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Original Article

A diagnostic prediction model for the early detection of heart failure following primary percutaneous coronary intervention in patients with ST-elevation myocardial infarction

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Abstract: Background: In this study, we aimed to construct a robust diagnostic model that can predict the early onset of heart failure in patients with ST-elevation myocardial infarction (STEMI) following a primary percutaneous coronary intervention (PCI). This diagnostic model can facilitate the early stratification of high-risk patients, thereby optimizing therapeutic management. Methods: We performed a retrospective analysis of 664 patients with STEMI who underwent their inaugural PCI. We performed logistic regression along with optimal subset regression and identified important risk factors associated with the early onset of heart failure during the time of admission. Based on these determinants, we constructed a predictive model and confirmed its diagnostic precision using a receiver operating characteristic (ROC) curve. Results: The logistic and optimal subset regression analyses revealed the following three salient risk factors crucial for the early onset of heart failure: the Killip classification, the presence of renal insufficiency, and increased troponin T levels. The constructed prognostic model exhibited excellent discriminative ability, which was indicated by an area under the curve value of 0.847. The model's 95% confidence interval following 200 Bootstrap iterations was found to be between 0.767 and 0.925. The Hosmer-Lemeshow test revealed a chi-square value of 3.553 and a *p*-value of 0.938. Notably, the calibration of the model remained stable even after 500 Bootstrap evaluations. Furthermore, decision curve analysis revealed a substantial net benefit of the model. Conclusion: We have successfully constructed a diagnostic prediction model to predict the incipient stages of heart failure in patients with STEMI following primary PCI. This diagnostic model can revolutionize patient care, allowing clinicians to quickly identify and create individualized interventions for patients at a higher risk.

Keywords: Diagnostic prediction model, heart failure, primary percutaneous coronary intervention, ST-elevation myocardial infarction, ROC curve

Introduction

ST-segment elevation myocardial infarction (STEMI) is a prevalent complication of coronary heart disease, with high incidence and mortality rates [1]. The diagnosis of STEMI mainly depends on the changes in electrocardiogram (ECG) and increased levels of cardiac biomarkers. Furthermore, STEMI is an important medical emergency. Due to the recent advancements in medical technology, percutaneous coronary intervention (PCI) has become the gold standard treatment for STEMI [2] mainly due to its ability to quickly restore coronary circulation and alleviate myocardial damage. Adjunctive therapies, such as antiplatelet and beta-blockers, can complement PCI in improving patient outcomes.

Nevertheless, even with the widespread use of PCI, many patients with STEMI may still face the risk of heart failure, which can potentially wors-

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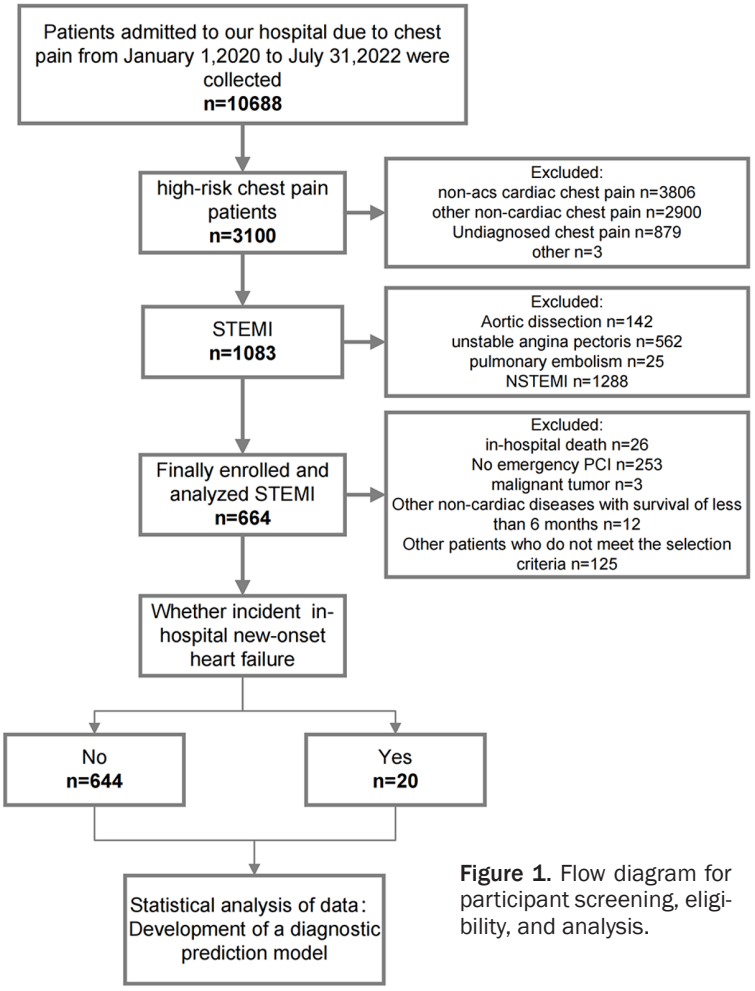

Figure 1. Flow diagram for participant screening, eligibility, and analysis.

en their prognosis and overall quality of life [3, 4]. This highlights the variability in long-term outcomes for such patients, which is influenced by different factors including the extent of myocardial damage and the timing of treatment. Early and quick identification of patients with STEMI at an increased risk of heart failure, followed by appropriate intervention, is important to improve survival outcomes and overall quality of life [5-8]. Major prognostic indicators, such as left ventricular function and arrhythmia occurrence, are important in evaluating these risks.

Based on the aforementioned concerns, reliable diagnostic and prognostic tools are warranted for evaluating the risk of heart failure in patients with STEMI post-PCI. Although many models are available and are in active use, their precision and broad applicability are questionable [9-11].

In this study, we aimed to construct an innovative and highly accurate predictive algorithm individualized for patients with STEMI undergoing PCI. We hope that this model will be a robust tool for early risk stratification, allowing clinicians to establish more individualized and quick therapeutic interventions.

Methods

Study design and participants

We conducted a retrospective analysis by examining the medical records of 664 patients diagnosed with STEMI. These patients underwent their first PCI at the Cardiology Department of Xiangtan Central Hospital between January 1, 2020, and July 31, 2022 (**Figure 1**). The inclusion criteria were as follows: patients older than 18 years of age with a definitive diagnosis of STEMI [1], no previous PCI procedures, and a complete set of medical records. The exclusion criteria were as follows: patients with other severe cardiovascular complications, missing clinical and laboratory documentation, or a history of surgical procedures on coronary arteries.

Data collection

Patient data were obtained from both the integrated electronic health record system of the hospital and the China Chest Pain Center data platform. The obtained data included the following patient information: demographics, antecedent medical history prior to admission, levels of salient biochemical markers during the time of admission, pharmacological interventions, and relevant details regarding PCI.

Follow-up and outcome events

All study participants were followed continuously until January 31, 2023. A multidisciplinary team was set up, including five experienced

cardiologists and two specialized nurses. Data on patient outcomes were collected via a combination of outpatient consultations, telephone follow-ups, and community-based outreach visits. The primary outcome metric focused on the emergence of heart failure symptoms during the patient's hospital stay. This was clinically defined by the onset of new symptomatic manifestations, clinical signs, and pertinent diagnostic changes, indicating heart failure.

Ethics and informed consent

This study was approved by the Ethics Committee of Xiangtan Central Hospital, Xiangtan, China (Reference number: 2023-02-001). This study was performed in accordance with the ethical principles of the Declaration of Helsinki. Because this is a retrospective study that only included clinical data of the patients and did not affect their treatment outcomes, the requirement of informed consent was waived.

Statistical analysis

Baseline characteristics and analysis: Data categorization was performed using the *CompareGroups* package. Based on data distribution, intergroup variances were evaluated by performing the independent *t*-test, chi-square test, or Mann-Whitney U test. The normality of continuous variables was determined by performing the Kolmogorov-Smirnov test. Normally distributed continuous variables are presented as mean ± standard deviation, whereas skewed distributions are presented as median with interquartile range. Furthermore, categorical variables are presented as n (%).

Identifying risk factors associated with heart failure during hospital admission: The *glm* package was used for multivariate logistic regression, and the *bestglm* package was used for subset selection.

Construction and evaluation of the model: Selected variables were integrated into the analytical model. Diagnostic efficacy was estimated using receiver operating characteristic (ROC) curves from the *pROC* package. Model calibration was performed on the *calibrate* and *val.prob* functions of the *rms* package, which was confirmed by performing the Hosmer-Lemeshow test using the *HLtest* package. Bootstrap replicates were used for internal validation.

Net benefit and analysis of model performance: The *dca.R* package was used to determine net benefits by performing decision curve analysis (DCA). Diagnostic nomograms for the quick identification of in-hospital heart failure were established using the *rms* package.

Comparative analysis of models: The structural integrity of models resulting from logistic and optimal subset regressions was compared with those developed using the *glmnet* package by performing the LASSO regression analysis (min and 1se criteria). Evaluation of ROC curve models was performed using the *ROCR* package, with DCA contrasts managed through *Dcurves*.

All statistical tests were two-sided, with a significance level set at $P < 0.05$. These methodologies support our evaluation of model construction, discriminative potency, and calibration ability. All computations were performed using R software (Version 4.2.0; http://www.R-project.org).

Results

Baseline characteristics

This study included 664 patients with STEMI, of whom 20 (3.0%) patients developed new-onset heart failure. Male patients comprised 77.6% of the study population; however, in the heart failure subset, their representation slightly decreased to 70.0% compared with 77.8% in the non-heart failure subset ($P = 0.417$). Patients in the heart failure subset had a mean age of 70.0 years, which was significantly older than those in the non-heart failure subset ($P = 0.024$). Furthermore, renal impairment was significantly higher in the heart failure subset (50.0%) compared with 13.7% in the non-heart failure subset ($P < 0.001$). Regarding the Killip classification, 55.0% of patients in the heart failure subset were categorized as class IV, which was only 12.1% in the non-heart failure subset ($P < 0.001$). Increased levels of biochemical markers, especially NT-proBNP and troponin T (TNT), were discernible in the heart failure subset ($P < 0.001$). Regarding PPCI determinants, 55.0% of patients in the heart failure subset predominantly exhibited the involvement of the left main artery, which was

Table 1. Baseline characteristics for the development of the diagnostic model for in-hospital new-onset heart failure in STEMI patients undergoing PPCI

	[Total Patients] N = 664	In-Hospital New-Onset Heart Failure		*P-value*
		[No] N = 644	[Yes] N = 20	
Demographics				
Male, N (%)	515 (77.6%)	501 (77.8%)	14 (70.0%)	0.417
Age, years	63.0 [55.0; 71.0]	63.0 [54.8; 71.0]	70.0 [62.0; 76.8]	0.024
Obesity	184 (27.7%)	180 (28.0%)	4 (20.0%)	0.597
Medical history, N (%)				
Anemia	115 (17.3%)	109 (16.9%)	6 (30.0%)	0.135
Hyperlipidemia	253 (38.1%)	247 (38.4%)	6 (30.0%)	0.600
Hypertension	382 (57.5%)	371 (57.6%)	11 (55.0%)	0.998
Atrial fibrillation	52 (7.83%)	48 (7.45%)	4 (20.0%)	0.063
Diabetes	186 (28.0%)	180 (28.0%)	6 (30.0%)	1.000
Stroke	86 (13.0%)	82 (12.7%)	4 (20.0%)	0.313
Heart valve disease	103 (15.5%)	99 (15.4%)	4 (20.0%)	0.533
Cardiomyopathy	26 (3.92%)	25 (3.88%)	1 (5.00%)	0.555
COPD	87 (13.1%)	84 (13.0%)	3 (15.0%)	0.737
Renal insufficiency	98 (14.8%)	88 (13.7%)	10 (50.0%)	< 0.001
Clinical conditions at admission				
Killip Classification				< 0.001
I	402 (60.5%)	399 (62.0%)	3 (15.0%)	
II	158 (23.8%)	153 (23.8%)	5 (25.0%)	
III	15 (2.26%)	14 (2.17%)	1 (5.00%)	
IV	89 (13.4%)	78 (12.1%)	11 (55.0%)	
Hemoglobin, g/dL	135 [124; 146]	135 [124; 147]	133 [128; 143]	0.700
NT-proBNP/100, pg/ml	5.68 [1.33; 17.6]	5.60 [1.27; 16.9]	33.3 [11.0; 79.6]	< 0.001
TnT, ng/mL	4.68 [1.64; 8.73]	4.63 [1.57; 8.46]	10.0 [4.19; 10.0]	< 0.001
C-reactive protein, mg/L	19.6 [5.02; 55.4]	19.0 [5.01; 54.0]	43.5 [15.4; 96.9]	0.040
Sodium, mmol/L	137 [135; 139]	137 [135; 139]	137 [135; 140]	0.609
Uric acid, mg/dL	331 [271; 395]	331 [270; 394]	342 [280; 412]	0.484
Creatinine, mg/dL	76.0 [64.0; 91.0]	76.0 [64.0; 91.0]	77.5 [65.5; 103]	0.887
LVEF, %	50.0 [46.0; 57.0]	50.0 [46.0; 57.0]	48.0 [43.2; 51.2]	0.044
PPCI related situation				
Main diseased vessel				0.001
LAD	311 (46.8%)	303 (47.0%)	8 (40.0%)	
LCX	65 (9.79%)	63 (9.78%)	2 (10.0%)	
RCA	282 (42.5%)	275 (42.7%)	7 (35.0%)	
LM	6 (0.90%)	3 (0.47%)	3 (15.0%)	
Stenosis degree				0.935
90-99%	188 (28.3%)	183 (28.4%)	5 (25.0%)	
100%	476 (71.7%)	461 (71.6%)	15 (75.0%)	
Preoperative TIMI				0.582
0	478 (72.0%)	463 (71.9%)	15 (75.0%)	
1	24 (3.61%)	24 (3.73%)	0 (0.00%)	
2	107 (16.1%)	105 (16.3%)	2 (10.0%)	
3	55 (8.28%)	52 (8.07%)	3 (15.0%)	
Complication				
Shock	40 (6.02%)	33 (5.12%)	7 (35.0%)	< 0.001
Infect	103 (15.5%)	98 (15.2%)	5 (25.0%)	0.218

CPC quality control index, min				
D to B	66.0 [50.0; 84.0]	66.0 [50.0; 84.0]	66.0 [56.8; 83.2]	0.520
Total ischemic time	252 [171; 431]	252 [171; 429]	300 [176; 483]	0.700
CL activation time	14.0 [5.00; 20.0]	14.0 [5.00; 20.0]	16.0 [4.75; 22.2]	0.532

Categorical variables were presented as n (%). Values for continuous variables are given as means ± SD or medians with interquartile ranges. Abbreviations: STEMI: ST-segment elevation myocardial infarction; COPD: Chronic Obstructive Pulmonary Disease; NT-proBNP: N-terminal pro-B type natriureti peptide; TnT: Troponin T; LVEF: left ventricular ejection fraction; PPCI: primary percutaneous coronary intervention; LAD: left anterior descending artery; LCX: left circumflex artery; RCA: right coronary arter; LM: Left Main; TIMI: thrombolysis in myocardial infarction; CPC: chest pain center; D-to-B: door-to-balloon; CL: catheter lab.

significantly different from those in the non-heart failure subset (P = 0.001). The incidence of shock in the heart failure subset was 35.0%, which was significantly higher than the 5.12% observed in the non-heart failure subset (P < 0.001). No significant difference was observed between the groups in terms of the Door-to-Balloon (D2B) duration, overall ischemic time, and CL activation period (**Table 1**).

Univariate logistic regression analysis

After evaluating the included characteristics, the following variables exhibited a robust association with in-hospital new-onset heart failure: Killip classification (OR: 2.478, 95% CI: 1.74-3.642, P < 0.001), renal dysfunction (OR: 6.318, 95% CI: 2.524-15.82, P < 0.001), onset of shock (OR: 9.97, 95% CI: 3.546-26.09, P < 0.001), levels of C-reactive protein (OR: 1.01, 95% CI: 1.004-1.016, P = 0.001), TNT (OR: 1.258, 95% CI: 1.097-1.473, P = 0.002), NT-proBNP/100 (OR: 1.009, 95% CI: 1.003-1.015, P = 0.002), age (OR: 1.047, 95% CI: 1.007-1.091, P = 0.024), left ventricular ejection fraction (OR: 0.954, 95% CI: 0.914-0.999, P = 0.037), and the occurrence of atrial fibrillation (OR: 3.104, 95% CI: 0.864-8.864, P = 0.050).

Many variables, including anemia, predominant lesion vessels, presence of infections, uric acid levels, duration of CL activation, incidence of stroke, gender, obesity status, hyperlipidemia, cardiac valvular abnormalities, sodium concentration, D2B duration, extent of stenosis, hemoglobin concentration, chronic obstructive pulmonary disease, cardiomyopathy, hypertension, diabetes, antecedent thrombolysis in MI score, cumulative ischemic duration, and serum creatinine, were not significantly associated with the emergence of in-hospital heart failure (**Table 2**).

LASSO regression analysis

Through the LASSO regression model, we found the following seven salient predictors associated with the risk of in-hospital heart failure in patients with STEMI undergoing PPCI: age, renal insufficiency, shock, Killip classification, TNT, C-reactive protein, and NT-proBNP/100 (**Figure 2**). A congruous outcome was obtained from the differential analysis (Supplementary Table 1).

Multivariable logistic and optimal subset regression analyses

Based on the variables identified by the LASSO regression, our multivariable logistic regression analysis revealed that the following factors are significantly relevant to the onset of heart failure in patients with STEMI post-PPCI (**Table 3**): Killip classification: OR: 2.061, 95% CI: 1.4-3.106, P < 0.001; coefficient = 0.7232. Renal insufficiency: OR: 2.796, 95% CI: 1.026-7.549, P = 0.041; coefficient = 1.0281. TNT: OR: 1.161, 95% CI: 1.01-1.361, P = 0.046; coefficient = 0.1493. For the intercept: OR: 0.002, 95% CI: 0-0.007, P < 0.001; coefficient = -6.3528.

The findings obtained by the optimal subset regression are consistent with those obtained by the multivariable logistic regression, highlighting the identical three variables (Supplementary Table 2).

In the derived model, three determinants identified from multivariable logistic regression and best subset regression were integrated. The ROC of the model yielded an area under the curve (AUC) of 0.847 (95% CI: 0.767-0.925; **Figure 3A**). **Figure 3C** shows an improved AUC for the comprehensive model compared with that for the individual variables. DCA revealed the net benefits of the model (**Figure 3B**). As

Table 2. Univariate logistic regression analysis of in-hospital new-onset heart failure in STEMI patients receiving PPCI

Characteristics	SE	OR (95% CI)	Z	*P-value*
Killip Classification	0.18569	2.478 (1.74-3.642)	4.886	**< 0.001**
Renal insufficiency	0.46166	6.318 (2.524-15.82)	3.993	**< 0.001**
Shock	0.50171	9.97 (3.546-26.09)	4.583	**< 0.001**
C-reactive protein	0.00310	1.01 (1.004-1.016)	3.279	**0.001**
TNT	0.07403	1.258 (1.097-1.473)	3.096	**0.002**
NT-proBNP/100	0.00292	1.009 (1.003-1.015)	3.117	**0.002**
Age	0.02027	1.047 (1.007-1.091)	2.259	**0.024**
LVEF	0.02259	0.954 (0.914-0.999)	-2.090	**0.037**
Atrial fibrillation	0.57880	3.104 (0.864-8.864)	1.957	0.050
Anemia	0.49914	2.104 (0.731-5.37)	1.490	0.136
Main diseased vessel	0.24058	1.362 (0.856-2.227)	1.283	0.199
Infect	0.52792	1.857 (0.593-4.917)	1.173	0.241
Uric acid	0.00210	1.002 (0.998-1.006)	1.123	0.261
CL activation time	0.01824	1.017 (0.977-1.051)	0.943	0.346
Stroke	0.57138	1.713 (0.482-4.807)	0.942	0.346
Sex	0.49707	0.666 (0.262-1.909)	-0.818	0.414
Obesity	0.56586	0.644 (0.183-1.785)	-0.776	0.437
Hyperlipidemia	0.49463	0.689 (0.241-1.741)	-0.754	0.451
Heart valve disease	0.56959	1.376 (0.388-3.845)	0.561	0.575
Sodium	0.06764	0.963 (0.844-1.1)	-0.553	0.580
D to B	0.00822	1.003 (0.986-1.018)	0.345	0.730
Stenosis degree	0.52373	1.191 (0.454-3.704)	0.334	0.739
Hemoglobin	0.01284	0.996 (0.972-1.022)	-0.290	0.772
COPD	0.63706	1.176 (0.271-3.595)	0.255	0.799
Cardiomyopathy	1.04606	1.303 (0.071-6.686)	0.253	0.800
Hypertension	0.45648	0.899 (0.367-2.26)	-0.232	0.816
Diabetes	0.49579	1.105 (0.386-2.8)	0.201	0.841
Preoperative TIMI	0.21532	1.042 (0.653-1.546)	0.190	0.849
Total ischemic time	0.00046	1 (0.999-1.001)	-0.111	0.912
Creatinine	0.00334	1 (0.988-1.004)	0.004	0.997

Bold represent significant values (P < 0.05). Abbreviations: SE: Standard Error; OR: Odds Ratio; CI: Confidence Interval; Z: Z-Score or Z-Value; Other abbreviations can be found in **Table 1**.

shown in **Figure 3D**, a sentiment echoed where the integrated model surpassed singular variables in net benefits. These analyses were confirmed by 200 iterations of bootstrap resampling.

Consistency was observed in the calibration curve after 500 iterations of bootstrap resampling, signifying the robustness of the model (**Figure 4**). Further validation with the Hosmer-Lemeshow test showed a chi-square value of 3.552964 and a *p*-value of 0.9383024. Concurrently, a *p*-value exceeding 0.05 suggests a satisfactory model fit.

The plots scores attributed to the three prognostic variables are shown in **Figure 5**. We found that increasing scores are associated with increased risks of in-hospital heart failure. As shown in **Figure 6**, the models derived from multivariate logistic regression and the best subset regression were compared with those derived from LASSO regression. Model A, representing the 1se criterion of the LASSO regression, only incorporates the Killip classification and has an AUC of 0.791. Model B, generated from both multivariate logistic regression and the best subset regression, includes the following variables: Killip classification, renal insuffi-

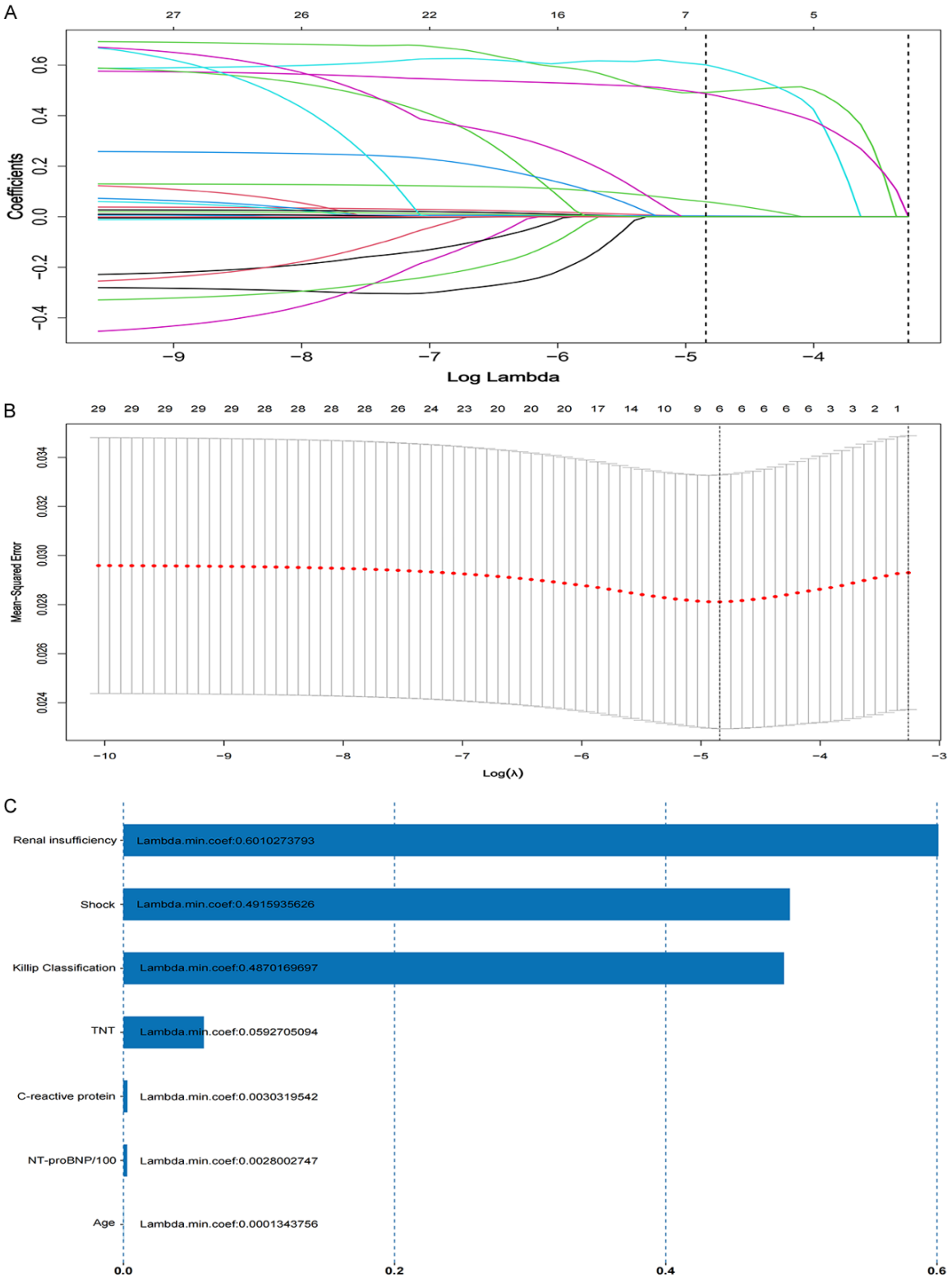

Figure 2. LASSO regression coefficient path and CV LASSO regression coefficient path. A: LASSO regression coefficient path. B: CV LASSO regression coefficient path. C: LASSO regression's min criterion.

ciency, and TNT, with an AUC of 0.847. Model C, generated from the min criterion of LASSO regression, includes all the following seven variables: Killip classification, renal insufficiency,

Table 3. Multivariate logistic regression analysis of in-hospital new-onset heart failure in STEMI patients receiving PPCI

Characteristics	SE	OR (95% CI)	Z	*P*-value	Coefficients
Killip Classification	0.20080	2.061 (1.4-3.106)	3.602	< 0.001	0.7232
Renal insufficiency	0.50389	2.796 (1.026-7.549)	2.040	0.041	1.0281
TNT	0.07476	1.161 (1.01-1.361)	1.997	0.046	0.1493
(Intercept)	0.75716	0.002 (0-0.007)	-8.390	< 0.001	-6.3528

Call: glm (formula = In-hospital new-onset heart failure~Renal insufficiency + Killip Classification + TNT, family = binomial(logit), data = dfglm). Degrees of Freedom: 663 Total (i.e. Null); 660 Residual. Null Deviance: 179.5. Residual Deviance: 144.2. Abbreviations: Abbreviations can be found in **Tables 1** and **2**.

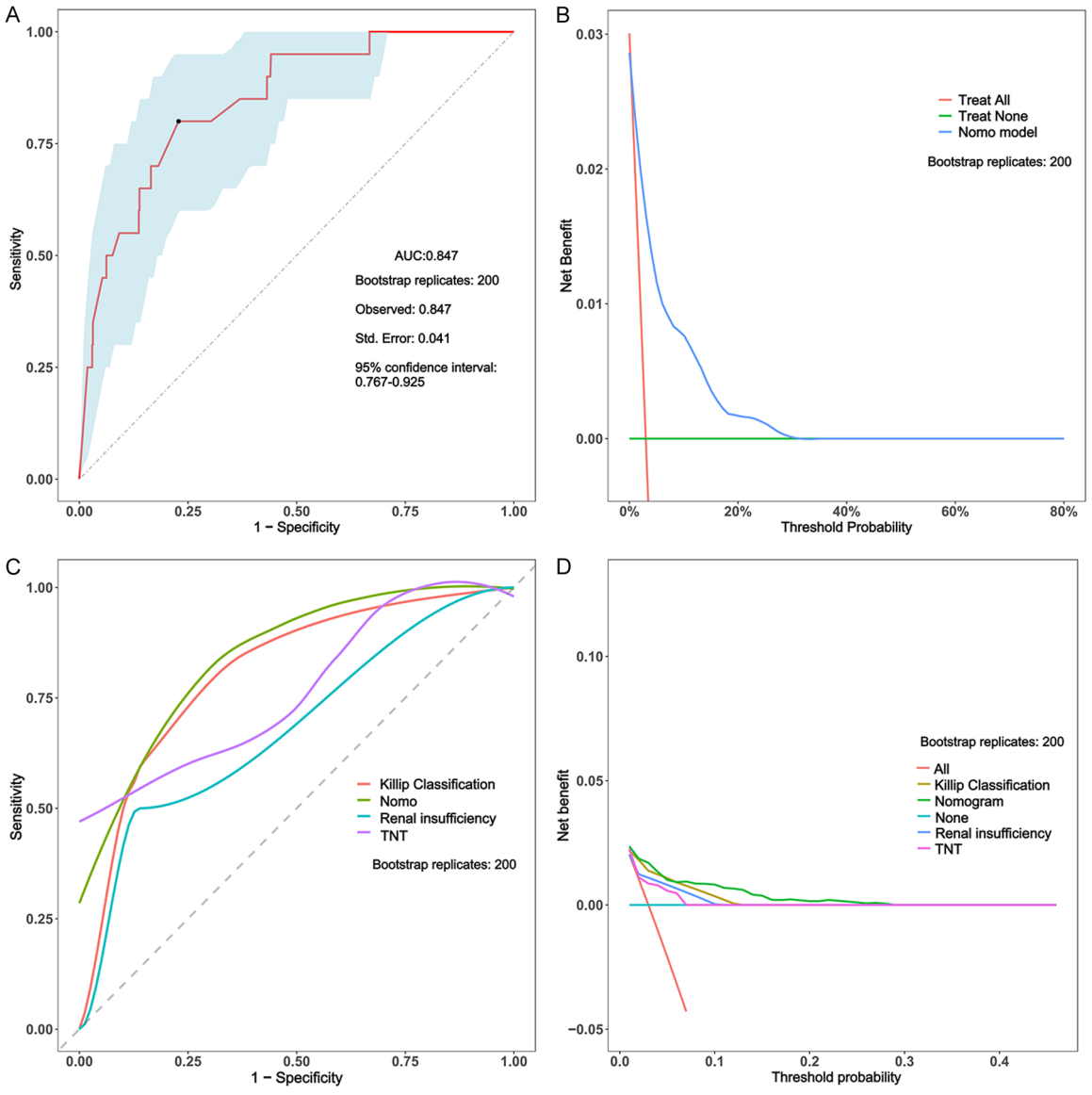

Figure 3. Model development, evaluation, net benefit, and performance analysis. A: Area under the ROC curve of the constructed model. B: Net benefit of the established model: Decision Curve Analysis (DCA). C: Comparison of AUC between the constructed model and individual variables. D: Comparison of net benefit between the constructed model and individual variables: Decision Curve Analysis (DCA).

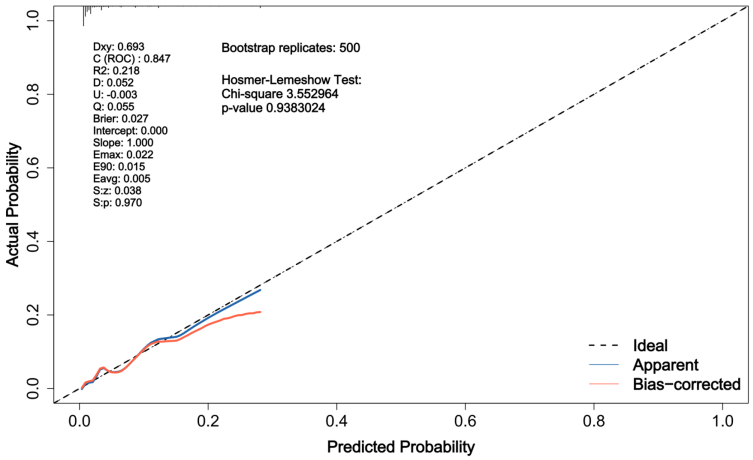

Figure 4. Calibration curves.

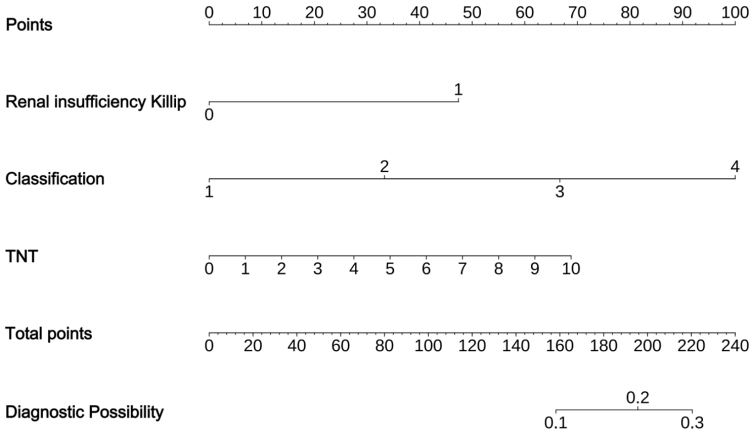

Figure 5. Nomogram for in-hospital onset of heart failure diagnostic prediction.

TNT, shock, C-reactive protein, NT-proBNP/100, and age, with an AUC of 0.849. **Figure 6A** and **6B** show that Model B surpasses Model A in terms of AUC and DCA metrics. Furthermore, Model C did not exhibit any distinct difference from Model B, and Model B was notably more simplified and convenient compared with Model C. As shown in **Figure 6C**, the calibration curve further indicates the stability of the models.

Discussion

In our recent study, we thoroughly evaluated the predictors of new-onset heart failure in patients with STEMI following primary PCI. We performed both logistic and optimal subset regression analyses and identified the following three important predictive variables: the Killip classification, renal insufficiency, and TNT levels. The model we constructed exhibited strong discriminatory capability, with an AUC value of 0.847. A Hosmer-Lemeshow test result with a *p*-value > 0.05 suggests a satisfactory fit of our model.

Our findings regarding the risk factors post-MI heart failure are consistent with the existing literature and also provide novel insights. The Killip classification has been recognized as a crucial prognostic tool for post-MI outcomes. This classification was first introduced by Killip and Kimball, which plays an important role in predicting adverse outcomes post-MI [12]. Consistent with this, studies by Sathvik and Sasaki et al. further focused on the importance of Killip classification in STEMI prognosis [13, 14].

The role of renal insufficiency in predicting heart failure after STEMI is of immense significance. Notably, Schefold et al. reported a strong association between renal insufficiency and the incidence and prognosis of heart failure [15], which is consistent with the recent findings of Beldhuis et al. [16].

TNT is considered a hallmark biomarker of myocardial injury. TNT levels can reveal the risk of heart failure post-MI. Jaffe et al. delved deeply into the association between TNT and heart failure in their seminal work [17], revealing the importance of evaluating the levels of TNT for the diagnosis, management, and prognosis of heart failure. Many studies have further confirmed the significance of TNT in patients with heart failure [18, 19].

Furthermore, while our study is consistent with many prognostic models for post-MI heart failure in some aspects [9, 10, 20], our model integrates innovative variables and amalgamations, thus improving the predictive accuracy of heart failure in the aftermath of an MI.

In the therapeutic paradigm, the diligent early use of specific agents, such as β-blockers,

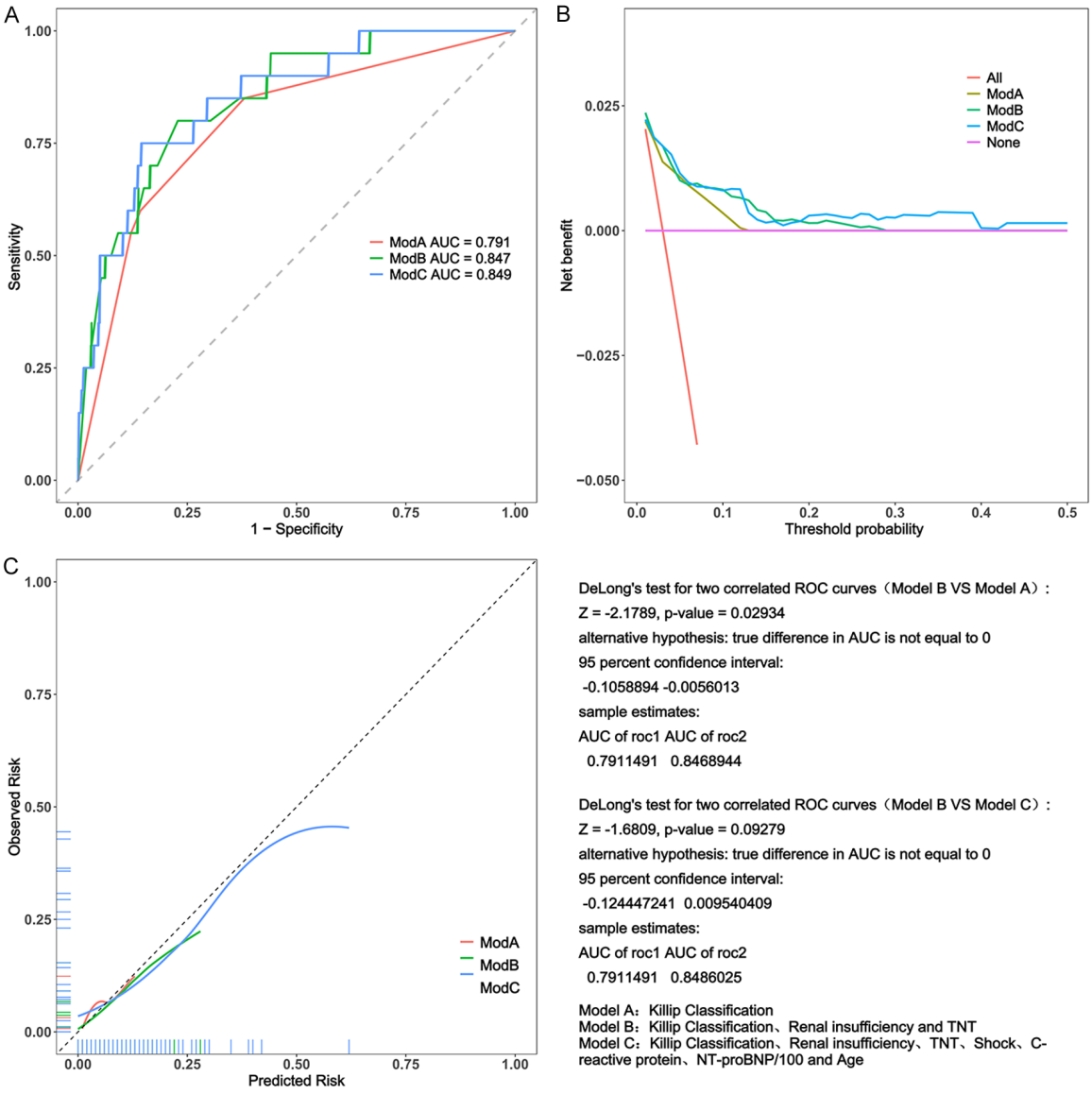

Figure 6. Comparative analysis of multiple models. A: Comparison of AUC among three models. B: Comparison of DCA among three models. C: Comparison of calibration curves among three models.

ACEIs, ARBs, ARNIs, and statins, can notably mitigate the incidence and subsequent prognosis of heart failure following MI [5-8, 21-23]. These findings support our consolidated therapeutic directives.

Limitations

Geographical limitations: The cohort included in this study primarily belonged to a distinct geographical region. Hence, this regional constraint may not include the larger variability of global populations, potentially limiting the universality of the model.

Retrospective design: Due to the retrospective nature of the study, inherent selection biases may have occurred, especially in obtaining antecedent medical data and important patient information.

Data comprehensiveness: Even though the model included a large variety of variables, some influential factors may not have been included that can affect its accuracy.

Variability in treatment protocols: The heterogeneity in therapeutic interventions among patients may contribute to the differential risk outcomes of post-MI heart failure.

Validation limitation: The model did not undergo external validation. Limitations due to the number of positive outcomes impeded stratified validation. Therefore, we mainly depended on iterative bootstrap resampling for internal validation.

Future prospects

Considering the increasing prevalence of MI and consequential heart failure, stringent exploration in this domain remains crucial. Therefore, we propose the following research trajectories:

Collaborative multicentric studies: Engaging in cooperative endeavors with diverse healthcare institutions can facilitate the acquisition and analysis of highly expansive and varied datasets, which can further improve the robustness and external validity of the model.

Elaboration on risk determinants: Understanding the lifestyle factors and genomic determinants associated with heart failure due to post-MI can streamline the predictive efficacy of such models.

Technological integration: Leveraging advanced machine learning modalities can help yield deeper insights from the available datasets, revealing a large number of latent risk determinants.

Exploration of therapeutic modalities: Innovative therapeutic interventions or pharmacological agents can improve the clinical outcomes of patients with heart failure post-MI.

Conclusion

In this study, we successfully constructed an efficacious and accurate model predicting the onset of heart failure in patients who underwent primary PCI due to STEMI. Our model mainly functions on the following three important risk factors: the Killip Classification, renal insufficiency, and TNT. The establishment of this predictive model holds immense clinical significance, allowing the quick identification and assessment of high-risk patients and ensuring their timely and personalized treatment.

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Disclosure of conflict of interest

None.

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Supplementary Table 1. Differential analysis of in-hospital new onset heart failure incidence in STEMI patients receiving PPCI

	[Total Patients] N = 664	In-Hospital New-Onset Heart Failure		OR (95% CI)	*P-value*
		[No] N = 644	[Yes] N = 20		
Sex	0.78 (0.42)	0.78 (0.42)	0.70 (0.47)	0.67 [0.25; 1.76]	0.472
Age	62.3 (12.1)	62.1 (12.0)	68.4 (12.1)	1.05 [1.01; 1.09]	0.034
Obesity	0.28 (0.45)	0.28 (0.45)	0.20 (0.41)	0.64 [0.21; 1.95]	0.405
Anemia	0.17 (0.38)	0.17 (0.38)	0.30 (0.47)	2.10 [0.79; 5.60]	0.233
Shock	0.06 (0.24)	0.05 (0.22)	0.35 (0.49)	9.97 [3.73; 26.7]	0.013
Infect	0.16 (0.36)	0.15 (0.36)	0.25 (0.44)	1.86 [0.66; 5.23]	0.341
Hyperlipidemia	0.38 (0.49)	0.38 (0.49)	0.30 (0.47)	0.69 [0.26; 1.82]	0.443
Hypertension	0.58 (0.49)	0.58 (0.49)	0.55 (0.51)	0.90 [0.37; 2.20]	0.824
Atrial fibrillation	0.08 (0.27)	0.07 (0.26)	0.20 (0.41)	3.10 [1.00; 9.65]	0.190
Diabetes	0.28 (0.45)	0.28 (0.45)	0.30 (0.47)	1.10 [0.42; 2.92]	0.849
Stroke	0.13 (0.34)	0.13 (0.33)	0.20 (0.41)	1.71 [0.56; 5.25]	0.442
Heart valve disease	0.16 (0.36)	0.15 (0.36)	0.20 (0.41)	1.38 [0.45; 4.20]	0.624
Cardiomyopathy	0.04 (0.19)	0.04 (0.19)	0.05 (0.22)	1.30 [0.17; 10.1]	0.827
COPD	0.13 (0.34)	0.13 (0.34)	0.15 (0.37)	1.18 [0.34; 4.10]	0.816
Renal insufficiency	0.15 (0.35)	0.14 (0.34)	0.50 (0.51)	6.32 [2.56; 15.6]	0.005
Killip Classification	1.69 (1.03)	1.64 (1.00)	3.00 (1.21)	2.48 [1.72; 3.57]	<0.001
Hemoglobin	135 (17.5)	135 (17.5)	134 (17.4)	1.00 [0.97; 1.02]	0.774
NT-proBNP/100	17.3 (35.7)	16.4 (35.1)	47.3 (42.4)	1.01 [1.00; 1.01]	0.004
TNT	4.97 (3.57)	4.89 (3.55)	7.60 (3.26)	1.26 [1.09; 1.45]	0.002
C-reactive protein	37.3 (46.3)	36.2 (44.7)	73.4 (77.1)	1.01 [1.00; 1.02]	0.045
Sodium	137 (3.35)	137 (3.35)	137 (3.61)	0.96 [0.84; 1.10]	0.613
Uric acid	341 (98.3)	340 (97.1)	365 (134)	1.00 [1.00; 1.01]	0.415
Creatinine	85.8 (67.7)	85.8 (68.4)	85.9 (40.1)	1.00 [0.99; 1.01]	0.994
LVEF	50.8 (8.84)	51.0 (8.77)	46.8 (10.2)	0.95 [0.91; 1.00]	0.083
Main diseased vessel	1.97 (0.96)	1.97 (0.96)	2.25 (1.16)	1.36 [0.85; 2.18]	0.293
Stenosis degree	0.72 (0.45)	0.72 (0.45)	0.75 (0.44)	1.19 [0.43; 3.32]	0.738
Preoperative TIMI	0.61 (1.03)	0.61 (1.03)	0.65 (1.18)	1.04 [0.68; 1.59]	0.870
D to B	69.6 (26.9)	69.5 (27.0)	71.6 (24.7)	1.00 [0.99; 1.02]	0.712
Total ischemic time	420 (521)	420 (524)	407 (386)	1.00 [1.00; 1.00]	0.884
CL activation time	13.8 (10.6)	13.7 (10.5)	16.0 (13.5)	1.02 [0.98; 1.05]	0.465

Abbreviations: Abbreviations can be found in **Tables 1** and **2**.

Supplementary Table 2. Results of best subset regression

	Estimate	Std. Error	z-value	Pr(>|z|)
(Intercept)	-6.3527552	0.7571593	-8.390249	4.85E-17
Renal insufficiency	1.028113	0.5038875	2.040362	4.13E-02
Killip Classification	0.7231886	0.2007974	3.601584	3.16E-04
TNT	0.1492672	0.0747564	1.996715	4.59E-02

Bayesian Information Criterion based on q (BICq) equivalent for q in (0.774134184858393, 0.923498631445638). Abbreviations: Abbreviations can be found in **Tables 1** and **2**.