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RESEARCH

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# Microcirculatory resistance based on a single angiographic view in ST-segment elevation myocardial infarction patients

Zhe Zhang[1†], Qing Dai[2†], Xinlin Zhang[2†], Shiyang Qiao[2], Xue Bao[2], Kun Wang[2], Peng Xue[2], Yuan Gao[2], Xuemei Guo[2], Yanan Xue[2], Zhonghai Wei[1,2*], Biao Xu[1,2*] and Lina Kang[1,2*]

## Abstract

**Background** Angio-based microvascular resistance (AMR) was proposed as a tool to quantitatively assess coronary microvascular based on single angiographic projection. The aims of this study are to assess the diagnostic accuracy and prognostic significance of AMR in ST-segment elevation myocardial infarction (STEMI) patients.

**Methods** AMR was measured (Of these, 22 patients measured index of microvascular resistance (IMR)) in 70 STEMI patients after primary percutaneous coronary intervention (pPCI). ST-segment resolution (STR) was assessed 2 h after pPCI simultaneously. Transthoracic echocardiography was performed within 1 day and approximately 1 year after pPCI. STEMI patients underwent pPCI were followed up for 7.3 years and the primary endpoint was the major adverse cardiac and cerebral events (MACCEs).

**Results** AMR showed significant correlations with IMR ($R=0.334$, $P=0.005$). AMR has good predictive power for STR after pPCI (area under the curve: 0.889, sensitivity: 94.59%, specificity: 75.76%) in receiver operating characteristic (ROC) curve. Low-AMR patients showed markedly improved left ventricular ejection fraction (LVEF) 1 year after pPCI (42(40–49) vs. 41(39–44), $P=0.041$). High-AMR patients showed higher risk for MACCEs than those with Low-AMR (HR = 3.90, $P=0.02$). In multivariate cox regression analysis, AMR was considered an independent predictor of MACCEs (HR: 1.153, $P=0.020$).

**Conclusions** AMR is a reliable tool for the estimation of microvascular resistance and prognosis in the absence of intracoronary pressure-temperature sensor wire and adenosine based on single angiographic projection.

**Keywords** ST-segment elevation myocardial infarction, Myocardial microcirculation, Coronary microvascular dysfunction, Angio-based microvascular resistance, Major adverse cardiac and cerebral events

†Zhe Zhang, Qing Dai and Xinlin Zhang contributed equally to this work.

*Correspondence:
Zhonghai Wei
weizhonghai@nju.edu.cn
Biao Xu
xubiao62@nju.edu.cn
Lina Kang
kanglina@njglyy.com
[1]Department of Cardiology, Nanjing Drum Tower Hospital, Nanjing Drum Tower Hospital Clinical College of Nanjing Medical University, Nanjing 210008, China
[2]Department of Cardiology, Nanjing Drum Tower Hospital, The Affiliated Hospital of Nanjing University Medical School, Nanjing 210008, Jiangsu, China

## Introduction

In patients with ST-segment elevation myocardial infarction (STEMI), prompt coronary revascularization via primary percutaneous coronary intervention (pPCI) is widely recognized as the standard of care [1]. However, despite effective interventional therapy, some patients experience unsatisfactory outcomes due to coronary microvascular dysfunction (CMD) [2]. Previous researches have consistently shown that CMD significantly increases the risk of heart failure (HF), major adverse cardiovascular events (MACE), and all-cause mortality [3, 4]. The index of microvascular resistance (IMR) has emerged as a reliable diagnostic tool for assessing CMD in STEMI patients, with its calculation based on distal coronary pressure and mean transit time at maximum hyperemia, as measured using a pressure-temperature sensor wire. Nevertheless, its clinical application remains limited due to the potential side effects of adenosine, the risk of wire injury, and the associated increased costs [5–7].

The quantitative flow ratio (QFR) has been introduced as a novel angiography-based index metric, demonstrating a strong correlation with fractional flow reserve (FFR). Subsequent studies have shown that QFR offers excellent diagnostic accuracy, and the lesion selection guided by QFR can improve clinical outcomes in patients 1 year after PCI [8–12]. De Maria et al. and Mejia-Renteria et al. separately reported on angiography-derived IMR and Angio-IMR, both of which eliminated the need for a pressure-temperature sensor wire and adenosine, while demonstrating excellent diagnostic performance for CMD [13, 14]. However, the current angiography-derived IMR requires at least two angiographic projections, and its diagnostic capacity and prognostic significance in patients with STEMI remain unproven.

Angio-based microvascular resistance (AMR) is an adenosine-free, wire-free, angiography-derived IMR based on a single angiographic projection. The objectives of this study were to evaluate the diagnostic accuracy of AMR and assess the long-term clinical outcomes of STEMI patients stratified according to AMR-based management.

## Method

### Study population

This study consecutively included 122 patients with STEMI who underwent pPCI in Nanjing Drum Tower Hospital from May 2014 to December 2015 and agreed to be retrospectively analyze the AMR of the culprit vessel. According to the 2017 ESC guidelines, patients with chest discomfort or other symptoms indicating ischemia, high cardiac troponin, and at least two consecutive leads with ST-segment elevation are diagnosed as STEMI [15]. The following patients were excluded: (1) Age > 85 years ($n = 4$); (2) Lack of echocardiographic data ($n = 9$); (3) Patients with incomplete files or records ($n = 3$); (4) Insufficient angiographic view for CTFC ($n = 3$); (5) AMR could not be analyzed due to incorrect vessel identification or poor angiographic quality ($n = 12$); (6) Cardiac shock ($n = 2$); (7) Patients who have been lost or refused follow-up ($n = 19$). Seventy STEMI patients were included in the final analysis. All procedures were performed in accordance with the Declaration of Helsinki. Nanjing Drum Tower Hospital approved the protocol of this study (No.2022-060-02).

### Angio-based microvascular resistance

The coronary angiography video of the successful recanalization of the culprit vessel was uploaded to the Pulse Medical software platform, where the images were analyzed by the researchers blinded to the patient's history and clinical outcome. According to a single angiographic image, the QFR of the target vessel can be calculated by the software (Angio Plus Core, version V3, Shanghai Pulse Medical Technology Inc., Shanghai, China) (Fig. 1). Then the blood flow velocity at maximum hyperemia ($V_{(Flow)}$) was calculated through software simulation and set the average aortic pressure (Pa) at this time was 86 mmHg, as previously reported [8]. Finally, the formula was used to compute AMR, in mmHg*s/cm.

$$AMR = Pa \times QFR/V_{(Flow)}$$

### Index of coronary microvascular resistance

Ohm's law simplifies the detection approach, which is described by multiplying the distal intravascular pressure (Pd) of the culprit vessel's stenosis by the transit time, in mmHg * S or U. The guide wire was inserted into the lesion's distal vessel to measure the aortic pressure (Pa) and Pd. Subsequently, to achieve maximum hyperemia, adenosine triphosphate (ATP) (140 ug/kg/min) was drip-fed into the peripheral vein and then 3 ml of room temperature normal saline was quickly injected into the coronary artery with a guiding catheter to obtain thermodilution curve. Obtain mean transit time at maximum hyperemia ($Tmn_{hyp}$) after repeated three injections. Pd, Pa, and FFR can be obtained from modified Pressure Wire-4 (Radi Medical Systems, Sweden). IMR is calculated according to the formula, i.e.

$$IMR = Pd \times Tmn_{hyp}$$

### ST segment resolution and corrected TIMI frame count

At the time of admission and 120 min after the culprit vessel had been restored, a 12-lead ECG was taken, both times at a speed of 25 mm/s and a calibration of

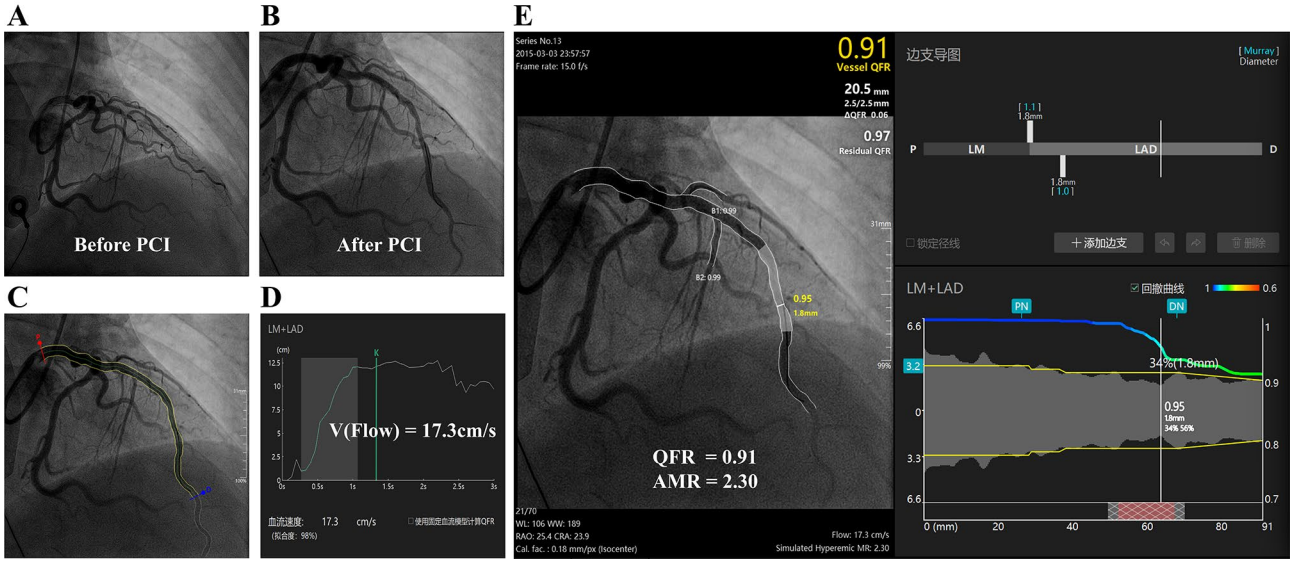


**Fig. 1** Analysis Process of AMR. The figure demonstrates the AMR analysis process of a STEMI patient. Figures (**A**) and (**B**) are the coronary angiography images of the patient before and after PCI. Selection of target vessels on coronary angiography images after PCI (**C**) and automatic analysis of coronary blood flow velocity (**D**). Finally, the computer software calculates the values of QFR and AMR according to the formula (**E**)

1.0 mV/10 mm. ST-segment resolution (STR) was determined by subtracting mean post-PCI ST-segment elevation from mean pre-PCI ST-segment elevation divided by mean pre-PCI ST-segment elevation. STR is represented as a percentage, i.e.

$$STR = ((ST_{pre-PCI} - -ST_{post-PCI}) / ST_{pre-PCI}) \times 100\%$$

STR < 70% is defined as failure to achieve complete STR [16].

The cine angiographic examination was recorded at a frame rate of 30 per second. As reported by Gibson et al. TIMI frame counts (TFC) have been reported to start from the first frame when the contrast media in the infarct-related arteries is fully incorporated and continue until the frame where the contrast media reaches normalized distal coronary landmarks [17]. Three projections where the culprit vessel was best observed were used to calculate the TFC, and the results were averaged. The TFC of the left anterior descending coronary (LAD) was divided by 1.7 to get the corrected TIMI frame count (CTFC) of LAD.

### Echocardiographic measurement

For the 70 patients, transthoracic echocardiography within 1 day and approximately 1 year after pPCI were performed and analyzed respectively by two experienced cardiologists blinded to the coronary physiology or clinical information of the patients. Left ventricular end-diastolic diameter (LVEDD) was measured at end-diastole on parasternal views. From the parasternal long-axis view, left ventricular end-systolic diameter (LVESD) was measured at end-systole. According to the modified Simpson's rule, left ventricular ejection fraction (LVEF) could be measured from the two-chamber and four-chamber areas in the parasternal long-axis view.

### Clinical outcomes

Clinical outcomes were retrospectively collected during follow-up. The primary endpoint was MACCEs, defined as a composite of all-cause death, worsening heart failure, non-fatal acute myocardial infarction, or non-fatal ischemic stroke. STEMI patients were followed up by outpatient and/or telephone every month in the first year and every three months after the first year.

### Statistical analysis

Perform normality analysis on continuous variables using Kolmogorov Smirnov test. Normally distributed data were expressed as mean ± SD, and non-normally distributed data were presented as median (interquartile ranges). The frequency and percentage of categorical variables are used in their expression. And Categorical variables between two groups were tested by chi-square test or Fisher's exact test. Paired t-test and Wilcoxon signed-rank were used to test paired comparisons. Pearson or Spearman was used to determine the correlation between two continuous variables using Pearson or Spearman. In the receiver-operating characteristic (ROC) curve, the best cutoff value, area under the curve (AUC), 95% confidence intervals (95%CI), sensitivity, and specificity for AMR prediction of STR < 70% or IMR > 40U were calculated [16, 23]. The cumulative event-free survival rate was calculated using log-rank test and

visualized by Kaplan-Meier plot. The collected baseline data were incorporated respectively into the Cox univariate analysis, and the value with $P<0.1$ was considered statistically significant. The baseline characteristics with $P<0.1$ in the univariate analysis were subjected to a multivariate Cox proportional hazards model analysis. The association between AMR and the incidence of MACCEs was determined by multivariate Cox proportional hazards model. Before these analyses, Schoenfeld residuals were interpreted intuitively to test the assumption of proportional hazards. A two-sided $P<0.05$ was considered significant. All statistical analysis was carried out using Windows version of R version 4.2.2, GraphPad Prism version 9.4.1, and SPSS version 26, all statistical analysis was carried out.

## Result

### Baseline characteristics

This study enrolled a total of 70 STEMI patients underwent pPCI. Male accounted for 87.14%, and the average age was 60.07 ± 11.44 years. The mean AMR was 2.40 ± 0.49. The patients were divided into two groups based on an AMR prediction cut-off of STR < 70%. Forty-three patients (61.43%) with AMR > 2.21 were divided into the High-AMR group, and the rest (38.57%) were divided into the Low-AMR group. Baseline characteristics for patients grouped according to AMR cutoff values are listed in Table 1. There was no significant difference between the two groups in baseline clinical characteristics, laboratory examination, admission situation, or angiographic characteristics (Table 1).

### Diagnostic accuracy of AMR

In this study, 70 patients were assessed for AMR, STR, and CTFC. Amongst, 22 patients were applied pressure-temperature sensor wire to assess FFR and IMR. There was not a systemic bias between FFR and QFR as indicated by the Bland-Altman diagram (Fig. 2C). But a significant linear correlation between them ($R=0.6515$; $P<0.001$) (Fig. 2A). AMR showed significant correlations ($R=0.3344$; $P=0.0048$) with IMR and had a cut-off value of 2.25 to predict IMR > 40 U in ROC curve (AUC, 0.821; 95% CI: 0.601–0.950; sensitivity, 100%; specificity, 71.43%) (Fig. 2B and E). In all patients, AMR had a cut-off value of 2.21 to predict STR < 70% and showed an AUC of 0.889 in ROC curve (sensitivity, 94.59%; specificity, 75.76%; 95%CI: 0.792–0.952) (Fig. 2F). And AMR also had a significant linear correlation with CTFC ($R=0.5753$, $P<0.0001$) (Fig. 2D).

### Change in echocardiography in patients with different AMR subgroups

The values of LVESD and LVEF were no significant differences between the two subgroups at 1 day or 1 year. However, the value of LVEDD in the High-AMR group was significantly higher than that in the Low-AMR group at 1 year (Table 2). The value of LVEF in the Low-AMR group at 1-year follow-up was higher than that within 24 h after pPCI (42(40–49) vs. 41(39–44), Z = -2.048, $P=0.041$). However, there was no significant improvement of LVEF in the High-AMR group (42(39–45) vs. 42(38–48), $P=0.382$). The value of LVEDD in the High-AMR group at 1-year follow-up was significantly higher than that within 24 h after pPCI (5.75 ± 0.40 vs. 5.41 ± 0.32, $P<0.001$). No significant improvement of LVEDD was observed in the Low-AMR group (5.52 ± 0.39 vs. 5.47 ± 0.38, $P=0.477$). (Fig. 3).

### The prognostic significance of AMR

During a median follow-up of 7.3 (IQR 7.1–7.6) years, there were 13 (18.57%) MACCEs, including 1 (1.43%) all-cause death, 6 (8.57%) worsening heart failure, 5 (7.14%) non-fatal acute myocardial infarction, and 1 (1.43%) non-fatal ischemic stroke. During the 7.3-year follow-up after the pPCI, the High-AMR group patients showed significantly higher risk for MACCEs than did those in the Low-AMR group (Log-Rank $P=0.02$; HR = 3.90; 95%CI: 1.23–12.31). (Fig. 4) In univariate and multivariate cox regression analysis, AMR (per 0.1) was an independent predictor of MACCEs (multivariate analysis: HR = 1.153, 95%CI: 1.022-1.300; $P=0.020$). (Fig. 5)

## Discussion

This study evaluated the diagnostic accuracy of AMR in STEMI patients after PCI and the long-term prognostic significance of AMR in STEMI patients. The main findings are as follows: (1) AMR demonstrated a linear correlation with both IMR and CTFC. And the increase of AMR was correlated with STR < 70% and IMR > 40u; (2) At 1 year after pPCI, the value of LVEF in patients with Low-AMR recovered significantly and the value of LVEDD in patients with High-AMR apparently increased; (3) The risk of MACCEs significantly elevated in patients with High-AMR. And AMR was proved to be an independent predictor of MACCEs in the multivariable cox regression analysis.

Patients with STEMI who undergo PCI may experience suboptimal myocardial perfusion or even microvascular obstruction (MVO), suggesting that PCI may restore only epicardial coronary flow without ensuring adequate myocardial perfusion [18]. The Thrombolysis in Myocardial Infarction (TIMI) flow grade (TFG), TIMI myocardial perfusion grade (TMPG), myocardial blush grade (MBG), and STR are commonly used to assess myocardial perfusion; however, these indicators are semiquantitative and exhibit considerable measurement variability [19–21]. The IMR is an invasive metric based on pressure-temperature sensor wires and adenosine, offering the advantage

**Table 1** Baseline characteristics of the study population

| Patient characteristics | High-AMR ($n=43$) | Low-AMR ($n=27$) | *P* value |
|---|---|---|---|
| **Demographics** | | | |
| Age (years) | 60.65 ± 10.43 | 59.15 ± 13.06 | 0.60 |
| Sex (n, %) | 36(83.72%) | 25(92.59%) | 0.48 |
| Current smoking (n, %) | 29(67.44%) | 16(59.26%) | 0.49 |
| Hypertension (n, %) | 21(48.84%) | 14(51.85%) | 0.81 |
| Diabetes (n, %) | 14(32.56%) | 7(25.96%) | 0.56 |
| Dyslipidemia (n, %) | 14(32.56%) | 10(37.04%) | 0.70 |
| Previous angina (n, %) | 16(37.21%) | 11(40.74%) | 0.77 |
| **Admission Situation** | | | |
| Killip class (n, %) | | | |
| I | 31(72.09%) | 20(74.07%) | 0.86 |
| II | 10(23.26%) | 6(22.22%) | - |
| III | 1(2.33%) | 1(3.70%) | - |
| IV | 1(2.33%) | 0(0%) | - |
| SBP (mmHg) | 132.68 ± 18.66 | 135.83 ± 21.97 | 0.54 |
| DBP (mmHg) | 80.50(75.25–95.5) | 90.00(73.25-92.00) | 0.43 |
| D2B (min) | 75.50(50.75–89.25) | 70(60–141) | 0.38 |
| P2B (min) | 305(200–590) | 210(140–320) | 0.09 |
| **Laboratory Examination** | | | |
| Hb (g/L) | 145 ± 16.03 | 144.26 ± 20.18 | 0.87 |
| HbA1c (%) | 5.90(5.30–6.50) | 5.85(5.40–6.35) | 0.96 |
| TG (mmol/L) | 1.39(0.93–2.17) | 1.35(0.96–1.83) | 0.82 |
| TC (mmol/L) | 4.58(3.62–5.41) | 4.72(4.16–5.81) | 0.40 |
| LDL (mmol/L) | 2.54 ± 0.75 | 2.78 ± 1.11 | 0.33 |
| HDL (mmol/L) | 0.96(0.89–1.20) | 1.16(0.75–1.33) | 0.34 |
| Peak CK-MB (U/L) | 297.50(162.75–310.50) | 294.0(219.00-384.50) | 0.37 |
| Peak troponin t(ug/L) | 7.30(4.54–9.90) | 5.17(4.07–8.31) | 0.08 |
| BNP (pg/ml) | 239.00(122.50-427.5) | 230.00(58.40-479.50) | 0.68 |
| Neutrophils (10^9/L) | 8.30(6.90–10.10) | 7.70(6.65–10.68) | 0.78 |
| Lymphocytes (10^9/L) | 1.60(1.00–2.00) | 1.45(0.98–2.10) | 0.91 |
| Monocytes (10^9/L) | 0.70(0.60–0.90) | 0.70(0.50–0.83) | 0.48 |
| PLT (10^9/L) | 167.00(138.00-221.00) | 198.50(172.00-227.25) | 0.14 |
| **Angiographic Characteristics** | | | |
| TFG (pre-PCI) | | | |
| 0 | 36(83.72%) | 22(81.48%) | 1.00 |
| 1 | 6(13.95%) | 5(18.52%) | - |
| 2 | 1(2.33%) | 0(0%) | - |
| TFG (post-PCI) | | | |
| 3 | 40(93.02%) | 25(92.59%) | 1.00 |
| 2 | 3(6.98%) | 2(7.41%) | - |
| CTFC | 37.74 ± 6.92 | 22.89 ± 6.37 | < 0.001 |
| QFR | 0.94(0.92–0.96) | 0.90(0.84–0.93) | < 0.001 |
| Culprit vessel (n, %) | | | |
| LAD | 36(83.72%) | 22(81.48%) | 1.00 |
| LCX | 2(4.65%) | 1(3.70%) | - |
| RCA | 5(11.63%) | 4(14.81%) | - |
| Number of stents (n, %) | | | |
| 1 | 30(69.77%) | 18(66.67%) | 0.79 |
| > 1 | 13(30.23%) | 9(33.33%) | - |
| Total length of stents(mm) | 29(18–42) | 30(18–47) | 0.86 |
| **Medication** | | | |
| Aspirin (n, %) | 43(100%) | 27(100%) | - |
| Clopidogrel (n, %) | 27(62.79%) | 16(59.26%) | 0.77 |

**Table 1** (continued)

| Patient characteristics | High-AMR ($n=43$) | Low-AMR ($n=27$) | *P* value |
|---|---|---|---|
| Ticagrelor (n, %) | 16(37.21%) | 11(40.74%) | 0.77 |
| β-blocker (n, %) | 39(90.70%) | 27(100%) | 0.27 |
| ACEI/ARB (n, %) | 35(81.40%) | 24(88.89%) | 0.62 |
| Statins (n, %) | 43(100%) | 27(100%) | - |

Values are mean ± SD, number (%), or median (interquartile range)

of quantitatively assessing coronary microcirculation with high reproducibility. It is specific to microcirculation and unaffected by hemodynamic changes [5, 22]. Previous studies have demonstrated that elevated IMR is a strong negative predictor of infarct volume, LVEF measured by CMR, and significantly increases the risk of rehospitalization or death due to HF when IMR > 40 [7, 23].

Despite the evidence supporting the use of IMR to evaluate coronary microcirculation and stratify patient risk, its clinical application remains limited. A prior study identified several factors contributing to physicians' reluctance to utilize invasive physiological assessments, including physician attitudes (e.g., the belief that clinical and angiographic data are sufficient), environmental barriers such as reimbursement issues, and the additional demand for medical resources, including hyperemic agents [31]. In contrast, AMR offers an alternative approach by eliminating the need for pressure-temperature sensor wires and adenosine. Clinicians can use computer software to measure the QFR of target vessels through a single angiographic projection, simulate blood flow velocity under maximal hyperemia, and calculate AMR using established formulas. However, the diagnostic efficacy and prognostic implications of AMR require further clinical research for validation.

This study demonstrated that AMR is comparable to IMR in evaluating microvascular resistance in STEMI patients undergoing pPCI. Furthermore, a significant correlation was found between AMR and STR < 70% at 2 h after pPCI. Previous studies have highlighted the efficacy of STR in diagnosing CMD in STEMI patients and its favorable prognostic implications [16]. The strong correlation between AMR and STR not only supports the diagnostic performance of AMR but also suggests its potential prognostic value in STEMI patients. Unlike angiography-derived IMR, which requires two angiographic views separated by at least 25° with minimal vessel foreshortening and overlap [13, 14], AMR can assess microvascular resistance using a single angiographic projection. This results in reduced contrast media usage and shorter PCI procedure times. Physicians in catheterization laboratories are actively seeking effective treatments to prevent CMD and adverse clinical outcomes following PCI, including vasodilatory agents or low-dose intracoronary thrombolytics [24–26]. By enabling accurate and rapid assessment of coronary microcirculation and risk stratification, AMR may facilitate intra-operative interventions.

In addition to accurately assessing coronary microvascular resistance in STEMI patients undergoing PCI, the correct identification of patients at high risk for cardiac deterioration or adverse clinical events is crucial. Research by Scarsini et al. demonstrated that STEMI patients with IMR > 40 after PCI had more than a 4-fold increased risk of adverse outcomes at long-term follow-up [27]. Previous studies have shown that patients with low IMR, as measured by echocardiography, experienced significant improvement in left ventricular function at 3 months following PCI [28, 29]. The present study found that patients with low-AMR exhibited improvements in LVEF during follow-up, suggesting that AMR may offer similar predictive capabilities as IMR. Furthermore, in multivariate Cox regression analysis, AMR independently predicted MACCEs, indicating that AMR may serve as an effective tool for risk stratification in clinical models for predicting adverse outcomes in STEMI patients.

This study preliminarily demonstrated the diagnostic value and prognostic significance of AMR, offering the potential to assess coronary microcirculation function in STEMI patients using a single angiographic image. However, A larger sample size is required to calculate the cut-off value for AMR diagnosis of microcirculation dysfunction and prediction of clinical outcome.

### Limitations

This study has several limitations. First, it was a retrospective analysis with a small sample size, meaning that the AMR cut-off value model developed is not a true predictive model but rather represents only internal validation. Second, IMR were not measured on all patients, and the small sample size might limit the representativeness of the data, potentially affecting the determination of key AMR cut-off values and the accuracy of other indicators used to assess microcirculatory dysfunction. Third, microvascular resistance in the culprit vessels can fluctuate dynamically after coronary revascularization; [30] however, AMR was not measured dynamically, which complicated the identification of the optimal time point for assessment. This limitation restricts our ability to accurately evaluate a patient's microcirculatory function. Fourth, AMR was measured offline after pPCI, and a

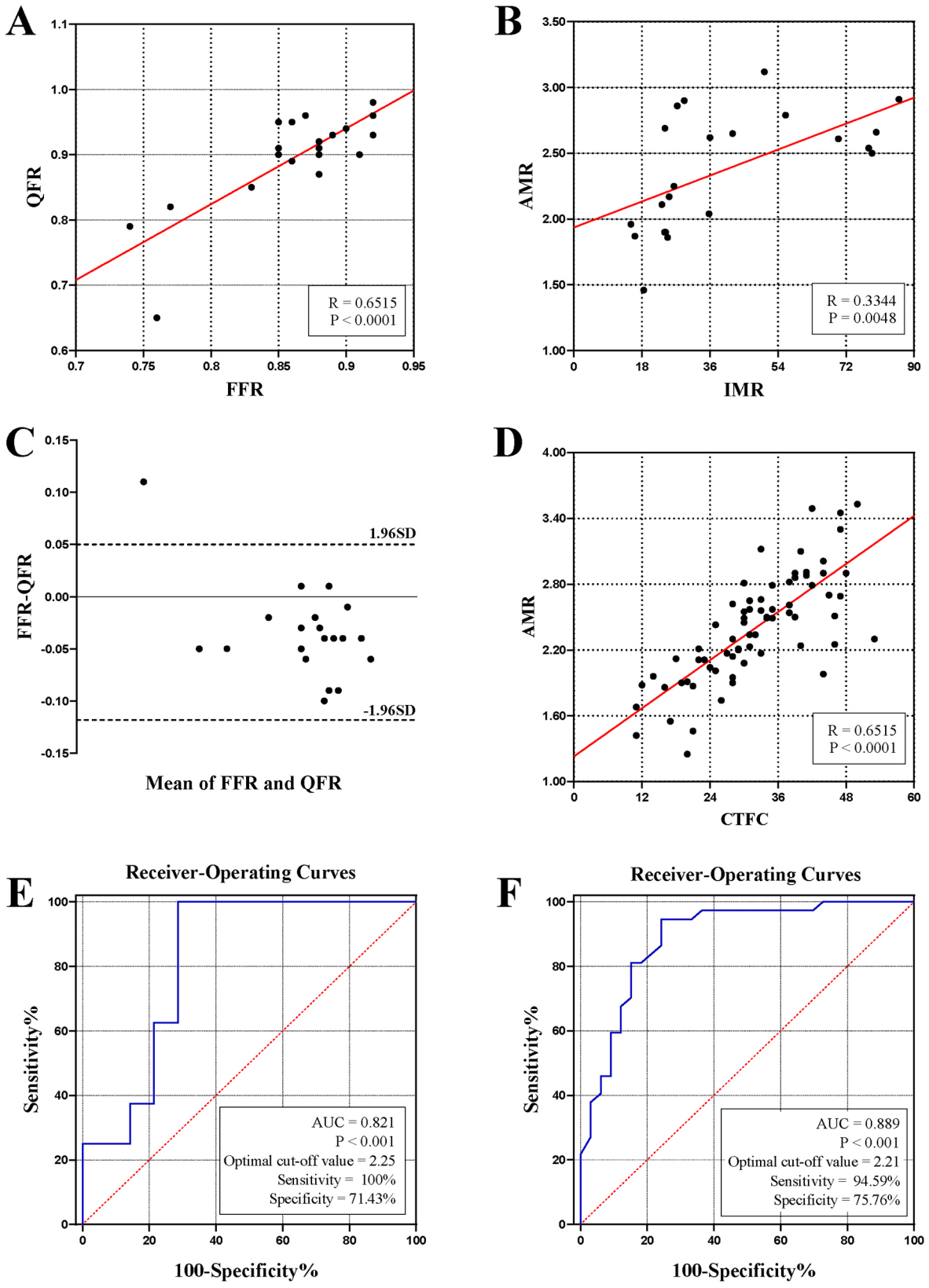


**Fig. 2** The Diagnostic Accuracy of AMR. Scatter plots summarize the correlation between QFR and FFR (**A**), IMR, and AMR (**B**). Bland–Altman plots (**C**) summarize the agreement between FFR and QFR. Receiver-Operating Curves analysis (**E**) for AMR predicts IMR > 40U. Receiver-Operating Curves analysis (**F**) for AMR predicts STR ≤ 70%. Scatter plots summarize the correlation between AMR and CTFC (**D**). Abbreviations: AUC: area under curve

**Table 2** The echocardiography within 24 h after pPCI and 1 year after Follow-up

| | Post-PCI | | |
|---|---|---|---|
| | **High-AMR(*n* = 43)** | **Low-AMR(*n* = 27)** | ***P* value** |
| LVEDD (cm) | 5.41 ± 0.32 | 5.47 ± 0.38 | 0.485 |
| LVESD (cm) | 4.29 ± 0.32 | 4.40 ± 0.38 | 0.235 |
| LVEF (%) | 42(39–45) | 41(39–44) | 0.593 |
| | **Follow-up** | | |
| | **High-AMR(*n* = 43)** | **Low-AMR(*n* = 27)** | ***P*value** |
| LVEDD (cm) | 5.75 ± 0.40 | 5.52 ± 0.39 | 0.021 |
| LVESD (cm) | 4.56 ± 0.49 | 4.37 ± 0.54 | 0.164 |
| LVEF (%) | 42(38–48) | 42(40–49) | 0.454 |

Values are mean ± SD or median (interquartile range)

small fraction of lesions could not be assessed due to suboptimal angiographic image quality. Finally, the diagnostic accuracy and prognostic significance of AMR require further investigation in different patient subgroups through prospective trials.

## Conclusions

Estimation of the coronary microvascular resistance based on AMR in STEMI patients underwent pPCI is feasible in the absence of intracoronary pressure-temperature sensor wire and adenosine. The lower AMR is associated with significantly improved left ventricular function, whereas the higher AMR is independently associated with adverse prognosis.

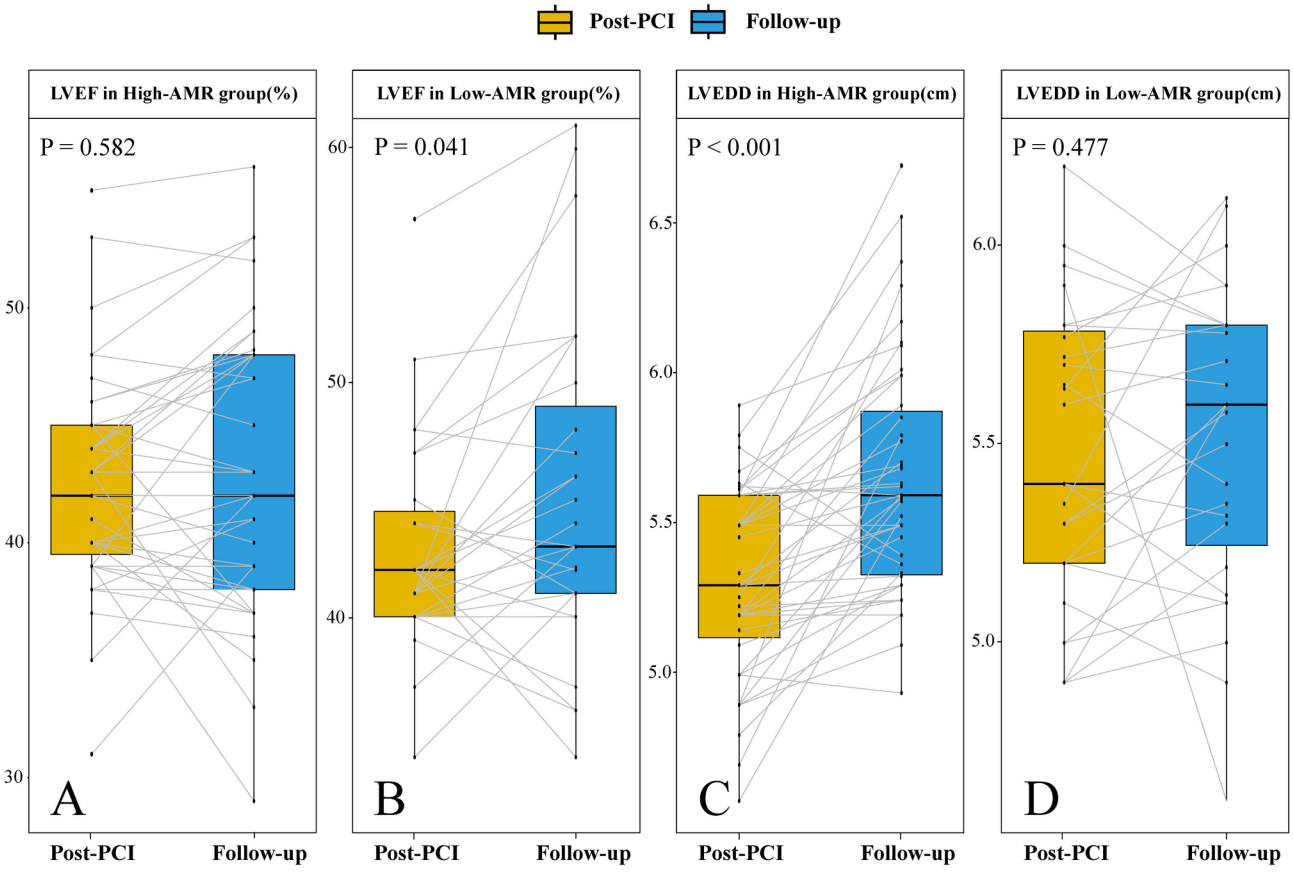


**Fig. 3** The LVEDD and LVEF Variations after pPCI and after Follow-up
All patients completed echocardiography within 24 h after pPCI and within 1 year of follow-up, and the recorded indicators are compared using paired t-test or Wilcoxon signed-rank test

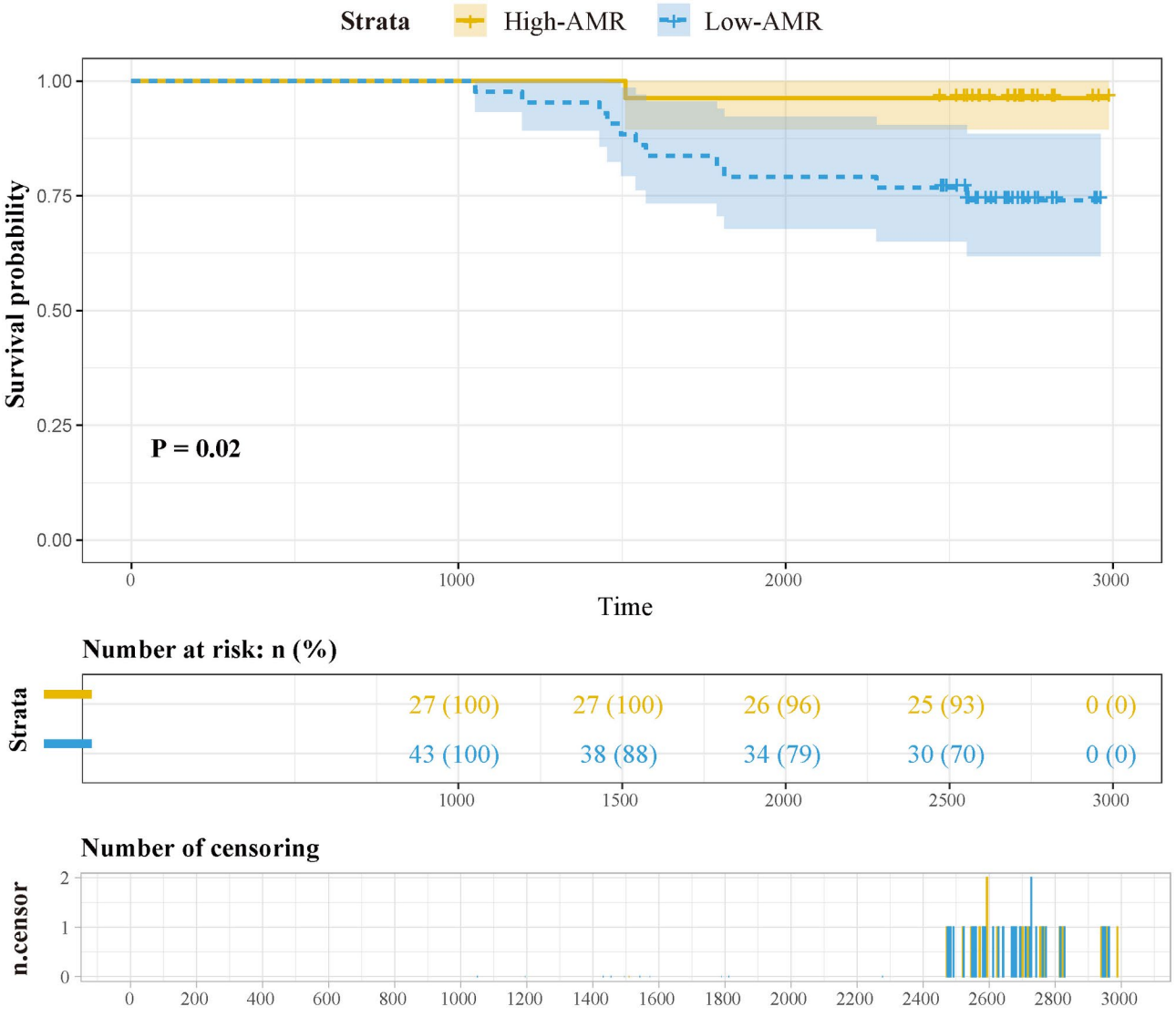


**Fig. 4** Comparison of Long-Term Risk of MACCEs between High-AMR and Low-AMR among STEMI Patients Underwent pPCI

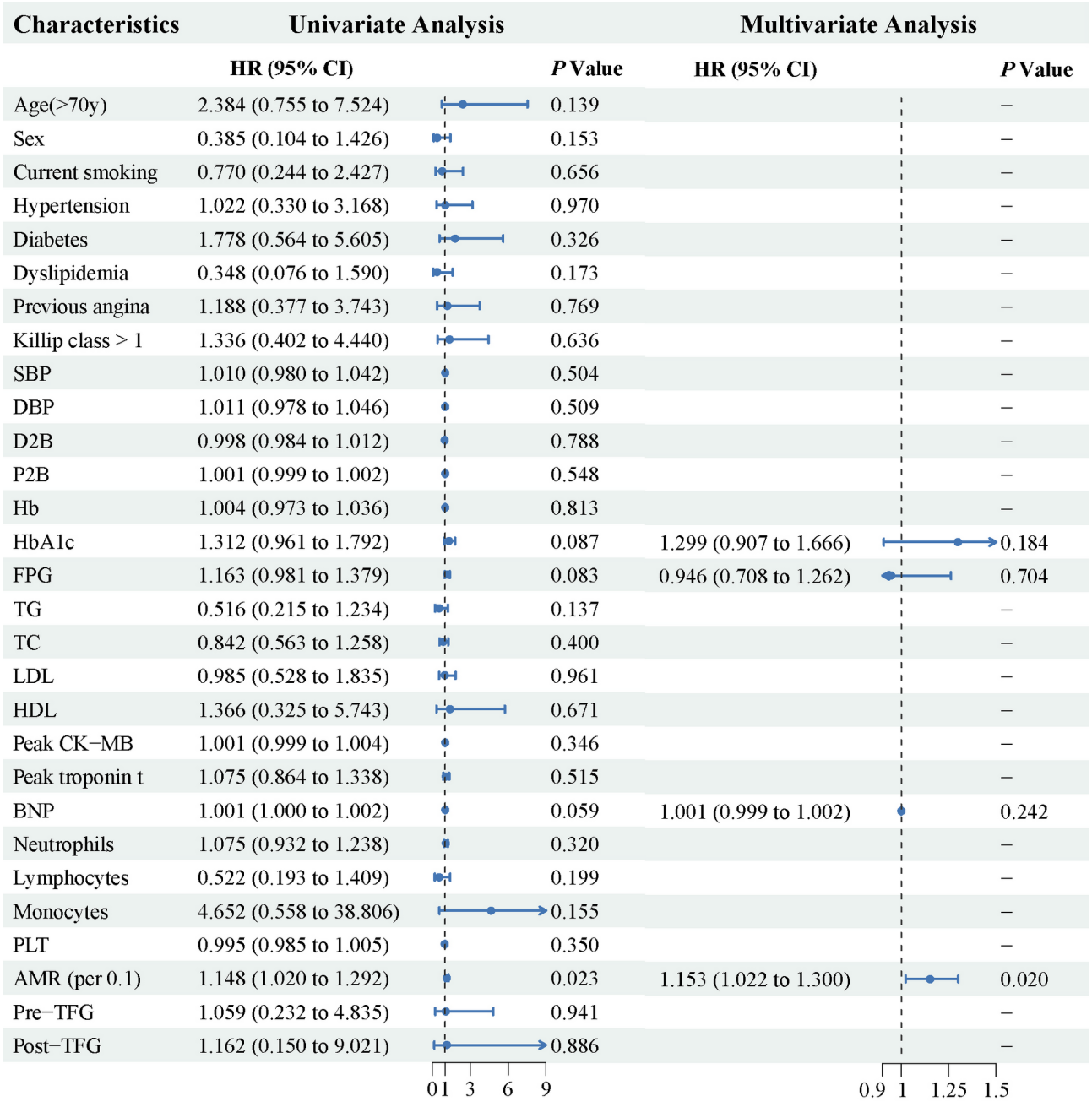


**Fig. 5** Forest Map of the Univariate and Multivariate Cox Regression Analyses

**Abbreviations**

| | |
|---|---|
| STEMI | ST-segment elevation myocardial infarction |
| PCI | Percutaneous coronary intervention |
| QFR | Quantitative flow ratio |
| AMR | Angio-based microvascular resistance |
| STR | ST-segment resolution |
| CTFC | Corrected TIMI frame count |
| FFR | Fractional flow reserve |
| IMR | Index of microvascular resistance |
| SBP | Systolic blood pressure |
| DBP | Diastolic blood pressure |
| FPG | Fasting plasma glucose |
| D2B | Door-to-balloon time |
| P2B | Pain-to-balloon time |
| Hb | Hemoglobin |
| HbA1c | Glycosylated hemoglobin |
| TG | Triglyceride |
| TC | Cholesterol |
| LDL | Low-density lipoprotein |
| HDL | High-density lipoprotein |
| Peak CK-MB | Peak creatine kinase-myocardial band |
| PLT | Platelet |
| TFG | Thrombolysis In Myocardial Infarction flow grade |
| BNP | B-type natriuretic peptide |
| LAD | Left anterior descending coronary artery |
| LCX | Left circumflex artery |
| RCA | Right coronary artery |
| ACEI | Angiotensin-converting enzyme inhibitor |
| ARB | Angiotensin receptor inhibitor. LVEDD: Left ventricular end-diastolic diameter |

| | |
|---|---|
| LVESD | Left ventricular end-systolic diameter |
| LVEF | Left ventricular ejection fraction |
| AUC | area under curve |
| CI | confidence interval |
| HR | hazard ratio |
| MVO | microvascular obstruction |

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None.

**Author contributions**
LNK, ZHW, BX, and ZZ contributed to the conception and design of the study; ZZ, QD, XLZ contributed to manuscript writing; SYQ, PX, and QD contributed to data collection and management; QD, YG, XMG, and YNX participated in the patient follow-up; QD, KW, and XB contributed to the statistical analysis; LNK, BX, and ZHW contributed to manuscript revision and data review. All authors have read and approved the manuscript.

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**Data availability**
The information and data of the study population were extracted from the hospital information system. The deidentified participant data will be shared upon reasonable request. Data are however available from the corresponding author on a reasonable request.

## Declarations

**Ethics approval and consent to participate**
The data collection for the study was approved by the Medical Ethics Committee of Nanjing Drum Tower Hospital (No.2022-060-02). Clinical trial number: not applicable. All participants provided written informed consent participate.

**Ethical guidelines**
The study protocol was conducted in accordance with the declaration of Helsinki.

**Consent for publication**
Not applicable.

**Competing interests**
The authors declare no competing interests.

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