

Emergency Department Evaluation of NLR, PLR, and LMR for Predicting Contrast-Induced Nephropathy in Acute Coronary Syndrome Patients: A Retrospective Observational Study

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Abstract

Aim: Contrast-induced nephropathy (CIN) is a significant complication following contrast exposure, especially in patients with acute coronary syndrome (ACS). Inflammation plays a critical role in CIN pathogenesis. This study aimed to evaluate the prognostic value of hematologic inflammation indices-neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-monocyte ratio (LMR), platelet-to-lymphocyte ratio (PLR) and the lactate-to-albumin ratio (LAR) in predicting CIN development in emergency department patients diagnosed with ACS.

Materials and Methods: This retrospective, single-center study included ACS patients who underwent contrast-enhanced imaging or percutaneous coronary intervention between July and December 2024. Inflammatory indices were calculated from complete blood count on admission and biochemical parameters. Patients were categorized as CIN (+) or CIN (–) based on Kidney Disease Improving Global Outcomes criteria. ROC curve analysis was used to assess diagnostic performance.

Results: Among 814 patients, CIN developed in 89 (10.9%). Compared to the non-CIN group, patients with CIN were older (71 vs. 61 years, $p<0.001$) and had significantly higher NLR (3.3 vs. 2.6, $p<0.001$), PLR (143 vs. 117, $p=0.006$), and lower LMR (2.8 vs. 3.3, $p=0.016$). ROC analysis showed that age had the highest area under the curve (0.697), followed by NLR (0.615), PLR (0.590), and LMR (0.578). LMR showed the highest sensitivity (86%), while NLR had the highest specificity (84%). LAR was not significantly associated with CIN ($p=0.208$).

Conclusion: Hematologic inflammation indices such as NLR, LMR, and PLR may serve as cost-effective, accessible tools for early CIN risk stratification in ACS patients. These biomarkers may complement established clinical risk scores and assist in guiding nephroprotective strategies in the emergency setting.

Keywords: Acute coronary syndrome, acute kidney injury, neutrophils, lymphocytes, monocytes, platelets

Introduction

Acute coronary syndrome (ACS) is one of the leading causes of cardiovascular mortality worldwide and represents a critical clinical condition frequently encountered in emergency departments, where diagnosis and treatment are time-sensitive. To confirm the diagnosis of ACS and evaluate concomitant coronary artery disease, imaging modalities involving contrast media are often used. However, the use of intravenous contrast agents, particularly in patients with comorbidities, increases the risk of developing contrast-induced nephropathy (CIN), which

may result in prolonged hospital stay, increased healthcare costs, and worsened clinical outcomes (1).

Although several clinical scoring systems have been proposed to predict the development of CIN, many of these tools are complex and time-sensitive, limiting their applicability in emergency settings. As a result, there is an increasing need for practical, rapid, cost-effective, and objective biomarkers to support timely clinical decision-making. In this context, hematologic inflammatory parameters derived from complete blood count (CBC) data have gained prominence. Notably, indices such as the neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-monocyte ratio (LMR), and



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platelet-to-lymphocyte ratio (PLR) have demonstrated prognostic value as surrogate markers of systemic inflammation in various cardiovascular, neoplastic, and infectious conditions (2-4).

NLR increases in response to acute inflammatory processes due to neutrophilia and lymphocyte suppression, making it a reliable marker of inflammation severity. Elevated NLR has been significantly associated with the development of CIN in patients diagnosed with ACS undergoing percutaneous interventions, and has been identified as an independent predictor in previous studies (5). Similarly, LMR reflects inflammatory activity through increased monocyte levels and decreased lymphocyte counts, both of which play pivotal roles in the regulation of immune responses. Low LMR has been reported to be associated with an increased risk of CIN (6). On the other hand, PLR serves as a marker that reflects both the pro-inflammatory role of platelets (PLTs) and the immunomodulatory function of lymphocytes. A growing body of literature supports the correlation between elevated PLR and adverse cardiovascular outcomes, as well as renal dysfunction, highlighting its potential prognostic utility (7,8).

Beyond hematologic indices, we also evaluated the lactate-to-albumin ratio (LAR). This parameter combines two routinely measured laboratory values: lactate, which reflects tissue hypoperfusion and metabolic stress, and albumin, which indicates nutritional and inflammatory status as a negative acute-phase reactant. Considering that both markers are commonly associated with systemic illness and organ dysfunction, we hypothesized that LAR might also serve as a potential predictor of CIN in patients with ACS.

In conclusion, hematologic inflammatory indices such as NLR, LMR, and PLR, which are easily calculated, cost-free, and widely accessible, may serve as practical tools for predicting CIN in patients presenting to the emergency department with ACS and undergoing contrast-enhanced procedures. Evaluating the predictive utility of these biomarkers may facilitate more effective risk stratification in clinical practice. The findings of this study may contribute to the development of more informed and preventive strategies, particularly in high-risk individuals. Accordingly, the aim of this study is to investigate the prognostic value of hematologic inflammatory markers, including NLR, LMR, PLR, and the LAR, in predicting the development of CIN in ACS patients exposed to contrast media.

Materials and Methods

Study Design and Setting

This manuscript was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology guidelines

to ensure transparent and comprehensive reporting. The study was designed as a single-center, retrospective, descriptive and analytical observational study. Ethical approval was obtained from the Clinical Research Ethics Committee of University of Health Sciences Türkiye, Ankara Etlik City Hospital (decision number: AEŞH-BADEK1-2025-174, date: 07.05.2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Since all participants were anonymized, informed consent was not required.

Study Population and Definitions

The target population of this study consisted of patients who presented to the emergency department with suspected ACS between July 1, 2024, and December 31, 2024, whose diagnosis was confirmed, and who underwent diagnostic or interventional imaging with contrast media.

ACS is a cardiovascular emergency that requires prompt diagnosis and treatment, and encompasses unstable angina pectoris, ST-elevation myocardial infarction (STEMI), and non-STEMI, all of which result from myocardial ischemia (9). The diagnosis of ACS was based on clinical symptoms, electrocardiogram (ECG) changes, and elevated cardiac biomarkers.

CIN was defined as an absolute increase in serum creatinine of ≥ 0.5 mg/dL or a relative increase of $\geq 25\%$ from baseline within 72 hours after contrast administration (10).

Inclusion Criteria

- Age ≥ 18 years
- Confirmed diagnosis of ACS based on clinical presentation, ECG findings, or elevated troponin levels
- Undergoing a diagnostic or interventional procedure involving contrast media
- Availability of serum creatinine measurements at presentation and within 72 hours after contrast exposure

Exclusion Criteria

- Presence of active infection or clinical signs of sepsis
- History of chronic inflammatory or autoimmune disease
- Patients with acute or chronic liver disease
- Patients undergoing chronic medical treatment with steroids or non-steroidal anti-inflammatory drugs, or with a history of organ transplantation
- Use of systemic corticosteroids within the past 2 weeks
- Exposure to contrast media within the past 2 weeks

- History of infection within the past 2 weeks (e.g., respiratory tract infection, urinary tract infection, dental abscess, etc.)
- History of hematologic malignancy or immunosuppressive therapy
- Patients undergoing emergency surgical procedures
- Presence of pre-existing renal failure prior to contrast administration (eGFR <30 mL/min/1.73 m²)
- Patients who received nephroprotective agents such as N-acetylcysteine prior to contrast exposure
- Cases with missing laboratory data
- Patients with multiple exposures to contrast media within 24 hours
- Patients without serum creatinine re-evaluation within 72 hours after contrast administration
- Pregnant patients

Data Collection

Within the specified study period, patients presenting to the emergency department were screened using ICD-10 codes to identify those diagnosed with ACS. Among these, patients that underwent contrast-enhanced imaging procedures were selected. Data were retrospectively retrieved from the hospital information management system. Variables evaluated in the study included demographic, clinical, hematologic, and biochemical parameters. Demographic data included age and sex. Clinical variables comprised heart rate, presence of STEMI, anemia, diabetes mellitus (DM), hypertension (HT), and prior history of percutaneous coronary intervention (PCI). Cardiac rhythm was documented using a standard 12-lead ECG.

In the hematologic evaluation, CBC data were used to assess hemoglobin (HGB), PLT count, mean PLT volume, neutrophil, lymphocyte, and monocyte levels. From these parameters, LMR, NLR, and PLR were calculated.

For the biochemical evaluation, serum glucose, creatinine, albumin, and lactate levels were recorded, and LAR was calculated accordingly. Venous blood samples were obtained within 10 minutes of presentation to the emergency department and prior to coronary angiography. Hematologic parameters, including monocyte, lymphocyte, and neutrophil counts, and HGB, were analyzed within 30 minutes of sampling using an automated hematology analyzer. Biochemical analyses-including creatinine, lactate, albumin, and glucose levels-were performed using standard laboratory techniques. These laboratory results were

documented for all patients. Serum creatinine was re-measured in all patients within 72 hours following contrast exposure.

CIN was evaluated according to the diagnostic criteria defined by the Kidney Disease Improving Global Outcomes guidelines. Accordingly, patients who demonstrated an absolute increase in serum creatinine of ≥ 0.5 mg/dL, or a relative increase of $\geq 25\%$ from baseline within 72 hours following contrast administration, were classified as CIN-positive; those who did not meet these thresholds were categorized as CIN-negative.

To ensure patient confidentiality, all data were processed in a de-identified format. A unique code was assigned to each individual, and personally identifiable information was excluded from the analysis. Missing data were checked on a variable-by-variable basis. Patients with any missing clinical or laboratory parameters were excluded from the study in accordance with the predefined exclusion criteria.

To ensure data accuracy, parallel data entry was performed independently by two researchers. A random 10% sample was selected for cross-checking. Inconsistent entries were verified and corrected through re-examination of the hospital database.

The data collection process was coordinated by the principal investigator (V.S.) and all clinical, laboratory, and diagnostic variables were systematically compiled according to the predefined data collection protocol.

Statistical Analysis

All statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA) and Jamovi version 2.5.7 software. Descriptive statistics for categorical variables were presented as frequencies and percentages, while continuous variables were expressed as medians with [interquartile ranges (IQR), 25th-75th percentiles]. The normality of distribution for continuous variables was assessed using the Kolmogorov–Smirnov test and histogram analysis.

Comparisons of categorical variables were conducted using the chi-square test. For continuous variables with normal distribution, the Student's t-test and Welch's t-test were used depending on the homogeneity of variance, which was evaluated by Levene's test. The Mann–Whitney U test was employed for comparing non-normally distributed continuous variables.

To assess the predictive performance of inflammatory indices for CIN, ROC curve analysis was conducted. From this analysis, threshold values, sensitivity, specificity, positive predictive value, negative predictive value (NPV), positive likelihood ratio, and negative likelihood ratio were calculated. The Youden index was used to determine the optimal cut-off points.

The DeLong test was applied to compare the diagnostic performance [area under the curve, (AUC)] of different biomarkers. A p value of <0.05 was considered statistically significant.

Results

A total of 1.326 patients were initially identified based on ICD-10 codes. Of these, 398 patients were excluded due to missing data. The remaining 928 patients were evaluated for eligibility. Subsequently, patients were excluded for the following reasons: history of contrast media exposure within the past two weeks (n=6); liver disease (n=8); pre-existing renal failure prior to contrast administration (n=13); lack of follow-up creatinine measurement within 72 hours (n=57); multiple exposures to contrast media within 24 hours (n=11); active infection or a history of infection within the past two weeks (n=19). After applying these exclusion criteria, a total of 814 patients were included in the final analysis (Figure 1).

The median age of the participants was 62 years (IQR: 53-71), and 72.4% were male. The most common comorbidity was HT, observed in 52.9% of patients, followed by DM (38.1%), history of PCI (33.7%) and anemia (7.9%). CIN developed in 10.9% of patients. Additionally, 16.5% of the study population was diagnosed with STEMI (Table 1).

The median age of patients who developed CIN was significantly higher than those who did not (p<0.001). HT was more prevalent among patients with CIN (74.2%) than those without CIN (50.3%)

Table 1. Distribution of patients’ demographic characteristics, comorbidities, laboratory parameters, and clinical features	
Age, years, median (IQR)	62 (53-71)
Sex, n (%)	
Male	589 (72.4%)
Female	225 (27.6%)
Hypertension, n (%)	431 (52.9%)
Diabetes mellitus	310 (38.1%)
Anemia	64 (7.9%)
History of PCI	274 (33.7%)
Heart rate, median (IQR)	76 (66-89)
Laboratory parameters, median (IQR)	
Hemoglobin, g/dL	14.4 (12.9-15.5)
Platelet, 109/L	255 (214-300)
Neutrophil, 109/L	5.8 (4.5-7.8)
Lymphocyte, 109/L	2.1 (1.5-2.8)
Monocyte, 109/L	0.67 (0.52-0.83)
Mean platelet volume, fL	10.3 (9.7-11.0)
Blood glucose, mg/dL	130 (104-182)
Creatinine, mg/dL	0.94 (0.81-1.11)
Post-anjio creatinine, mg/dL	0.93 (0.79-1.10)
Lactate, mmol/L	1.7 (1.3-2.4)
Albumin, g/L	40 (38-43)
Lactate / albumin ratio	4.3 (3.3-5.8)
LMR	3.3 (2.4-4.4)
NLR	2.7 (1.8-4.3)
PLR	119 (89-163)
CIN, n (%)	89 (10.9%)
STEMI, n (%)	134 (16.5%)

IQR: Interquartile range, PCI: Percutaneous coronary intervention, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, LMR: Lymphocyte-to-monocyte ratio, CIN: Contrast-induced nephropathy, STEMI: ST-elevation myocardial infarction

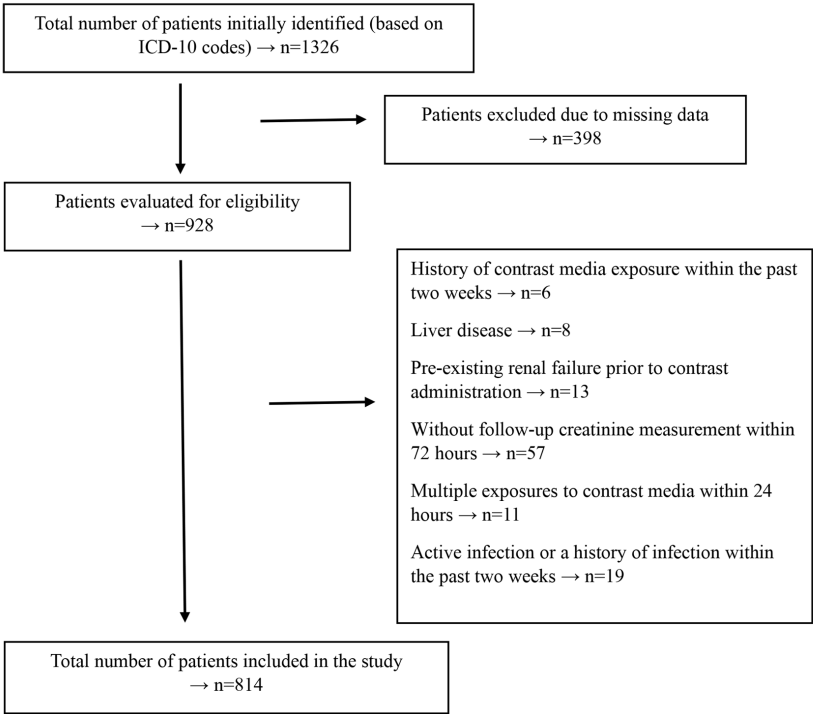


Figure 1. Patient flow diagram

($p<0.001$). Additionally, a prior history of PCI was significantly more common in the CIN group ($p=0.017$) (Table 2).

Median heart rate was higher in patients with CIN ($p=0.020$). HGB levels were significantly lower in the CIN group ($p=0.010$), while PLT was lower ($p=0.029$) (Table 2).

Patients with CIN had significantly lower lymphocyte counts ($p<0.001$) and higher blood glucose levels ($p=0.014$). Both baseline and post-procedural creatinine levels were significantly elevated in the CIN group (Table 2).

The albumin level was slightly lower in the CIN group ($p=0.019$). Moreover, LMR, NLR, and PLR were significantly higher in patients who developed CIN (LMR: $p=0.016$; NLR: $p<0.001$; PLR: $p=0.006$) (Table 2).

Diagnostic Accuracy of Age, NLR, PLR, and LMR in Predicting CIN

As shown in Table 3, the cut-off values determined for predicting CIN were >64 for age, >5.2 for NLR, >137 for PLR, and <2.0 for LMR. Among these, age had the highest AUC: 0.697, followed

by NLR (AUC: 0.615), PLR (AUC: 0.590), and LMR (AUC: 0.578) (Figure 2).

Notably, LMR demonstrated the highest sensitivity [(86%; 95% confidence interval (CI): 83-88)], although its specificity remained low (29%; 95% CI: 20-40). In contrast, NLR showed the highest specificity (84%; 95% CI: 81-86) with relatively low sensitivity (36%; 95% CI: 26-47). NPV were consistently high across all markers, ranging from 0.91 to 0.95 (Table 3).

Pairwise comparisons using the DeLong test revealed that the predictive accuracy of age was significantly superior to that of NLR (AUC difference = 0.082; 95% CI: 0.001-0.163; $p=0.046$), PLR (AUC difference = 0.107; 95% CI: 0.031-0.183; $p=0.006$), and LMR (AUC difference = 0.119; 95% CI: 0.044-0.194; $p=0.002$) (Table 3).

These results indicate that while age exhibited the best overall discriminatory performance, LMR, and NLR may also serve as supplementary inflammatory markers in predicting the risk of CIN.

Table 2. Association of patients' demographic characteristics, comorbidities, laboratory parameters, and clinical features with the development of CIN

	CIN+ (n=89)	CIN- (n=725)	p value
Age, years, median (IQR)	71 (64-76)	61 (52-70)	$<0.0011^{***}$
Sex, n (%)			
Male	58 (65.2%)	58 (65.2%)	0.1082
Female	31 (34.8%)	31 (34.8%)	
Comorbidities, n (%)			
Hypertension, n (%)	66 (74.2%)	365 (50.3%)	$<0.0012^{***}$
Diabetes mellitus	37 (41.6%)	273 (37.7%)	0.4732
Anemia	8 (9.0%)	56 (7.7%)	0.6762
History of PCI	40 (44.9%)	234 (32.3%)	0.0172*
Heart rate, median (IQR)	81 (70-93)	75 (66-88)	0.0203*
Laboratory parameters, median (IQR)			
Hemoglobin, g/dL	13.4 (11.7-15.2)	14.4 (13.1-15.6)	0.0101*
Platelet, 109/L	234 (194-293)	257 (215-302)	0.0291*
Neutrophil, 109/L	6.1 (4.8-8.4)	5.8 (4.5-7.7)	0.0873
Lymphocyte, 109/L	1.7 (1.2-2.6)	2.1 (1.6-2.9)	$<0.0013^{***}$
Monocyte, 109/L	0.67 (0.51-0.79)	0.67 (0.52-0.84)	0.2703
Mean platelet volume, fL	10.4 (9.8-11.0)	10.3 (9.7-11.0)	0.2891
Blood glucose, mg/dL	152 (114-210)	129 (103-181)	0.0143*
Creatinine, mg/dL	1.07 (0.94-1.40)	0.93 (0.80-1.09)	$<0.0013^{***}$
Post-anjio creatinine, mg/dL	1.43 (1.21-1.87)	0.91 (0.78-1.05)	$<0.0013^{***}$
Lactate, mmol/L	1.7 (1.4-2.5)	1.7 (1.3-2.3)	0.4153
Albumin, g/L	41 (38-42)	41 (38-43)	0.0193*
Lactate / albumin ratio	4.3 (3.5-6.6)	4.3 (3.3-5.6)	0.2083
LMR	2.8 (1.8-4.1)	3.3 (2.5-4.5)	0.0163*
NLR	3.3 (2.0-6.9)	2.6 (1.7-4.1)	$<0.0013^{***}$
PLR	143 (101-203)	117 (88-159)	0.0063**
STEMI, n (%)	14 (15.7 %)	120 (16.6 %)	0.8442

Statistical comparisons were made using the independent samples t-test[†], chi-square test[‡], and Mann-Whitney U test[§] as appropriate. *** $p<0.001$, ** $p<0.01$, * $p<0.05$
IQR: Interquartile range, PCI: Percutaneous coronary intervention, CIN: Contrast-induced nephropathy, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, LMR: Lymphocyte-to-monocyte ratio, STEMI: ST-elevation myocardial infarction

Table 3. Cut-off values for age, NLR, PLR and LMR in predicting CIN

	Cut-off	AUC (95% CI)	SEN (95% CI)	SPE (95% CI)	PPD (95% CI)	NPD (95% CI)	+LR (95% CI)	-LR (95% CI)
Age	>64	0.697 (0.642-0.753)	0.78 (0.67-0.86)	0.58 (0.55-0.62)	0.19 (0.17-0.21)	0.95 (0.93-0.97)	1.9 (1.6-2.1)	0.4 (0.3-0.6)
NLR	>5.2	0.615 (0.550-0.680)	0.36 (0.26-0.47)	0.84 (0.81-0.86)	0.21 (0.16-0.27)	0.91 (0.90-0.93)	2.2 (1.6-3.1)	0.8 (0.6-0.9)
PLR	>137	0.590 (0.523-0.657)	0.55 (0.44-0.66)	0.65 (0.60-0.67)	0.16 (0.13-0.19)	0.92 (0.90-0.94)	1.5 (1.2-1.9)	0.7 (0.5-0.9)
LMR	<2.0	0.578 (0.513-0.643)	0.86 (0.83-0.88)	0.29 (0.20-0.40)	0.91 (0.90-0.92)	0.20 (0.15-0.27)	1.2 (1.1-1.4)	0.5 (0.3-0.7)

CIN: Contrast-induced nephropathy, NLR: Neutrophil-to-lymphocyte ratio, PLR: Platelet-to-lymphocyte ratio, LMR: Lymphocyte-to-monocyte ratio, AUC: Area under the curve, PPV: Positive predictive value, NPV: Negative predictive value, SEN: Sensitivity, SPE: Specificity, +LR: Positive likelihood ratio, -LR: Negative likelihood ratio, NPD: New predictive determinant, PPD: Pre-procedural data, CI: Confidence interval

Delong test pairwise comparisons results

Age vs NLR: AUC Difference =0.082 (%95 CI: 0.001, 0.163), p=0.046

Age vs PLR: AUC Difference =0.107 (%95 CI: 0.031, 0.183), p=0.006

NLR vs PLR: AUC Difference =0.025 (%95 CI: -0.020, 0.069) p=0.278

Age vs LMR: AUC Difference =0.119 (%95 CI: 0.044, 0.194) p=0.002

NLR vs LMR: AUC Difference =0.037 (%95 CI: -0.015, 0.088) p=0.165

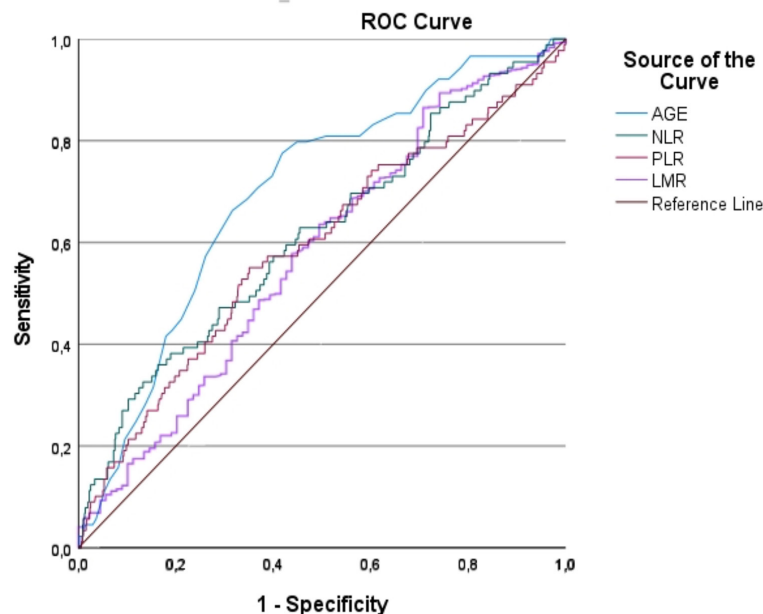


Figure 2. ROC curves of age, NLR, PLR and LMR in predicting CIN

ROC: Receiver operating characteristic, NLR: Neutrophil-to-lymphocyte ratio, LMR: Lymphocyte-to-monocyte ratio, PLR: Platelet-to-lymphocyte ratio, CIN: Contrast-induced nephropathy

Discussion

In this retrospective observational study, we evaluated the prognostic utility of NLR, LMR, PLR, and LAR in predicting the development of CIN in patients diagnosed with ACS and exposed to contrast media in the emergency department setting. According to our findings, elevated NLR and PLR levels, as well as decreased LMR values, were significantly associated with the development of CIN. Moreover, among all evaluated parameters, age was identified as the strongest independent predictor.

CIN is a significant iatrogenic complication, particularly among individuals with cardiovascular comorbidities. It is believed to

develop through mechanisms such as impaired renal perfusion, inflammation, and oxidative stress (11,12). This underlying pathophysiology supports the rationale for using inflammatory biomarkers as potential predictors of CIN.

In this context, NLR, which can be easily calculated from routine CBC parameters, has emerged as a widely used and practical marker of systemic inflammation in clinical practice. Previous studies have reported that NLR is independently associated with the development of CIN in patients who experience myocardial infarction and undergo PCI (3,13,14). Consistent with these findings, our study also demonstrated a significant association between elevated NLR levels (>5.2) and CIN development (13,15).

Notably, the high specificity (84%) and NPV (91%) observed in our analysis suggest that patients with low NLR values are at a considerably lower risk for CIN.

LMR reflects both the cellular and immunologic aspects of the inflammatory response by indicating monocyte elevation and lymphocyte suppression. Previous evidence has shown that low LMR levels are associated with various clinical conditions, including sepsis, cardiovascular disease, and chronic kidney disease (6,16,17). In our study, an LMR threshold of <2.0 demonstrated high sensitivity (86%) for predicting CIN, suggesting that LMR may serve as a valuable screening tool for identifying patients at high risk for CIN.

PLR serves as a biomarker positioned at the intersection of inflammation and thrombosis, as it reflects both the pro-inflammatory activity of PLTs and the immunoregulatory function of lymphocytes (18). In our study, a PLR value greater than 137 was found to be significantly associated with the development of CIN. This finding is consistent with previous literature demonstrating the association of elevated PLR with cardiovascular mortality and renal injury (19-21).

LAR, which has been increasingly used in the literature as a prognostic biomarker for mortality in sepsis and critically ill patients (22-24), was not found to be significantly associated with CIN development in our study. This discrepancy may be attributed to the relatively lower degree of systemic inflammation in ACS patients compared to more severe inflammatory conditions such as sepsis.

Another important factor influencing the development of CIN is the variability in contrast media exposure between diagnostic and interventional coronary procedures. While diagnostic coronary angiography usually involves relatively low volumes, interventional procedures such as PCI typically require substantially higher amounts, particularly in complex or multi-vessel interventions. Since our study did not include standardized data on the type or amount of contrast administered, this procedural heterogeneity may have contributed to CIN risk and should be acknowledged as a limitation.

According to the ROC analysis, age demonstrated the highest AUC: 0.697 when compared to hematologic indices such as NLR, LMR, and PLR. The association between advanced age and increased CIN risk may be explained by age-related physiological changes, including a decline in glomerular filtration rate, heightened susceptibility to oxidative damage, and endothelial dysfunction—all of which contribute to age as an independent and significant risk factor for CIN (25,26).

Although the AUC values of NLR, PLR, and LMR reached statistical significance, their relatively low magnitude restricts their discriminatory power. Therefore, these indices alone are unlikely to substantially influence clinical decision-making. Rather, they should be interpreted as adjunctive markers, supporting established risk scores such as the Mehran score, to improve the overall accuracy of CIN risk prediction.

Our findings suggest that hematologic parameters such as NLR, LMR, and PLR may contribute to CIN risk stratification in clinical practice. These biomarkers offer several advantages, including wide availability, low cost, repeatability, and ease of calculation. However, relying solely on these indices for clinical decision-making is insufficient. Instead, they should be used as complementary tools alongside established risk prediction models, such as the Mehran score, to enhance the accuracy of CIN risk assessment (11).

Nevertheless, the retrospective and single-center nature of our study restricts external validity, and the findings should be interpreted with caution when applied to different populations or healthcare settings.

Study Limitations

Although this study possesses several strengths, certain limitations should be acknowledged. First, its retrospective and single-center design restricts the ability to establish causal relationships and limits the generalizability of the findings to wider populations. Second, hematologic inflammatory parameters were measured only once at the time of emergency department admission, whereas inflammation is a dynamic process; therefore, the lack of serial measurements may have reduced the prognostic value of these biomarkers. Third, despite excluding patients with active infection, chronic inflammatory disease, or recent corticosteroid use, the possibility of subclinical inflammation or other systemic processes could not be entirely ruled out. Finally, details regarding nephroprotective interventions, hydration protocols, contrast agent type, and dose, and other procedural factors were not standardized or fully recorded, and these uncontrolled variables may have acted as potential confounders influencing CIN development. Similarly, the type and volume of contrast media varied across diagnostic and interventional procedures, with the latter typically requiring substantially higher contrast doses. This lack of standardization limited our ability to assess the impact of contrast exposure in detail and may have influenced the incidence of CIN.

Conclusion

In conclusion, hematologic inflammation indices such as NLR, LMR, and PLR may serve as useful adjuncts in predicting CIN in patients with ACS. While age emerged as the strongest independent predictor, these indices may contribute to early risk stratification and help guide the implementation of nephroprotective strategies in clinical practice.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Clinical Research Ethics Committee of Ankara Etlik City Hospital (decision number: AEŞH-BADEK1-2025-174, date: 07.05.2025).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: V.S., Concept: V.S., B.K., Design: V.S., S.A., B.K., Data Collection or Processing: V.S., K.Y., M.B.Ö., M.S.D., Analysis or Interpretation: V.S., S.A., B.K., Literature Search: K.Y., M.B.Ö., M.S.D., Writing: V.S.

Conflict of Interest: No conflict of interest was declared by the authors.

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