Development and Validation of a Risk Prediction Model for Contrast-Induced Acute Kidney Injury Following Percutaneous Coronary Intervention in Patients with Acute Myocardial Infarction: A Postprint

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Abstract

Background Early reperfusion therapy for acute myocardial infarction (AMI) is an effective method to reduce mortality in AMI patients. Percutaneous coronary intervention (PCI) is one of the reperfusion treatment modalities, and the occurrence of contrast-induced acute kidney injury (CI-AKI) after PCI has become one of the common causes of acute kidney injury.

Objective To investigate the risk factors for CI-AKI in AMI patients after PCI, establish a risk prediction model for CI-AKI based on these risk factors, and evaluate its effectiveness.

Methods Clinical data of 1,274 patients initially diagnosed with AMI and undergoing PCI at the Affiliated Hospital of Xuzhou Medical University from 2019 to 2021 were consecutively collected. According to the order of admission time, patients were divided into a training group (January 2019–March 2021, 900 cases) and a validation group (April–December 2021, 374 cases) at a ratio of approximately 7:3; and based on the diagnostic criteria for CI-AKI, patients were divided into CI-AKI and non-CI-AKI groups. Univariate Logistic regression, Lasso regression, cross-validation, and multivariate Logistic regression were used to screen independent risk factors, and a CI-AKI risk nomogram was constructed. The discriminative ability, calibration, and clinical utility were evaluated by calculating the C-statistic and drawing calibration curves and decision curves.

Results Among the 900 patients in the training group, 109 (12.1%) developed CI-AKI after PCI; among the 374 patients in the validation group, 27 (7.2%)

developed CI-AKI. Multivariate Logistic regression analysis showed that left ventricular ejection fraction (LVEF) [OR=0.903, 95%CI (0.873, 0.934)], platelet distribution width [OR=1.158, 95%CI (1.053, 1.274)], mean platelet volume to lymphocyte count ratio (MPVLR) [OR=1.047, 95%CI (1.016, 1.079)], neutrophil to high-density lipoprotein ratio (NHR) [OR=1.072, 95%CI (1.021, 1.124)], serum creatinine (Scr) [OR=1.006, 95%CI (1.002, 1.011)], and diuretics [OR=2.321, 95%CI (1.452, 3.709)] were independent influencing factors for CI-AKI after PCI in AMI patients (P<0.05). A prediction model incorporating the six risk factors of LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretics was established, and a CI-AKI risk nomogram was drawn. The C-statistic was 0.794 [95%CI (0.766, 0.820)] for the training group and 0.799 [95%CI (0.774, 0.855)] for the validation group. Calibration plots showed good consistency between predicted and actual results; decision curves and clinical impact curves indicated that the nomogram had clinical utility.

Conclusion The CI-AKI risk prediction model includes LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretics. This prediction model has good discriminative ability and accuracy, can intuitively and independently screen high-risk populations, and has high predictive value for the occurrence of CI-AKI after PCI in AMI patients.

Full Text

Development and Validation of a Risk Prediction Model for Contrast-Induced Acute Kidney Injury after Percutaneous Coronary Intervention in Patients with Acute Myocardial Infarction

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Abstract

Background: Early reperfusion therapy for acute myocardial infarction (AMI) is an effective approach to reduce mortality in AMI patients. Percutaneous coronary intervention (PCI) is one of the reperfusion therapy modalities, and contrast-induced acute kidney injury (CI-AKI) after PCI has become one of the common causes of AKI.

Objective: To investigate the risk factors for the development of CI-AKI in AMI patients after PCI, establish a risk prediction model for CI-AKI based on these risk factors, and evaluate its validity.

Methods: The clinical data of 1,274 patients who were consecutively admitted to the Affiliated Hospital of Xuzhou Medical University between 2019 and 2021 with a primary diagnosis of AMI and underwent PCI were collected. Patients were divided into a training group (January 2019 to March 2021, 900 cases) and a validation group (April 2021 to December 2021, 374 cases) in a chronological order at a ratio of approximately 7:3. They were further categorized into CI-AKI and non-CI-AKI groups according to CI-AKI diagnostic criteria. Independent risk factors were screened using univariate Logistic regression, Lasso regression, cross-validation, and multivariate Logistic regression analysis. A nomogram for predicting CI-AKI risk was constructed, and its discriminatory power, calibration ability, and clinical application value were evaluated by calculating the C-statistic and plotting calibration and decision curves.

Results: Among the 900 patients in the training group, 109 (12.1%) developed CI-AKI after PCI; among the 374 patients in the validation group, 27 (7.2%) developed CI-AKI. Multivariate Logistic regression analysis showed that left ventricular ejection fraction (LVEF) [OR=0.903, 95%CI (0.873, 0.934)], platelet distribution width [OR=1.158, 95%CI (1.053, 1.274)], mean platelet volume to lymphocyte ratio (MPVLR) [OR=1.047, 95%CI (1.016, 1.079)], neutrophil to high-density lipoprotein ratio (NHR) [OR=1.072, 95%CI (1.021, 1.124)], serum creatinine (Scr) [OR=1.006, 95%CI (1.002, 1.011)], and diuretic use [OR=2.321, 95%CI (1.452, 3.709)] were independent influencing factors for CI-AKI after PCI in AMI patients ($P<0.05$). A prediction model incorporating these six risk factors was constructed, and a nomogram for CI-AKI risk was developed. The C-statistic was 0.794 [95%CI (0.766, 0.820)] for the training group and 0.799 [95%CI (0.774, 0.855)] for the validation group. Calibration plots demonstrated good consistency between predicted and actual outcomes, while decision curve and clinical impact curve analyses indicated the nomogram had clinical utility.

Conclusion: The CI-AKI risk prediction model, which includes LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretic use, demonstrates good discrimination and accuracy. This model can intuitively and independently identify high-risk populations and has high predictive value for CI-AKI occurrence after PCI in AMI patients.

Keywords: Acute myocardial infarction; Percutaneous coronary intervention; Acute kidney injury; Contrast-induced acute kidney injury; Nomograms; Risk prediction model

1. Methods

1.1 Study Population

This retrospective, observational study consecutively enrolled 1,274 patients initially diagnosed with acute myocardial infarction (AMI) who underwent percutaneous coronary intervention (PCI) at the Affiliated Hospital of Xuzhou Medical University between 2019 and 2021. The inclusion criteria were: (1) meeting the AMI diagnostic criteria published by the American College of Cardiology/American Heart Association (ACC/AHA) [**?**]; (2) meeting the CI-AKI diagnostic criteria published by the European Society of Urogenital Radiology (ESUR) [**?**]; and (3) having indications for PCI and successfully undergoing the procedure. Exclusion criteria included: (1) incomplete basic information; (2) undergoing hemodialysis or having an estimated glomerular filtration rate (eGFR) <15 ml $\cdot$ min^{-1} $\cdot$ (1.73 m^2)$^{-1}$; (3) having autoimmune diseases; (4) recent contrast agent use (within 3 days); (5) recent use (within 72 hours before or after surgery) of potentially nephrotoxic drugs; (6) malignancy; and (7) death. The study protocol was approved by the Ethics Committee of the Affiliated Hospital of Xuzhou Medical University (Ethics No.: XYFY2022-KL123-01).

1.2 Research Methods

This study retrospectively analyzed clinical data to identify risk factors for CI-AKI and construct a prediction model for early identification of high-risk patients, enabling timely intervention to prevent CI-AKI occurrence.

1.2.1 Data Collection General patient information was recorded in detail, including age, sex, smoking history, underlying diseases, heart rate, left ventricular ejection fraction (LVEF), and medication use. Serum creatinine (Scr) levels were measured 48-72 hours postoperatively, and the difference between preoperative and postoperative Scr values was calculated.

1.2.2 Diagnostic Criteria The diagnostic criteria for AMI [**?**] included: (1) Clinical presentation: chest, upper abdominal, arm, wrist, or jaw discomfort at rest or during activity, typically lasting at least 20 minutes; (2) Electrocardiogram: signs of myocardial ischemia, particularly ST-segment and T-wave changes, and signs of myocardial necrosis, particularly QRS changes; and (3) Biomarkers of myocardial necrosis: sensitive and specific biomarkers such as cardiac troponin >99th percentile of the upper reference limit or creatine kinase-MB >99th percentile of the upper reference limit in the setting of acute ischemia.

The diagnostic criteria for CI-AKI [**?**] were: acute kidney injury occurring 48-72 hours after contrast administration, excluding other possible causes of renal damage, defined as Scr level increase $ $0.3 mg/dL (26.5 mol/L) or $ $1.5 times the baseline level.

1.2.3 Blood Sample Measurement All patients underwent venous blood collection before PCI. Anticoagulated blood samples (2 mL) were sent to the clinical laboratory, centrifuged at 1,000-1,200×g (3,000 r/min) for 5-10 minutes to extract middle-lower layer blood cells for detection of white blood cell count, neutrophils, lymphocytes, monocytes, C-reactive protein (CRP), red blood cells, hemoglobin, hematocrit, red cell distribution width, platelet count, platelet distribution width, and mean platelet volume. Coagulation biochemical samples (3 mL) were centrifuged under the same conditions to extract upper serum specimens for analysis of N-terminal pro-brain natriuretic peptide (NT-proBNP), triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), small dense LDL, uric acid, blood urea nitrogen, Scr, fasting blood glucose, and glycated hemoglobin. Anticoagulated coagulation samples (2 mL) were processed similarly for analysis of fibrinogen, D-dimer, and antithrombin III (AT III). All blood specimens were tested in the hospital's central laboratory with unified reports. NHR was calculated as neutrophil count divided by HDL, and MPVLR as mean platelet volume divided by lymphocyte count.

1.2.4 PCI Procedure PCI was performed by interventional cardiologists via radial artery access according to standard clinical practice. All patients received aspirin (loading dose 300 mg), clopidogrel (loading dose 300 mg) or ticagrelor (180 mg) at presentation, followed by aspirin (100 mg/d), clopidogrel (75 mg/d) or ticagrelor (180 mg/d). Low-osmolar non-ionic contrast agents with osmolarity of 600-800 mOsm/kg were used. Postoperative hydration was administered according to patients' baseline physical condition.

1.2.5 Grouping Methods Patients were divided into: (1) a training group (January 2019 to March 2021, 900 cases) and a validation group (April 2021 to December 2021, 374 cases) in a 7:3 ratio based on admission chronology; and (2) CI-AKI and non-CI-AKI groups according to CI-AKI diagnostic criteria.

1.3 Statistical Analysis

Statistical analysis was performed using SPSS 26.0 and R software (version 4.2.1). Categorical data were expressed as frequencies and compared using the ^{2} test. Normally distributed continuous data were expressed as mean ± standard deviation and compared using independent samples t-test. Non-normally distributed continuous data were expressed as median (P25, P75) and compared using Mann-Whitney U test. Univariate Logistic regression, Lasso regression, and cross-validation were used to screen risk factors for CI-AKI after PCI in AMI patients. Variables with statistical significance in univariate analysis were included in multivariate Logistic regression to identify independent risk factors. The Hosmer-Lemeshow test was used to assess model goodness-of-fit. The "rms" package was used to construct a visual nomogram prediction model. The "proc," "car," and "rmda" packages were used for receiver operating characteristic (ROC) curve, calibration curve, and decision curve analysis (DCA). All tests

were two-sided, with P<0.05 considered statistically significant.

2. Results

2.1 Comparison of Clinical Data in the Training Group

Among the 900 patients in the training group, 109 (12.1%) developed CI-AKI after PCI. There were no statistically significant differences between the CI-AKI and non-CI-AKI groups in terms of sex, smoking history, hypertension, white blood cell count, monocytes, CRP, red blood cells, hemoglobin, hematocrit, red cell distribution width, platelet count, fibrinogen, D-dimer, AT III, TC, LDL, small dense LDL, fasting blood glucose, glycated hemoglobin, blood urea nitrogen, aspirin use, β-blockers, ACEI/ARB, statins, calcium channel blockers (CCB), nitrates, or low molecular weight heparin (P>0.05). However, significant differences were observed between groups in age, diabetes, chronic kidney disease, heart rate, LVEF, neutrophils, lymphocytes, platelet distribution width, mean platelet volume, MPVLR, lnNT-proBNP, TG, HDL, NHR, uric acid, Scr, and diuretic use (P<0.05) .

2.2 Screening for Independent Risk Factors of CI-AKI

Based on intergroup comparisons, variables with mutual interference or inclusion were excluded (neutrophils, lymphocytes, mean platelet volume, HDL). The remaining risk factors were analyzed using univariate Logistic regression, with continuous variables (age, heart rate, LVEF, platelet distribution width, MPVLR, lnNT-proBNP, NHR, TG, uric acid, Scr) entered as actual measured values and categorical variables (diabetes, chronic kidney disease, diuretic use) coded as 1 if present/used and 0 otherwise. To further screen risk factors and prevent model overfitting due to high dimensionality and multicollinearity, variables with statistical significance in univariate analysis {age [OR=1.027, 95%CI (1.010, 1.044)], diabetes [OR=1.693, 95%CI (1.105, 2.593)], chronic kidney disease [OR=6.178, 95%CI (2.383, 16.015)], diuretics [OR=3.302, 95%CI (2.143, 5.089)], LVEF [OR=0.892, 95%CI (0.864, 0.921)], platelet distribution width [OR=1.126, 95%CI (1.033, 1.227)], MPVLR [OR=1.061, 95%CI (1.030, 1.092)], LnNT-proBNP [OR=1.771, 95%CI (1.511, 2.075)], NHR [OR=1.082, 95%CI (1.031, 1.135)], TG [OR=0.695, 95%CI (0.519, 0.932)], uric acid [OR=0.997, 95%CI (0.994, 0.999)], Scr [OR=1.006, 95%CI (1.002, 1.011)]} were imported into RStudio. Lasso regression and cross-validation were performed using the "glmnet" and "caret" packages to identify the most important features: LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretic use. These significant risk factors were then included in multivariate Logistic regression analysis, which confirmed them as independent influencing factors for CI-AKI after PCI in AMI patients (P<0.05) .

2.3 Nomogram Development

Based on the multivariate Logistic regression analysis, we selected LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretic use to construct a prediction model and develop a nomogram for CI-AKI risk [Figure 1: see original paper]. Each independent predictor is projected upward to the top "Points" scale to obtain a value between 0-100, and the total score is used to accurately predict the risk of CI-AKI in AMI patients. Higher total scores indicate greater risk of CI-AKI. The Hosmer-Lemeshow test yielded ^{2}=9.229 (P=0.323), confirming good model fit. The C-statistic, equivalent to the area under the ROC curve (AUC), was 0.794 [95%CI (0.766, 0.820)] for the training group [Figure 2: see original paper]A, demonstrating good discriminatory ability. The calibration curve confirmed strong agreement between predicted and actual risks [Figure 3: see original paper]A.

2.4 Internal Validation

In the validation group, 27 patients (7.2%) developed CI-AKI. The Hosmer-Lemeshow test result was ^{2}=5.026 (P=0.755). The AUC for predicting CI-AKI in AMI patients was 0.799 [95%CI (0.774, 0.855)] [Figure 2: see original paper]B, indicating good model discriminability. The calibration curve demonstrated excellent consistency and goodness-of-fit for the nomogram prediction model [Figure 3: see original paper]B.

2.5 Decision Curve Analysis

Decision curve analysis (DCA) demonstrated the predictive capability of the nomogram for CI-AKI. The horizontal line represents no intervention with zero net benefit, while the diagonal line represents intervention for all patients. The DCA showed a wide range of high-risk threshold probabilities applicable to both training and validation groups, indicating clinical utility of the nomogram [Figure 4: see original paper]. In [Figure 5: see original paper], the solid curve shows the number of subjects classified as positive (high-risk number) at each threshold probability, while the dashed curve (high-risk number of events) shows the true positive number at each threshold probability, indicating good agreement between actual and predicted distributions.

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3. Discussion

This study investigated risk factors for CI-AKI after PCI in AMI patients and identified six independent risk factors: LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretic use. Using these independent risk factors, we constructed a prediction model and developed a visual nomogram using R software. External validation confirmed that the prediction model has good discriminatory ability, consistency, and effectiveness.

Inflammatory response is an important risk factor for CI-AKI. Neutrophils and lymphocytes are systemic inflammatory markers that mediate early inflammatory responses. Neutrophils release cytotoxic substances and promote reactive oxygen species release, leading to local ischemia, plaque instability, and thrombosis [**?**]. HDL has strong anti-atherosclerotic functions and exhibits anti-inflammatory and antioxidant capabilities in healthy individuals, promoting endothelial function and repair. HDL also serves as a systemic signaling mechanism facilitating rapid inter-organ communication during physiological stress, and previous studies have confirmed its association with CI-AKI [**?**], consistent with our findings. Research indicates HDL can regulate activated neutrophil function, while activated neutrophils can also affect HDL composition and function [**?**]. NHR, a simple ratio of neutrophils to HDL, may be more effective and reliable than single indicators and represents a potential novel lipid biomarker that quantitatively reflects inflammatory status and lipid distribution. Recent studies have found NHR can predict retinal artery occlusion [**?**], metabolic syndrome [**?**], acute ischemic stroke [**?**], and assess inflammatory processes in Parkinson's disease [**?**]. It has also been widely used in cardiovascular diseases, and our results confirm NHR as an independent risk factor for CI-AKI.

Lymphocytes are involved not only in physiological stress and inflammatory responses but also are inseparable from atherosclerotic plaque growth, development, rupture, and thrombosis [**?**]. Increased MPV and platelet distribution width caused by platelet activation are potentially useful markers for early diagnosis of thromboembolic diseases. Larger platelets accelerate coronary thrombosis and exacerbate systemic inflammatory responses, playing important roles in the pathophysiology of AMI [**?**]. Platelet distribution width represents platelet size variability and provides more information about platelet reactivity than MPV alone [**?**]. MPVLR, combining MPV and lymphocyte count, integrates the advantages of both markers and is a novel marker reflecting thrombosis and inflammation. Previous studies have confirmed MPVLR as an independent predictor of CI-AKI [**?**], consistent with our findings.

Although eGFR is considered the best indicator for evaluating renal function, its measurement largely depends on Scr. The most classic CI-AKI risk prediction model, the Mehran score, includes preoperative Scr level, which not only reflects baseline renal function but is also an independent predictor of CI-AKI, as confirmed in previous guidelines [**?**]. LVEF is a dynamic indicator closely associated with hemodynamic instability and adverse outcome risk. In this study, patients with low LVEF were more susceptible to CI-AKI, possibly due to low cardiac output state, severe cardiac dysfunction, hemodynamic instability, and reduced effective renal blood flow, which accelerates renal function impairment after contrast administration. Additionally, reduced effective renal blood flow triggers renin-angiotensin system stagnation, activates sympathetic nervous system, and increases inflammatory factors and oxygen free radical levels, thereby increasing CI-AKI risk [**?**].

Our data revealed that patients receiving diuretics during treatment had higher CI-AKI incidence, and diuretic use was independently associated with CI-AKI. Diuretics primarily affect renal tubular and collecting duct reabsorption and secretion, promoting sodium, chloride, and water excretion to reduce cardiac burden, but they also decrease renal perfusion, causing transient renal function impairment and increasing CI-AKI incidence [**?**].

A nomogram is a visual diagram composed of lines of varying lengths used to predict clinical event incidence. This study incorporated six predictors (LVEF, platelet distribution width, MPVLR, NHR, Scr, and diuretic use) to establish a nomogram. The nomogram demonstrated good discriminatory power in both training and validation cohorts, confirming the model's validity and applicability to some extent. Clinically, healthcare providers can predict CI-AKI incidence after PCI in AMI patients based on the sum of scores for each risk factor, allowing for enhanced management of modifiable factors such as adequate hydration therapy or combined medication strategies to actively prevent CI-AKI after PCI.

This study has several limitations: (1) It is a single-center retrospective observational study; (2) Some risk factors mentioned in previous studies were unavailable; and (3) Although internal validation was performed using data from different time periods within the same hospital, multi-center studies with larger sample sizes are needed to further evaluate the model's clinical predictive value.

Author Contributions

WANG Zhen: data curation, writing, statistical analysis, revision. SHEN Guoqi: data collection and curation. LI Yanan, ZHU Yinghua, QIU Hang: data collection. ZHENG Di, XU Tongda: research guidance. LI Wenhua: research guidance, revision.

Conflict of Interest: None declared.

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