

Evaluation of CK-MB, Troponin, Lipid Profile in Diabetic Patients**M. Sangamithra¹, P V Susheel Kumar², K. Divya Teja³, M. Prasanth⁴, M. Keerthi Thej⁵**¹Associate Professor, Department of Biochemistry, ACSR, GMC, Nellore²Assistant Professor, Department of Hospital Administration, ACSR, GMC, Nellore³Assistant Professor, Department of Pathology, ACSR, GMC, Nellore⁴Assistant Professor, Department of Biochemistry, ACSR, GMC, Nellore⁵Assistant Professor, Department of Biochemistry, ACSR, GMC, Nellore

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Abstract**Background:** Patients with diabetes mellitus (DM) are at heightened risk for myocardial infarction (MI). While routine lipid panels often fall within or near conventional target thresholds, underlying atherogenic dyslipidemia may remain uncaptured.**Methods:** This retrospective study evaluated 100 diabetic patients admitted with acute MI (67 STEMI, 33 NSTEMI) at ACSR Government Medical College, Nellore. Analysis encompassed admission cardiac biomarkers (cTnI, CK-MB) alongside retrospective longitudinal lipid profiles retrieved at 18, 12, and 6 months prior to index admission, from which secondary atherogenic lipid indices were derived.**Results:** Admission cTnI and CK-MB confirmed acute myocardial injury. Over the 18-month pre-infarction observation window, routine lipid levels demonstrated mild numerical increases (6-month mean Total Cholesterol: 192.96 mg/dL, Triglycerides: 183.70 mg/dL, LDL-C: 119.32 mg/dL, HDL-C: 36.00 mg/dL), while derived ratios (TC/HDL-C: 5.38, LDL-C/HDL-C: 3.32, Non-HDL-C/HDL-C: 4.38, source-reported log(TG/HDL-C): 0.71) were consistently elevated. Bivariate correlation showed no statistically significant linear associations between 6-month lipid indices and admission cardiac markers. Exploratory comparisons revealed no statistically significant differences between STEMI and NSTEMI subgroups.**Conclusion:** Long term elevation of derived atherogenic lipid indices was observed prior to acute MI presentation despite conventional lipid parameters remaining near reference targets, describing a pattern of chronic dyslipidemia in diabetic individuals.**DOI:** 10.25258/ijcpr.18.8.341

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Introduction

Patients with Diabetes Mellitus (DM) face a significantly elevated risk of developing cardiovascular complications, primarily driven by accelerated atherogenesis and micro and macrovascular structural changes. Myocardial Infarction (MI) remains a major cause of morbidity and mortality in diabetic populations [1]. In acute emergency settings, early and precise diagnosis of myocardial injury is achieved through clinical presentation, electrocardiographic (ECG) changes, and cardiac biomarkers, specifically Creatine Kinase-MB (CK-MB) and Cardiac Troponin I (cTnI) [2]. While cardiac troponins serve as sensitive markers for detecting acute myocardial necrosis, understanding the silent progression of coronary artery disease prior to an acute event remains a crucial clinical priority. Traditionally, routine lipid panels measuring Total Cholesterol (TC), Triglycerides (TG), High-Density

Lipoprotein Cholesterol (HDL-C), and Low-Density Lipoprotein Cholesterol (LDL-C) are utilized to evaluate cardiovascular risk [1]. However, standard lipid profiles which yields near normal or borderline values in diabetic individuals, may practically fail to reflect a increased atherogenic risk characterized by small, dense LDL particles and lipoprotein dysfunctions [3]. To complement the standard lipid panels, derived metric ratios known as atherogenic lipid indices have been studied. Metrics such as Castelli Risk Index I (TC/HDL-C), Castelli Risk Index II (LDL-C/HDL-C), Atherogenic Coefficient (AC = (TC-HDL-C)/HDL-C), Non-HDL Cholesterol (Non-HDL-C), and Lipoprotein Combined Index (LCI) provide complementary risk assessment regarding particle distribution and relative atherogenicity [4,5]. This study evaluates a retrospective cohort of 100 diabetic patients admitted to the hospital with

confirmed acute Myocardial Infarction, supported by elevated troponin I and CK-MB levels. Retrospective longitudinal historical lipid data from these patients was extracted across three serial time points, six months, one year, and one and a half years prior to the event and demonstrate routine lipid parameters. Concurrently, derived atherogenic lipid indices remained consistently elevated over the 18-month pre infarction period. This pattern indicates that subclinical atherogenic dyslipidemia can coexist alongside near normal or borderline lipid profile values that appear more assuring than routine screening, highlighting the utility of integrating secondary atherogenic lipid indices for complementary cardiovascular risk assessment in diabetic management.

Materials and Methods

Study Design and Setting: This retrospective study analyzed clinical and laboratory data obtained from the medical records of 100 hospitalized patients admitted to ACSR Government Medical College and Hospital, Nellore, Andhra Pradesh, India, between July 2025 and January 2026. All included patients had a documented history of diabetes mellitus. Upon admission, clinical classification into acute myocardial infarction (AMI) subgroups was documented based on primary clinical presentation, electrocardiographic (ECG) findings, and elevated cardiac biomarkers, as per institutional protocol:

- **ST-Segment Elevation Myocardial Infarction (STEMI):** n = 67
- **Non-ST-Segment Elevation Myocardial Infarction (NSTEMI):** n = 33

Diagnostic classifications (STEMI vs. NSTEMI) were recorded directly as documented in the primary clinical records.

Study Population and Clinical Variables: Extracted demographic, clinical, and laboratory parameters included:

- **Demographics:** Age and sex.
- **Glycemic Profile:** Fasting plasma glucose (FPG), postprandial plasma glucose (PPG), HbA1c levels, and source-defined HbA1c categories (Normal [$<5.7\%$]: n=40; Near Normal [$5.7\text{--}6.4\%$]: n=20; Slightly Above [$\geq 6.5\%$]: n=40).
- **Cardiac Biomarkers:** Admission levels of Cardiac Troponin I (cTnI) and Creatine Kinase-MB (CK-MB).
- **Longitudinal Lipid Profiles:** Retrospective extraction of historical serial measurements of Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), and Low-Density Lipoprotein Cholesterol (LDL-C) recorded at three sequential historical time points: 18 months, 12 months, and 6 months prior to index MI admission.

Derived Lipid Indices

To evaluate atherogenic dyslipidemia alongside standard lipid parameters, secondary lipid indices were calculated:

- **Non-HDL Cholesterol (Non-HDL-C):** Calculated as $TC - HDL-C$.
- **Atherogenic Ratios:** Atherogenic lipid indices were calculated as ****Castelli Risk Index-I (CRI-I)****, using the ratio of total cholesterol (TC) to high-density lipoprotein cholesterol (HDL-C); ****Castelli Risk Index-II (CRI-II)****, using the ratio of low-density lipoprotein cholesterol (LDL-C) to HDL-C; and the ****Atherogenic Coefficient (AC)****, calculated as the ratio of non-HDL cholesterol (Non-HDL-C) to HDL-C. Since $AC = (TC - HDL-C)/HDL-C$ is mathematically equivalent to $Non-HDL-C/HDL-C$, the latter representation was used to avoid redundancy.

Source-reported $\log(TG/HDL-C)$ ratio: The conventional Atherogenic Index of Plasma (AIP) is calculated as $\log_{10}[TG \text{ (mmol/L)} / HDL-C \text{ (mmol/L)}]$. In the extracted dataset, the reported 6-month mean value of 0.7077 corresponds to $\log_{10}[183.70/36.00]$, calculated using triglyceride and HDL-C values expressed in mg/dL. As individual-level molar conversion data were unavailable, this measure was not considered conventional AIP and is therefore reported throughout as the source-reported $\log(TG/HDL-C)$ ratio.

Cardiac Biomarkers Analysis: Admission cardiac biomarkers (cTnI and CK-MB) were evaluated upon initial hospital presentation. Analytical assay platforms, detection limits, clinical cut-off limits, and sampling protocols were maintained as recorded in primary medical records.

Statistical Analysis: Continuous variables were expressed as mean $\pm$ standard deviation (SD), while categorical variables were summarized using frequencies and percentages (n, %). Pearson correlation coefficients (r) evaluated linear associations between 6-month historical lipid parameters and admission cardiac biomarkers. Two-tailed Welch's independent-samples t-tests were conducted for exploratory cross-sectional comparisons between STEMI and NSTEMI subgroups.

Methodological Note: While the study dataset comprises serial longitudinal observations at 18, 12, and 6 months, patient-level raw paired data were unavailable in primary records; thus, summary trajectory trends are reported descriptive of aggregate population means. A two-sided P-value < 0.05 was considered statistically significant.

Results

Demographic and Baseline Clinical

Characteristics: The study cohort comprised 100 hospitalized diabetic patients admitted with acute myocardial infarction (50% male, 50% female).

The mean age was 60.37 years (range: 55–65 years). Baseline glycemic parameters demonstrated a mean HbA1c of 6.11%, mean fasting plasma glucose (FPG) of 106.16 mg/dL, and mean postprandial plasma glucose (PPG) of 146.89 mg/dL.

Table 1: Demographic and Baseline Clinical Characteristics (N = 100)

Characteristic	Overall Value
Sample Size (N)	100
Sex (Male / Female), n (%)	50 (50%) / 50 (50%)
Age (years), Mean (Range)	60.37 (55–65)
HbA1c (%), Mean	6.11
Fasting Plasma Glucose (mg/dL), Mean	106.16
Postprandial Plasma Glucose (mg/dL), Mean	146.89
Source Reported HbA1c Categories, n (%)	
• Normal (< 5.7%)	40 (40%)
• Near Normal (5.7–6.4%)	20 (20%)
• Slightly Above (>= 6.5%)	40 (40%)
Clinical MI Presentation, n (%)	
• STEMI	67 (67%)
• NSTEMI	33 (33%)

Longitudinal Profile of Standard Lipids and Derived Atherogenic Indices: Serial historical lipid panels evaluated at 18, 12, and 6 months prior to index MI presentation demonstrated numerical increases in conventional lipid parameters alongside progressive elevation of derived atherogenic lipid ratios. To relate these observations to the established clinical guidelines (e.g., NCEP ATP III / AHA targets: Total

Cholesterol < 200 mg/dL, Triglycerides < 150 mg/dL, LDL-C < 100 mg/dL [or < 70 mg/dL in high-risk diabetes], HDL-C > 40 mg/dL for men / > 50 mg/dL for women, TC/HDL-C < 5.0, LDL-C/HDL-C < 3.5, Non-HDL-C/HDL-C < 3.0), absolute routine lipid means remained near standard target limits, whereas derived lipid indices exceeded general desirable thresholds across the pre-infarction period.

Table 2: Longitudinal Profile of Standard Lipids and Derived Indices Prior to Admission

Lipid Parameter / Index	18 Mos	12 Mos	6 Mos	Reference Guidelines
Total Cholesterol (TC) [mg/dL]	184.96	188.96	192.96	< 200 mg/dL (Desirable)
Triglycerides (TG) [mg/dL]	169.70	176.70	183.70	< 150 mg/dL (Normal); 150–199 (Borderline)
HDL Cholesterol (HDL-C) [mg/dL]	38.00	37.00	36.00	> 40 mg/dL (Men) / > 50 mg/dL (Women)
LDL Cholesterol (LDL-C) [mg/dL]	113.32	116.32	119.32	< 100 mg/dL (Optimal); < 70 mg/dL (High Risk)
TC / HDL-C Ratio	4.8829	5.1103	5.3783	< 5.0 (Desirable); < 3.5 (Optimal)
LDL-C / HDL-C Ratio	2.9896	3.1456	3.3238	< 3.5 (Desirable); < 2.5 (Optimal)
Non-HDL-C / HDL-C Ratio	3.8829	4.1103	4.3783	< 3.0 (Desirable Target)
Source-Reported log(TG/HDL-C) Ratio	0.64947	0.67834	0.70773	Evaluated as continuous mass ratio log

Admission Cardiac Biomarkers: Acute admission cardiac biomarkers confirmed myocardial necrosis across the cohort. Mean admission Cardiac Troponin I (cTnI) concentration was 4.413 +/- 1.792 ng/mL (range: 1.35–7.54 ng/mL; diagnostic reference cut-off > 0.04 ng/mL). Mean Creatine Kinase-MB (CK-MB) level was 35.95 +/- 14.96 ng/mL (range: 12.0–62.1 ng/mL; diagnostic reference cut-off > 5.0 ng/mL).

Table 3: Admission Cardiac Biomarkers

Biomarker	Mean +/- SD	Range	Unit	Clinical Cut-off
Cardiac Troponin I (cTnI)	4.413 +/- 1.792	1.35 – 7.54	ng/mL	< 0.04 ng/mL
CK-MB	35.95 +/- 14.96	12.0 – 62.1	ng/mL	< 5.0 ng/mL

Bivariate Correlation Analysis: Pearson correlation analysis (r) between 6-month pre-admission lipid parameters/indices and admission cardiac necrosis markers revealed no statistically significant linear associations. Standard lipid measures and derived ratios showed r values ranging from 0.002 to 0.146 with cTnI and CK-MB (all P > 0.14).

Table 4: Bivariate Correlation Between 6-Month Lipid Parameters and Admission Biomarkers

Variable	Troponin I (r)	P-value	CK-MB (r)	P-value
Source-Reported log(TG/HDL-C)	0.002	0.986	0.133	0.188
TC / HDL-C	0.014	0.893	0.146	0.146
LDL-C / HDL-C	0.033	0.742	0.113	0.261
Non-HDL-C / HDL-C	0.014	0.893	0.146	0.146
Total Cholesterol (TC)	0.023	0.819	0.136	0.176
Triglycerides (TG)	0.038	0.710	0.097	0.338
HDL Cholesterol (HDL-C)	0.034	0.735	0.097	0.335
LDL Cholesterol (LDL-C)	0.012	0.903	0.059	0.557

Exploratory Comparison Between STEMI and NSTEMI Subgroups: Exploratory cross-sectional comparisons between STEMI (n=67) and NSTEMI (n=33) subgroups using Welch's t-test demonstrated no statistically significant differences across lipid fractions, derived atherogenic indices, or admission cardiac markers.

Table 5: Exploratory Comparison Between STEMI and NSTEMI Subgroups

Variable	STEMI (n=67)	NSTEMI (n=33)	P-value
Source-Reported log(TG/HDL-C)	0.708	0.707	0.844
TC / HDL-C	5.378	5.380	0.978
LDL-C / HDL-C	3.318	3.335	0.774
Non-HDL-C / HDL-C	4.378	4.380	0.978
Total Cholesterol (TC) [mg/dL]	193.015	192.848	0.908
Triglycerides (TG) [mg/dL]	184.090	182.909	0.617
HDL Cholesterol (HDL-C) [mg/dL]	36.030	35.939	0.834
LDL Cholesterol (LDL-C) [mg/dL]	119.209	119.545	0.840
Troponin I (ng/mL)	4.395	4.450	0.887
CK-MB (ng/mL)	35.966	35.918	0.988

Discussion

The present retrospective study evaluated conventional lipid parameters and derived atherogenic lipid indices at 6, 12, and 18 months before admission with acute myocardial infarction in patients with diabetes mellitus. The principal finding was that conventional lipid parameters, when considered individually, appeared relatively normal or mildly abnormal during the preadmission period. Total cholesterol remained below 200 mg/dL at all three time points, while LDL-C remained close to desirable level of 100 mg/dL. However, triglycerides were consistently borderline to high and HDL-C remained below the desirable concentration. Thus, conventional lipid concentrations alone did not fully reflect the overall atherogenic risk.

In contrast, the derived indices demonstrated a consistently abnormal pattern. The TC/HDL-C ratio increased from 4.88 at 18 months to 5.38 at 6

months, exceeding the stated desirable level of 5.0 at 6 months. Similarly, LDL-C/HDL-C increased from 2.99 to 3.32, while Non-HDL-C/HDL-C increased from 3.88 to 4.38. The source-reported log(TG/HDL-C) ratio also increased progressively from 0.649 to 0.708. These findings indicate that the atherogenic risk may not be fully reflected by individual lipid concentrations alone.

The clinical relevance of lipid ratios has been described previously. Castelli et al. demonstrated the value of lipid indices, including TC/HDL-C and LDL-C/HDL-C, in assessing coronary heart disease risk [6]. Lipid indices have also been evaluated as cardiovascular risk markers in type 2 diabetes [2]. Niroumand et al. reported an association between Atherogenic Index of Plasma (AIP) and cardiovascular risk [3], while Cai et al. demonstrated an independent association between AIP and coronary artery disease [4]. Frohlich and Dobiášová reported an association between

TG/HDL-C and atherogenic lipoprotein characteristics [5], while Dobiášová and Frohlich described $\log(\text{TG}/\text{HDL-C})$ in relation to lipoprotein particle size and cholesterol esterification [6-9]. Similar relationships between AIP and metabolic complications have also been reported in type 2 diabetes [10-14]. Non-HDL-C reflects cholesterol carried by all major atherogenic lipoproteins and may therefore provide additional information beyond LDL-C alone. Meta-analytic evidence has shown that non-HDL-C, LDL-C, and apolipoprotein B are associated with cardiovascular events [15-17]. In the present study, the elevated Non-HDL-C/HDL-C ratio further highlighted an altered balance between atherogenic and protective lipid fractions, which was not fully evident from total cholesterol levels alone.

Notably, the lipid profile findings do not establish the risk of myocardial infarction. Rather, their persistent elevation of derived lipid indices across the 18, 12, and 6 month follow up intervals, suggesting that the derived lipid indices may provide additional information regarding atherogenic risk that surpasses traditional lipid profile parameters. Additional prospective studies are required to determine their clinical applicability for improved early cardiovascular risk stratification in patients with diabetes. The absence of significant associations between preadmission lipid indices and acute Troponin I or CK-MB levels is biologically plausible because lipid indices reflect long-term atherogenic and metabolic risk, whereas these biomarkers indicate acute myocardial injury.

Similarly, the lack of significant differences in lipid indices between STEMI and NSTEMI patients (all $P > 0.05$) suggests that the underlying atherogenic lipid pattern was broadly similar in both types of myocardial infarction. Thus, derived lipid indices may be more useful for identifying underlying cardiovascular risk than for assessing the severity or clinical type of acute myocardial infarction. However, their clinical usefulness needs to be confirmed in larger prospective studies.

Conclusion

This retrospective study demonstrated that diabetic patients admitted with acute myocardial infarction exhibited elevated derived atherogenic lipid indices (TC/HDL C, LDL C/HDL C, Non HDL C/HDL C, and source reported $\log(\text{TG}/\text{HDL C})$) over an 18month pre infarction observation period, while routine absolute lipid values remained near normal levels. These findings describe a pattern where routine lipid profiles alone may in complete depict underlying lipid particle distribution. Integrating derived atherogenic lipid indices alongside standard lipid panels offers a practical, complementary approach for cardiovascular risk assessment in routine diabetic management.

Limitations of the Study

1. Retrospective single-center design: The study was conducted retrospectively at a single institution, limiting the generalizability of the findings.
2. Absence of a non-MI control group: As only acute MI patients were included, diagnostic performance of lipid indices, including sensitivity, specificity, and ROC analysis, could not be assessed.
3. Incomplete historical data: Detailed treatment information and patient-level molar TG and HDL-C values were unavailable, limiting adjustment for potential confounders and independent calculation of standard molar AIP.

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