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Original Article

The Association Between LDL Levels and Heart Failure Incidence in Patients with Acute Myocardial Infarction: Observational Study

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Abstract

Background: Acute Myocardial Infarction (AMI) is one of the leading causes of cardiovascular morbidity and mortality worldwide. A serious complication that can arise from AMI is heart failure, which can significantly worsen the patient's prognosis. Low Density Lipoprotein (LDL) is recognized as a major risk factor for atherosclerosis and plays a critical role in the pathophysiology of AMI. This study aims to determine whether there is an association between LDL levels and the incidence of heart failure in patients with acute myocardial infarction.

Methods: This observational study used medical records from Purwokerto Islamic Hospital (January 2022-December 2024) relating to patients diagnosed with acute myocardial infarction, regardless of the presence of heart failure. LDL levels were categorized as optimal or non-optimal using a cut-off level of 100 mg/dL. Bivariate analysis was performed using RStudio, while baseline characteristics that were classified by the presence or absence of heart failure status were examined with SPSS software platform.

Results: Statistical analysis using the Chi-square test revealed a significant association between LDL levels and the incidence of heart failure in patients with acute myocardial infarction at Islamic Hospital Purwokerto, with a p-value of 3.52e-10/ < 0.05.

Conclusions: Higher LDL levels are significantly associated with an increased risk of heart failure in AMI patients, highlighting the importance of LDL control. Further studies should consider additional factors like infarct size, myocardial injury, hypertension, diabetes, ejection fraction, and the role of inflammation for a more comprehensive risk assessment.

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Introduction

Acute Myocardial Infarction (AMI) constitutes a critical and potentially fatal coronary incident linked with Sudden Cardiac Death (SCD), representing the most extreme clinical expression of coronary artery disease. This condition can be classified into two subtypes: ST-Elevation Myocardial Infarction (STEMI) and non-ST-Elevation Myocardial Infarction (NSTEMI). Given that unstable angina often precedes the onset of AMI, it is also recognized as a form of Acute Coronary Syndrome (ACS).[1]

Myocardial Infarction (MI) is a major contributor to cardiovascular diseases, which are the world's leading cause of death. In 2016, they caused 17.9 million deaths, making up 31% of global deaths. Over 75% of these deaths occurred in developing countries, with low- and middle-income nations accounting for 82% of all cardiovascular disease deaths.[2]

AMI frequently results in heart failure, an undesirable side effect. Depending on patient characteristics, treatment techniques, and follow-up period, the cumulative incidence of Heart Failure (HF) following AMI in contemporary cohorts can range from 10% to over 30%. According to Jenča et al.'s comprehensive meta-analysis and review, for example, the development of HF following MI significantly lowers the prognosis and continues to be a major worldwide health burden.[3]

Low-Density Lipoprotein Cholesterol (LDL-C) is a crucial factor in the context of lipid metabolism in atherosclerosis, activating endothelial cells to produce pro-inflammatory molecules, impairing vasodilation, increasing monocyte infiltration, promoting foam cell formation, and stimulating smooth muscle cell proliferation. One obvious risk factor for cardiovascular disease is elevated LDL-C levels, with a direct correlation between LDL-C levels and the inflammatory burden in individuals who have suffered an AMI.[4-5] the division of LDL levels can be categorized into optimal and non-optimal, with a threshold value of 100 mg/dL.[6]

AMI leads to HF through myocardial damage, ischemia, mechanical complications (e.g., papillary muscle rupture), cardiomyocyte changes, progressive necrosis, reperfusion-induced damage via Reactive Oxygen Species (ROS), thrombotic embolization causing microvascular dysfunction, and inflammation from myocyte death.[3] Despite established links between Low-Density Lipoprotein (LDL) levels and cardiovascular diseases, the specific association among patients with AMI, elevated LDL levels, and the incidence of HF remains underexplored, leaving a critical gap in understanding how lipid profiles, particularly LDL, influence HF development. This study aims to fill this gap, potentially guiding more targeted interventions to prevent HF in high-risk patients, ultimately improving patient outcomes and reducing healthcare burdens.

Methods

A cross-sectional, observational, analytic, quantitative design was used in this study to determine the relationship between LDL levels and the incidence of HF among patients with AMI at Purwokerto Islamic Hospital. The location of this study was Purwokerto Islamic Hospital in Banyumas Regency, Central Java. This study was conducted in January-February 2025 using patients' electronic medical records. This research has received ethical approval from the Purwokerto Islamic Hospital.

The study population consisted of inpatients diagnosed with AMI. Consecutive sampling was used to select participants based on defined inclusion and exclusion criteria. The sample size was determined using GPower 3.1.9.7 software (Heinrich Heine University Düsseldorf, Germany)[7], with an effect size of 0.3 (medium), a significance level of $p < 0.05$, and a statistical power of 84%. Based on these parameters, the final sample size was 97.

Inclusion criteria for this study are patients diagnosed with AMI, identified from retrospective data collected from January 2022 to December 2024. There is data on the results of the first LDL level examination in the patient's electronic medical record, and there are complications in the form of HF. In this study, we confirmed the complications of HF based on data written in the electronic medical record by the doctor in charge of the patient, wrote the degree of HF based on Killip criteria, attached chest x-ray images, and administered furosemide therapy. Exclusion criteria included patients who were diagnosed with HF with a cause other than AMI.

We dichotomize the LDL research variable as optimal (<100 mg/dL) and non-optimal (>100 mg/dL), using an ordinal scale. The LDL cut-off value of 100 mg/dL was chosen based on the 2019 Indonesian Guidelines for the Management of Dyslipidemia.[6] The LDL cut-off value selected for this study was based on prior studies and clinical judgment.

Although the 100 mg/dL threshold was chosen for convenience, it is important to note that this value exceeds current clinical recommendations for

patients with AMI and HF. According to the 2018 American College of Cardiology/American Heart Association (ACC/AHA) and Indonesian Society of Endocrinology (Perkumpulan Endrokinologi Indonesia, PERKENI) guidelines, patients with AMI or HF are classified as very high risk, with an LDL target level of less than 70 mg/dL. Some recent guidelines even suggest an LDL target as low as 55 mg/dL. The 100 mg/dL cutoff value used in this study exceeds the recommended target; however, it was selected for practical reasons and based on available data.

This choice reflects the researcher's discretion in defining cutoff points based on available clinical evidence and data constraints.[8] Thus, we employed 100 mg/dL as a clinically relevant, evidence-based cutoff to categorize in our analysis. For HF in patients with AMI, we categorize the presence or absence of HF and report it in the form of a nominal variable.

We used RStudio software (RStudio, PBC, Boston, MA, USA)[9] to perform bivariate analysis between LDL level categories and the presence or absence of HF complications using Chi-square statistical analysis/Fisher's exact test, while for baseline characteristic analysis, we categorize based on the presence or absence of HF, and we analyze using SPSS software (IBM Corp., Armonk, NY, USA).[10]

A multivariate logistic regression analysis was performed using SPSS to examine the relationship between LDL levels and the incidence of HF in patients with AMI. The analysis was adjusted for potential confounding factors, including age, gender, comorbidities (e.g., hypertension, diabetes), and other variables. Statistical significance was determined at $p < 0.05$.

Results

The study population consisted of 97 patients with a mean age of 62.61 years (Standard Deviation [SD] 10.40) in the HF+ group and 63.60 years (SD 9.96) in the HF- group. Males dominated the gender distribution, comprising 73.1% of patients in the HF+ group and 26.9% in the HF- group. Regarding LDL levels, the mean value in the HF+ group was 143.61 mg/dL (SD 54.46), and in the HF- group, it was 72.68 mg/dL (SD 25.17). The population was divided into two groups: 72 patients in the HF+ group and 25 patients in the HF- group. In addition to age, gender, and LDL levels, other baseline characteristics, such as hypertension and diabetes, were evaluated. The detailed distribution of these characteristics is shown in Table 1.

Table 1. Baseline patient characteristics based on heart failure.

Variable	HF+ (N=72)	HF- (N=25)	P-value[2]
Age	62.61 (10.40)[1]	63.60 (9.96)[1]	0.467
Diagnosis			
NSTEMI	24 (80%)	6 (20.0%)	0.377
STEMI	48 (71.6%)	19 (28.4%)	
Diabetes			
Diabetes+	22 (73.3%)	8 (26.7%)	0.893
Diabetes-	50 (74.6%)	17 (25.4%)	
Hypertension			
Hypertension+	37 (74.0%)	13 (26.0%)	0.958
Hypertension-	35 (74.5%)	12 (25.5%)	
Systolic	137.68 (26.19)[1]	130.52 (38.91)[1]	0.011*
Diastolic	86.25 (16.28)[1]	79.92 (24.14)[1]	0.120
Heart Rate	87.86 (19.29)[1]	78.36 (21.99)[1]	0.217
Respiration Rate	23 (20.24)[2]	22 (20.24)[2]	0.929
Temperature	36.4 (36.2, 36.73)[2]	36.6 (36.3, 36.6)[2]	0.178
LDL	143.61 (54.46) [1]	72.68 (25.17)[1]	0.017*
LDL Level			
Non-Optimal	59 (95.2%)	3 (4.8%)	0.000***
Optimal	13 (37.1%)	22 (62.9%)	

HDL	38.5 (33.00, 51.25)[2]	43 (37.00, 54.00)[2]	0.644
TG	128.89 (46.87)[1]	126.40 (61.12)[1]	0.557
Gender			
Female	23 (76.7%)	7 (23.3%)	0.711
Male	49 (73.1%)	18 (26.9%)	
Smoking History			
Present	49 (73.1%)	18 (26.9%)	0.711
Absent	23 (76.7%)	7 (23.3%)	

[1]Mean (SD) [2]Median (Q1, Q3); n (%)
[2]*p<0.05; **p<0.01; ***p<0.001
Notes: HF: Heart Failure; NSTEMI: Non–ST-Elevation Myocardial Infarction; STEMI: ST-Elevation Myocardial Infarction; LDL: Low-Density Lipoprotein; HDL: High-Density Lipoprotein; TG: Triglycerides.

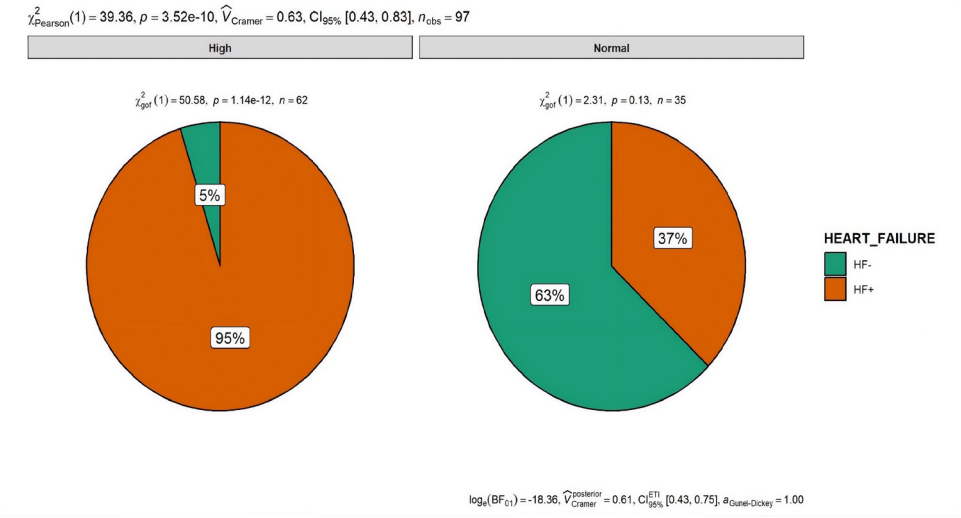

Figure 1. Bivariate analysis of association between LDL levels and heart failure.

In Figure 1, the analysis indicates a clear association between LDL levels and the incidence of HF. Among patients with high LDL levels, 95% developed HF, compared to only 37% of those with normal LDL levels. The association between high LDL levels and HF was statistically significant with a P-value <0.05, suggesting a strong link between elevated LDL levels and the increased risk of HF. On the other hand, individuals with normal LDL levels did not exhibit a significant association with HF (p = 0.13), as 63% of them did not develop HF. This indicates that while high LDL levels significantly contribute to HF incidence, normal LDL levels do not show the same effect. Overall, the findings reinforce the significant impact of high LDL levels on HF risk, while normal LDL levels have little influence.

Table 1 presents the baseline characteristics. The study population consisted of 97 patients, with a mean age of 62.61 years in the heart failure (HF+) group and 63.60 years in the non-heart failure (HF-) group, showing no significant difference in age (p = 0.467). The distribution of diagnoses showed no significant association between the type of diagnosis and the incidence of HF (p = 0.377), with 80% of the HF+ group diagnosed with NSTEMI and 28.4% of the HF- group diagnosed with STEMI. Regarding comorbidities, diabetes was present in 73.3% of the HF+ group and 26.7% of the HF- group, with no significant difference (p = 0.893). Hypertension affected 74% of the HF+ group and 26% of the HF- group (p = 0.958). Systolic Blood Pressure (SBP) differed significantly between the two groups (p = 0.011), with lower values in the HF- group. Diastolic Blood Pressure (DBP), heart rate, respiratory rate, and body temperature did not differ significantly between the two groups (p = 0.120, 0.217, 0.929, and 0.178, respectively). LDL levels were significantly higher in the HF+ group (mean 143.61 mg/dL) than in the HF- group (mean

72.68 mg/dL). Of the HF+ group, 95.2% had high LDL levels, which were strongly correlated with HF ($p = 0.000$). No significant differences were found in HDL levels, triglyceride levels, gender distribution, or smoking history between the two groups ($p = 0.644$, 0.557, 0.711, and 0.711, respectively).

After adjusting for other factors, multivariate logistic regression identified LDL levels as the only variable significantly associated with HF ($p < 0.001$; Table 2). The model demonstrated good overall performance and a strong fit to the data ($p = 0.804$), indicating reliable prediction of HF occurrence among patients with AMI. Higher LDL levels were strongly linked to an increased risk of HF, with each 1 mg/dL increase in LDL associated with a 6.9% higher probability of HF (odds ratio = 1.069, $p < 0.001$). In contrast, other clinical variables, such as age, blood pressure, diabetes, and hypertension, were not significantly associated ($p > 0.05$ for all). These findings suggest that elevated LDL is an independent and dominant predictor of HF in this population.

Discussion

In this study, we looked into the association between the occurrence of HF in individuals who had an AMI and their LDL cholesterol levels. Our main finding indicates that there is a substantial correlation between elevated or non-optimal LDL levels and a higher risk of HF incidence in this group of participants. Additionally, SBP was the only significant baseline characteristic independently associated with the development of HF. These results highlight the interplay between lipid metabolism and hemodynamic stress in post-AMI outcomes, with implications for risk stratification and management.

Our observation that elevated LDL levels correlate with HF incidence aligns with the understanding that dyslipidemia exacerbates ischemic injury and promotes adverse cardiac remodeling.[11] LDL cholesterol contributes to endothelial dysfunction, plaque instability, and microvascular obstruction during AMI, potentially extending infarct size and impairing left ventricular function. This mechanistic pathway is supported by several studies.

Table 2. Multivariate analysis result.

Variable	B	Standard Error (SE)	P-value (Sig.)	Odds Ratio
Age	-0.024	0.040	0.553	0.976
Diagnosis	-0.317	0.885	0.720	0.728
Diabetes	0.641	0.945	0.498	1.899
Hypertension	-0.846	1.233	0.493	0.429
Systole	0.017	0.027	0.516	1.018
Diastole	-0.008	0.035	0.828	0.992
HR	0.025	0.023	0.267	1.025
RR	0.093	0.129	0.469	1.098
Temperature	0.114	0.977	0.907	1.121
LDL	0.067	0.015	<.001	1.069
HDL	-0.016	0.033	0.621	0.984
Triglycerides	-0.010	0.008	0.236	0.990
Gender	0.143	0.889	0.872	1.154

HR: Heart Rate; RR: Respiration Rate; LDL: Low-Density Lipoprotein; HDL: High-Density Lipoprotein.

The CANTOS trial demonstrated that inflammatory pathways driven by atherosclerosis (where LDL is a key mediator) increase HF risk post-AMI.[12] Lowering LDL via targeted therapies reduced HF hospitalizations, underscoring LDL's role in post-infarct complications.[12-13]

The DYSIS II study highlights that individuals suffering from coronary heart disease, such as those with ACS, often present with high LDL cholesterol levels, and many do not meet the recommended target of LDL < 70 mg/dL. In addition, ACS patients have a very high cardiovascular risk, and greater LDL levels have been linked to worse clinical outcomes, such as an increased chance of developing HF. This finding is especially relevant.[14]

Adverse myocardial remodeling, or alterations in the size, shape, and function of the heart muscle, can result from high SBP, especially after an AMI. Increased SBP can worsen left ventricular dysfunction, increasing the likelihood of developing HF. Based on this data, high blood pressure is an established risk factor for HF, strokes, and other cardiovascular diseases.

In AMI patients, those with a history of hypertension are at higher risk of developing HF post-infarction, largely due to the combined effects of high blood pressure on myocardial function and vascular integrity. Studies mentioned in the article, like the GISSI-2 trial, found that hypertensive patients experienced higher mortality rates and worse clinical outcomes after AMI compared to those without hypertension.[15]

The CORONA trial[16] found no HF benefit from LDL lowering in chronic HF patients with ischemic etiology, suggesting that once HF is established, lipid modulation may not reverse remodeling. Contrary to the view from our study that elevated LDL cholesterol levels significantly contribute to HF development, in line with recent research, LDL might not directly contribute to the occurrence of HF, particularly in those who have experienced an AMI.

For instance, research indicates that in certain populations, elevated LDL levels may not significantly affect the development of HF, with other factors, such as blood pressure, infarct size, and comorbidities, being more predictive of HF outcomes.[17] Studies have shown that lowering LDL cholesterol with statin therapy does not always imply a significant reduction in HF. Incidence or mortality, suggesting that LDL levels alone may not be as pivotal in the development of HF as previously thought.[16]

A FOURIER trial sub-analysis noted that while LDL reduction prevented atherosclerotic events, HF risk reduction was less pronounced, implying alternative pathways (e.g., fibrosis, metabolic dysfunction) may dominate HF pathogenesis in stable CAD.[18]

Despite substantial evidence supporting the role of LDL in ischemic injury and adverse cardiac remodeling, recent studies, such as the CORONA and FOURIER trials[16,18], highlight that once HF is established, lipid modulation may have limited efficacy in reversing cardiac remodeling and reducing the incidence of HF.

One of the study's limitations is that the use of lipid-lowering medications, including statins, in the sample was not recorded and therefore not accounted for. To further strengthen the study's findings and reduce potential bias, future researchers should be more precise in identifying which individuals have received statins or other lipid-lowering medications and which have not. A limitation of this study is the use of 100 mg/dL as the LDL cutoff for patients with AMI. This cutoff value exceeds the current clinical guidelines' recommended LDL target of <70 mg/dL for very high-risk patients. Although the chosen cutoff value is based on prior studies and available data, it should be acknowledged that it exceeds the optimal LDL target for this population. This higher cutoff may limit the generalizability of the findings to clinical practice, particularly for cardiovascular risk management.

Future studies may consider using the recommended LDL targets to align with the current guidelines. Another limitations of our study include the observational design, potential confounding factors, and the need for longer follow-up to assess long-term outcomes. Further randomized controlled trials are necessary to more definitively clarify the role of LDL reduction in preventing HF in post-AMI patients and to explore the interplay between lipid metabolism, blood pressure, and myocardial remodeling in this high-risk population.

Conclusion

In conclusion, this investigation discovered a strong association between elevated LDL levels and the increased incidence of HF in AMI patients, with SBP also playing a crucial role. These results highlight the importance of managing both lipid levels and blood pressure in patients with AMI.

List of Abbreviations

ACC	American College of Cardiology
ACS	Acute Coronary Syndrome
AHA	American Heart Association
AMI	Acute Myocardial Infarction
DBP	Diastolic Blood Pressure
HDL	High-Density Lipoprotein
HF	Heart Failure
HR	Heart Rate
ICU	Intensive Care Unit
LDL	Low-Density Lipoprotein
LDL-C	Low-Density Lipoprotein Cholesterol
MI	Myocardial Infarction
NSTEMI	Non–ST-segment Elevation Myocardial Infarction
PERKENI	Indonesian Society of Endocrinology
ROS	Reactive Oxygen Species
RR	Respiratory Rate
SBP	Systolic Blood Pressure
SCD	Sudden Cardiac Death
SD	Standard Deviation
STEMI	ST-segment Elevation Myocardial Infarction

Ethical Clearance

This study has received ethical approval from the Chair of the Health Research Ethics Committee at RSI Purwokerto No. No. 27/ND/KEPK/RSIP/XI/2024.

Publication Approval

All authors are consent to the publication of this manuscript.

Authors Contributions

Ghossan Faisol contributed to the conception and design of the study. Ensuring the consistency and coherence of the theoretical framework was also his responsibility, as was performing the data analysis and interpretation and drafting the manuscript. Sofina Kusnadi, Joriandhita Surya Ramadhan, and Erdiansyah Zulyadaini served as subject matter experts, critically reviewing the manuscript to ensure conceptual accuracy, theoretical consistency, and intellectual coherence, as well as revising it for important intellectual content.

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Conflict of Interest

None.

Availability of Data and Materials

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Generative AI and AI-Assisted Technologies in the Writing Process

The authors declare that they did not use generative artificial intelligence (AI) or AIassisted technologies in writing, editing, or preparing this manuscript. The authors conducted all aspects of the manuscript, including conceptualization, data composition, solely.

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