Journal of Biomedical Sciences and Health
e-ISSN: 3047-7182 | p-ISSN: 3062-6854
Available online at ejournal.unkaha.ac.id/index.php/jbsh

OPEN ACCESS

Vol. 3, No. 2, 2026 DOI: 10.34310/jbsh.v3.i2.360 Pages 114-124

Original Research

Association of neutrophil-to-lymphocyte ratio and low-density lipoprotein cholesterol with admission troponin I status in coronary heart disease: a cross-sectional study

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ABSTRACT

Background: Coronary heart disease involves chronic lipid burden and acute inflammation; troponin I is central to diagnosing myocardial injury. **Objective:** This study evaluated whether the neutrophil-to-lymphocyte ratio (NLR) and low-density lipoprotein (LDL) cholesterol differed by admission troponin I status and characterized each marker's discrimination for this threshold. **Methods:** This cross-sectional study used de-identified data from 38 coronary heart disease patients at a tertiary hospital in Central Java, Indonesia (January-December 2025). The first admission troponin I value was classified as below (<50 ng/L) or at/above (≥50 ng/L) the hospital's clinical decision threshold for a conventional troponin I assay. NLR and LDL were compared between groups, with separate ROC analyses. **Results:** Mean NLR was higher in the at/above-threshold group than the below-threshold group (4.21 ± 1.10 vs 2.28 ± 0.67; $p < 0.001$), whereas mean LDL did not differ significantly (103.47 ± 32.69 vs 106.32 ± 32.79 mg/dL; $p = 0.791$). NLR showed within-sample discrimination for at/above-threshold troponin I (AUC 0.798, 95% CI 0.640-0.955; $p = 0.002$); an exploratory, data-derived threshold of ≥2.92 yielded 84.2% sensitivity and 89.5% specificity. LDL showed no discrimination beyond chance (AUC 0.478, 95% CI 0.290-0.665; $p = 0.815$). **Conclusion:** NLR was higher at/above the local decision threshold and showed fair within-sample discrimination, whereas LDL did not. Because ROC curves were not formally compared and the NLR threshold was derived in the same cohort, these findings are exploratory. NLR warrants validation as a potential adjunctive inflammatory marker but should not replace or delay guideline-recommended troponin testing.

Keywords: coronary heart disease; neutrophil-to-lymphocyte ratio; low-density lipoprotein; troponin I; inflammation

Received: 31 July 2026 | **Revised:** 22 August 2026 | **Accepted:** 24 August 2026 | **Published:** 27 August 2026

Cite this article: Ramadhani, E. A., Afrianti, D., & Wahyudi (2026). Association of neutrophil-to-lymphocyte ratio and low-density lipoprotein cholesterol with admission troponin i status in coronary heart disease: a cross-sectional study. *Journal of Biomedical Sciences and Health*, *3*(2), 114-124. https://doi.org/10.34310/jbsh.v3.i2.360

1. INTRODUCTION

Cardiovascular disease killed an estimated 19.8 million people in 2022, accounting for roughly 32% of deaths recorded globally, and ischemic heart disease alone remains the single largest contributor to that toll (World Health Organization, 2025). In Indonesia, the 2023 national health survey conducted by the Ministry of Health put heart disease prevalence at 0.85% of the population, corresponding to a substantial national burden of diagnosed heart disease and underscoring the need for accessible laboratory-based risk stratification tools (Kementerian Kesehatan Republik Indonesia, 2023).

Coronary heart disease (CHD), the dominant form of ischemic heart disease, develops when atherosclerotic plaque narrows the coronary lumen and starves the myocardium of oxygen. Two processes sit at the center of that narrowing: lipid deposition, principally by low-density lipoprotein (LDL) cholesterol, and a chronic inflammatory response that both initiate and destabilize the plaque (Libby et al., 2019; Khatana et al., 2020). Once LDL particles cross into the arterial intima, they are prone to oxidative modification. Oxidized LDL is taken up by macrophage scavenger receptors in an unregulated manner, and the resulting foam cells form the structural core of the atherosclerotic lesion (Khatana et al., 2020). This mechanism explains why LDL functions well as a marker of cumulative, long-term cardiovascular risk. It provides limited information, however, about whether a plaque has ruptured today or a week ago, because the lipid burden of an artery changes over years, not hours (Shaya et al., 2022).

Cardiac troponin I is a cardiac-specific biomarker of myocardial injury released when cardiomyocytes are damaged. Under the Fourth Universal Definition of Myocardial Infarction, an acute myocardial infarction diagnosis requires a rise and/or fall in cardiac troponin with at least one value above the assay-specific 99th-percentile upper reference limit together with clinical evidence of myocardial ischemia (Thygesen et al., 2019). The 2023 European Society of Cardiology guidelines for acute coronary syndromes likewise place high-sensitivity cardiac troponin at the center of rapid rule-in and rule-out pathways using serial measurements (Byrne et al., 2024). Troponin kinetics depend on assay characteristics, timing from symptom onset, and infarct size; consequently, a single admission value may be below a clinical decision threshold early in the course of an acute event. Access to high-sensitivity assays may also be uneven in resource-constrained settings, motivating interest in inexpensive complementary markers obtainable from routine laboratory testing.

One such marker is the neutrophil-to-lymphocyte ratio (NLR), calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. A healthy-adult study reported an NLR reference interval of 0.78-3.53 (Forget et al., 2017), although such reference values are population- and method-dependent and should not be treated as a universal diagnostic cutoff. During acute inflammation, circulating neutrophils may increase while lymphocytes decrease, so NLR can summarize two components of the systemic inflammatory response (Balta et al., 2016; Buonacera et al., 2022). A 2018 meta-analysis pooling 38 studies and more than 76,000 participants found that higher NLR was associated with acute coronary syndrome and coronary artery disease (Angkananard et al., 2018). More recently, a population-based analysis of more than 13,000 adults in the United States National Health and Nutrition Examination Survey reported an association between higher log-transformed NLR and coronary heart disease after adjustment for lipid profile and traditional risk factors (Wang et al., 2024). A biologically plausible link also exists between acute myocardial injury and changes in circulating leukocyte populations: neutrophils are recruited early after myocardial infarction, while acute stress can contribute to transient lymphopenia (Ma, 2021; Ma et al., 2022).

Prior studies have examined NLR in acute coronary syndromes, but the clinical questions and outcomes have varied. Ahmad et al. (2024) reported higher NLR with greater coronary disease severity among patients with myocardial infarction. Zunardi et al. (2024) proposed an NLR cutoff of 4.5 for identifying NSTEMI in an Indonesian emergency-department cohort, while Ji et al. (2021) evaluated NLR for in-hospital mortality after acute myocardial infarction. These studies address diagnosis, severity, or prognosis rather than the narrower question of how NLR and LDL each relate to a first

admission troponin I value. LDL is an established marker and causal factor in long-term atherosclerotic risk, whereas NLR reflects systemic inflammatory activity over a much shorter time scale. Their relationships with a single admission troponin I measurement therefore need not be similar.

This study therefore examined whether NLR and LDL cholesterol differed according to admission troponin I status in patients with coronary heart disease and characterized the separate ROC-based discrimination of each marker for the hospital-defined troponin I threshold. The study was not designed to establish causality, to diagnose myocardial infarction from NLR or LDL, or to test formal statistical superiority of one ROC curve over the other.

2. METHODS

2.1 Study Design

This was an observational, analytic study with a cross-sectional design. Data were drawn from the medical records and laboratory information system of a tertiary public referral hospital in Klaten, Central Java, Indonesia, covering patients treated between January and December 2025. The study used de-identified secondary data and did not involve direct contact with participants. Because the study was retrospective and relied entirely on pre-existing, anonymized laboratory and clinical records, ethical clearance was obtained from the Health Research Ethics Committee, Universitas Muhammadiyah Purwokerto, prior to data analysis for the use of de-identified secondary data (registration number KEPK/UMP/401/V/2025), and institutional permission was granted by the hospital's research and development unit. Patient identifiers were replaced with numeric codes throughout data handling and analysis.

2.2 Participants

The source population comprised patients diagnosed with coronary heart disease who underwent troponin I, complete blood count, and lipid profile testing during the study period. Thirty-eight records met the eligibility criteria and were included by consecutive inclusion of all eligible records available for analysis. Inclusion required age >18 years, a documented diagnosis of coronaryheart disease, and complete laboratory data for NLR, LDL, and troponin I. Patients with pre-specified conditions likely to substantially alter leukocyte counts or inflammatory status - active infection, hematologic malignancy, or advanced renal failure - were excluded. No a priori sample size calculation was performed; the analyzed sample was determined by the number of eligible records available during the study period. Accordingly, estimates, particularly ROC-based estimates and the data-derived NLR threshold, should be interpreted with attention to their confidence intervals and limited sample size.

2.3 Interventions/Procedures

No study intervention was administered because this was a retrospective observational study. NLR was derived from the admission complete blood count by dividing the absolute neutrophil count by the absolute lymphocyte count. For descriptive categorization only, NLR values were split at 3.53, the upper bound of a reference interval reported in a healthy adult population by Forget et al. (2017); this value was not treated as a validated diagnostic cutoff for acute coronary syndromes. LDL cholesterol was measured in serum by an enzymatic photometric method on a clinical chemistry analyzer, using the earliest lipid panel obtained after admission. Troponin I was measured by chemiluminescent immunoassay on an Abbott ARCHITECT i1000SR analyzer (Abbott Diagnostics, Abbott Park, IL, USA) using the ARCHITECT STAT Troponin-I reagent, a conventional (non-high-sensitivity) assay.

The first troponin I value obtained during the admission diagnostic work-up was used for all analyses and dichotomized as below (<50 ng/L) or at/above (≥50 ng/L) the hospital laboratory clinical decision threshold of 50 ng/L. A 50 ng/L diagnostic threshold has been used with the same contemporary ARCHITECT STAT troponin I assay under local laboratory conditions in a prior clinical cohort (Shah et al., 2015). The present study therefore used the local laboratory decision threshold and did not treat 50 ng/L as the assay manufacturer's 99th-percentile upper reference limit. Troponin I status was the grouping variable, while NLR and LDL were analyzed as continuous candidate markers.

2.4 Data Collection

Data were obtained from medical records and the laboratory information system. The first admission troponin I value obtained during the diagnostic work-up was used for the primary grouping; LDL cholesterol was derived from the earliest lipid panel obtained after admission, and NLR was derived from the admission complete blood count. Records were de-identified, and patient identifiers were replaced with numeric codes during data handling and analysis.

2.5 Statistical Analysis

Continuous variables were summarized as mean ± standard deviation and range, with 95% confidence intervals for group means reported in Table 3. Categorical variables were compared by Fisher's exact test for 2×2 tables; for the 2×3 clinical-subtype table, in which more than 20% of expected cell counts were below 5, the Fisher-Freeman-Halton exact test was used instead of the Pearson chi-square approximation. Distributional assumptions were assessed with the Shapiro-Wilk test. Because no statistically significant departure from normality was detected for NLR or LDL within either troponin subgroup, between-group comparisons used independent-samples t-tests; when Levene's test indicated unequal variances, the Welch correction was applied. Statistical significance was set at $p < 0.05$.

Separate ROC curves were constructed for NLR and LDL against at/above-threshold admission troponin I status. AUCs were reported with standard errors, 95% confidence intervals, and p-values testing the null hypothesis AUC = 0.5. No formal pairwise statistical comparison of the two AUCs was performed. The NLR threshold maximizing the Youden index (sensitivity + specificity - 1) was identified exploratorily, and exact binomial 95% confidence intervals were calculated for sensitivity and specificity. Because the same dataset was used to select and evaluate this threshold, these operating characteristics are apparent (in-sample) estimates and were not internally or externally validated. All analyses were performed in IBM SPSS Statistics version 27.

3. RESULTS

3.1 Participant Characteristics

Of the 38 patients included, 19 (50.0%) had admission troponin I below 50 ng/L and 19 (50.0%) had admission troponin I at/above 50 ng/L. Baseline characteristics are shown in Table 1. Patients in the at/above-threshold group were older on average (64.8 ± 6.4 vs 60.1 ± 7.1 years; $p = 0.035$) and had a markedly different clinical-subtype distribution ($p < 0.001$): all patients in the at/above-threshold group were classified as STEMI or NSTEMI, whereas most patients in the below-threshold group were classified as unstable angina. Sex, hypertension, diabetes mellitus, smoking status, and statin use did not show statistically significant between-group differences, although the small sample limits the precision of these comparisons.

Table 1. Baseline characteristics of patients by troponin I status (n = 38)

Characteristic	Below-threshold troponin I (n = 19)	At/above-threshold troponin I (n = 19)	p-value
Age, years, mean ± SD	60.1 ± 7.1	64.8 ± 6.4	0.035a
Male sex, n (%)	13 (68.4)	14 (73.7)	1.000b
Hypertension, n (%)	12 (63.2)	15 (78.9)	0.476b
Diabetes mellitus, n (%)	6 (31.6)	11 (57.9)	0.191b
Current smoker, n (%)	8 (42.1)	11 (57.9)	0.517b
Statin use, n (%)	12 (63.2)	15 (78.9)	0.476b
Clinical subtype, n (%)			<0.001c
STEMI	0 (0.0)	7 (36.8)	
NSTEMI	5 (26.3)	12 (63.2)	
Unstable angina	14 (73.7)	0 (0.0)	

Note. [a]Independent samples t-test. [b]Fisher's exact test. [c]Fisher-Freeman-Halton exact test (2×3 table). SD, standard deviation; STEMI, ST-elevation myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction.

3.2 NLR and LDL According to Admission Troponin I Status

Table 2. Distribution of NLR and LDL categories by troponin I status (n = 38)

Troponin I status	NLR ≤3.53 n (%)	NLR >3.53 n (%)	LDL <100 mg/dL, n (%)	LDL ≥100 mg/dL, n (%)	Total n (%)
Below threshold (<50 ng/L)	17 (89)	2 (11)	8 (42)	11 (58)	19 (100)
At/above threshold (≥50 ng/L)	5 (26)	14 (74)	9 (47)	10 (53)	19 (100)
Total	22 (58)	16 (42)	17 (45)	21 (55)	38 (100)

Note: The NLR split at 3.53 is used for descriptive purposes only and is based on the upper bound of a reference interval reported in a healthy adult population (Forget et al., 2017); it is not a validated acute coronary syndrome diagnostic cutoff. The 100 mg/dL LDL cut-point is likewise a study-defined descriptive split and does not represent a universal normal/high clinical category.

Descriptively, 17 of 19 patients (89%) with below-threshold troponin I had NLR ≤3.53, whereas 14 of 19 patients (74%) with at/above-threshold troponin I had NLR >3.53. LDL categories did not show a comparable pattern: approximately half of each troponin subgroup had LDL ≥100 mg/dL. These categorical splits were descriptive; the primary inferential comparisons used continuous NLR and LDL values.

Table 3. Descriptive statistics of NLR and LDL cholesterol by troponin I status

Variable	Troponin I status	Minimum	Maximum	Mean ± SD	95% CI
NLR	Below threshold	1.26	3.75	2.28 ± 0.67	1.96-2.61
	At/above threshold	2.35	5.79	4.21 ± 1.10	3.68-4.75
LDL (mg/dL)	Below threshold	55	164	106.32 ± 32.79	90.51-122.12
	At/above threshold	57	175	103.47 ± 32.69	87.72-119.23

Note: SD, standard deviation; CI, confidence interval for the mean.

Figure 1. Mean (± SD) NLR (panel A) and LDL cholesterol (panel B) in patients with below-threshold versus at/above-threshold admission troponin I. Brackets denote the independent-samples t-test p-value for each comparison.

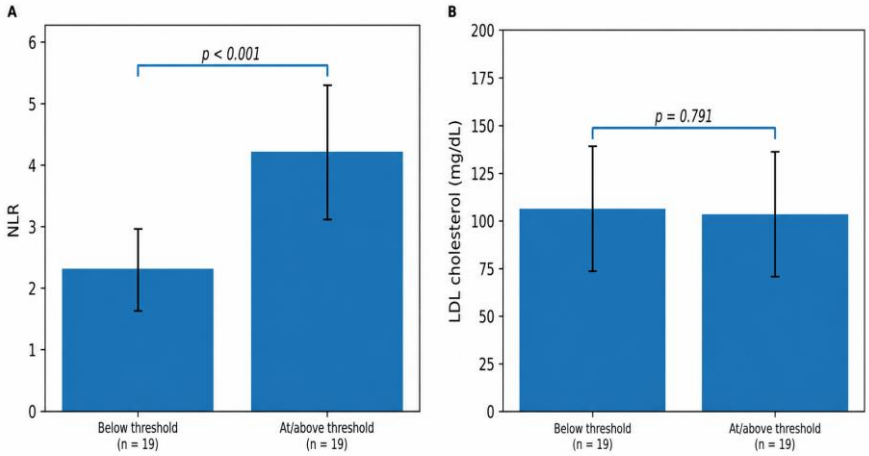

Figure 1. Mean (± SD) NLR (panel A) and LDL cholesterol (panel B) in patients with below-threshold versus at/above-threshold admission troponin I. Brackets denote the independent-samples t-test p-value for each comparison.

Shapiro-Wilk tests did not detect statistically significant departures from normality for either variable in either subgroup (NLR: p = 0.389 for below-threshold troponin I and p = 0.153 for at/above-threshold troponin I; LDL: p = 0.702 and p = 0.373, respectively). Independent-samples t-tests were therefore used as specified in the analysis plan, with Welch correction for NLR because Levene's test indicated unequal variances (Table 4).

Table 4. Independent samples t-test comparing NLR and LDL between troponin I groups

Variable	Levene's F (p)	t	df	Mean difference	p-value
NLR	7.552 (0.009)	-6.509	29.6[a]	-1.93	< 0.001
LDL (mg/dL)	0.035 (0.853)	0.268	36	2.85	0.791

Note: Mean difference computed as below-threshold-troponin-I group minus elevated-troponin-I group. [a]Welch-corrected degrees of freedom owing to unequal variances (Levene's p = 0.009).

3.3 ROC Analysis

In separate ROC analyses, NLR showed fair within-sample discrimination for admission troponin I at/above 50 ng/L (AUC 0.798, 95% CI 0.640-0.955; p = 0.002), whereas LDL had an AUC close to 0.5 (0.478, 95% CI 0.290-0.665; p = 0.815) (Table 5). These p values test each AUC against 0.5; they do not constitute a direct statistical comparison between the NLR and LDL ROC curves.

Table 5. Receiver operating characteristic analysis of NLR and LDL for at/above-threshold admission troponin I status

Test variable	AUC	SE	95% CI	p-value
NLR	0.798	0.080	0.640-0.955	0.002
LDL	0.478	0.096	0.290-0.665	0.815

Note. AUC, area under the receiver operating characteristic curve; SE, standard error. AUC = 0.5 represents chance discrimination. The reported p-values tested AUC = 0.5 separately for each marker; no pairwise comparison of the NLR and LDL AUCs was performed.

The exploratory Youden-index maximum occurred at NLR ≥2.92. At this data-derived threshold, apparent sensitivity was 84.2% (16/19; exact 95% CI 60.4-96.6%) and apparent specificity was 89.5% (17/19; exact 95% CI 66.9-98.7%), corresponding to a Youden index of 0.737. Because threshold selection and performance estimation used the same 38 patients, these values are potentially optimistic and require validation in an independent cohort.

Figure 2. Proportion of patients with NLR ≤3.53 versus NLR >3.53 (panel A) and LDL <100 mg/dL versus LDL ≥100 mg/dL (panel B), stratified by admission troponin I status.

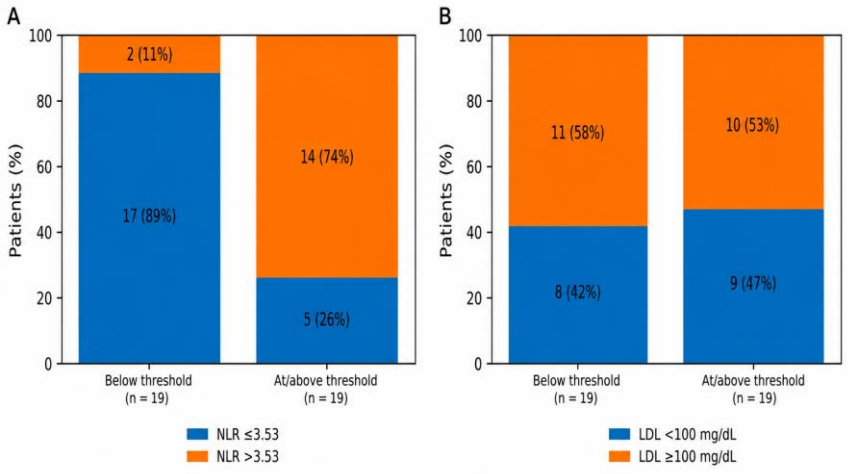

Figure 2. Proportion of patients with NLR ≤3.53 versus NLR >3.53 (panel A) and LDL <100 mg/dL versus LDL ≥100 mg/dL (panel B), stratified by admission troponin I status.

4. DISCUSSION

The principal finding was that NLR differed substantially according to the hospital-defined admission troponin I threshold, whereas LDL did not show evidence of a comparable between-group difference. Mean NLR was nearly twofold higher in the at/above-threshold group (4.21 vs 2.28), and its within-sample ROC AUC was 0.798. However, the 95% CI was wide (0.640-0.955), the ROC analysis was based on only 19 patients per group, and no formal pairwise comparison of the NLR and LDL AUCs was performed. The exploratory NLR threshold of 2.92 was selected and evaluated in the same dataset and was lower than both the upper bound of the healthy-adult reference interval reported by Forget et al. (2017) and the cutoff of 4.5 proposed by Zunardi et al. (2024) for a different diagnostic question. These values are not interchangeable, and the present threshold should be regarded as cohort-specific and hypothesis-generating rather than clinically validated.

The observed NLR pattern is biologically plausible in the context of acute myocardial injury. Neutrophils participate in the early inflammatory response after myocardial infarction, while acute physiological stress can contribute to reduced circulating lymphocyte counts (Ma, 2021; Ma et al., 2022). Because NLR combines both cell populations, a higher value can reflect systemic inflammatory activation accompanying more severe acute coronary presentations. The cross-sectional design, however, cannot determine whether inflammatory activation precedes, follows, or simply co-occurs with the myocardial injury reflected by troponin I.

The direction of the NLR finding is broadly consistent with prior reports, but direct numerical comparisons require caution because populations, assays, outcomes, and timing differ. Ahmad et al. (2024) associated higher NLR with greater coronary disease severity among patients with myocardial infarction, and Zunardi et al. (2024) proposed an NLR threshold of 4.5 for identifying NSTEMI in an Indonesian emergency-department cohort. Ji et al. (2021) evaluated a different endpoint and found NLR useful for predicting in-hospital mortality after acute myocardial infarction. That prognostic AUC cannot be compared directly with the present cross-sectional AUC for admission troponin I status. The current results therefore support an association between NLR and admission troponin category in this sample, but they do not establish diagnostic equivalence to troponin, independent prognostic value, or clinical utility.

LDL cholesterol showed little separation between troponin groups: mean values were similar (106.32 vs 103.47 mg/dL), the mean difference was small relative to its variability, and the ROC AUC was 0.478. These data do not provide evidence that a single admission LDL value discriminates the local troponin I threshold in this cohort. Importantly, this finding

does not challenge LDL's established causal role in atherosclerosis or its importance for long-term cardiovascular risk management (Khatana et al., 2020; Shaya et al., 2022). Rather, LDL and troponin reflect different biological time scales: serum LDL is primarily relevant to chronic atherogenic exposure, whereas troponin reflects myocardial injury around an acute clinical event.

Lipid-lowering therapy may also have attenuated any cross-sectional LDL difference. Statin use was common in both groups (63.2% below threshold and 78.9% at/above threshold), which could narrow the distribution of measured LDL values. Because treatment intensity, duration, adherence, and pre-treatment LDL concentrations were not available for analysis, the present data cannot determine the extent to which therapy influenced the null LDL result.

A major interpretive limitation is the strong imbalance in clinical subtype across troponin groups. The at/above-threshold group consisted entirely of STEMI and NSTEMI presentations, whereas the below-threshold group was predominantly unstable angina (Table 1; $p < 0.001$). Clinical subtype and admission troponin are therefore closely intertwined in this dataset, and both may track the severity and timing of the acute coronary presentation. Age also differed significantly between groups, while diabetes was numerically more common in the at/above-threshold group. With only 38 patients, multivariable adjustment for these factors was not feasible. Consequently, the observed NLR-troponin association should not be interpreted as evidence that NLR contributes information independent of clinical phenotype, age, diabetes, or other unmeasured determinants of inflammatory response.

The interpretation of troponin status also depends on the assay and cutoff used. The study used the hospital laboratory's 50 ng/L clinical decision threshold for the conventional ARCHITECT STAT Troponin-I assay. Shah et al. (2015) likewise described 50 ng/L as a diagnostic threshold for the contemporary ARCHITECT STAT assay under local laboratory conditions, while reporting a manufacturer 99th-percentile upper reference limit of 28 ng/L for that assay. Thus, the present 50 ng/L cutoff should not be equated with the assay-specific 99th-percentile upper reference limit required by the Universal Definition to define myocardial injury. Five patients in the below-threshold admission group were nevertheless classified clinically as NSTEMI. Because only the first admission troponin I value was analyzed, this discordance could reflect early sampling before a subsequent rise, use of additional clinical information, or other features of the final diagnostic process. Serial troponin trajectories, electrocardiographic findings, and angiographic data were not systematically analyzed, so the dataset cannot determine the explanation for these cases. The outcome examined here is therefore biochemical status at presentation relative to a local laboratory threshold, not final adjudicated myocardial infarction status.

4.1 Limitations

Several limitations constrain interpretation. First, the study included only 38 patients from a single center, with no a priori sample size calculation. This limits precision, particularly for ROC estimates and the LDL comparison, and restricts generalizability across populations and assay platforms. A non-significant LDL result should therefore not be interpreted as proof of no association. Second, the cross-sectional retrospective design evaluates measurements around one admission and cannot establish temporal direction or causality. Third, no multivariable adjustment was performed because of the small sample; the marked clinical-subtype imbalance, the age difference, diabetes, treatment patterns, timing from symptom onset, and other unmeasured factors may confound the NLR-troponin relationship.

Fourth, troponin status was defined using a single first-admission value and a local 50 ng/L decision threshold for a conventional assay rather than serial high-sensitivity troponin measurements referenced to an assay-specific 99th percentile. Fifth, the NLR cutoff was selected and evaluated in the same dataset without internal resampling or external validation, so its apparent sensitivity and specificity are susceptible to optimism. Finally, NLR and LDL were each measured once; longitudinal trajectories, pre-admission lipid levels, statin intensity, renal function beyond the exclusion criterion, and other inflammatory biomarkers were not available for analysis.

4.2 Clinical Implications

The present findings are best viewed as hypothesis-generating rather than as evidence for an NLR-based diagnostic pathway. NLR is inexpensive and routinely available from a complete blood count, and its association with the admission troponin category may justify further study as a contextual inflammatory marker. However, NLR should not be used to rule in or rule out myocardial infarction, replace cardiac troponin, or delay guideline-recommended serial troponin testing. Future prospective, multicenter studies should use high-sensitivity troponin assays, record symptom-to-sampling time and serial troponin values, adjust for clinical subtype and other confounders, and validate any NLR threshold in an independent sample before clinical implementation is considered.

5. CONCLUSION

In this 38-patient cross-sectional cohort, NLR was higher among patients whose first admission troponin I value was at/above the hospital laboratory's 50 ng/L decision threshold and showed fair within-sample discrimination (AUC 0.798). LDL cholesterol showed neither a statistically significant mean difference nor evidence of discrimination beyond chance (AUC 0.478). These findings do not establish that NLR is statistically superior to LDL because the ROC curves were not directly compared, and they do not establish independent diagnostic value because clinical subtypes differed markedly between troponin groups and multivariable adjustment was not performed. The data-derived NLR threshold of ≥2.92 requires independent validation before clinical use. NLR may merit further evaluation as an adjunctive inflammatory marker, while LDL remains central to chronic atherogenic risk assessment and secondary prevention. Larger prospective multicenter studies using serial high-sensitivity troponin measurements and prespecified validation strategies are needed.

DECLARATION

Acknowledgments

The authors gratefully acknowledge the laboratory and medical records staff of Dr. Soeradji Tirtonegoro General Hospital, Klaten, for their assistance in facilitating access to the laboratory and medical record data required for this study. The authors also thank the Department of Medical Laboratory Technology, Poltekkes Kemenkes Semarang, for its academic guidance and institutional support throughout the research and manuscript preparation.

Author Contributions

Conceptualization: E.A.R. and D.A.; Methodology: E.A.R. and D.A.; Formal analysis: E.A.R.; Investigation and data curation: E.A.R.; Validation: W.; Writing - original draft: E.A.R.; Writing - review and editing: E.A.R.; Supervision: D.A. and W. All authors read and approved the final manuscript.

Ethics Approval

This study was approved by the Health Research Ethics Committee, Universitas Muhammadiyah Purwokerto (registration number KEPK/UMP/401/V/2025). This approval was granted for the use of de-identified secondary data from existing medical records.

Conflict of Interest

The authors declare no conflict of interest.

Funding

This research received no external funding.

Data Availability

The datasets analyzed during the current study are available from the corresponding author upon reasonable request, subject to the institutional data-sharing policy.

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