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Original Article

Analysis of risk factors for PCI no-reflow in coronary heart disease and construction of related prediction models

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Abstract: Objective: To analyze the risk factors of percutaneous coronary intervention (PCI) no-reflow in patients with coronary heart disease (CHD) and construct a predictive nomogram model. Methods: This retrospective study included 260 patients with CHD who underwent PCI in the Third Affiliated Hospital of Chongqing Medical University from January 2022 to December 2023. The subjects were divided into a PCI no-reflow group (n = 86) and normal reflow group (n = 174) based on thrombolysis in myocardial infarction (TIMI) blood flow grading. General data, PCI related data and laboratory indexes of patients were collected. Logistic regression was used to analyze the risk factors of no-reflow after PCI in CHD patients. Based on the significant variables from regression analysis, a nomogram prediction model was constructed by using R language. The accuracy of the model was evaluated by receiver operating characteristic (ROC) curve and calibration curve, and the decision curve was drawn to clarify the clinical utility of the model. Model performance metrics included area under the curve (AUC), accuracy, sensitivity and specificity. Results: Multivariate logistic regression analysis showed that hypertension, cystatin C (Cys-C), hypersensitive c-reactive protein (hs-CRP) and platelet-to-lymphocyte ratio (PLR) were risk factors for no-reflow after PCI in CHD patients (OR > 1, P < 0.001), while ADAM metallopeptidase with thrombospondin type 1 motif 13 (ADAMTS-13) and lymphocyte (LYM) were protective factors (OR < 1, P < 0.001). The nomogram prediction model based on the above risk factors showed good predictive value. The AUC of the nomogram prediction model in the training set was 0.967 (95% CI: 0.946-0.989), with a specificity of 0.923 and a sensitivity of 0.908. In the validation set, the AUC was 0.894 (95% CI: 0.817-0.971), with a specificity of 0.807 and a sensitivity of 0.857. The calibration curve indicated good agreement between the predicted and actual probabilities, and the decision curve showed clinical benefit across a range of threshold probabilities in both the training and validation sets (0.0-0.99). Conclusion: The risk factors affecting the occurrence of no-reflow after PCI in patients with CHD include hypertension, serum Cys-C, hs-CRP, PLR, ADAMTS-13 and LYM levels. The nomogram risk prediction model based on the above factors is valuable for identifying patients with high risk of no-reflow after PCI.

Keywords: Coronary heart disease, percutaneous coronary intervention, no reflow, influencing factors, nomogram model

Introduction

The acceleration of the aging process has gradually increased the prevalence and mortality of cardiovascular diseases in China. Current estimates suggest that China has approximately 330 million patients with cardiovascular disease, and its mortality rate ranks the first, surpassing other chronic non-communicable diseases [1]. In 2018, cardiovascular diseases accounted for 46.66% of deaths in rural areas and 43.81% in urban areas [2]. Among cardiovascular diseases, coronary heart disease (CHD) is a significant contributor, comprising 3.45% of all cases [3]. Besides, it is also a leading cause of death and disability, imposing great pressure to modern medical treatment [4].

With advancements in science and technology, our understanding of the internal mechanism and influencing factors of CHD pathogenesis and progression has significantly improved. Globally standardized guidelines have been

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established for CHD [5], including comprehensive protocols for percutaneous coronary intervention (PCI), which has become the primary minimally invasive treatment for chronic CHD patients. PCI aims to achieve coronary revascularization and timely improve myocardial ischemia [6]. The incidence of adverse cardiovascular and cerebrovascular events such as myocardial infarction, heart failure, and stroke caused by CHD is high, and even PCI does not represent a cure [7]. Study [8] has highlighted that CHD patients undergoing interventional therapy are at risk of no-reflow, a phenomenon manifested as dyspnea and chest pain, worsening the patient's condition and compromising treatment effect. According to statistics [9], the incidence of coronary no-reflow in elective PCI is 3-6%, which can escalate to approximately 30% in emergency PCI. Therefore, early assessment of the risk of no-reflow after PCI in CHD patients and the establishment of an effective risk prediction model for no-reflow after PCI are crucial to guide clinical treatment and enhance patient prognosis.

The nomogram model is characterized by its intuitive, simple and visual nature, making it a valuable tool for disease risk prediction and widely adopted in the medical field in recent years [10]. According to relevant literature [11], establishing a nomogram prediction model for coronary artery disease risk in elderly patients with acute myocardial infarction offers intuitive research methods and clinical insights to prevent disease progression. Based on this, this study analyzed clinical data to identify risk factors for PCI no-reflow in patients with CHD based, and constructed a nomogram prediction model, hoping to provide reference for clinical prevention and treatment.

Data and methods

Research subjects

This study was approved by the Ethics Committee of the Third Affiliated Hospital of Chongqing Medical University. A retrospective analysis was conducted on 260 patients with CHD who underwent PCI in the Third Affiliated Hospital of Chongqing Medical University from January 2022 to December 2023.

Inclusion criteria: (1) Patients meeting the diagnostic criteria for CHD [12] (coronary angiography findings showing ≥ 50% diameter stenosis in the left main coronary artery, three major branches of the anterior descending branch, right coronary artery, or their main branches. Additionally, patients admitted with acute coronary syndrome were diagnosed based on electrocardiogram and serum enzymology criteria); (2) Patients with indications for PCI and underwent the procedure for the first time; (3) Patients who voluntarily requested treatment and signed informed consent; (4) Patients who had not received other therapies in the past month. Exclusion criteria: (1) Patients with severe infection, immune disease, malignant tumor or cachexia; (2) Patients with severe valvular heart disease, or congenital heart disease; (3) Patients with severe hepatic and renal insufficiency; (4) Patients lost to follow-up or died during follow-up.

Methods

No-reflow evaluation and grouping method: The evaluation and grouping method for no-reflow after percutaneous coronary intervention (PCI) utilized the Thrombolysis in Myocardial Infarction (TIMI) flow grade [13]. Two experienced doctors assessed the post-interventional blood flow. Grade 3: The contrast agent completely fills and clears at the distal end of the stenosis artery, resembling normal blood flow; Grade 2: The narrow artery is fully filled at the distal end, but filling is slow and contrast agent clearance is delayed; Grade 1: Partial passage of contrast agent, with incomplete filling at the distal end of the vessel; Grade 0: No blood perfusion.

Grouping method: Based on the TIMI blood flow classification, patients with TIMI 0-2 were included in the PCI no-reflow group (n = 86), while those with TIMI grade 3 were included in the normal group (n = 174).

Data collection: Baseline demographic data: gender, age, BMI, smoking history, drinking history, diabetes, hypertension, stroke, chronic kidney disease, and acute coronary syndrome were recorded in both groups. PCI-related data: Gensini score, number of diseased vessels, number of coronary artery lesions, left ventricular ejection fraction (LVEF), duration of chest pain, and maximum ST segment changes were documented for both groups. Laboratory indicators: 5 mL of peripheral venous blood was

Table 1. Basic situation analysis of the two groups [n (%), (mean ± SD)]

Index	No-reflow group (n = 86)	Normal group (n = 174)	χ^2/t	*P*
Gender (male/female)	56/30	94/80	2.902	0.088
Age (year)	59.42±3.67	59.32±4.12	0.196	0.845
BMI (kg/m^2)	24.77±2.86	24.84±2.50	0.196	0.845
Smoking history (yes/no)	48/38	74/100	3.082	0.079
Drinking history (yes/no)	45/41	72/102	2.786	0.095
Diabetes (yes/no)	48/38	80/94	2.228	0.136
Hypertension (yes/no)	66/20	68/106	32.688	< 0.001
Stroke (yes/no)	38/48	57/117	3.241	0.072
Chronic kidney disease (yes/no)	40/46	60/114	3.518	0.061
Acute coronary syndrome (yes/no)	45/41	75/99	1.970	0.160

BMI, body mass index.

collected from each patient on admission and after an overnight fast, and centrifuged at 3000 rpm for 10 min at a radius of 15 cm. The supernatant was used for analysis. The levels of high density lipoprotein cholesterol (HDL-C), total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), triglyceride (TG), serum creatinine (Scr), glutamic pyruvic transaminase (GPT),and cystatin C (Cys-C) were measured by automatic biochemical analyzer (Olympus AU2700, Japan). The levels of hs-CRP, von Willebrand factor cleaving protease (ADAM metallopeptidase with thrombospondin type 1 motif 13 (ADAMTS-13)) and Hb were measured by enzyme-linked immunosorbent assay. Another 5 mL of peripheral venous blood was collected in a vacuum anticoagulant tube, and analyzed for WBC, PLT and lymphocyte (LYM) using an XE 100 blood cell analyzer (Shisen Meikang Company, Japan). Platelet-to-lymphocyte ratio (PLR) was calculated.

Outcome index: Taking no-reflow after PCI in patients with CHD as the end point of the study [14], 70% of the patients were randomly selected as the training set (n = 182), and 30% of the patients were used as the validation set (n = 78). The training set was used to construct the prediction model, and the validation set was used for model validation.

Statistical analysis

SPSS 26.0 was used for data analysis. Measurement data were expressed as mean ± SD and analyzed using t-tests. Categorical variables were expressed as rates (%) and analyzed using chi-square tests. Logistic regression was used to determine the risk factors associated with no-reflow after PCI in patients with CHD. Significant variables identified in the regression analysis were utilized to construct the nomogram prediction model using R 4.3.0. The receiver operating characteristic (ROC) curve, area under the ROC curve (AUC), accuracy, sensitivity, specificity, calibration curve and decision curve were used to evaluate the predictive performance of the model. $P < 0.05$ was considered statistically significant.

Results

Basic situation analysis

The prevalence of hypertension in no-reflow group was 76.74%, and that in normal group was 39.08% ($P < 0.05$). Other data showed no significant differences between the two groups (all $P > 0.05$, **Table 1**).

Analysis of PCI related data in the two groups

While the Gensini score was higher in the no-reflow group compared to the normal group, this difference was not statistically significant ($P > 0.05$). Specific analysis of diseased vessels revealed that there was no difference in the number of diseased vessels between the no-reflow group and the normal group ($P > 0.05$). Similarly, there were no significant differences in the number of coronary artery lesions, LVEF, duration of chest pain and maximum ST segment changes between the two groups (all $P > 0.05$). See **Table 2**.

Analysis of laboratory indicators in the two groups

Comparisons of laboratory indicators between the two groups showed similar levels of HDL-C,

Table 2. Basic situation analysis of the two groups [n (%), (mean ± SD)]

Index	No-reflow group (n = 86)	Normal group (n = 174)	χ^2/t	*P*
Gensini Score	57.24±4.70	56.95±5.03	0.183	0.855
Diseased blood vessels			2.482	0.648
LM	11 (12.79)	31 (17.82)		
LAD	27 (31.39)	45 (25.86)		
LCX	20 (23.26)	37 (21.26)		
RCA	17 (19.77)	31 (17.82)		
D1/D2	11 (12.79)	30 (17.24)		
Number of coronary lesions			3.295	0.193
Single	28 (32.56)	75 (43.10)		
Double	37 (43.02)	57 (32.76)		
Multi-branch	21 (24.42)	42 (24.14)		
LVEF (%)	39.60±4.25	39.49±4.38	0.203	0.839
Persistent chest pain duration (h)	6.15±1.24	6.18±1.35	0.145	0.885
The maximum ST segment change (mm)	3.23±0.81	3.15±0.68	0.870	0.385

LVEF, left ventricular ejection fraction.

Table 3. Analysis of laboratory indexes in the two groups [n (%), (mean ± SD)]

Index	No-reflow group (n = 86)	Normal group (n = 174)	χ^2/t	*P*
HDL-C (mmol/L)	1.15±0.26	1.10±0.24	1.472	0.142
TC (mmol/L)	5.06±0.85	5.12±0.78	0.570	0.569
LDL-C (mmol/L)	2.58±0.65	2.62±0.75	0.458	0.647
TG (mmol/L)	1.78±0.42	1.80±0.45	0.354	0.724
SCr (μmol/L)	103.69±12.43	102.33±13.15	0.787	0.432
GPT (U/L)	27.23±5.27	26.76±5.36	0.666	0.506
Cys-C (mg/L)	0.95±0.22	0.68±0.25	8.878	< 0.001
hs-CRP (mg/L)	51.47±9.46	43.15±7.23	7.180	< 0.001
ADAMTS-13 (mg/L)	156.35±13.73	173.43±19.34	8.198	< 0.001
Hb (g/L)	140.42±10.33	141.39±10.72	0.693	0.489
WBC ($\times 10^9$/L)	10.66±2.15	10.82±2.46	0.519	0.605
PLT ($\times 10^9$/L)	213.59±20.35	211.16±19.74	0.925	0.356
LYM ($\times 10^9$/L)	1.55±0.25	1.93±0.32	10.508	< 0.001
PLR	175.83±18.57	165.29±10.75	4.880	< 0.001

HDL-C, high density lipoprotein cholesterol; TC, total cholesterol; LDL-C, low density lipoprotein cholesterol; TG, triglyceride; Scr, serum creatinine; GPT, glutamic pyruvic transaminase; Hb, hemoglobin; WBC, white blood cell count; PLT, platelet count; PCI, percutaneous coronary intervention; Cys-C, Cystatin C; hs-CRP, hypersensitive c-reactive protein; PLR, platelet-to-lymphocyte ratio; ADAMTS-13, ADAM metallopeptidase with thrombospondin type 1 motif 13; LYM, lymphocyte.

TG, LDL-C, TC, SCr, GPT, Hb, WBC, and PLT (all P > 0.05). However, significant differences were observed in the levels of serum Cys-C, hypersensitive c-reactive protein (hs-CRP), platelet-to-lymphocyte ratio (PLR), ADAMTS-13 and lymphocyte (LYM) between the two groups (all P < 0.05). See **Table 3**.

Analysis of risk factors

Univariate regression analysis showed hypertension, Cys-C, hs-CRP, ADAMTS-13, LYM and PLR as significant factors affecting no-reflow after PCI in patients with CHD. The reflow after PCI was used as the dependent variable (no reflow = 1, reflow = 0), and the assignment of other independent variables (Cys-C, hs-CRP, ADAMTS-13, LYM and PLR) is shown in **Table 4**. The results showed that hypertension, Cys-C, hs-CRP and PLR were hazard factors for no-reflow after PCI in patients with CHD (all odd ratio (OR) > 1, P < 0.001), see **Table 5**.

Table 4. Assignment of related variables

Risk Factor	Variable	Assignment
Reflow after PCI	Y	No reflow = 1, reflow = 0
Hypertension	X1	Yes = 1, no = 0
Cys-C	X2	Actual numerical value
hs-CRP	X3	Actual numerical value
ADAMTS-13	X4	Actual numerical value
LYM	X5	Actual numerical value
PLR	X6	Actual numerical value

PCI, percutaneous coronary intervention; Cys-C, cystatin C; hs-CRP, hypersensitive c-reactive protein; PLR, platelet-to-lymphocyte ratio; ADAMTS-13, ADAM metallopeptidase with thrombospondin type 1 motif 13; LYM, lymphocyte.

Model construction

According to the results of clinical data analysis, statistically significant indicators including hypertension, Cys-C, hs-CRP, ADAMTS-13, LYM and PLR were used as variables to construct the model with R language. The nomogram algorithm for no-reflow risk prediction is shown in **Figure 1**.

In order to further understand the predictive performance of the nomogram model, the ROC curves of the training set and the validation set were drawn respectively (as shown in **Figure 2**). The AUC of the nnomogram model in the training set was 0.967 (95% CI: 0.946-0.989) with a specificity of 0.923 and a sensitivity of 0.908. The AUC of the model in the validation set was 0.894 (95% CI: 0.817-0.971) with a specificity of 0.807 and a sensitivity of 0.857, indicating robust predictive ability.

The calibration curve drawn in this study shows close alignment between predicted and actual probabilities in both the training and validation sets, suggesting good calibration of the model (**Figure 3**). The Hosmer-Lemeshow test yielded a statistic of $\chi^2 = 4.19$, with a corresponding *P*-value of 0.839 ($P > 0.05$), suggesting excellent model fit.

The decision curve analysis (**Figure 4**) of the nomogram model showed that the threshold probabilities of the training set and the validation set were both 0.0-0.99, suggesting good clinical utility of the nomogram model.

Discussions

No-reflow in patients with CHD after interventional therapy is mainly manifested as the obstruction of the blood supply vessels after PCI, where myocardial blood flow fails to fully recover or ceases shortly after initial restoration. This condition often induces chest pain and dyspnea, and leads to a variety of serious complications, affecting the effect of PCI treatment [15]. A study [16] has indicated that the occurrence of no-reflow significantly elevates the risk of rehospitalization and disease recurrence in patients undergoing interventional therapy. Therefore, identifying efficient predictors of no-reflow after PCI in CHD patients, in a straightforward, rapid, and effective manner, remains a pivotal focus in clinical practice to enable early intervention and evaluation.

Literature [17] has shown that no-reflow after interventional therapy in patients with CHD may be related to inflammatory response and thrombosis. Myocardial ischemia and hypoxia in patients with CHD lead to neutrophil and platelet adhesion and aggregation. During blood flow reperfusion, oxygen free radicals are generated to activate white blood cells and other ways to destroy the cell membrane, leading to mitochondrial swelling and dissolution, causing vascular smooth muscle contraction, accelerating vascular endothelial injury, and resulting in platelet aggregation and vascular thrombosis [18]. The results of this study showed that the incidence of no-reflow after PCI was 33.08%, with a higher prevalence observed in hypertensive individuals (76.74% in the no-reflow group vs. 39.08% in the normal group). Significant differences were also noted in serum levels of Cys-C, hs-CRP, PLR, ADAMTS-13 and LYM between the two groups, highlighting increased inflammatory response in the no-reflow group after PCI. Considering the multifaceted mechanism of no-reflow, including ischemia-reperfusion injury, vascular endothelial injury, platelet aggregation, microvascular embolism and oxidative stress, along with factors such as underlying diseases, medications, inflammatory state, coagulation parameters, surgical operation and vascular characteristics [19, 20], it is crucial to further identify influential factors.

In this study, Cys-C, hs-CRP and PLR emerged as risk factors for no-reflow after PCI in patients with CHD, while ADAMTS-13 and LYM were identified as protective factors. Analysis indicates that prolonged hypertension leads to vascular endothelial damage, reduced elasticity, increased tension, and exacerbated athero-

Table 5. Multivariate Logistic regression analysis

Risk Factor	B	S.E.	Wald value	OR value	95% CI	*P* value
Hypertension	2.025	0.496	16.664	7.580	2.866-20.045	< 0.001
Cys-C	4.510	1.005	20.140	90.888	12.681-651.398	< 0.001
hs-CRP	0.097	0.029	11.181	1.102	1.041-1.166	< 0.001
ADAMTS-13	-0.037	0.013	7.880	0.963	0.938-0.989	0.005
LYM	-3.750	0.848	19.538	0.024	0.004-0.124	< 0.001
PLR	0.065	0.016	16.954	1.067	1.035-1.101	< 0.001
Constant	-8.633	3.963	4.745	-	-	0.029

Cys-C, cystatin C; hs-CRP, hypersensitive c-reactive protein; PLR, platelet-to-lymphocyte ratio; ADAMTS-13, ADAM metallopeptidase with thrombospondin type 1 motif 13; LYM, lymphocyte.

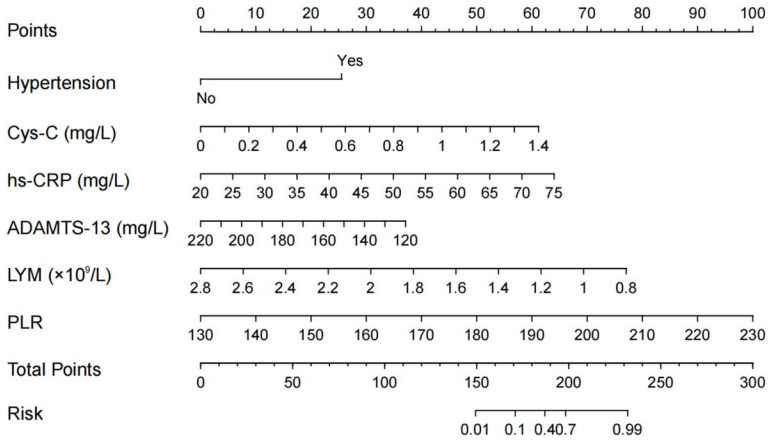

Figure 1. Nomogram prediction model for predicting no-reflow risk in patients with coronary heart disease (CHD) after PCI. PCI, Percutaneous coronary intervention; Cys-C, cystatin C; hs-CRP, hypersensitive c-reactive protein; PLR, platelet-to-lymphocyte ratio; ADAMTS-13, ADAM metallopeptidase with thrombospondin type 1 motif 13; LYM, lymphocyte.

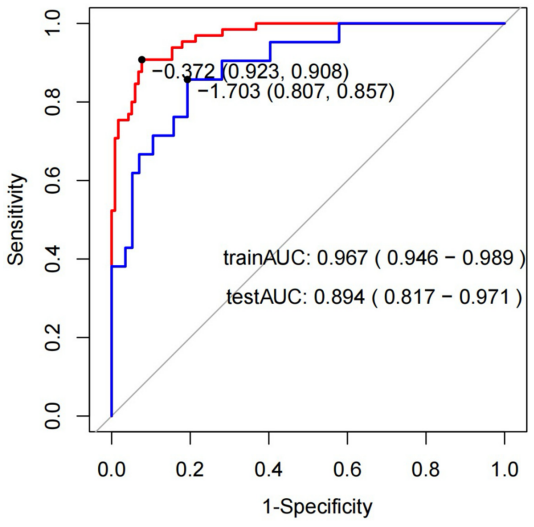

Figure 2. ROC curve of nomogram model (training set - red, test set - blue).

sclerosis, predisposing patients to increased risk of no-reflow post-PCI [21]. Elevated Cys-C level can trigger inflammatory mediator release, promoting WBC accumulation in microvascular sites and thrombosis formation [22]. ADAMTS-13, synthesized by vascular endothelial cells, is a sensitive marker of inflammatory response and plays an important role in thrombosis regulation by enhancing von Willebrand factor (vWF) adhesion and activity modulation [23]. Decreased ADAMTS-13 levels correlates with heightened thrombosis risk [24]. In summary, the levels of serum Cys-C and ADAMTS-13 in patients with CHD may be related to the occurrence of no-reflow after interventional therapy. Moreover, heightened hs-CRP levels indicate systemic inflammation, contributing to white blood cell aggregation in microvascular sites and increasing the likelihood of no-reflow after PCI in CHD patients [25]. PLR, reflecting platelet-to-lymphocyte ratio, is significantly increased in patients with CHD. More importantly, PLT activity plays a pivotal role in the progression of CHD [26], and lymphocytes depletion enhances platelet activation and thrombosis, exacerbating no-reflow risk post-PCI. A study [27] has underscored hypertension, dyslipidemia, obesity, and smoking as significant risk factors for no-reflow during PCI across various CHD presentations. Clinical awareness of these risk factors informs targeted strategies to optimize

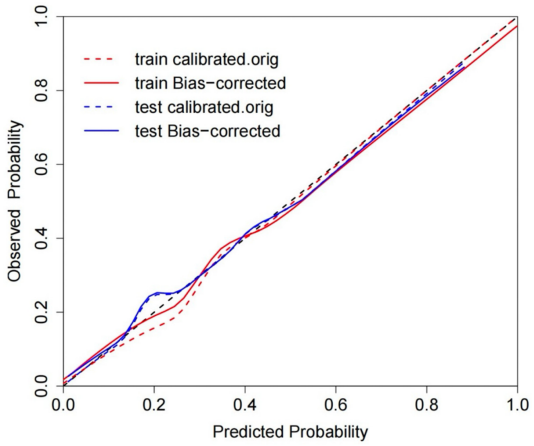

Figure 3. Calibration curve of nomogram model.

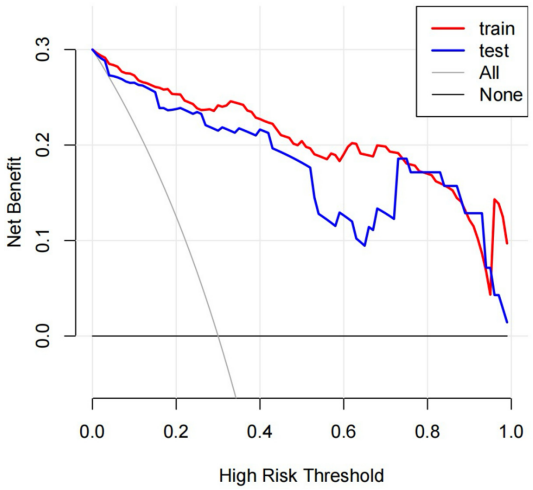

Figure 4. Decision curve of nomogram model.

patient outcomes. Further research [28, 29] highlights platelet activity, lymphocyte count, and other inflammatory markers as pivotal in predicting no-reflow and long-term prognosis post-PCI. Vigilance in monitoring Cys-C, hs-CRP, PLR levels, and ADAMTS-13 expression can guide early anti-inflammatory and antiplatelet interventions, potentially reducing the incidence of PCI-related no-reflow in CHD patients.

The traditional logistic regression model can identify prognostic factors for cardiovascular and coronary artery disease [30]. However, the prediction efficiency can be limited in datasets with small sample sizes. In contrast, machine learning algorithms, such as nomogram model, can offer enhanced capability to process complex data and deliver robust predictive performance. The nomogram model has been widely used in clinical prediction of the risk of disease or adverse outcomes in recent years due to its advantages of easy understanding, good accuracy, and individualized assessment of the occurrence of certain adverse events [31, 32]. In this study, a nomogram prediction model was established based on the risk factors screened by multivariate Logistic regression analysis. The results demonstrated the model's strong predictive value for assessing the risk of no-reflow after PCI in patients with CHD. The accuracy and discriminative ability of the risk prediction model were rigorously evaluated through ROC curves and calibration plots. The AUC values of 0.967 for the training set and 0.894 for the validation set indicated excellent predictive performance, with calibration curves showing close alignment with the ideal curve, affirming the model's reliability. The findings underscore the nomogram model's utility as an effective tool in predicting no-reflow risk post-PCI in CHD patients. Leveraging this predictive model in clinical practice can facilitate targeted interventions to mitigate relevant risk factors, thereby reducing the incidence of no-reflow and improving treatment outcomes. Such proactive measures not only enhance patient satisfaction but also contribute to alleviating the economic burden associated with medical management of CHD.

Compared with traditional statistical methods machine learning algorithms offer higher accuracy in predicting the risk of no-reflow after PCI in CHD patients. This study innovatively used hypertension, serum Cys-C, hs-CRP, PLR, ADAMTS-13, and LYM levels to construct a nomogram prediction model for CHD patients post-PCI, providing valuable insights for assessing the risk of no-reflow. Clinically, these risk factors can be evaluated to formulate effective prevention and treatment strategies. However, the study is limited by its small sample size and the use of clinical samples from a single center. The complex nature of CHD warrants further research with larger, high-quality datasets to deepen understanding of its mechanisms and establish more robust prediction models that can better guide clinical decision-making.

In summary, hypertension, serum Cys-C, hs-CRP, PLR, ADAMTS-13 and LYM levels are crucial factors influencing the occurrence of no-reflow after PCI in CHD patients. The nomogram

risk prediction model based on the above factors holds significant promise in clinical practice for identifying high-risk patients and implementing targeted interventions to mitigate the risk of no-reflow post-PCI.

Disclosure of conflict of interest

None.

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