

Dyslipidemia And Early Lipid Profile Changes in Patients with Acute Myocardial Infarction: A Prospective Observational Study

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Abstract

Dyslipidemia is an important modifiable risk factor for atherosclerotic cardiovascular disease and is closely associated with the development of coronary artery disease and acute myocardial infarction (AMI). Total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides and high-density lipoprotein cholesterol (HDL-C) provide important information regarding the lipid-related cardiovascular risk of patients. However, lipid concentrations may undergo short-term changes following an acute myocardial infarction because of acute-phase and metabolic responses. Therefore, understanding the behavior of lipid parameters during the early period following AMI is important for appropriate interpretation and clinical management.

Aim: To evaluate the lipid profile and its early changes in patients with acute myocardial infarction during the first 12 hours after hospital admission.

Materials and Methods: This prospective observational study was conducted in the Department of Biochemistry in collaboration with the Departments of Cardiology and Emergency Medicine at MGM Medical College and Hospital, Navi Mumbai, from August 2024 to April 2026. The study included 110 patients diagnosed with myocardial infarction on the basis of clinical, electrocardiographic and biochemical findings. Patients aged 18–80 years of either gender who provided written informed consent were included. Patients with recent surgery or trauma, pregnant or lactating women, patients outside the specified age range and those unwilling to provide consent were excluded. Blood samples were collected at admission (0 hour) and 12 hours after admission. Serum total cholesterol (TC), triglycerides (TG), HDL-C, LDL-C and VLDL-C were assessed using standard biochemical methods. LDL-C and VLDL-C were calculated using standard formulas. Statistical analysis was performed using the paired t-test, and $p < 0.05$ was considered statistically significant.

Results: The mean TC concentration was 211.9985 ± 13.2163 mg/dL at admission and 207.4628 ± 26.5348 mg/dL at 12 hours. Mean TG concentration was 169.9974 ± 13.3914 mg/dL at admission and 172.0111 ± 22.3236 mg/dL at 12 hours. Mean HDL-C was 38.0034 ± 4.6943 mg/dL and 36.3220 ± 14.2705 mg/dL, respectively. Mean LDL-C was 139.9957 ± 12.7235 mg/dL at admission and 136.7387 ± 30.2242 mg/dL at 12 hours, while VLDL-C was 33.9995 ± 2.6784 mg/dL and 34.4022 ± 4.4648 mg/dL, respectively. None of the lipid parameters demonstrated a statistically significant difference between admission and 12 hours ($p > 0.05$).

Conclusion: Patients with acute myocardial infarction demonstrated an adverse lipid profile characterized by elevated mean total cholesterol and LDL-C concentrations together with relatively low HDL-C concentrations. However, TC, TG, HDL-C, LDL-C and VLDL-C did not change significantly during the first 12 hours after admission. These findings suggest that lipid assessment performed during the early phase of AMI may provide useful information regarding the patient's lipid status, although the present study cannot determine the pre-infarction lipid profile or establish a causal relationship between individual lipid parameters and AMI.

Keywords: Acute myocardial infarction; dyslipidemia; lipid profile; total cholesterol; LDL cholesterol; HDL cholesterol; triglycerides; VLDL cholesterol; coronary artery disease.

1. Introduction

Acute myocardial infarction (AMI) is one of the major clinical manifestations of atherosclerotic cardiovascular disease and remains an important cause of morbidity and mortality worldwide. Atherosclerosis develops over a prolonged period and is strongly influenced by the concentration and duration of exposure to atherogenic lipoproteins. Among the conventional lipid parameters, low-density lipoprotein cholesterol (LDL-C) has a particularly important role in the development and progression of atherosclerotic cardiovascular disease. Evidence

from genetic studies, prospective epidemiological studies and randomized clinical trials has demonstrated a consistent relationship between the magnitude of LDL-C exposure and the risk of atherosclerotic cardiovascular disease.¹ LDL particles enter the arterial wall and can undergo retention and modification, contributing to endothelial dysfunction, inflammation, macrophage lipid accumulation and atherosclerotic plaque development. The cumulative exposure to atherogenic lipoproteins is therefore an important determinant of long-term cardiovascular risk.² The European Atherosclerosis Society has emphasized that LDL is not merely associated with atherosclerotic cardiovascular disease but plays a causal role in its development.^{1,2}

Although LDL-C is the principal conventional lipid target, other components of the lipid profile also contribute to cardiovascular risk. Triglyceride-rich lipoproteins and their remnants have received increasing attention because epidemiological and genetic evidence suggests that elevated triglycerides and remnant cholesterol are associated with cardiovascular disease.^{3,4} In particular, triglyceride-rich lipoprotein remnants can penetrate the arterial wall and contribute to atherogenesis and vascular inflammation.⁴

HDL-C has traditionally been considered protective because HDL participates in reverse cholesterol transport. However, the relationship between HDL-C concentration and cardiovascular disease is more complex than previously thought. Studies have suggested that HDL function, particularly cholesterol efflux capacity, may provide information beyond HDL-C concentration alone.⁵ Consequently, a low HDL-C concentration should be interpreted as part of the overall metabolic and cardiovascular risk profile rather than as an isolated therapeutic target.

Other atherogenic lipoproteins may also contribute to cardiovascular risk. Lipoprotein(a) [Lp(a)] is an apoB-containing lipoprotein with a substantial genetic component and has been identified as an independent causal risk factor for atherosclerotic cardiovascular disease through atherogenic, inflammatory and potentially prothrombotic mechanisms.⁶ Although Lp(a) was not measured in the present study, its importance illustrates why the conventional lipid profile represents only part of the total spectrum of lipid-associated cardiovascular risk.

The lipid profile of a patient with AMI is clinically important not only because dyslipidemia contributes to the underlying atherosclerotic process but also because lipid concentrations may change following an acute cardiovascular event. Acute illness may produce alterations in cholesterol and lipoprotein concentrations through inflammatory, metabolic and hormonal mechanisms. Serial studies in AMI have demonstrated reductions in total cholesterol, LDL-C and HDL-C, with changes in triglycerides during the days following infarction.⁷

The present study therefore focuses specifically on the lipid profile of patients with AMI and evaluates whether total cholesterol, triglycerides, HDL-C, LDL-C and VLDL-C demonstrate significant changes between admission and 12 hours after admission. Determining the stability of these conventional lipid parameters during this early interval may help clarify the usefulness of early lipid assessment in patients presenting with AMI.

2. Aim

To evaluate the lipid profile and early changes in lipid parameters among patients with acute myocardial infarction during the first 12 hours after hospital admission.

3. Objectives

1. To estimate serum total cholesterol, triglycerides, HDL-C, LDL-C and VLDL-C in patients with acute myocardial infarction at the time of admission (at 0 hour).
2. To estimate the same lipid parameters at 12 hours after hospital admission.
3. To compare lipid profile parameters between at the time of admission (at 0 hour) and at 12 hours after admission.
4. To assess whether significant short-term changes occur in lipid parameters during the first 12 hours following AMI.

4. Materials and Methods

4.1 Study design

This was a prospective observational study conducted in the Department of Biochemistry in collaboration with the Departments of Cardiology and Emergency Medicine at MGM Medical College and Hospital, Navi Mumbai, Maharashtra, India.

The study was conducted from August 2024 to April 2026 and was approved by the Institutional Ethics Committee

before commencement. Written informed consent was obtained from all participants.

4.2 Study population

A total of 110 patients diagnosed with myocardial infarction were enrolled.

Diagnosis of MI was based on the clinical presentation, electrocardiographic findings and biochemical evidence as determined by the treating clinical team.

4.3 Inclusion criteria

Patients fulfilling the following criteria were included:

- Age 18–80 years.
- Either gender.
- Diagnosed cases of myocardial infarction.
- Willingness to provide written informed consent.

4.4 Exclusion criteria

Patients were excluded if they had:

- Recent surgery.
- Recent trauma.
- Conditions likely to influence serum biochemical parameters.
- Age below 18 years or above 80 years.
- Pregnancy or lactation.
- Inability or unwillingness to provide written informed consent.

4.5 Sample collection

Blood samples were collected at two time points:

- **At 0 hour:** At the time of hospital admission.
- **At 12 hours:** Twelve hours after admission.

Blood was collected in plain vacutainers. Serum was separated by centrifugation and used for biochemical analysis.

4.6 Lipid profile estimation

The following lipid parameters were assessed:

i.Total cholesterol: Total cholesterol was estimated using the cholesterol oxidase-peroxidase (CHOD-PAP) method.

ii.Triglycerides: Triglycerides were estimated using the GPO-Trinder method.

iii.HDL cholesterol: HDL-C was estimated using the phosphotungstic acid method.

iv.LDL cholesterol: LDL-C was calculated using a standard calculation formula.

v.VLDL cholesterol: VLDL-C was calculated using a standard formula.

4.7 Statistical analysis

Data were expressed as mean $\pm$ standard deviation (SD). Lipid parameters measured at the time of admission (at 0 hour) and 12 hours after admission were compared using the paired t-test. A p-value <0.05 was considered statistically significant.

5. Results

A total of 110 patients with myocardial infarction were included in the analysis. Lipid profile parameters were evaluated at the time of admission (at 0 hour) and at 12 hours after admission.

Table 1. Lipid profile at the time of admission (at 0 hour) and At 12 hours after admission

Lipid parameter	At the time of admission (at 0 hour) Mean $\pm$ SD	At 12 hours after admission Mean $\pm$ SD	Statistical significance
Total cholesterol (mg/dL)	211.9985 $\pm$ 13.2163	207.4628 $\pm$ 26.5348	p > 0.05

Triglycerides (mg/dL)	169.9974 ± 13.3914	172.0111 ± 22.3236	p > 0.05
HDL-C (mg/dL)	38.0034 ± 4.6943	36.3220 ± 14.2705	p > 0.05
LDL-C (mg/dL)	139.9957 ± 12.7235	136.7387 ± 30.2242	p > 0.05
VLDL-C (mg/dL)	33.9995 ± 2.6784	34.4022 ± 4.4648	p > 0.05

Total cholesterol: The mean total cholesterol concentration decreased numerically from 211.9985±13.2163 mg/dL at admission to 207.4628±26.5348 mg/dL at 12 hours. This difference was not statistically significant (p>0.05).

Triglycerides: The mean triglyceride concentration increased slightly from 169.9974±13.3914 mg/dL at admission to 172.0111±22.3236 mg/dL at 12 hours. The difference was not statistically significant (p>0.05).

HDL cholesterol: The mean HDL-C concentration decreased from 38.0034±4.6943 mg/dL at admission to 36.3220±14.2705 mg/dL at 12 hours. The difference was not statistically significant (p>0.05).

LDL cholesterol: The mean LDL-C concentration decreased from 139.9957±12.7235 mg/dL at admission to 136.7387±30.2242 mg/dL at 12 hours. The difference was not statistically significant (p>0.05).

VLDL cholesterol: The mean VLDL-C concentration increased slightly from 33.9995±2.6784 mg/dL at admission to 34.4022±4.4648 mg/dL at 12 hours. This change was not statistically significant (p>0.05).

Overall, none of the five conventional lipid parameters demonstrated a statistically significant change between at the time of admission and at 12 hours after admission.

6. Discussion

Dyslipidemia is an established component of cardiovascular risk and plays an important role in the development of atherosclerotic coronary artery disease. The present study evaluated five conventional lipid parameters-TC, TG, HDL-C, LDL-C and VLDL-C-in 110 patients with AMI and compared their concentrations at the time of admission and at 12 hours after admission.

The principal finding of the present study was that none of the lipid parameters changed significantly during the first 12 hours. Total cholesterol showed a small numerical reduction, while triglycerides and VLDL-C showed small numerical increases. HDL-C and LDL-C also demonstrated modest numerical reductions. However, all differences were statistically nonsignificant.

Previous studies have reported that lipid concentrations can change following AMI, although the magnitude and timing of these changes vary. A study of patients with AMI reported no significant change in total cholesterol during the hospital stay, while other lipid fractions, particularly HDL-C and triglycerides, demonstrated more evident changes later in the clinical course.⁸

Similarly, Shrivastava et al. reported significant changes in lipid concentrations between days 1, 2 and 7 following AMI, supporting the concept that the timing of measurement is important.⁷ Their study also suggested that lipid assessment within 24 hours of infarction provides a useful opportunity to assess lipid status before more pronounced acute-phase changes occur.

Ference et al. demonstrated a dose-dependent relationship between cumulative LDL-C exposure and atherosclerotic cardiovascular disease risk across genetic, epidemiological and randomized clinical evidence.¹ Borén et al. subsequently summarized pathophysiological, genetic and therapeutic evidence supporting the causal role of LDL in atherosclerotic cardiovascular disease.²

Therefore, the elevated LDL-C observed in the present AMI population may be clinically relevant as an indicator of underlying atherogenic risk, although causal conclusions cannot be drawn from the present observational dataset. Previous studies have demonstrated that HDL-C may decline following AMI, particularly after the first day.^{7, 8} The absence of a significant change in the present study may therefore be related to the relatively short observation period of only 12 hours.

The biological role of HDL is complex. Although low HDL-C has traditionally been associated with cardiovascular risk, increasing HDL-C pharmacologically has not consistently produced cardiovascular benefit. Research has therefore increasingly focused on HDL function, particularly cholesterol efflux capacity, rather than HDL-C concentration alone.⁵ Rohatgi et al. demonstrated an association between cholesterol efflux capacity and incident cardiovascular events, highlighting the importance of HDL function beyond its circulating concentration.

Previous studies of AMI have reported an increase in triglyceride concentrations during the days following

infarction.^{7,8,11} The lack of significant change during the first 12 hours in the present study may indicate that substantial acute-phase alterations had not yet developed during the short observation period.

The finding therefore supports the importance of considering the timing of lipid measurement when interpreting triglyceride concentrations after AMI.

VLDL particles are triglyceride-rich lipoproteins and contribute to the pool of atherogenic remnant particles. Remnant cholesterol has been investigated as a potential causal risk factor for ischemic heart disease.^{12,13}

6.1 Early lipid assessment following AMI

One of the important clinical considerations in AMI is the timing of lipid measurement. Acute illness can influence lipid metabolism and may cause changes in serum lipid concentrations. Historical studies and more recent serial investigations have demonstrated that lipid concentrations may change progressively during the days following myocardial infarction.^{7,8,9,10}

The present study observed no statistically significant differences in TC, TG, HDL-C, LDL-C or VLDL-C between at the time of admission and at 12 hours after admission. These findings indicate that, within this particular cohort, conventional lipid parameters remained relatively stable during the initial 12-hour period.

This observation is consistent with the broader evidence that lipid assessment should be performed early following AMI rather than being unnecessarily delayed. A previous Indian study concluded that lipid assessment within 24 hours of AMI was preferable because subsequent changes could influence interpretation.⁸

6.2 Clinical relevance of lipid assessment

Lipid assessment remains important in patients with AMI because AMI represents a very high-risk manifestation of atherosclerotic cardiovascular disease. Identification of elevated LDL-C and other atherogenic lipid abnormalities can support long-term cardiovascular risk management.

Current lipid-management recommendations emphasize intensive LDL-C lowering in patients with established atherosclerotic cardiovascular disease, with additional lipid-lowering therapies considered when LDL-C remains above the desired level despite maximally tolerated statin therapy.^{14,15}

The present study therefore supports the practical value of obtaining a lipid profile early during hospitalization for AMI. However, treatment decisions should be based on the complete clinical context and applicable contemporary guidelines rather than on the lipid values from this study alone.

7. Conclusion

The present prospective observational study demonstrated that patients with acute myocardial infarction had a lipid profile characterized by a relatively high mean total cholesterol and LDL-C concentration together with relatively low HDL-C concentration.

Despite these findings, none of the conventional lipid parameters-total cholesterol, triglycerides, HDL-C, LDL-C or VLDL-C-showed a statistically significant change between at the time of admission and at 12 hours after hospital admission.

These findings indicate that the conventional lipid profile remained relatively stable during the first 12 hours in this cohort. Early lipid assessment during AMI hospitalization may therefore provide useful information for identifying underlying lipid abnormalities and planning subsequent cardiovascular risk management.

8. References

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