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Neutrophil-to-lymphocyte ratio as an independent risk factor for slow flow/no-reflow after percutaneous coronary intervention in patients with acute myocardial infarction: a single-center retrospective cohort study

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Abstract

Background

Slow flow/no-reflow after percutaneous coronary intervention (PCI) is a common complication of acute myocardial infarction (AMI) and is associated with larger infarct size and worse outcomes, yet simple tools for pre-procedural risk estimation remain limited. We investigated whether the neutrophil-to-lymphocyte ratio (NLR) independently predicts slow flow/no-reflow after PCI in patients with AMI, and whether adding high-sensitivity cardiac troponin I (hs-cTnI) and culprit-vessel location improves prediction.

Methods

This single-center retrospective study included consecutive patients with AMI who underwent PCI between January 2023 and December 2025. Slow flow/no-reflow was defined as a post-procedural Thrombolysis in Myocardial Infarction (TIMI) flow grade of 2 or less in the culprit vessel. Multivariable logistic regression identified independent risk factors. Discrimination was assessed with the area under the receiver operating characteristic curve (AUC); the combined model was compared with NLR alone using paired bootstrap testing of the AUC difference and a nested likelihood-ratio test. Internal validation used bootstrap optimism correction and 10-fold cross-validation.

Results

Of 305 patients, 53 (17.4%) developed slow flow/no-reflow. Log-transformed NLR (odds ratio [OR] 1.88, 95% confidence interval [CI] 1.22–2.88, P = 0.004) and log-transformed hs-cTnI (OR 1.24, 95% CI 1.01–1.53, P = 0.043) were independent risk factors, whereas a left anterior descending culprit vessel, including the left main, was inversely associated with slow flow/no-reflow (OR 0.44, 95% CI 0.22–0.86, P = 0.016). The combined model achieved an AUC of 0.735 (95% CI 0.659–0.805) versus 0.674 for NLR alone (ΔAUC 0.061, 95% CI 0.003–0.123, P = 0.039); the optimism-corrected AUC was 0.719 and the 10-fold cross-validated AUC 0.722, and calibration was good (Hosmer–Lemeshow P = 0.101). Adding thrombus burden, left ventricular ejection fraction, AMI type, age, or sex did not improve discrimination (ΔAUC ≤ 0.002).

Conclusions

NLR is an independent risk factor for slow flow/no-reflow after PCI in patients with AMI. Combining NLR with hs-cTnI and culprit-vessel location adds discriminative value that is retained after internal validation. NLR is easy to obtain at admission and may support pre-procedural risk stratification, but external validation is needed before clinical use.

Background

Slow flow/no-reflow is a common and clinically important complication after percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI). Its pathological basis is coronary microvascular obstruction, involving distal microembolization, ischemia–reperfusion injury, microvascular spasm, and inflammatory microthrombosis, and it is independently associated with larger infarct size and worse long-term outcomes [1–3]. The Thrombolysis in Myocardial Infarction (TIMI) flow grade is the standard method for assessing epicardial reperfusion [4]. Although intracoronary imaging and endothelial glycocalyx studies have advanced the understanding of microvascular injury [5,6], a simple, low-cost test that reliably flags pre-procedural risk is still missing from everyday practice [2,5].

Growing evidence links inflammatory markers to slow flow/no-reflow. The neutrophil-to-lymphocyte ratio (NLR) has attracted particular attention because it is cheap, easy to obtain, and available in the emergency setting [7–10]. Other ratios, such as the C-reactive protein to albumin ratio [11] and the stress hyperglycemia ratio [12], have also been associated with no-reflow. Most earlier studies, however, were single-center ST-elevation myocardial infarction (STEMI) cohorts with limited sample sizes, and few combined inflammatory status, myocardial injury, and culprit-vessel anatomy in one model with formal internal validation [7,10].

In this study we asked two questions: whether NLR independently predicts slow flow/no-reflow after PCI in patients with AMI (both STEMI and non-ST-elevation myocardial infarction [NSTEMI]), and whether adding hs-cTnI and culprit-vessel location improves prediction.

Methods

Study design and population

We retrospectively identified consecutive patients with AMI who underwent PCI at Chongqing General Hospital between January 2023 and December 2025 (Fig. 1). Inclusion criteria were: (1) age ≥ 18 years; (2) diagnosis of AMI according to the Fourth Universal Definition of Myocardial Infarction [13]; (3) emergency or elective PCI; and (4) essentially complete clinical, laboratory, and angiographic data. Exclusion criteria were previous coronary artery bypass grafting, active infection, autoimmune or hematologic disease, malignancy, and intraprocedural death precluding flow assessment [confirm final list against hospital records]. After exclusions, 305 patients remained for analysis. The study was approved by the Ethics Committee of Chongqing General Hospital [approval number to be added]; the requirement for informed consent was waived because of the retrospective design.

Figure 1. Patient flow diagram. TIMI, Thrombolysis in Myocardial Infarction. Screening total and exclusion details to be completed from hospital records.

Outcome definition

The outcome was slow flow/no-reflow, defined as a post-PCI TIMI flow grade ≤ 2 in the culprit vessel; TIMI grade 3 was considered normal flow [4]. Angiograms were independently assessed by two interventional

cardiologists who were blinded to laboratory results and not involved in patient management; disagreements were resolved by consensus [inter-rater agreement, kappa, to be added].

Data collection and variable definitions

Demographic data, cardiovascular risk factors, AMI type, culprit vessel, procedural data, laboratory data, and in-hospital outcomes were extracted from the electronic medical records. Inflammatory indices were defined as follows: NLR = neutrophil count / lymphocyte count; derived NLR (dNLR) = neutrophil count / (white blood cell count − neutrophil count); systemic immune-inflammation index (SII) = platelet count × neutrophil count / lymphocyte count. hs-cTnI was the admission value; values above the upper limit of the assay (25,000 pg/mL) were handled as censored at that limit. The culprit vessel was categorized as LAD (including the left main [LM] and LAD-complex lesions such as LAD/diagonal and LM-LAD), right coronary artery (RCA), or LCX/other.

Statistical analysis

Continuous variables were tested for normality using the Shapiro–Wilk test: normally distributed variables are presented as mean ± SD and compared using the independent-samples t test; non-normally distributed variables are presented as median (interquartile range, IQR) and compared using the Mann–Whitney U test. Categorical variables are presented as n (%) and compared using the chi-square test (with Yates' continuity correction) or Fisher's exact test when expected frequencies were < 5. Variables with $P < 0.10$ in univariate analysis or with established clinical relevance were entered into multivariable logistic regression (forward stepwise) to identify independent risk factors; odds ratios (OR) with 95% confidence intervals (CI) were calculated. Discrimination was assessed using the AUC with bootstrap 95% CIs (2,000 resamples). The ΔAUC between the combined model and NLR alone was tested with paired bootstrap (3,000 resamples) and a nested likelihood-ratio test. Internal validation used bootstrap optimism correction (2,000 resamples) and 10-fold cross-validation. Calibration was assessed with the Hosmer–Lemeshow test and clinical utility with decision curve analysis (DCA). All analyses were carried out in R 4.3 (Python statsmodels/scipy) and SPSS 26.0, and the two implementations were used to cross-check each other. A two-sided $P < 0.05$ was considered statistically significant.

Results

Baseline characteristics and univariate analysis

Of the 305 patients, 53 (17.4%) had slow flow/no-reflow and 252 (82.6%) had normal flow. Age, sex, BMI, cardiovascular risk factors, lipid profile, renal function, and LVEF were similar between the two groups (Table 1). Patients with slow flow/no-reflow had higher neutrophil counts ($P = 0.005$), NLR ($P = 0.001$), hs-cTnI ($P = 0.002$), CK-MB ($P = 0.009$), a higher rate of reperfusion arrhythmia ($P = 0.002$), and longer hospital stay ($P = 0.043$); white blood cell count was borderline ($P = 0.050$). LAD culprit vessels (including LM) were less common in the slow flow/no-reflow group than in the normal-flow group (32.1% vs 48.8%, $P = 0.038$).

Table 1
Baseline characteristics and univariate comparison of patients with and without slow flow/no-reflow.

Variable	Slow flow/no-reflow (n = 53)	Normal flow (n = 252)	Test	P
Age, years	62.00 (52.00–76.00)	61.50 (53.75–73.00)	Mann–Whitney U	0.743
Male	41/53 (77.4%)	199/252 (79.0%)	Chi-square	0.940
Smoking	32/51 (62.7%)	179/252 (71.0%)	Chi-square	0.314
Hypertension	30/53 (56.6%)	150/252 (59.5%)	Chi-square	0.811
Diabetes mellitus	21/53 (39.6%)	88/252 (34.9%)	Chi-square	0.623
Stroke/TIA history	9/53 (17.0%)	45/252 (17.9%)	Chi-square	1.000
STEMI	35/53 (66.0%)	131/252 (52.0%)	Chi-square	0.086
Culprit vessel LAD (incl. LM)	17/53 (32.1%)	123/252 (48.8%)	Chi-square	0.038
WBC, ×10⁹/L	10.21 (8.02–13.11) (n = 52)	9.20 (7.11–11.69) (n = 250)	Mann–Whitney U	0.050
Neutrophils, ×10⁹/L	8.54 (5.94–11.23) (n = 52)	6.56 (4.91–8.95) (n = 250)	Mann–Whitney U	0.005
Lymphocytes, ×10⁹/L	1.31 (0.87–2.02) (n = 52)	1.54 (1.08–2.12) (n = 250)	Mann–Whitney U	0.096
NLR	6.57 (4.13–10.78) (n = 52)	4.23 (2.51–6.78) (n = 250)	Mann–Whitney U	0.001
CRP, mg/L	6.25 (1.88–16.00) (n = 44)	3.98 (1.52–10.94) (n = 216)	Mann–Whitney U	0.320
Albumin, g/L	40.60 (36.75–44.30) (n = 51)	41.10 (38.52–44.18) (n = 242)	Mann–Whitney U	0.625
Glucose, mmol/L	7.24 (5.91–12.05) (n = 40)	7.14 (5.93–9.39) (n = 171)	Mann–Whitney U	0.563
HbA1c, %	6.10 (5.70–7.40) (n = 41)	6.10 (5.60–7.80) (n = 161)	Mann–Whitney U	0.813
Total cholesterol, mmol/L	4.52 (4.02–5.50) (n = 50)	4.56 (3.74–5.22) (n = 239)	Mann–Whitney U	0.483
Triglycerides, mmol/L	1.33 (0.83–1.98) (n = 50)	1.52 (1.02–2.32) (n = 239)	Mann–Whitney U	0.121
HDL-C, mmol/L	1.06 (0.93–1.28) (n = 50)	1.04 (0.89–1.27) (n = 239)	Mann–Whitney U	0.447
LDL-C, mmol/L	2.74 ± 0.86 (n = 50)	2.66 ± 0.95 (n = 239)	t test	0.551

Variable	Slow flow/no-reflow (n = 53)	Normal flow (n = 252)	Test	P
Creatinine, μmol/L	78.05 (64.30–90.20) (n = 52)	82.60 (68.10–93.90) (n = 249)	Mann–Whitney U	0.239
eGFR, mL/min/1.73 m²	90.58 (72.62–99.96) (n = 52)	85.75 (68.57–99.75) (n = 244)	Mann–Whitney U	0.297
hs-cTnI, pg/mL †	17282.30 (9318.80–25000.00) (n = 51)	8658.00 (1414.35–25000.00) (n = 246)	Mann–Whitney U	0.002
CK-MB, ng/mL †	50.98 (9.22–141.94) (n = 51)	19.21 (2.60–94.75) (n = 246)	Mann–Whitney U	0.009
NT-proBNP, pg/mL	632.27 (151.00–2031.00) (n = 53)	702.93 (192.00–2070.73) (n = 227)	Mann–Whitney U	0.747
LVEF, %	52.00 (43.00–58.00) (n = 53)	55.00 (45.00–60.00) (n = 251)	Mann–Whitney U	0.199
Hospital stay, days	7.00 (6.00–10.00) (n = 53)	6.00 (5.00–9.00) (n = 252)	Mann–Whitney U	0.043
Reperfusion arrhythmia	11/53 (20.8%)	16/252 (6.3%)	Fisher's exact	0.002

Data are median (IQR) or n (%). † hs-cTnI and CK-MB values above the assay upper limit were censored (hs-cTnI upper limit 25,000 pg/mL). BMI, body mass index; LVEF, left ventricular ejection fraction; WBC, white blood cell; STEMI, ST-elevation myocardial infarction; LAD, left anterior descending; LM, left main; NLR, neutrophil-to-lymphocyte ratio; CRP, C-reactive protein; HbA1c, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate; hs-cTnI, high-sensitivity cardiac troponin I; CK-MB, creatine kinase-MB; NT-proBNP, N-terminal pro-B-type natriuretic peptide; TIA, transient ischemic attack.

Multivariable logistic regression

Variables with P < 0.10 in univariate analysis (neutrophil count, NLR, white blood cell count, hs-cTnI, CK-MB, reperfusion arrhythmia, LAD culprit vessel, STEMI type) and clinically relevant variables (age, sex) were entered into the multivariable model. The final model retained three variables (Table 2): log-NLR (OR 1.88, 95% CI 1.22–2.88, P = 0.004) and log hs-cTnI (OR 1.24, 95% CI 1.01–1.53, P = 0.043) were independent risk factors, whereas LAD culprit vessel (including LM) was inversely associated with slow flow/no-reflow (OR 0.44, 95% CI 0.22–0.86, P = 0.016). Forest plots of the multivariable odds ratios of the final model are provided in Additional file 1, and a sensitivity analysis using the original LAD coding (LM excluded from LAD) is provided in Additional file 2.

Table 2
Multivariable logistic regression for slow flow/no-reflow (final three-variable model).

Variable	β	SE	OR	95% CI	P
NLR (log)	0.629	0.218	1.88	1.22–2.88	0.004
hs-cTnI (log)	0.216	0.107	1.24	1.01–1.53	0.043
Culprit vessel LAD (incl. LM)	−0.830	0.344	0.44	0.22–0.86	0.016
Intercept	−4.236	1.014	0.014	—	< 0.001

Final model: log-NLR + log(hs-cTnI) + LAD culprit vessel (including LM); n = 294, events = 50, AUC = 0.735; Hosmer–Lemeshow test P = 0.101. ORs are per 1 natural-log unit increase (continuous) or for the presence vs absence of the category (LAD). LAD coding: culprit vessel containing LAD or LM (LM/LM-LAD assigned to the LAD group). AUC, area under the receiver operating characteristic curve.

Under the original coding, in which LM/LM-LAD lesions (8 cases) were classified as non-LAD, the sensitivity model (n = 294, events = 50) had an AUC of 0.725 (95% CI 0.647–0.797), and the direction and significance of all associations were unchanged, excluding a coding artifact (Additional file 2).

Predictive performance of NLR and the combined model

Using the same cohort (n = 294, events = 50), NLR alone had an AUC of 0.674 (95% CI 0.590–0.753; Fig. 2). The combined model (NLR + hs-cTnI + LAD) had an AUC of 0.735 (95% CI 0.659–0.805; Fig. 3), an improvement of 0.061 over NLR alone (95% CI 0.003–0.123; paired bootstrap P = 0.039), and the nested likelihood-ratio test for the two added variables was significant (P = 0.006; Table 3). In the full cohort (n = 302), the AUCs of NLR, dNLR, and SII were 0.649, 0.659, and 0.636, respectively (Table 3).

Figure 2. Receiver operating characteristic (ROC) curve of NLR for predicting slow flow/no-reflow in the analysis set (n = 294). AUC, area under the curve; NLR, neutrophil-to-lymphocyte ratio.

Figure 3. ROC curves of the combined model (NLR + hs-cTnI + LAD) and NLR alone in the same analysis set (n = 294). AUC, area under the curve; hs-cTnI, high-sensitivity cardiac troponin I; LAD, left anterior descending; NLR, neutrophil-to-lymphocyte ratio.

Table 3
Model development and ROC performance.

Model	n	Events	AUC	95% CI	ΔAUC vs NLR	P
NLR (alone)	294	50	0.674	0.590–0.753	—	—
NLR + hs-cTnI	294	50	0.702	0.618–0.778	+ 0.028	—
NLR + hs-cTnI + LAD (final model)	294	50	0.735	0.659–0.805	+ 0.061	—
NLR + hs-cTnI + LAD + STEMI	294	50	0.733	0.656–0.802	+ 0.059	—
Full model (+ age, sex)	294	50	0.732	0.656–0.801	+ 0.058	—
Final model vs NLR alone (paired bootstrap ΔAUC)	294	50	0.735	+ 0.003 to + 0.123	+ 0.061	0.039
Nested LRT (log-hs-cTnI + LAD added)	294	50	—	—	$\Delta\chi^2$=10.29	0.006
Final model, optimism-corrected AUC	294	50	0.719	—	—	—
Final model, 10-fold cross-validated AUC	294	50	0.722	—	—	—
Single indices (full cohort)						
NLR	302	52	0.649	—	cutoff 5.23; 65.4%/62.0%	
dNLR	302	52	0.659	—	cutoff 4.28; 55.8%/75.6%	
SII	302	52	0.636	—	cutoff 1423.33; 55.8%/74.4%	

Model comparisons were performed within the same analysis set (n = 294, events = 50). ΔAUC was estimated by paired bootstrap (3,000 resamples); final model vs NLR alone: ΔAUC 0.061 (95% CI 0.003–0.123), two-sided P = 0.039; nested likelihood-ratio test for the combined addition of log-hs-cTnI and LAD: P = 0.006. Optimism-corrected AUC 0.719; 10-fold cross-validated AUC 0.722. Single-index ROC analyses were performed in the full cohort (n = 302); cutoff values maximize the Youden index (sensitivity/specificity). LRT, likelihood-ratio test; dNLR, derived NLR; SII, systemic immune-inflammation index.

Model validation and extension

After bootstrap optimism correction, the AUC of the combined model was 0.719; the 10-fold cross-validated AUC was 0.722, indicating moderate discrimination that was retained after internal validation. The

Hosmer–Lemeshow test showed good calibration (P = 0.101; Additional file 3), and decision curve analysis (Additional file 4) supported clinical utility across a range of threshold probabilities. Sequentially adding thrombus burden, LVEF, STEMI type, age, and sex to the final model did not materially improve discrimination (ΔAUC ≤ 0.002; Table 4), suggesting that the three-variable model adequately integrates inflammatory, myocardial injury, and anatomical information. Coefficients of the extended eight-variable model are provided in Additional file 5.

Table 4
Performance of the final model after adding candidate variables (sensitivity analysis).

Model	n	Events	AUC	95% CI	ΔAUC vs final model
NLR + hs-cTnI + LAD (final model)	294	50	0.735	0.659–0.805	—
+ Heavy thrombus burden	294	50	0.734	0.656–0.805	−0.000
+ LVEF	293	50	0.735	0.658–0.807	+ 0.000
+ Heavy thrombus burden + LVEF	293	50	0.735	0.660–0.806	+ 0.000
+ Heavy thrombus burden + LVEF + AMI type + age + sex	293	50	0.733	0.658–0.802	−0.002

ΔAUC values are differences from the final model (n = 294) evaluated on the same subset. Adding candidate variables did not materially improve discrimination (ΔAUC ≤ 0.002); the parsimonious three-variable model was retained. LVEF, left ventricular ejection fraction.

Subgroup analysis

Stratified by AMI type, the AUC of dNLR was 0.610 (95% CI 0.494–0.715) in the STEMI subgroup (n = 165, slow flow in 34 [20.6%]) and 0.729 (95% CI 0.589–0.850) in the NSTEMI subgroup (n = 137, slow flow in 18 [13.1%]); the difference was not statistically significant (P = 0.166), supporting the combined analysis of STEMI and NSTEMI (Table 5; Additional file 6).

Table 5
Subgroup analysis of dNLR by AMI type (sensitivity analysis).

Subgroup	n	Slow flow, n (%)	dNLR, slow flow	dNLR, normal flow	MW P	AUC	95% CI	Cutoff (sensitivity/specificity)
STEMI	165	34 (20.6%)	4.40 (2.89–6.77)	3.27 (2.05–5.01)	0.048	0.610	0.494–0.715	2.58 (82%/41%)
NSTEMI	137	18 (13.1%)	4.82 (2.74–6.83)	2.67 (1.84–3.87)	0.002	0.729	0.589–0.850	4.13 (67%/79%)

Full-cohort dNLR AUC 0.659. The difference between STEMI and NSTEMI subgroup AUCs was not statistically significant (bootstrap two-sided P = 0.166), supporting combined analysis. MW, Mann–Whitney; NSTEMI, non-ST-elevation myocardial infarction.

Discussion

NLR as an independent risk factor for slow flow/no-reflow

In this mixed STEMI/NSTEMI cohort, NLR was an independent risk factor for slow flow/no-reflow after PCI (OR 1.88 per log unit, P = 0.004), consistent with most previous studies [7–10]. Slow flow/no-reflow results from microvascular obstruction driven by distal microembolization, ischemia–reperfusion injury, microvascular spasm, and inflammatory microthrombosis involving neutrophil extracellular traps (NETs). NLR integrates the relative states of neutrophils (pro-inflammatory, pro-thrombotic) and lymphocytes (immunomodulatory, anti-inflammatory); a high NLR reflects an elevated systemic inflammatory burden and may promote microcirculatory no-reflow through neutrophil activation, NET release, endothelial injury, and capillary obstruction. The independent association we observed after multivariable adjustment supports the use of NLR as a marker obtainable at the emergency department for pre-procedural risk stratification.

Incremental value of the combined model

NLR alone discriminated only modestly (AUC ≈ 0.67). Adding hs-cTnI and culprit-vessel anatomy raised the AUC to 0.735 (ΔAUC 0.061, P = 0.039), and the improvement survived optimism correction (0.719) and 10-fold cross-validation (0.722). The three variables appear to carry complementary information about inflammation, myocardial injury, and anatomy. Thrombus burden, LVEF, age, and sex added nothing further (ΔAUC ≤ 0.002), so we kept the simpler model. Even so, the gain over NLR alone was small, with the lower 95% CI bound close to zero, and the clinical value of the added variables should be judged after external validation.

The inverse association between LAD culprit vessel and slow flow/no-reflow

Unlike most earlier STEMI-based studies, in which an LAD culprit vessel usually increases no-reflow risk, LAD culprit vessel (including LM) was inversely associated with slow flow/no-reflow in our cohort (OR 0.44). The slow flow/no-reflow group had a predominance of RCA culprit vessels (43.4%), and the RCA group showed higher rates of slow flow (23.0%), thrombus aspiration (17.3%), and reperfusion arrhythmia (13.0%) than the LAD group (12.1%, 11.7%, and 5.7%, respectively). The inverse association persisted after adjustment for STEMI type (OR 0.45, P = 0.014), which argues against confounding by AMI type. We read this result as a reflection of our case mix (mixed STEMI/NSTEMI, many RCA culprit vessels) and the higher thrombus burden and distal-embolization tendency of RCA lesions, not as evidence that the LAD is protective. The difference from earlier reports suggests the effect of culprit-vessel location on slow flow/no-reflow may depend on the population studied. We therefore treat this finding as exploratory and center-specific and would not give it a causal interpretation before it is replicated elsewhere.

Clinical implications

Both NLR and hs-cTnI are measured routinely at admission, and the culprit vessel is known as soon as angiography is done. The nomogram built from the three-variable model (Additional file 7) gives a quick way to identify patients at high risk of slow flow/no-reflow before emergency PCI, which could help operators prepare thrombus aspiration, step up antithrombotic therapy, or defer stenting. The model's discrimination is only moderate, however, and it has not been externally validated; it is not ready for direct use in clinical decisions.

Limitations

This study has several limitations. First, it is a single-center retrospective study with unmeasured confounding and limited capacity for causal inference. Second, the number of events was relatively small (50 events), giving an events-per-variable ratio of approximately 17:1 in the three-variable model, which is acceptable but precludes adding further predictors. Third, hs-cTnI values above the assay upper limit (25,000 pg/mL) were censored, which may have attenuated the estimated association. Fourth, CRP and other inflammatory markers had high rates of missing data and were not adjusted for, so residual confounding cannot be excluded. Fifth, the inverse LAD association is a center-specific finding of unknown external validity. Sixth, long-term follow-up outcomes were not collected, and the impact of slow flow/no-reflow on long-term prognosis was beyond the scope of this study.

Conclusions

NLR is an independent risk factor for slow flow/no-reflow after PCI in patients with AMI, in line with the evidence linking inflammation to microvascular injury. Combining NLR with hs-cTnI and culprit-vessel location adds discriminative value, and the model stayed stable under internal validation. NLR is easy to obtain at admission and may be useful for pre-procedural risk stratification; multicenter prospective cohorts are needed to confirm its external validity.

Abbreviations

AMI
acute myocardial infarction
AUC
area under the receiver operating characteristic curve
BMI
body mass index
CI
confidence interval
CK-MB
creatine kinase-MB
CRP
C-reactive protein
DCA
decision curve analysis

dNLR
derived neutrophil-to-lymphocyte ratio
eGFR
estimated glomerular filtration rate
HbA1c
glycated hemoglobin
HDL-C
high-density lipoprotein cholesterol
hs-cTnI
high-sensitivity cardiac troponin I
IQR
interquartile range
LAD
left anterior descending
LCX
left circumflex
LDL-C
low-density lipoprotein cholesterol
LM
left main
LVEF
left ventricular ejection fraction
NLR
neutrophil-to-lymphocyte ratio
NSTEMI
non-ST-elevation myocardial infarction
NT-proBNP
N-terminal pro-B-type natriuretic peptide
OR
odds ratio
PCI
percutaneous coronary intervention
RCA
right coronary artery
ROC
receiver operating characteristic
SII
systemic immune-inflammation index
STEMI
ST-elevation myocardial infarction
TIMI

Thrombolysis in Myocardial Infarction
WBC
white blood cell

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki. The protocol was approved by the Ethics Committee of Chongqing General Hospital [approval number to be added]. Because of the retrospective design, the requirement for written informed consent was waived by the Ethics Committee of Chongqing General Hospital.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analysed during the current study are not publicly available because of hospital confidentiality policies, but are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Hua-yin Li, Ming-wei Yang and Bo-li Ran conceived and designed the study. Hua-yin Li, Fang-chao Wang and Chao-yi Chen collected the clinical and angiographic data. Hua-yin Li and Ming-wei Yang performed the statistical analyses and interpreted the data. Hua-yin Li, Ming-wei Yang and Bo-li Ran drafted the manuscript; Fang-chao Wang, Chao-yi Chen and Bo-li Ran revised it critically for important intellectual content. All authors read and approved the final manuscript.

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Figures

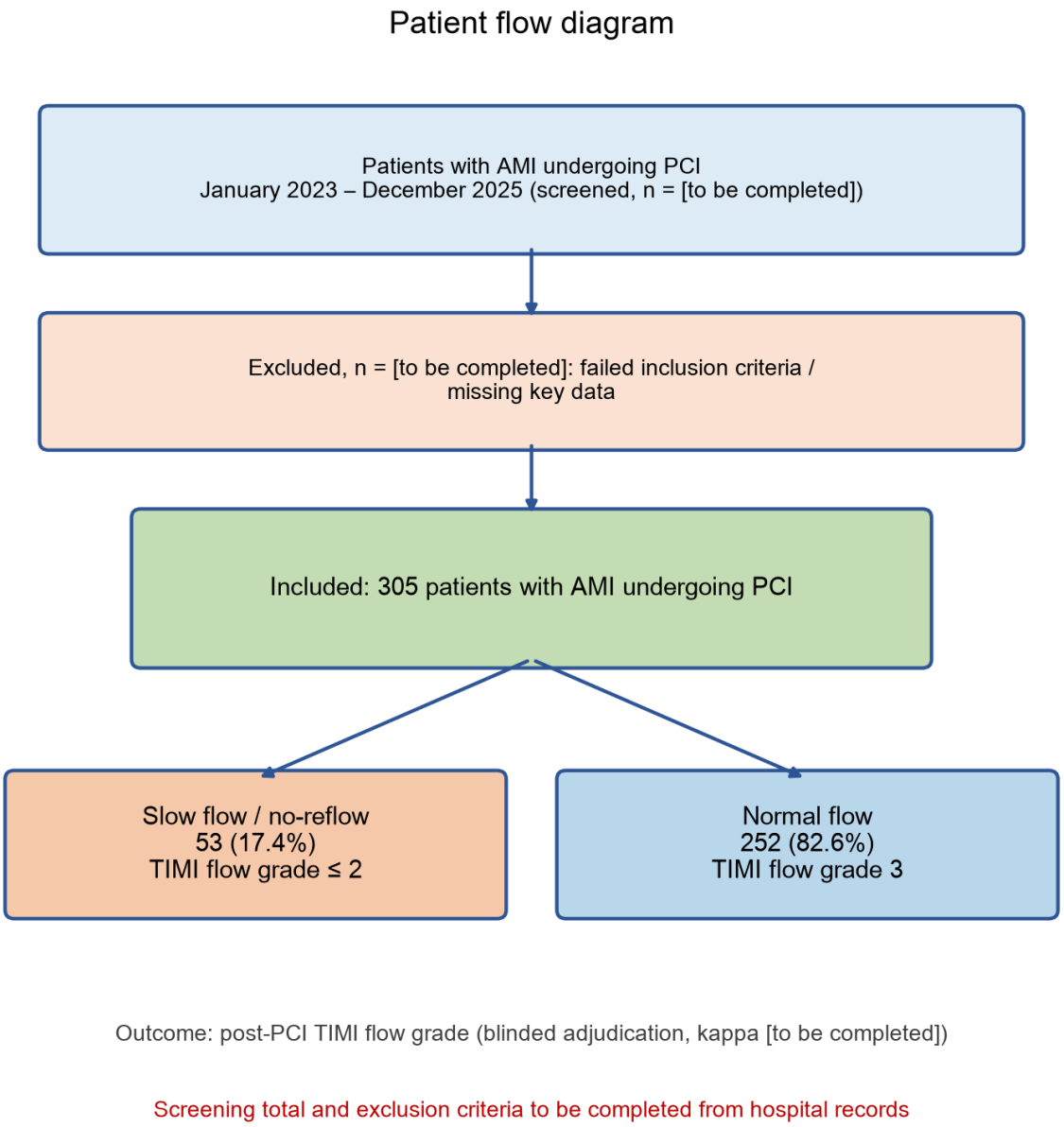


Figure 1

Patient flow diagram. TIMI, Thrombolysis in Myocardial Infarction. Screening total and exclusion details to be completed from hospital records.

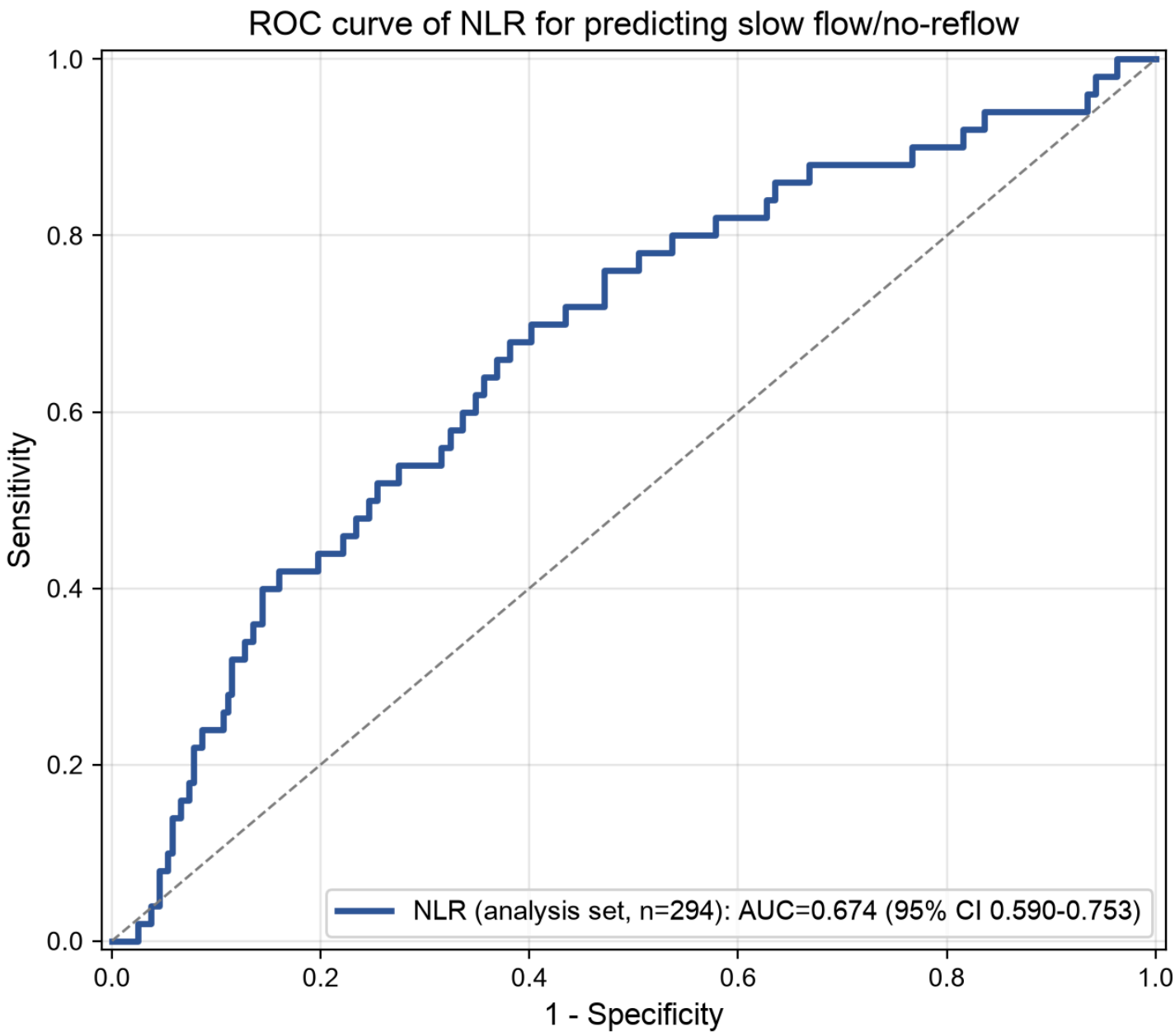


Figure 2

Receiver operating characteristic (ROC) curve of NLR for predicting slow flow/no-reflow in the analysis set (n=294). AUC, area under the curve; NLR, neutrophil-to-lymphocyte ratio.

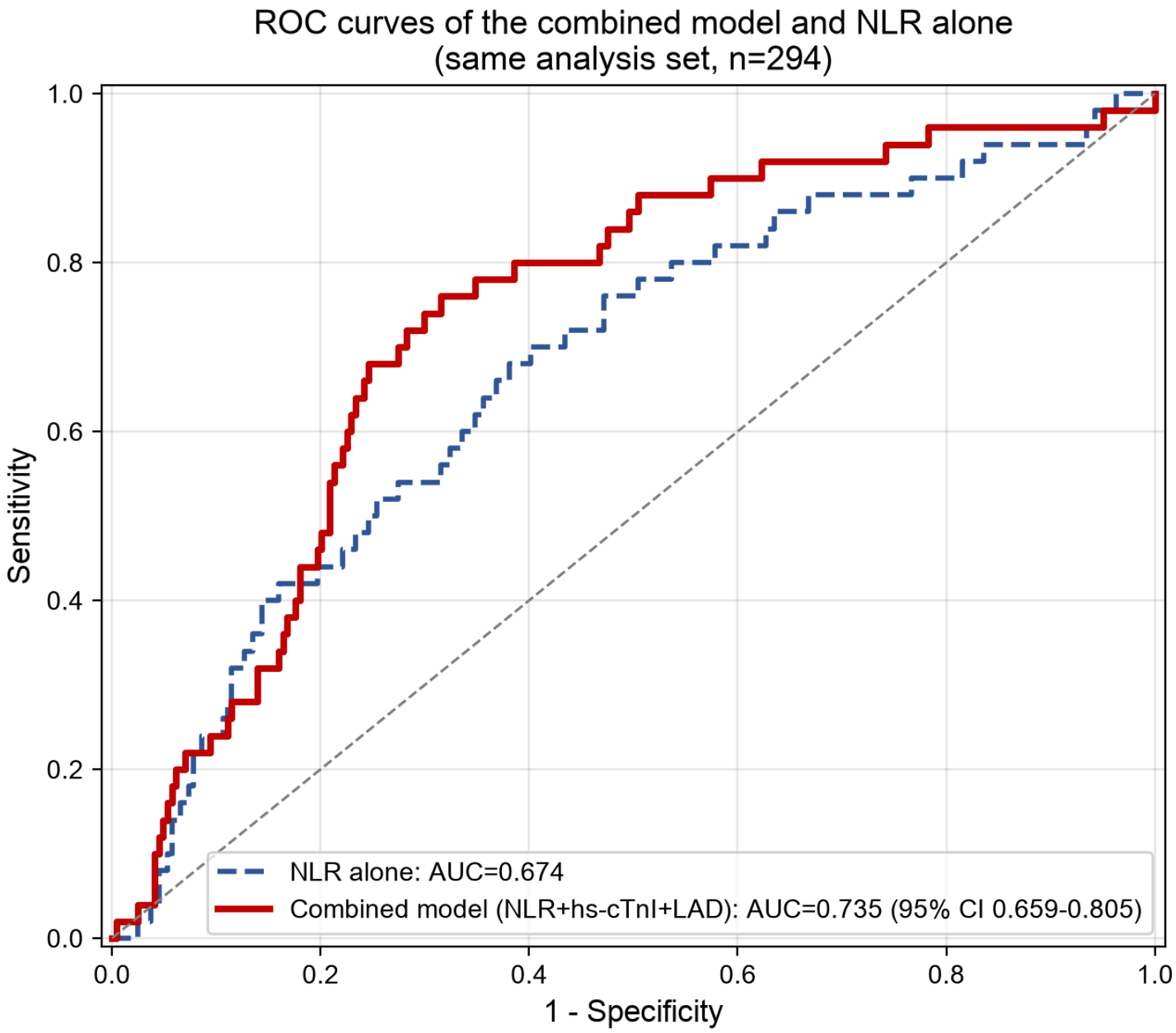


Figure 3

ROC curves of the combined model (NLR + hs-cTnI + LAD) and NLR alone in the same analysis set (n=294). AUC, area under the curve; hs-cTnI, high-sensitivity cardiac troponin I; LAD, left anterior descending; NLR, neutrophil-to-lymphocyte ratio.

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