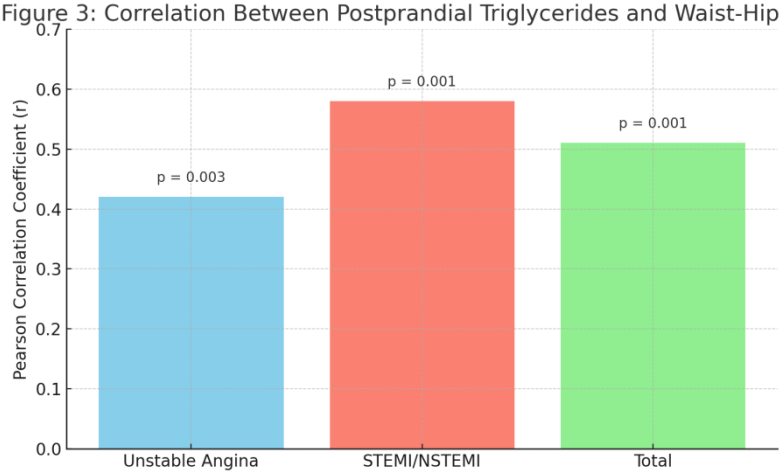

Table 7: Correlation Between Postprandial Triglycerides and Diabetes Mellitus

Diabetes Status	n	Mean TG (mg/dL)	SD	P-value
Yes	38	258.4	41.2	0.001
No	62	211.6	35.8	

Table 8: Logistic Regression Analysis of Postprandial Hypertriglyceridemia as an Independent Risk Factor for IHD

Variable	Odds Ratio (OR)	95% CI	P-value
Postprandial Hypertriglyceridemia	2.78	1.24–6.21	0.013
Age	0.94	0.89–0.99	0.022
Sex (Male vs. Female)	1.32	0.58–3.01	0.504
Diabetes Mellitus	1.15	0.51–2.59	0.732
Waist-Hip Ratio	1.89	0.82–4.37	0.134

Table 9: Echocardiographic, Clinical, and Laboratory Parameters by IHD Diagnosis

Parameter	Unstable Angina (Mean ± SD or %)	STEMI/NSTEMI (Mean ± SD or %)	P-value
Wall Motion Abnormality (%)	0 (0%)	51 (100%)	0.001
Ejection Fraction (%)	58.2 ± 2.1	47.6 ± 3.4	0.001
Pulse Rate (bpm)	81.4 ± 4.2	86.7 ± 3.9	0.001
Blood Pressure (mmHg)	140/88 ± 5/3	139/87 ± 6/4	0.632
Fasting Blood Sugar (mg/dL)	98.6 ± 8.4	118.2 ± 9.1	0.001
Hemoglobin (g/dL)	13.7 ± 0.5	13.9 ± 0.6	0.154

DISCUSSION

The present study evaluated **postprandial hypertriglyceridemia (PPHTG)** as an independent risk factor for ischemic heart disease (IHD), including unstable angina and acute myocardial infarction (STEMI/NSTEMI), in patients with normal fasting triglyceride and cholesterol levels. The findings underscore the clinical relevance of postprandial lipid testing in cardiovascular risk assessment, especially in populations with high cardiometabolic burden.

A significantly higher prevalence of postprandial hypertriglyceridemia was observed among STEMI/NSTEMI patients (74.5%) compared to those with unstable angina (53.1%) ($p = 0.013$), suggesting a strong association between elevated postprandial triglyceride levels and myocardial infarction severity. This supports previous evidence indicating that nonfasting triglyceride levels are more predictive of cardiovascular events than fasting levels [3,7]. The **mean postprandial triglyceride level** was also significantly higher in STEMI/NSTEMI patients (267.2 ± 38.6 mg/dL) compared to those with unstable angina (197.8 ± 32.4 mg/dL), aligning with prior studies on postprandial lipemia and plaque vulnerability [4,6].

The logistic regression analysis in this study revealed that postprandial hypertriglyceridemia had an **independent association with IHD** (OR = 2.78, p = 0.013), even after adjusting for age, sex, diabetes, and waist-hip ratio. This highlights the atherogenic potential of postprandial lipoprotein remnants, which may not be adequately reflected in fasting lipid profiles [5,8].

Anthropometric comparisons revealed that STEMI/NSTEMI patients had significantly higher **waist circumference, waist-hip ratio, and BMI**, all of which are established markers of visceral adiposity and metabolic dysfunction. Importantly, a significant **positive correlation was found between postprandial triglyceride levels and waist-hip ratio**, particularly among STEMI/NSTEMI patients (r = 0.58, p = 0.001), suggesting that central obesity may exacerbate postprandial lipemia and contribute to atherosclerosis [9].

Diabetes mellitus, another key cardiometabolic risk factor, was prevalent in both groups but did not significantly differ in distribution. However, patients with diabetes had significantly higher mean postprandial triglyceride levels compared to non-diabetics (258.4 vs. 211.6 mg/dL, p = 0.001), consistent with previous studies linking insulin resistance to impaired postprandial lipid clearance [2,6].

Echocardiographic findings further confirmed the clinical severity in the STEMI/NSTEMI group, with universal wall motion abnormalities and significantly lower ejection fractions compared to unstable angina patients. These functional impairments mirror the underlying structural myocardial damage caused by persistent ischemia and thrombotic occlusion [1,10].

Collectively, these findings support the concept that **postprandial hypertriglyceridemia is not merely a metabolic phenomenon but a critical contributor to coronary artery disease progression**. Current clinical guidelines typically focus on fasting lipid levels; however, growing evidence, including the results of this study, suggests that postprandial testing should be integrated into routine cardiovascular risk assessment, especially in high-risk populations [3,7,9].

Moreover, lifestyle interventions and pharmacologic therapies targeting postprandial lipid metabolism—such as omega-3 fatty acids, fibrates, and dietary fat modification—may have potential for **early intervention and secondary prevention** of cardiovascular events [6,8].

CONCLUSION

The study demonstrates that postprandial hypertriglyceridemia is significantly more prevalent in patients with STEMI/NSTEMI compared to those with unstable angina, despite normal fasting lipid levels. It was identified as an independent risk factor for ischemic heart disease, with a significant association observed through logistic regression analysis. Additionally, postprandial triglyceride levels showed strong correlations with waist-hip ratio and the presence of diabetes mellitus, indicating that metabolic factors play a key role in postprandial lipid dysregulation. These findings emphasize the clinical value of assessing postprandial triglycerides to unmask hidden cardiovascular risk and guide early intervention strategies, particularly in patients with central obesity or diabetes.

REFERENCES

1. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: From pathophysiology to practice. *J Am Coll Cardiol*. 2009;54(23):2129–2138.
2. Ceriello A. Postprandial hyperglycemia and diabetes complications: is it time to treat? *Diabetes*. 2005;54(1):1–7.
3. Nordestgaard BG, Benn M, Schnohr P, Tybjærg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death. *JAMA*. 2007;298(3):299–308.
4. Kolovou GD, Anagnostopoulou KK, Cokkinos DV. Pathophysiology of postprandial hypertriglyceridemia in humans. *Curr Vasc Pharmacol*. 2005;3(1):49–59.
5. Jackson KG, Poppitt SD, Minihane AM. Postprandial lipemia and cardiovascular disease risk. *Atherosclerosis*. 2012;220(1):22–33.
6. Lopez-Miranda J, Williams C, Lairon D. Dietary, physiological, and genetic impacts on postprandial lipid metabolism. *Nutr Rev*. 2007;65(6):S53–S59.
7. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. *JAMA*. 2007;298(3):309–316.
8. Kolovou GD, Anagnostopoulou KK, Kostakou PM, et al. Postprandial lipemia in men with metabolic syndrome, hypertensive and healthy subjects. *Lipids Health Dis*. 2005;4:21.
9. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. *J Clin Endocrinol Metab*. 2008;93(11):S9–S30.
10. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. *J Am Coll Cardiol*. 2012;60(16):1581–1598.