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Original Article

Predictors of right ventricular dysfunction and its clinical value after reperfusion of acute right coronary artery occlusion: a single-center retrospective analysis

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Abstract: Objective: To investigate independent predictors of right ventricular (RV) dysfunction following successful reperfusion by percutaneous coronary intervention (PCI) in patients with acute right coronary artery (RCA) occlusion, and to assess its association with short-term clinical outcomes. Methods: This single-center retrospective study enrolled consecutive patients with acute ST-segment elevation myocardial infarction (STEMI) who underwent emergency PCI for acute RCA occlusion. Based on right ventricular fractional area change (RVFAC) measured by echocardiography 24-72 hours post-PCI, patients were divided into an RV dysfunction group (RVFAC < 35%) and an RV normal function group (RVFAC ≥ 35%). Baseline characteristics, laboratory data, angiographic findings, and in-hospital and 30-day outcomes were compared. Multivariable logistic regression was used to identify independent predictors of RV dysfunction. Results: Among 136 patients, 29.4% had right ventricular (RV) dysfunction. These patients were older and had more cardiovascular risk factors. Independent predictors of RV dysfunction included an NLR ≥ 5, lactate ≥ 2 mmol/L, cardiogenic shock (Killip class IV) at admission, and age ≥ 65 years. A composite score of NLR and lactate demonstrated excellent predictive value for RV dysfunction. During hospitalization, patients with RV dysfunction experienced significantly higher rates of cardiogenic shock, malignant arrhythmias, acute kidney injury, and cardiac death. At 30 days, they also had a higher rate of readmission for heart failure. Conclusion: In patients with acute RCA occlusion undergoing successful PCI, elevated NLR (≥ 5), hyperlactatemia (≥ 2 mmol/L), cardiogenic shock at admission, and age ≥ 65 years are independent predictors of early postoperative RV dysfunction. RV dysfunction is strongly associated with adverse in−hospital outcomes and increased 30-day heart failure readmission. Early identification of these factors may guide risk stratification and individualized management.

Keywords: Right coronary artery occlusion, right ventricular dysfunction, percutaneous coronary intervention, predictors, echocardiography

Introduction

Right ventricular myocardial infarction (RVMI) is a common and critical complication of acute inferior ST-segment elevation myocardial infarction (STEMI), primarily attributed to acute occlusion of the right coronary artery (RCA) [1]. Epidemiological data indicate that in patients with acute inferior STEMI, the proportion involving right ventricular involvement is as high as 30-50%. These patients often experience a more complicated clinical course and significantly higher mortality rates [2, 3].

The right ventricle (RV), characterized by a thin wall and low pressure, relies heavily on continuous coronary perfusion and is extremely sensitive to changes in preload. Acute RCA occlusion leads to ischemia and necrosis of the free RV wall, resulting in acute deterioration of both systolic and diastolic functions. The severe consequences stem not only from the impairment of the RV's intrinsic pumping function but also from the mechanism of "ventricular interdependence" [4-7]. This involves the septum shifting toward the left ventricle, enhanced pericardial constraint, and reduced left ventricular filling, ultimately leading to systemic hypoperfusion and cardiogenic shock - the primary cause of death in these patients [8, 9].

With the advent of reperfusion therapy, emergency percutaneous coronary intervention (PCI) has become the cornerstone strategy for recan-

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alizing the culprit vessel and salvaging ischemic myocardium, significantly improving the overall prognosis of patients with acute myocardial infarction [10-13]. For myocardial infarction caused by RCA occlusion, successful PCI rapidly restores anterograde blood flow (TIMI grade 3) in the epicardial vessels, creating conditions for the recovery of the ischemic right ventricle [14]. However, clinical observations and studies indicate that anatomical revascularization does not equate to complete recovery of tissue-level perfusion or immediate restoration of contractile function [15]. A substantial number of patients still develop persistent or new-onset right ventricular dysfunction (RVD) in the early postoperative period despite successful RCA recanalization [16]. This persistent RVD is closely associated with refractory hypotension, high-grade atrioventricular block, and an increased risk of malignant arrhythmias during hospitalization [17-20]. It may also portend a longer length of stay, higher short-term mortality, and increased heart failure rehospitalization rates.

Currently, the predictive and evaluative system for RV functional recovery following acute RCA recanalization remains inadequate [21]. The recovery of RV function is a complex process modulated by multiple factors. In addition to the total ischemic time, microcirculatory perfusion status (e.g., no-reflow phenomenon), ischemic preconditioning, the severity of reperfusion injury, and the anatomical characteristics of the culprit lesion (e.g., occlusion site, collateral circulation) all play pivotal roles. Furthermore, systemic inflammatory response - one of the core pathological components of acute myocardial infarction - may also influence RV functional recovery by exacerbating microvascular dysfunction and myocardial stunning [22-24].

Although some studies have explored the relationship between single factors (e.g., ST-segment elevation in right precordial leads, peak myocardial enzymes) and RV function, research that systematically integrates admission hemodynamic status, laboratory inflammatory/perfusion markers, and coronary angiographic characteristics to provide an early, comprehensive prediction of post-recanalization RV dysfunction is relatively lacking [25, 26]. Such early risk stratification is crucial for clinical practice. It aids in identifying high-risk patient populations, potentially guiding more aggressive individualized interventions - such as optimizing volume management, early application of inotropic or vasopressor support, or even preemptive deployment of invasive hemodynamic monitoring - to break the vicious cycle and improve clinical outcomes.

Therefore, to bridge these knowledge gaps, this study aimed to systematically investigate the independent predictors of right ventricular (RV) dysfunction following successful reperfusion via emergency percutaneous coronary intervention (PCI) in patients with acute right coronary artery (RCA) total occlusion, through a single-center retrospective cohort analysis. We specifically evaluated the predictive value of admission clinical characteristics (e.g., cardiogenic shock), readily available laboratory markers (e.g., blood lactate, neutrophil-to-lymphocyte ratio), and angiographic parameters (e.g., preoperative TIMI flow, collateral circulation). Furthermore, this study aimed to clarify the association between early postoperative RV dysfunction and in-hospital complications, as well as 30-day short-term clinical outcomes (including mortality and heart failure rehospitalization). The findings of this study are expected to provide clinicians with a practical early risk assessment framework, facilitating the precise identification and stratified management of high-risk patients, ultimately improving prognosis.

Materials and methods

Clinical data

This study is a single-center, retrospective cohort study. We consecutively enrolled patients with acute ST-segment elevation myocardial infarction (STEMI) admitted to the Department of Cardiology at Nanfang Hospital, Southern Medical University, between January 2020 and December 2023. All patients underwent emergency coronary angiography, which confirmed acute total occlusion of the right coronary artery (RCA) (TIMI flow grade 0-1). All patients subsequently underwent successful percutaneous coronary intervention (PCI) with restoration of coronary flow (post-procedural TIMI flow grade 2-3). The study protocol was approved by the Ethics Committee of Nanfang

Hospital, Southern Medical University. Informed consent was waived due to the retrospective nature of the study and the use of de-identified data. All data were kept strictly confidential and managed in accordance with institutional data governance policies. This study was conducted in full compliance with the ethical principles of the Declaration of Helsinki (2013 revision).

Inclusion and exclusion criteria

This retrospective study enrolled consecutive patients admitted with acute ST-segment elevation myocardial infarction (STEMI). Patients were included if they met the following criteria: (1) age ≥ 18 years; (2) angiographically confirmed acute total occlusion of the right coronary artery (RCA) with TIMI flow grade 0-1; (3) successful emergency percutaneous coronary intervention (PCI) with post-procedural TIMI flow grade 2-3; and (4) completion of transthoracic echocardiography (TTE) within 1 week after PCI with images of analyzable quality.

The exclusion criteria were as follows: (1) history of prior right ventricular myocardial infarction, severe chronic right heart failure, or cor pulmonale; (2) concomitant severe valvular heart disease, cardiomyopathy, or pericardial disease; (3) admission due to non-cardiogenic shock (e.g., infection, hemorrhage); (4) missing clinical or imaging data > 20%; and (5) in-hospital death or discharge against medical advice (DAMA) preventing completion of echocardiography.

Data collection

Data were collected through the hospital's electronic medical record system, the cardiac catheterization laboratory records, and the echocardiographic image system.

Baseline characteristics: Age, sex, body mass index (BMI), cardiovascular risk factors (hypertension, diabetes, dyslipidemia, smoking history), past medical history (coronary artery disease, myocardial infarction, chronic heart failure, chronic kidney disease, atrial fibrillation, etc.), vital signs upon admission (heart rate, blood pressure), and Killip classification.

Treatment details: Time from symptom onset to first medical contact, door-to-balloon time, PCI procedural details (number and length of stents, thrombectomy, use of vasoactive agents, etc.), and the use of intra-aortic balloon pump (IABP) or temporary pacemaker during hospitalization.

Laboratory examinations: Peak values of myocardial injury markers (troponin I, CK-MB), complete blood count upon admission (to calculate neutrophil-to-lymphocyte ratio, NLR), renal function (estimated glomerular filtration rate, eGFR), N-terminal pro-B-type natriuretic peptide (NT-proBNP), and arterial blood lactate.

Imaging and electrocardiography: Coronary angiography: Culprit lesion location (proximal, middle, distal), preoperative TIMI flow grade, collateral circulation (Rentrop grade), and presence of multivessel disease.

Electrocardiography: ST-segment elevation in right precordial leads (V3R-V5R) and lead V1 upon admission, presence of complete right bundle branch block (RBBB), and the extent of ST-segment resolution after PCI.

Echocardiography: Performed 24-72 hours post-PCI by experienced sonographers using a Canon Aplio i800 (TUS-AI800) ultrasound system (Canon Medical Systems, Otawara, Japan) with a PST-25SX phased-array probe (1-5 MHz). The primary measurement was right ventricular fractional area change (RVFAC): In the apical four-chamber view, the endocardial border of the right ventricle was manually traced at end-diastole and end-systole. RVFAC was calculated as [(end-diastolic area - end-systolic area)/end-diastolic area] × 100%. Secondary parameters included tricuspid annular plane systolic excursion (TAPSE), right ventricular basal diameter, inferior vena cava (IVC) diameter, pulmonary artery systolic pressure (PASP), and septal wall motion.

Research methods

Group definition: Based on the RVFAC measured by the first postoperative echocardiogram, patients were divided into two groups using a cutoff value of 35%: the Right Ventricular Dysfunction group (RVFAC < 35%) and the Right Ventricular Normal Function group (RVFAC ≥ 35%).

Outcome measures: In-hospital outcomes: Cardiac death, cardiogenic shock, malignant arrhythmias requiring intervention, acute kid-

ney injury (per KDIGO criteria), and repeat revascularization.

Short-term follow-up outcomes: All-cause mortality, heart failure rehospitalization within 30 days post-discharge, and New York Heart Association (NYHA) functional class at discharge, ascertained by medical record review or telephone interview.

Statistical analysis

Statistical analysis was performed using SPSS 26.0 software. Normally distributed continuous variables are presented as mean ± standard deviation ($\bar{x}$ ± sd) and were compared using the independent samples *t*-test. Non-normally distributed continuous variables are presented as median (interquartile range) [M (P25, P75)] and were compared using the Mann-Whitney *U* test. Categorical variables are expressed as number (percentage) [n (%)], and group comparisons were performed using the χ^2 test or Fisher's exact test. Pearson or Spearman correlation analysis was used to explore correlations between key continuous variables. Variables with $P < 0.1$ in the univariate analysis were entered into a multivariate binary Logistic regression model (backward method) to identify independent predictors of right ventricular dysfunction; odds ratios (OR) and 95% confidence intervals (CI) were calculated. Receiver operating characteristic (ROC) curves were used to evaluate the discriminative ability of the predictors, and the area under the curve (AUC) was calculated. Kaplan-Meier curves were constructed to estimate 30-day heart failure-free survival, and the Log-rank test was used to compare differences between the groups. A two-sided $P < 0.05$ was considered statistically significant.

Results

Patient baseline characteristics and cardiovascular risk factors

A total of 136 patients with acute right coronary artery (RCA) occlusion who underwent emergency percutaneous coronary intervention (PCI) were included. Based on right ventricular fractional area change (RVFAC) measured by echocardiography 24-72 hours post-PCI, patients were divided into an RV dysfunction group (n = 40, 29.4%) and an RV normal function group (n = 96, 70.6%) (**Figure 1A**).

The RV dysfunction group was significantly older (P < 0.001). No significant differences were observed in sex distribution or body mass index (both P > 0.05) (**Table 1**; **Figure 1B**, **1C**). Regarding cardiovascular risk factors, the dysfunction group had significantly higher proportions of diabetes (P = 0.001) and smoking history (P < 0.001). The prevalence of hypertension, dyslipidemia, family history of coronary artery disease, and previous myocardial infarction did not differ significantly between the two groups (all P > 0.05) (**Figure 2A-F**; **Table 1**). Other medical histories, including chronic heart failure, chronic kidney disease, and atrial fibrillation, were also comparable (all P > 0.05) (**Table 1**).

The RV dysfunction group presented with worse Killip class (P < 0.001 overall; **Figure 1D**), significantly higher heart rate (P = 0.001), lower systolic and diastolic blood pressure (both P < 0.001), and a longer symptom-to-first-medical-contact time (P = 0.050). Door-to-balloon time was comparable between the two groups (P = 0.987) (**Table 1**).

Comparison of laboratory indicators

Patients in the RV dysfunction group exhibited significantly higher levels of inflammatory and myocardial injury markers, including neutrophil-to-lymphocyte ratio (NLR), blood lactate, NT-proBNP, peak troponin I, and peak CK-MB (all P < 0.001). Estimated glomerular filtration rate (eGFR) was significantly lower in the dysfunction group (P < 0.001). White blood cell count did not differ significantly between the two groups (P = 0.424) (**Table 2**; **Figure 3A-F**).

Imaging, electrocardiography, and treatment characteristics

There were no significant differences between the two groups in lesion location, preoperative TIMI flow grade 0, collateral circulation distribution, or multivessel disease (all P > 0.05). However, the RV dysfunction group had significantly higher rates of ST-segment elevation in leads V3R-V5R (P = 0.001) and V1 (P < 0.001), as well as complete right bundle branch block (P = 0.014). Post-PCI ST-segment resolution was similar between the two groups (P = 0.939). Regarding treatment, thrombus aspiration and vasoactive drug use were significantly more common in the dysfunction group (both P < 0.001), while the number and length of stents

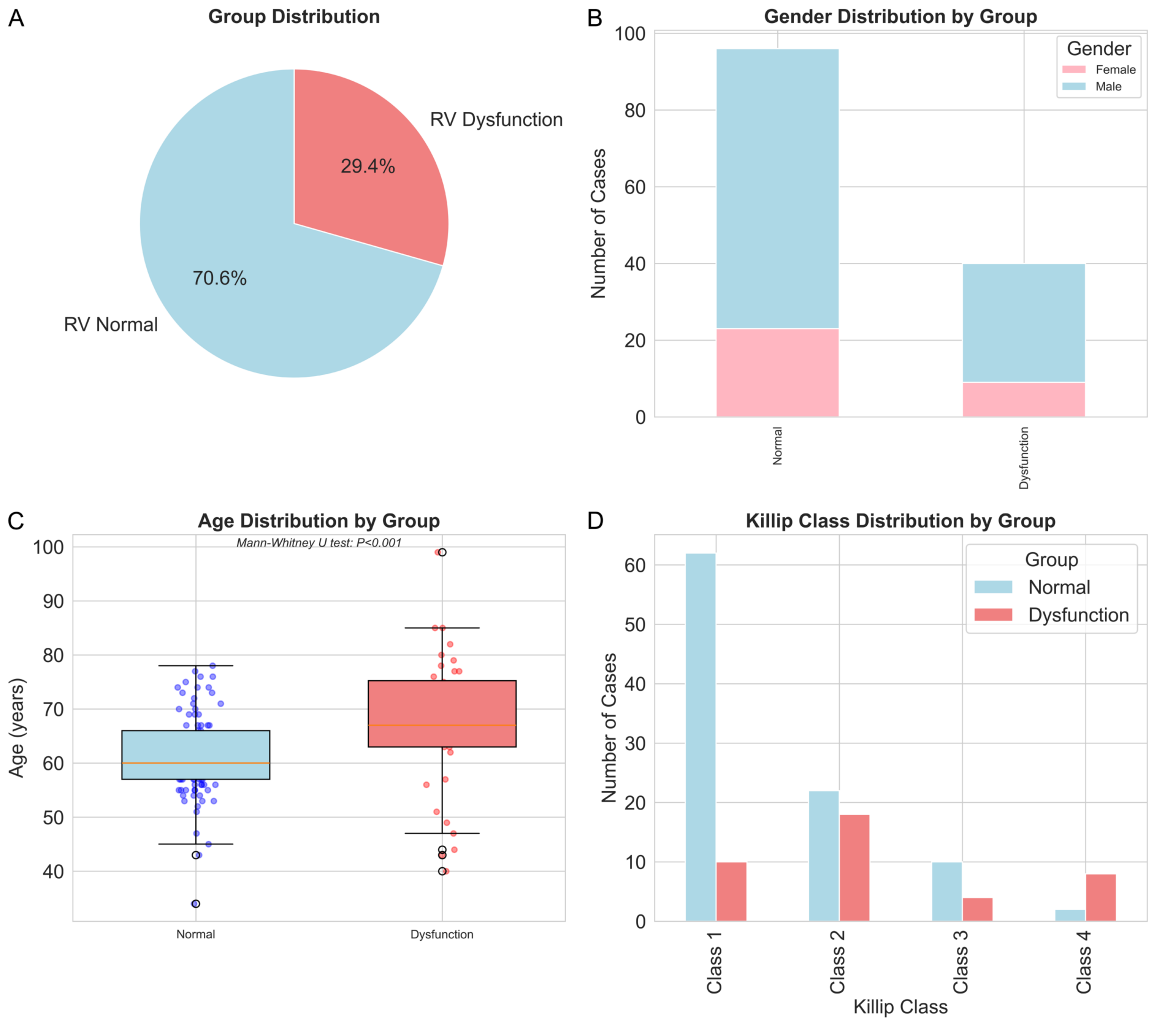

Figure 1. Baseline characteristics distribution. A. Group distribution showing proportion of patients with RV dysfunction (29.4%) and normal RV function (70.6%). B. Gender distribution was similar between the two groups. C. Age distribution: RV dysfunction group was significantly older (P < 0.001). D. Killip class distribution: RV dysfunction group had significantly worse Killip class (P < 0.001). RV, right ventricular.

implanted were comparable (both P > 0.05) (**Table 3**).

Right ventricular function and hemodynamic parameters

As expected, the RV dysfunction group had significantly lower RVFAC and TAPSE, and larger RV basal diameter (all P < 0.001). Inferior vena cava diameter and pulmonary artery systolic pressure were also significantly higher in the dysfunction group (both P < 0.001). Septal motion abnormality was significantly more prevalent (P = 0.001) (**Table 4**; **Figure 4A-F**). In line with these findings, the incidence of cardiogenic shock during hospitalization was significantly higher (P < 0.001), and the use of intra-aortic balloon pump, temporary pacemaker, diuretics, and inotropes was more frequent in the dysfunction group (all P ≤ 0.001) (**Table 4**).

Comparison of clinical outcomes

During hospitalization, patients with RV dysfunction had significantly higher rates of cardiogenic shock, malignant arrhythmias, acute kidney injury, and cardiac death (all P < 0.05). Repeat revascularization rates were similar between the two groups (P = 0.747). At 30-day follow-up, the dysfunction group showed a significantly higher rate of heart failure readmis-

Table 1. Comparison of baseline characteristics between patients with and without right ventricular dysfunction after acute right coronary artery occlusion reperfusion

Variable	Normal (n = 96)	Dysfunction (n = 40)	U/χ^2	*P* value
Age (years)	61.2 ± 7.7	67.1 ± 12.3	1201.000	< 0.001
Male	73 (76.0%)	31 (77.5%)	0.034	0.853
BMI (kg/m^2)	24.49 ± 2.79	25.42 ± 3.42	1589.000	0.581
Hypertension	50 (52.1%)	25 (62.5%)	0.853	0.356
Diabetes	27 (28.1%)	24 (60.0%)	10.918	0.001
Dyslipidemia	58 (60.4%)	28 (70.0%)	0.741	0.389
Smoking	38 (39.6%)	30 (75.0%)	12.785	0.000
Family history of CAD	16 (16.7%)	10 (25.0%)	0.786	0.375
Previous MI	8 (8.3%)	5 (12.5%)	0.187	0.665
Chronic heart failure	7 (7.3%)	3 (7.5%)	0.004	0.951
Chronic kidney disease	10 (10.4%)	9 (22.5%)	2.498	0.114
Atrial fibrillation	9 (9.4%)	5 (12.5%)	0.056	0.813
Heart rate (bpm)	84.27 ± 11.99	92.00 ± 11.49	1211.000	0.001
Systolic BP (mmHg)	118.54 ± 14.04	102.12 ± 17.17	2917.500	< 0.001
Diastolic BP (mmHg)	75.11 ± 10.53	63.40 ± 17.67	2776.000	< 0.001
Symptom to FMC (min)	194.31 ± 83.61	243.05 ± 136.93	1509.000	0.050
Door to balloon (min)	71.79 ± 18.94	70.42 ± 20.12	1916.000	0.987

BMI, body mass index; CAD, coronary artery disease; MI, myocardial infarction; CHF, chronic heart failure; CKD, chronic kidney disease; AF, atrial fibrillation; BP, blood pressure; FMC, first medical contact.

sion (P = 0.028), while all-cause mortality did not differ significantly (P = 0.194). Discharge NYHA functional class was significantly worse in the dysfunction group (P < 0.001) (**Table 5**; **Figure 5A**, **5B**).

Correlation analysis of key variables

Pearson correlation analysis (**Figure 6A**) revealed that RVFAC showed a strong positive correlation with TAPSE (P < 0.001). Conversely, RVFAC was negatively correlated with RV basal diameter, NT-proBNP, lactate, NLR, and PASP (all P < 0.001). Age showed a weak negative correlation with RVFAC (P = 0.011). NLR exhibited moderate positive correlations with RV basal diameter (P < 0.001) and lactate (P = 0.036). The correlation strengths are visualized in the heatmap (**Figure 6A**), and the correlations of RVFAC with other variables are highlighted in the bar plot (**Figure 6B**), where TAPSE demonstrated the strongest positive association, while RV basal diameter and NT-proBNP showed the most prominent negative associations.

Prediction model and survival analysis

Multivariable logistic regression identified four independent predictors of RV dysfunction (**Figure 7A**): NLR ≥ 5 (OR = 12.31, 95% CI: 4.01-37.79, P < 0.001), lactate ≥ 2 mmol/L (OR = 4.81, 95% CI: 1.78-13.01, P = 0.002), cardiogenic shock at admission (Killip class IV) (OR = 9.25, 95% CI: 1.11-76.73, P = 0.039), and age ≥ 65 years (OR = 3.12, 95% CI: 1.20-8.16, P = 0.020). Preoperative TIMI flow grade 0 and poor collateral circulation (Rentrop grade 0) were not significant in the multivariate model.

ROC curve analysis (**Figure 7B**) demonstrated that the composite score combining NLR and lactate yielded the highest AUC (0.893), outperforming NLR alone (AUC = 0.825) and lactate alone (AUC = 0.772). A graded relationship was observed between RVFAC quartiles and 30-day HF readmission (**Figure 7C**), with the lowest quartile having the highest readmission rate (P for trend < 0.05). The proportion of RV dysfunction increased with higher Killip class (P < 0.001 overall; **Figure 7D**). Kaplan-Meier analysis (**Figure 8**) revealed significantly lower 30-day HF readmission-free survival in the RV dysfunction group (log-rank P = 0.0135).

Discussion

Right ventricular myocardial infarction (RVMI), as a severe complication of acute inferior

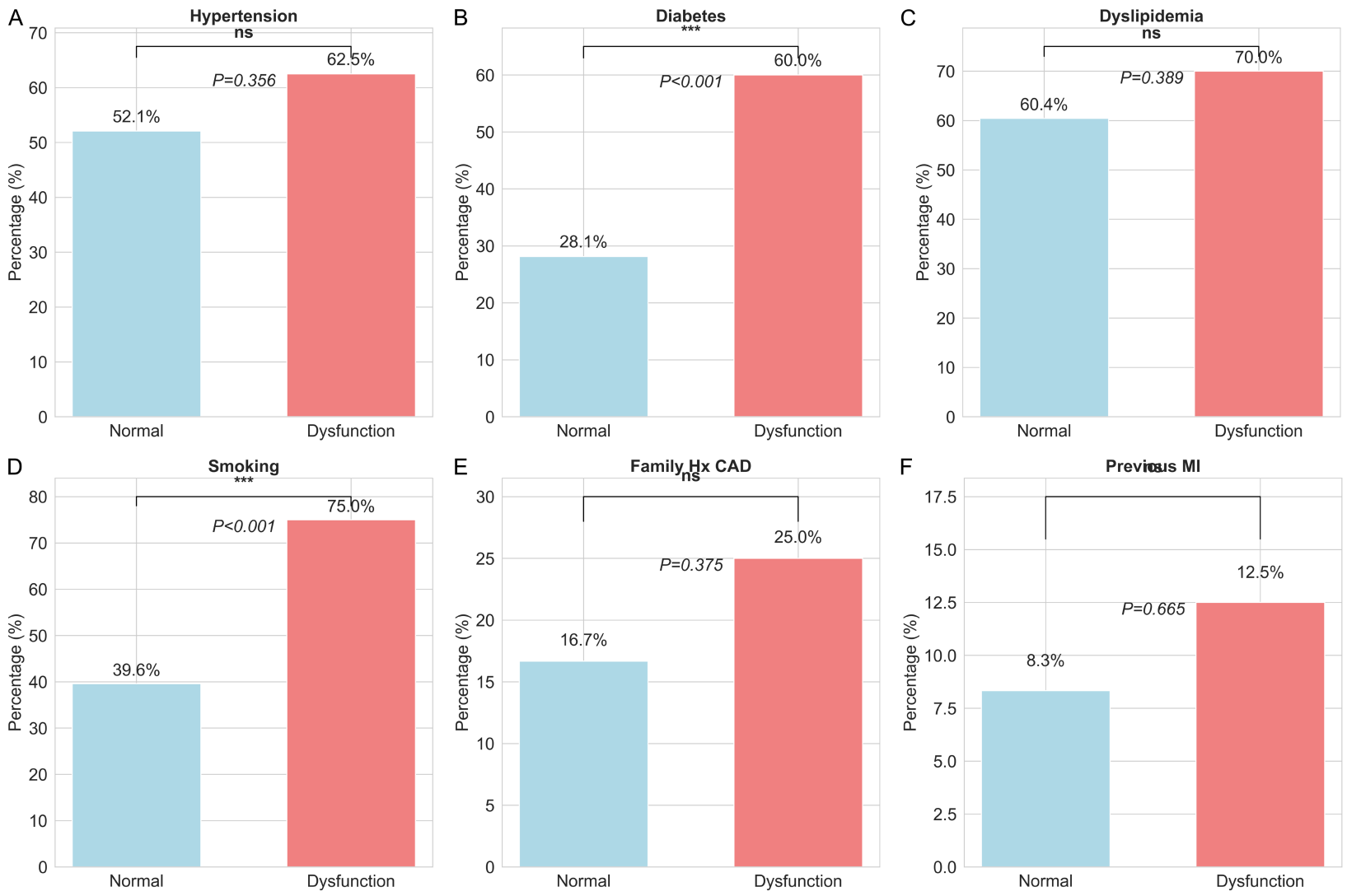

Figure 2. Comparison of cardiovascular risk factors. A. Hypertension prevalence was similar between the two groups (P = 0.356). B. Diabetes was significantly higher in the RV dysfunction group (P = 0.001). C. Dyslipidemia prevalence did not differ significantly (P = 0.389). D. Smoking history was significantly more frequent in the RV dysfunction group (P < 0.001). E. Family history of CAD was comparable between the two groups (P = 0.375). F. Previous myocardial infarction was similar between the two groups (P = 0.665). RV, right ventricular; CAD, coronary artery disease; MI, myocardial infarction.

Table 2. Comparison of laboratory parameters between patients with and without right ventricular dysfunction after acute right coronary artery occlusion reperfusion

Variable	Normal (n = 96)	Dysfunction (n = 40)	Statistics	*P* value
Troponin I peak (ng/mL)	17.88 [11.95-21.12]	21.67 [17.95-30.77]	1056.500	< 0.001
CK-MB peak (U/L)	192.00 [134.49-261.91]	326.31 [279.32-378.53]	639.000	< 0.001
White blood cell count ($\times 10^9$/L)	10.82 [9.02-12.71]	11.57 [8.73-13.39]	1752.000	0.424
Neutrophil-to-lymphocyte ratio	4.60 [3.25-5.67]	8.97 [6.05-11.00]	670.500	< 0.001
Blood lactate (mmol/L)	1.43 [1.01-1.99]	2.38 [1.75-3.39]	877.000	< 0.001
eGFR (mL/min/1.73 m^2)	79.24 [68.38-88.22]	61.44 [54.54-74.23]	2962.500	< 0.001
NT-proBNP (pg/mL)	1757.50 [1030.25-2290.50]	3420.00 [2692.50-4098.75]	741.500	< 0.001

CK-MB, creatine kinase-MB; WBC, white blood cell count; NLR, neutrophil-to-lymphocyte ratio; eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

ST-segment elevation myocardial infarction (STEMI), has long posed a significant challenge to cardiologists in terms of pathophysiology and clinical management [27-29]. Through retrospective analysis, this study found that among patients with acute right coronary artery (RCA) occlusion who successfully underwent emergency percutaneous coronary intervention (PCI), as high as 29.4% still exhibited early postoperative right ventricular (RV) dysfunction (defined as RVFAC < 35%) [30]. This proportion highlights that even in the current era of mature reperfusion therapy, the recovery of RV function remains an unresolved clinical issue [31].

Our study not only confirms the strong association between RV dysfunction and adverse short-term outcomes but, more importantly, identifies a set of independent predictors integrating hemodynamic, coronary anatomic, and systemic indices. This provides a multidimensional and practical tool for early risk stratification [32]. The recovery of RV function is determined by far more than the simple "opening" of the epicardial vessel; rather, it is a complex continuum involving ischemic injury, the reperfusion process, and systemic responses. The predictive factors identified in this study correspond precisely to pathophysiological mechanisms at different levels [33].

First, at the level of hemodynamics and tissue perfusion, the indicators of admission cardiogenic shock and hyperlactatemia serve as direct markers reflecting the severity of the initial injury. They indicate that patients were already in a state of systemic circulatory failure upon admission, having suffered extensive and severe ischemic damage to the RV myocardium, and the microvascular perfusion of which may have undergone irreversible impairment. This aligns with the concept of "time is muscle", but it also suggests that metabolic disturbances and cellular damage accumulated during profound shock may extend beyond the window salvageable by reperfusion.

At the level of coronary anatomy and regional perfusion, preoperative TIMI flow grade and collateral circulation are traditionally considered important determinants of myocardial salvage [34, 35]. However, in our multivariable analysis, neither TIMI flow grade 0 nor poor collateral circulation (Rentrop grade 0) emerged as independent predictors of RV dysfunction. This may be attributable to the relatively small sample size or to the fact that these angiographic features are partially reflected by other stronger markers of ischemic severity, such as elevated lactate and NLR. Nevertheless, the absence of significance in our model does not preclude their potential contribution in larger cohorts; the interplay between epicardial occlusion, collateral supply, and microvascular integrity remains complex and warrants further investigation.

Third, the biomarker reflecting the magnitude of the systemic inflammatory response - the elevated neutrophil-to-lymphocyte ratio (NLR) - demonstrated predictive value independent of the aforementioned factors, adding a novel perspective to our understanding of RV dysfunction (RVD) mechanisms. The robust inflammatory response triggered by acute myocardial infarction is a "double-edged sword". While it aids in clearing necrotic tissue, an excessive release of inflammatory mediators can exacerbate microvascular endothelial dysfunction, promote neutrophil plugging in the capillaries,

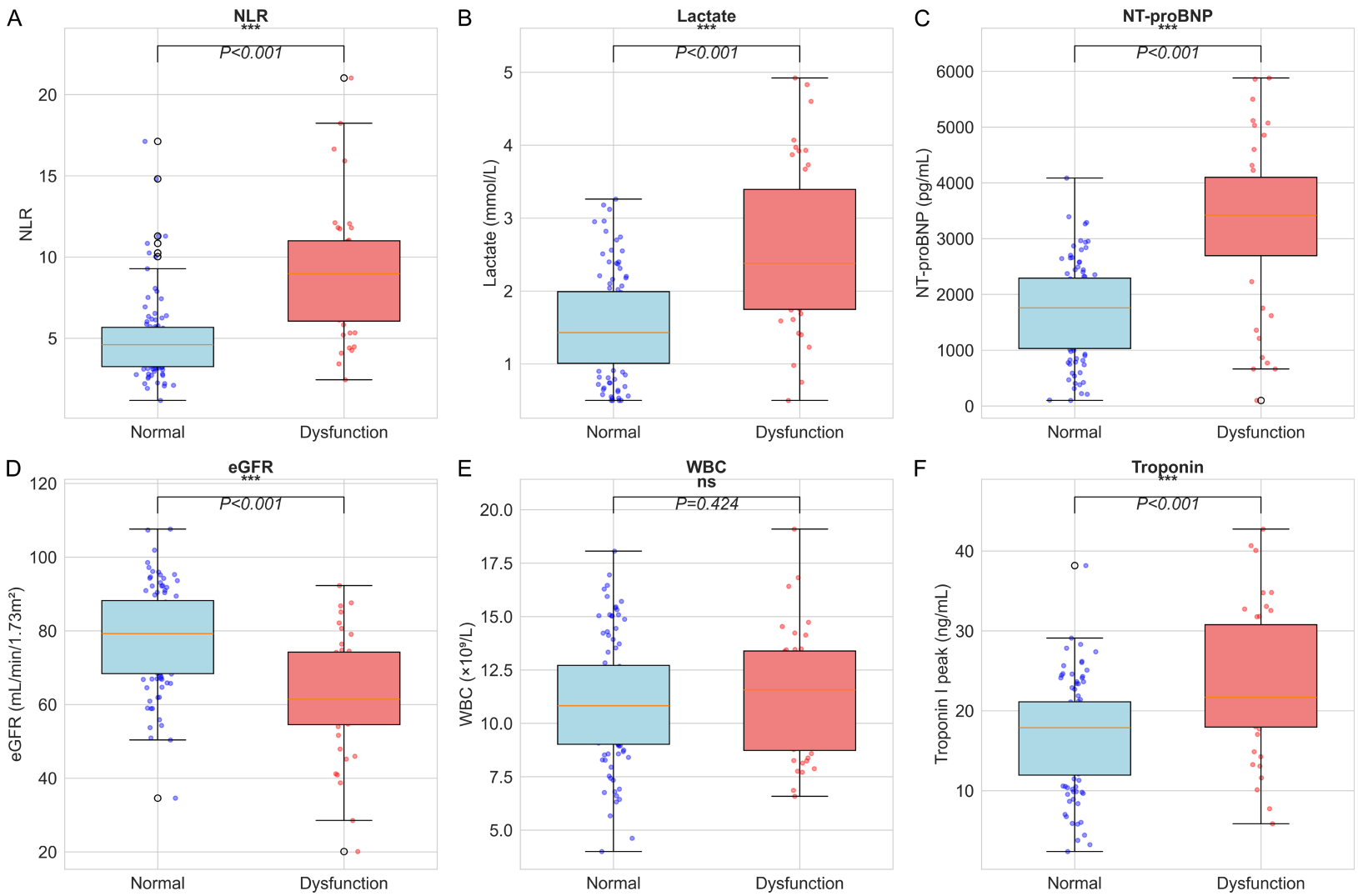

Figure 3. Comparison of laboratory metrics. (A) NLR, (B) lactate, (C) NT-proBNP, (D) eGFR, (E) WBC, (F) troponin I peak. RV dysfunction group exhibited significantly higher NLR, lactate, NT-proBNP, and troponin I (all $P < 0.001$), significantly lower eGFR ($P < 0.001$), and similar WBC count ($P = 0.424$). NLR, neutrophil-to-lymphocyte ratio; NT-proBNP, N-terminal pro-B-type natriuretic peptide; eGFR, estimated glomerular filtration rate; WBC, white blood cell count.

Table 3. Comparison of imaging, electrocardiographic, and treatment characteristics between patients with and without right ventricular dysfunction after acute right coronary artery occlusion reperfusion

Variable	Normal (n = 96)	Dysfunction (n = 40)	U/χ^2	*P* value
Lesion location			2.206	0.332
Proximal	44 (45.8%)	13 (32.5%)		
Mid	33 (34.4%)	16 (40.0%)		
Distal	19 (19.8%)	11 (27.5%)		
Pre-PCI TIMI 0 flow	54 (56.2%)	20 (50.0%)	0.228	0.633
Collateral circulation			1.328	0.723
Grade 0	31 (32.3%)	16 (40.0%)		
Grade 1	24 (25.0%)	11 (27.5%)		
Grade 2	24 (25.0%)	8 (20.0%)		
Grade 3	17 (17.7%)	5 (12.5%)		
Multivessel disease	40 (41.7%)	18 (45.0%)	0.028	0.867
V3R-V5R ST-segment elevation	44 (45.8%)	31 (77.5%)	10.202	0.001
V1 ST-segment elevation	43 (44.8%)	34 (85.0%)	16.984	< 0.001
Complete RBBB	6 (6.2%)	9 (22.5%)	6.032	0.014
Post-PCI ST-segment resolution			0.126	0.939
Complete	57 (59.4%)	23 (57.5%)		
Partial	26 (27.1%)	12 (30.0%)		
None	13 (13.5%)	5 (12.5%)		
Number of stents	1.25 ± 0.48	1.30 ± 0.56	1868.000	0.383
Total stent length (mm)	28.56 ± 9.87	27.34 ± 10.80	1989.000	0.774
Thrombus aspiration	23 (24.0%)	25 (62.5%)	16.717	< 0.001
Vasoactive drugs use	12 (12.5%)	28 (70.0%)	42.238	< 0.001

PCI, percutaneous coronary intervention; TIMI, Thrombolysis in Myocardial Infarction; RBBB, right bundle branch block.

Table 4. Comparison of right ventricular function and hemodynamic parameters between patients with and without right ventricular dysfunction after acute right coronary artery occlusion reperfusion

Variable	Normal (n = 96)	Dysfunction (n = 40)	t/U/χ^2	*P* value
RVFAC (%)	42.57 ± 5.03	27.20 ± 3.78	3840.000	< 0.001
TAPSE (mm)	19.84 ± 3.28	13.74 ± 3.04	3539.500	< 0.001
RV basal diameter (mm)	37.59 ± 3.90	46.64 ± 5.71	319.000	< 0.001
Septal motion abnormality	36 (37.5%)	28 (70.0%)	10.702	0.001
IVC diameter (mm)	17.98 ± 2.89	22.29 ± 3.35	652.000	< 0.001
PASP (mmHg)	35.21 ± 8.54	41.35 ± 8.30	1144.000	< 0.001
Cardiogenic shock	8 (8.3%)	19 (47.5%)	24.816	< 0.001
IABP use	5 (5.2%)	12 (30.0%)	13.681	< 0.001
Temporary pacemaker use	4 (4.2%)	10 (25.0%)	0.130	0.001
Diuretics use	22 (22.9%)	21 (52.5%)	10.102	0.001
Inotropes use	16 (16.7%)	18 (45.0%)	10.625	0.001

RVFAC, right ventricular fractional area change; TAPSE, tricuspid annular plane systolic excursion; RV, right ventricular; IVC, inferior vena cava; PASP, pulmonary artery systolic pressure; IABP, intra-aortic balloon pump.

thereby aggravating the "no-reflow" phenomenon, and hinder functional recovery via a "stunning" effect on viable myocardium. As a simple index integrating the dynamic changes of two key immune cells, an elevated NLR likely signifies a systemic inflammatory milieu that is unfavorable for myocardial repair, providing the systemic "soil" for the development of RVD. Beyond

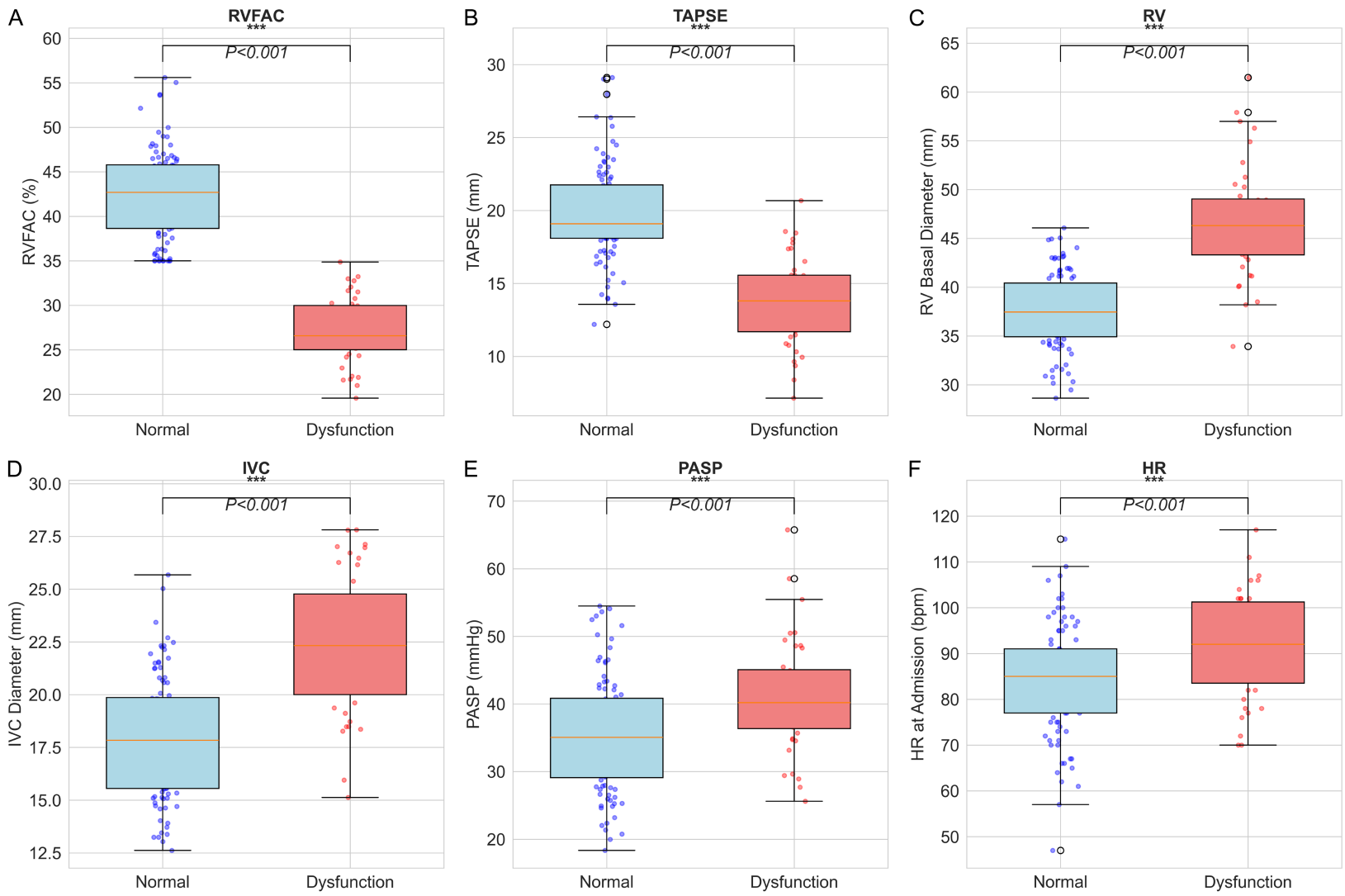

Figure 4. Comparison of echocardiographic and functional metrics. (A) RVFAC, (B) TAPSE, (C) RV basal diameter, (D) IVC diameter, (E) PASP, (F) heart rate at admission. RV dysfunction group had significantly lower RVFAC and TAPSE (both $P < 0.001$), and significantly larger RV basal diameter, IVC diameter, PASP, and higher heart rate (all $P < 0.01$). RVFAC, right ventricular fractional area change; TAPSE, tricuspid annular plane systolic excursion; RV, right ventricular; IVC, inferior vena cava; PASP, pulmonary artery systolic pressure; HR, heart rate.

Table 5. Comparison of clinical outcomes between patients with and without right ventricular dysfunction after acute right coronary artery occlusion reperfusion

Variable	Normal	Dysfunction	t/U/χ^2	*P* value
Cardiac death	1 (1.0%)	4 (10.0%)	0.095	0.026
Cardiogenic shock	8 (8.3%)	19 (47.5%)	24.816	0.039
Malignant arrhythmia	8 (8.3%)	14 (35.0%)	12.906	0.006
Acute kidney injury	5 (5.2%)	12 (30.0%)	13.681	0.061
Repeat revascularization	8 (8.3%)	4 (10.0%)	0.818	0.747
All-cause death	3 (3.1%)	4 (10.0%)	0.290	0.194
HF readmission	12 (12.5%)	12 (30.0%)	4.807	0.028
Discharge NYHA class			21.760	< 0.001
Class 1	45 (46.9%)	4 (10.0%)		
Class 2	43 (44.8%)	25 (62.5%)		
Class 3	8 (8.3%)	9 (22.5%)		
Class 4	0 (0.0%)	2 (5.0%)		

HF, heart failure; NYHA, New York Heart Association.

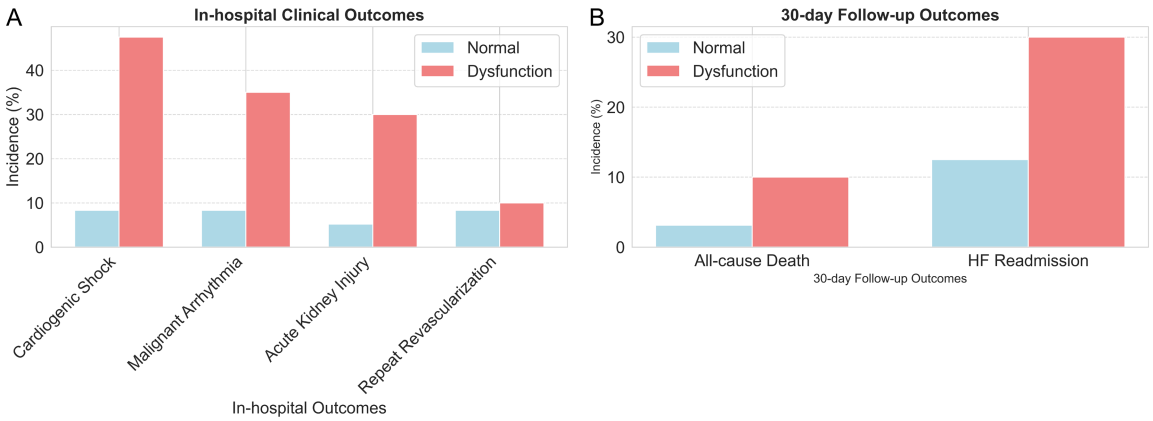

Figure 5. Comparison of clinical outcomes. A. In-hospital outcomes: RV dysfunction group had significantly higher rates of cardiogenic shock, malignant arrhythmias, and acute kidney injury (all P < 0.001). Repeat revascularization rates were similar (P = 0.747). B. 30-day follow-up outcomes: RV dysfunction group had significantly higher HF readmission rate (P = 0.028), while all-cause mortality was not significantly different (P = 0.194). HF, heart failure.

the factors discussed above, the right ventricle is also known to be sensitive to pharmacological interventions; for instance, anesthetic agents have been shown to modulate RV performance in surgical settings [36].

Our findings are consistent with previous literature while also offering new extensions. The consensus lies in our reaffirmation that RV dysfunction is a powerful predictor of poor prognosis in patients with acute inferior MI. The associations between RV dysfunction and in-hospital cardiogenic shock, malignant arrhythmias, and short-term heart failure rehospitalization are in line with the classic studies by Dubey et al. [37] and recent clinical observations. The extension of our study lies in our move beyond single predictors; instead, we constructed a multidimensional risk profile incorporating clinical (shock), laboratory (lactate, NLR), and angiographic (TIMI flow, collaterals) data. Notably, the independent predictive value of high lactate and high NLR - two indicators that are easily and rapidly obtainable in the emergency setting - provides the potential for early identification of high-risk patients, even prior to coronary angiography.

These findings have clear translational implications for clinical practice. For a patient presenting with acute inferior STEMI, if they present with cardiogenic shock, elevated blood lactate,

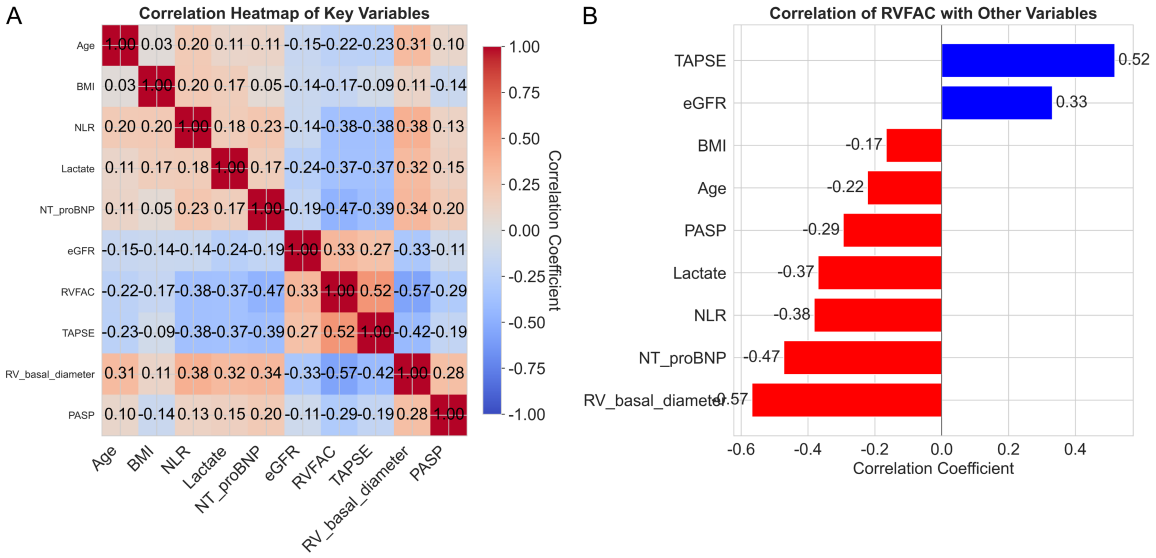

Figure 6. Correlation analysis of key variables. A. Heatmap displaying Pearson correlations among key clinical, laboratory, and echocardiographic variables. B. Bar plot showing correlation coefficients of RVFAC with other variables. RVFAC was strongly positively correlated with TAPSE ($r = 0.52$, $P < 0.001$) and negatively correlated with RV basal diameter, NT-proBNP, lactate, NLR, and PASP (all $P < 0.001$). Age showed a weak negative correlation with RVFAC ($P = 0.011$). NLR correlated positively with RV basal diameter and lactate (both $P < 0.05$). RVFAC, right ventricular fractional area change; TAPSE, tricuspid annular plane systolic excursion; RV, right ventricular; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NLR, neutrophil-to-lymphocyte ratio; PASP, pulmonary artery systolic pressure; BMI, body mass index.

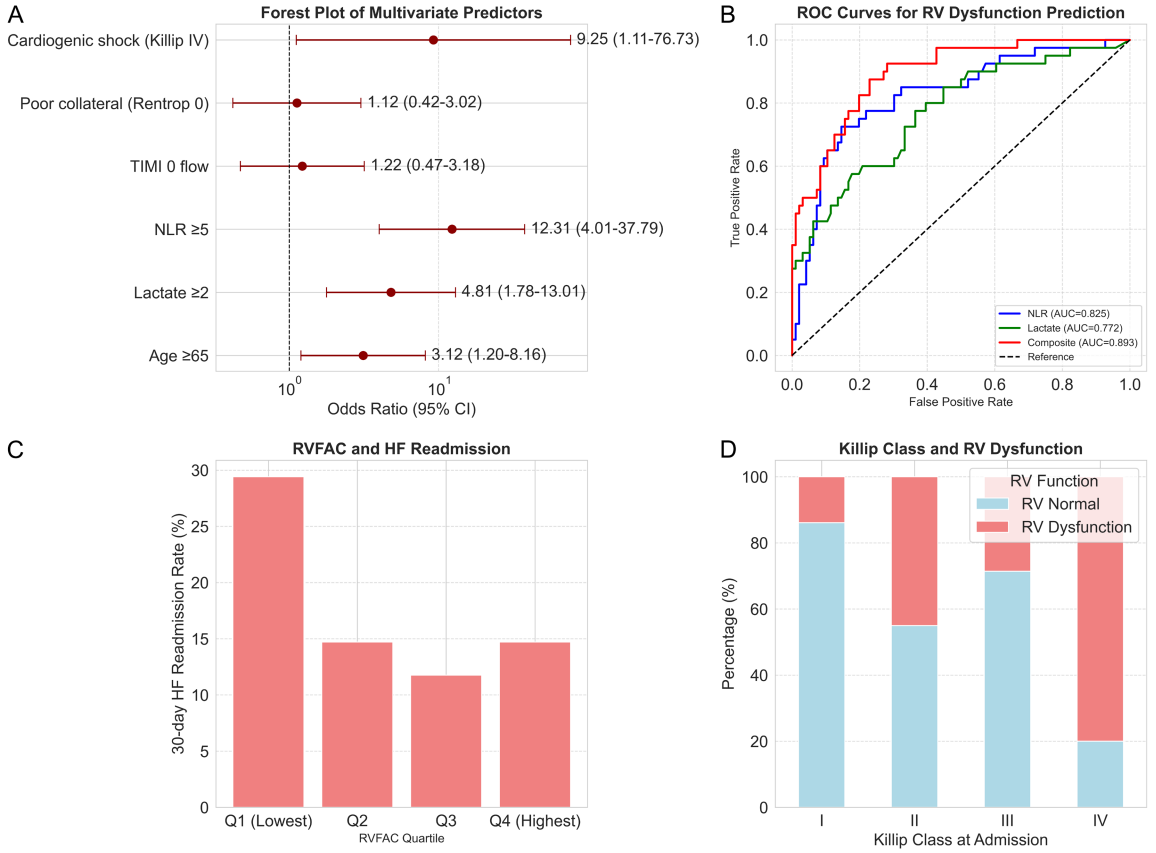

Figure 7. Predictive models and multivariate analysis. A. Forest plot of independent predictors of RV dysfunction from multivariable logistic regression. NLR ≥ 5 (OR = 12.31, 95% CI: 4.01-37.79, P < 0.001), lactate ≥ 2 mmol/L (OR = 4.81, 95% CI: 1.78-13.01, P = 0.002), cardiogenic shock at admission (Killip IV) (OR = 9.25, 95% CI: 1.11-76.73, P = 0.039), and age ≥ 65 years (OR = 3.12, 95% CI: 1.20-8.16, P = 0.020) were significant. B. ROC curves comparing predictive performance of NLR, lactate, and their composite score. The composite score showed the highest AUC (0.893). C. Relationship between RVFAC quartiles and 30-day HF readmission rate, with the lowest quartile having the highest rate (P for trend < 0.05). D. Proportion of RV dysfunction across Killip classes, increasing with higher Killip class (P < 0.001). RV, right ventricular; NLR, neutrophil-to-lymphocyte ratio; HF, heart failure; AUC, area under the curve; ROC, receiver operating characteristic.

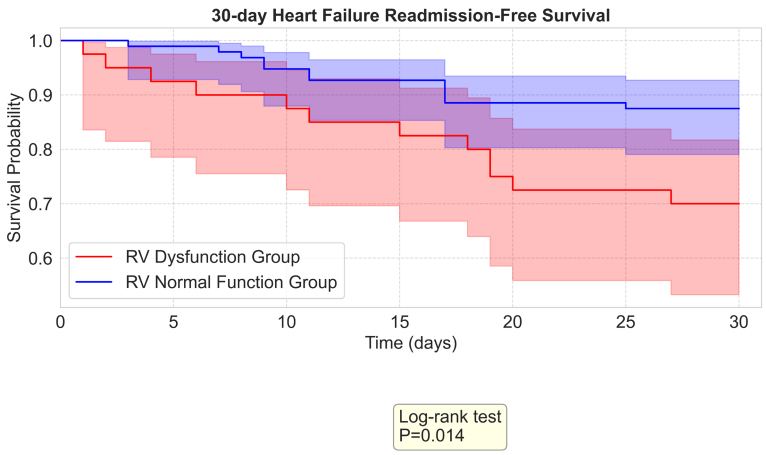

Figure 8. Kaplan-Meier survival analysis. Kaplan-Meier curves for 30-day HF readmission-free survival. Patients with RV dysfunction had significantly lower event-free survival (log-rank P = 0.014). HF, heart failure.

a significantly high NLR, and angiography reveals RCA total occlusion with poor collateral circulation, clinicians should be highly vigilant regarding the risk of persistent RV dysfunction. Such patients should be flagged as "extremely high-risk" and considered for more aggressive management strategies. These may include, but are not limited to: more intensive hemodynamic monitoring (e.g., arterial blood pressure, central venous pressure, or even pulmonary artery catheters), cautious volume management to optimize RV preload, early and potentially prolonged use of inotropic support (e.g., dobutamine) for RV function, and proactive monitoring and prevention of complications such as malignant arrhythmias and acute kidney injury. Our study provides direct evidence supporting such a risk-stratified, individualized precision management approach.

When interpreting our results, several limitations must be acknowledged. First, the single-center, retrospective design may introduce selection bias and cannot establish causality. Second, the limited sample size, particularly the small number of patients in the RV dysfunction group (n = 40), may affect the stability of the multivariate regression model and the power to analyze certain subgroups. Third, the assessment of RV function relied on echocardiographic RVFAC. While this is a commonly used and effective clinical parameter, it is still subject to intra- and inter-observer variability, and may not be as precise as cardiac magnetic resonance (CMR) in providing a comprehensive assessment of RV morphology. Fourth, the follow-up period was short, assessing only 30-day outcomes, thus providing no information on the long-term evolution of RV function (e.g., recovery, late heart failure) or long-term survival. Finally, while this study focused on identifying predictors and their associations, it did not validate whether intervention strategies based on these predictors can truly improve patient outcomes.

Based on the findings and limitations of this study, future research could proceed in the following directions: First, multicenter, prospective, and large-sample validation studies should be conducted to confirm the generalizability of the current predictive model and potentially develop a simple bedside risk scoring system. Second, more precise tools for evaluating right ventricular (RV) function, such as three-dimensional echocardiography or late gadolinium enhancement cardiovascular magnetic resonance (LGE-CMR), should be explored; the latter can simultaneously assess myocardial edema, infarct size, and microvascular obstruction, thereby providing more comprehensive information regarding the structure-function relationship. Third, interventional studies are needed to evaluate whether early implementa-

tion of intensified hemodynamic management strategies (e.g., standardized RV-guided treatment protocols) in the high-risk populations identified in this study can effectively reduce the incidence of RV dysfunction (RVD) and improve both short- and long-term clinical outcomes. Fourth, follow-up duration should be extended to clarify the persistent impact of early RVD on patients' long-term cardiac function and quality of life.

Conclusion

For patients with acute right coronary artery (RCA) occlusion who have undergone successful PCI, the presence of cardiogenic shock, elevated lactate, high NLR upon admission, and angiographic findings of TIMI flow grade 0 with poor collateral circulation are strong predictors of postoperative right ventricular dysfunction (RVD). Such RVD is significantly associated with worse short-term clinical outcomes. In clinical practice, these indicators should be integrated for early risk stratification, enabling individualized intensive management and follow-up for high-risk patients.

Disclosure of conflict of interest

None.

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