

Prediction of in-hospital mortality in acute myocardial infarction patients with left anterior descending artery occlusion

Chuan-De Zou

The Second Affiliated Hospital of Anhui Medical University

Lian-Lian Fang

The First Affiliated Hospital of Anhui Medical University

Dong-Lin Wang

The Second Affiliated Hospital of Anhui Medical University

Qian Tang

The Second Affiliated Hospital of Anhui Medical University

He-Ping Zuo

`efy120307@fy.ahmu.edu.cn`

The Second Affiliated Hospital of Anhui Medical University

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Abstract

Objective To identify potential risk factors for in-hospital mortality in acute myocardial infarction (AMI) patients with left anterior descending artery (LAD) occlusion and construct a nomogram for predicting their prognosis.

Methods The study dataset was randomly partitioned into a 70% training set and a 30% validation set. LASSO regression and random forest algorithms were used to screen potential risk factors among 32 candidate variables in the training set. Independent risk factors selected by multivariate logistic regression were used to construct a nomogram. The discriminatory performance, calibration performance and clinical utility of the nomogram were assessed using the receiver operating characteristic curve, calibration curve, and decision curve analysis.

Results A total of 454 AMI patients with LAD occlusion were enrolled in this study. Age, systolic blood pressure, heart rate, white blood cells, lactate dehydrogenase, fibrinogen, and albumin-to-globulin ratio were independent risk factors of in-hospital mortality in AMI patients with LAD occlusion. The nomogram showed good discriminatory performance with an area under the curve (AUC) of 0.953 (95%CI: 0.918-0.989) in the validation set. The calibration curve demonstrated that the nomogram exhibited satisfactory calibration performance for predicting in-hospital mortality in AMI patients with LAD occlusion. The decision curve analysis indicated favorable clinical utility of the predictive model.

Conclusion This nomogram can accurately predict in-hospital mortality in AMI patients with LAD occlusion.

Introduction

Acute myocardial infarction (AMI) is defined as myocardial necrosis resulting from acute occlusion of a coronary artery, including left main artery (LM), left anterior descending artery (LAD), left circumflex artery (LCX), and right coronary artery (RCA), and represents the most severe manifestation of cardiovascular emergencies[1]. Although the in-hospital mortality of AMI patients has decreased significantly with current treatment methods[2], such as percutaneous coronary intervention (PCI), thrombolytic therapy, and coronary artery bypass grafting (CABG), AMI is still a severe and fatal cardiovascular disease that deserves considerable attention. Previous studies have identified several factors, including hyperuricemia, admission serum calcium, body mass index, the synergistic effect of underweight and dementia, and an elevated apoB/apoA-1 ratio, as potential predictors of in-hospital mortality in patients with AMI[3–7]. For emergency physicians, prompt recognition and treatment of acute myocardial infarction are essential to lower in-hospital mortality in patients with AMI. Furthermore, it is critical to evaluate the prognosis of patients with AMI using available predictive indicators.

In recent years, nomograms derived from statistical prediction models have been widely used for the assessment of clinical events[8]. A well-performing nomogram can facilitate the evaluation of patient prognosis and inform clinical decision-making. Although numerous clinical risk models for in-hospital

mortality in AMI patients have been developed, few nomograms are specifically constructed to predict in-hospital mortality in AMI patients with LAD occlusion. This study aimed to identify potential risk factors for in-hospital mortality in AMI patients with LAD occlusion and construct a nomogram for predicting their prognosis.

Subjects and methods

Subjects

- According to the inclusion and exclusion criteria, 454 AMI patients with LAD occlusion admitted to the Second Affiliated Hospital of Anhui Medical University between January 2018 and December 2025 were retrospectively enrolled. All treatments for AMI patients in this study were performed in accordance with the latest published guidelines[9]. This study was approved by the Ethics Committee of the Second Affiliated Hospital of Anhui Medical University, and informed consent was waived owing to its retrospective design.
- The inclusion criteria were as follows: (1) conformance to the diagnostic criteria for acute myocardial infarction[9]; (2) presence of left anterior descending artery occlusion confirmed by coronary angiography. The exclusion criteria were as follows: (1) thrombolytic therapy before primary PCI in a local hospital; (2) chronic total occlusion of the LAD; (3) previous myocardial infarction with a history of LAD revascularization.

Data collection

- Clinical indicators of AMI patients at admission were extracted from the hospital's electronic medical record system. The collected data included age, gender, hypertension history, diabetes mellitus history, smoking history, drinking history, systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), symptom-to-door time, C-reactive protein (CRP), white blood cells (WBC), platelets, hemoglobin, random blood glucose, cardiac troponin I (cTnI), creatine kinase MB (CKMB), total cholesterol, triglyceride, high-density lipoprotein (HDL), low-density lipoprotein (LDL), carbon dioxide combining power (CO₂CP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin, blood urea nitrogen (BUN), creatinine, uric acid, potassium, lactate dehydrogenase (LDH), fibrinogen (FIB), albumin-to-globulin ratio (A/G ratio).

Statistical analysis

- SPSS 26.0 and R software (version 4.5.3) were used for statistical analysis. Normally distributed continuous variables were expressed as mean $\pm$ SD and compared with the independent samples t-test. Non-normally distributed continuous variables were expressed as medians and interquartiles range (IQRs) and compared with Mann-Whitney U-test. Categorical variables were expressed as frequency (percentage) and compared with the Chi-Squared test. The dataset was randomly divided

into a training set for nomogram construction and a validation set for model validation. LASSO regression and random forest algorithms were applied to screen potential risk factors among 32 candidate variables in training set. Variables screened by LASSO regression and RF algorithms were entered into multivariate logistic analysis with backward stepwise regression to construct the final model. A nomogram predicting in-hospital mortality in AMI patients with LAD occlusion was constructed with the 'regplot' package of R software. The performance of the nomogram was assessed by the receiver operating characteristic (ROC) curve, calibration curve, and decision curve analysis (DCA) in the validation set. A value of $P < 0.05$ was considered statistically significant.

Results

Patients characteristic

- A total of 549 AMI patients with LAD occlusion were screened for this study. Ninety-five patients were excluded for the following reasons: thrombolytic therapy before primary PCI in a local hospital ($n = 30$), chronic total occlusion of the LAD ($n = 20$), previous myocardial infarction with a history of LAD revascularization ($n = 45$). In total, 454 AMI patients with LAD occlusion were enrolled in this study (Fig. 1). The cohort was randomly divided into a training set (70%, $n = 318$) and a validation set (30%, $n = 136$). Statistical analyses comparing the training and validation sets were shown in Table 1. Patients in the training set exhibited a higher heart rate as well as lower levels of CKMB, HDL and ALT relative to those in the validation set ($p < 0.05$). The characteristics of the death and survival groups in the training set were compared in Table 2. Patients in the death group exhibited higher age, heart rate, symptom-to-door time, CRP, WBC, random blood glucose, cTnI, CKMB, LDL, ALT, AST, BUN, creatinine, uric acid, potassium and LDH, and lower levels of SBP, DBP, platelets, hemoglobin, CO2CP, FIB and A/G ratio compared with patients in the survival group ($p < 0.05$).

Table 1
Clinical characteristics between training and validation sets.

variables	Training set (n = 318)	Validation set (n = 136)	p-value
Age (years)	64.00(52.00–75.00)	65.00(53.00–77.00)	0.441
Gender, male (n, %)	170(53.46%)	74(54.41%)	0.918
Hypertension history (n, %)	192(60.38%)	84(61.76%)	0.834
Diabetes mellitus history (n, %)	82(25.79%)	40(29.41%)	0.421
Smoking history (n, %)	174(54.72%)	77(56.62%)	0.758
Drinking history (n, %)	166(52.20%)	73(53.68%)	0.838
SBP (mmHg)	120.00(107.00–135.00)	123.00(109.00–141.00)	0.137
DBP (mmHg)	78.00(66.00–90.00)	78.00(70.00–86.00)	0.419
Heart rate (b.p.m.)	85.00(75.00–101.00)	82.00(70.00–96.00)	0.026
Symptom-to-door time (h)	5.00(2.00–12.00)	6.00(3.00–16.00)	0.050
CRP(mg/L)	5.30(1.50–25.35)	3.65(0.80–26.63)	0.107
WBC (10 ⁹ /L)	10.97(8.92–13.81)	11.05(8.90–13.65)	0.829
Platelets (10 ⁹ /L)	209.00(161.00–254.00)	210.00(162.00–265.00)	0.812
Hemoglobin (g/L)	139.00(123.00–154.00)	141.00(114.00–154.00)	0.541
Random blood glucose (mmol/L)	8.38(7.02–13.20)	8.90(7.10–13.00)	0.921
cTnl (ng/ml)	3.56(0.12–24.03)	4.92(0.48–32.10)	0.103
CKMB(U/L)	67.00(26.00–147.00)	115.00(45.00–306.00)	< 0.001
Total cholesterol (mmol/L)	4.90(4.16–5.42)	4.96(4.31–5.41)	0.655
Triglyceride (mmol/L)	1.47(0.99–2.21)	1.38(0.97–2.02)	0.445
HDL (mmol/L)	1.06(0.92–1.25)	1.20(0.98–1.38)	0.001
LDL (mmol/L)	3.30 ± 0.99	3.28 ± 0.99	0.832
CO ₂ CP(mmol/L)	22.70(19.90–24.80)	22.70(19.70–25.20)	0.654
ALT(U/L)	40.00(25.00–72.00)	49.00(30.00–86.00)	0.048
AST(U/L)	65.00(31.00–253.00)	96.00(31.00–280.00)	0.386
Total bilirubin (μmol/L)	13.65(9.98–19.30)	13.45(9.33–18.70)	0.364

variables	Training set (n = 318)	Validation set (n = 136)	p-value
Blood urea nitrogen (mmol/L)	6.72(5.53–9.35)	6.77(5.05–9.11)	0.808
Creatinine (μmol/L)	80.00(66.00–103.00)	85.00(66.00–116.00)	0.324
Uric acid (μmol/L)	372.00(303.00–451.00)	357.00(302.00–450.00)	0.794
Potassium (mmol/L)	3.98(3.67–4.32)	4.01(3.69–4.37)	0.668
Lactate dehydrogenase (U/L)	327.00(154.00–551.00)	352.00(193.00–570.00)	0.257
Fibrinogen (g/L)	3.11(2.47–4.04)	3.14(2.67–3.94)	0.555
A/G ratio	1.54 ± 0.38	1.48 ± 0.33	0.154

Table 2
Clinical characteristics between death and survival groups in the training set.

variables	Death group (n = 118)	Survival group (n = 200)	p-value
Age (years)	74.0(65.0–81.0)	57.0(48.0–68.0)	< 0.001
Gender, male (n, %)	68(57.63%)	102(51.00%)	0.295
Hypertension history (n, %)	68(57.63%)	127(63.50%)	0.341
Diabetes mellitus history (n, %)	30(25.42%)	52(26.00%)	0.910
Smoking history (n, %)	61(51.69%)	113(56.50%)	0.417
Drinking history (n, %)	57(48.30%)	109(54.50%)	0.298
SBP (mmHg)	106.00(87.75–125.25)	125.50(112.25–141.00)	< 0.001
DBP (mmHg)	69.30 ± 18.58	82.90 ± 13.10	< 0.001
Heart rate (b.p.m.)	101.00(78.75–116.25)	82.00(73.25–92.75)	< 0.001
Symptom-to-door time (h)	12.00(5.00–30.75)	3.00(2.00–7.75)	< 0.001
CRP(mg/L)	25.30(5.28–70.85)	3.50(1.20–9.18)	< 0.001
WBC (10 ⁹ /L)	12.82(9.80–17.19)	10.27(8.43–12.40)	< 0.001
Platelets (10 ⁹ /L)	193.00(145.00–234.75)	217.50(179.00–260.75)	< 0.001
Hemoglobin (g/L)	124.00(109.00–140.00)	146.00(133.00–158.00)	< 0.001
Random blood glucose (mmol/L)	12.92(8.57–16.30)	7.73(6.74–9.42)	< 0.001
cTnI (ng/ml)	35.27(7.23–59.23)	0.62(0.01–4.91)	< 0.001
CKMB(U/L)	123.00(50.00–188.00)	40.00(25.00–121.00)	< 0.001
Total cholesterol (mmol/L)	5.02(4.09–5.34)	4.84(4.19–5.53)	0.842
Triglyceride (mmol/L)	1.46(1.04–2.13)	1.47(0.97–2.36)	0.563
HDL (mmol/L)	1.03(0.93–1.32)	1.08(0.89–1.22)	0.575
LDL (mmol/L)	3.89(2.75–4.22)	3.14(2.48–3.82)	0.001
CO ₂ CP(mmol/L)	19.90(16.08–23.13)	23.65(21.40–25.20)	< 0.001
ALT(U/L)	63.50(29.00–113.50)	35.00(24.00–52.75)	< 0.001
AST(U/L)	216.50(47.75–458.00)	45.50(29.00–120.75)	< 0.001
Total bilirubin (μmol/L)	14.95(10.15–20.48)	13.10(9.93–18.60)	0.186

variables	Death group (n = 118)	Survival group (n = 200)	p-value
Blood urea nitrogen (mmol/L)	9.34(6.64–12.11)	6.19(5.15–7.37)	< 0.001
Creatinine (μmol/L)	106.00(82.00–163.00)	73.00(62.00–86.00)	< 0.001
Uric acid (μmol/L)	411.00(337.00–515.00)	349.00(295.00–423.00)	< 0.001
Potassium (mmol/L)	4.13(3.79–4.58)	3.92(3.62–4.23)	< 0.001
Lactate dehydrogenase (U/L)	601.00(375.00–1004.00)	230.00(132.00–354.00)	< 0.001
Fibrinogen (g/L)	2.85(2.06–3.94)	3.24(2.63–4.04)	0.003
A/G ratio	1.27 ± 0.34	1.69 ± 0.30	< 0.001

Screening for potential risk factors

For LASSO regression, the optimal tuning parameter λ determined by 3-fold cross-validation was 0.0201 according to the one standard error of the minimum criteria. At this optimal λ value, 15 variables were identified as risk factors for in-hospital mortality in AMI patients with LAD occlusion (Fig. 2 and Table 3). The importance plot and arithmetic mean of accuracy and Gini of variables in the random forest model were shown in Fig. 3. The top 8 important variables were identified as potential risk factors for in-hospital mortality in AMI patients with LAD occlusion, including cTnI, HR, LDH, A/G ratio, SBP, CRP, creatinine and age. Potential risk factors screened by LASSO regression and RF algorithms were entered into multivariate logistic analysis with backward stepwise regression to identify the most reliable predictors. The results indicated that age, SBP, HR, WBC, LDH, FIB and A/G ratio were significant independent risk factors for prognosis in AMI patients with LAD occlusion (Table 4).

Table 3
Results of LASSO regression in training set.

variables	LASSO regression analysis	
	Assignment	Coefficient
Age (years)	Continuous variable	0.0337
SBP (mmHg)	Continuous variable	-0.0202
DBP (mmHg)	Continuous variable	
Heart rate (b.p.m.)	Continuous variable	0.0157
Symptom-to-door time (h)	Continuous variable	0.0048
CRP(mg/L)	Continuous variable	0.0094
WBC (10 ⁹ /L)	Continuous variable	0.0593
Platelets (10 ⁹ /L)	Continuous variable	
Hemoglobin (g/L)	Continuous variable	-0.0093
Random blood glucose (mmol/L)	Continuous variable	0.0648
cTnl (ng/ml)	Continuous variable	0.0184
CKMB(U/L)	Continuous variable	
LDL (mmol/L)	Continuous variable	0.1138
CO ₂ CP(mmol/L)	Continuous variable	
ALT(U/L)	Continuous variable	
AST(U/L)	Continuous variable	
Blood urea nitrogen (mmol/L)	Continuous variable	0.0349
Creatinine (μmol/L)	Continuous variable	
Uric acid (μmol/L)	Continuous variable	0.0007
Potassium (mmol/L)	Continuous variable	
Lactate dehydrogenase (U/L)	Continuous variable	0.0014
Fibrinogen (g/L)	Continuous variable	-0.2625
A/G ratio	Continuous variable	-1.7612

Table 4
Multivariate logistic analysis in the training set.

variables	<i>B</i>	<i>SE</i>	<i>P</i>	<i>OR</i>	<i>95%CI</i>
Age (years)	0.058	0.027	0.031	1.060	1.005–1.117
SBP (mmHg)	−0.039	0.012	0.001	0.961	0.939–0.985
Heart rate (b.p.m.)	0.043	0.015	0.004	1.044	1.014–1.075
Symptom-to-door time (h)	0.018	0.024	0.470	1.018	0.970–1.067
CRP(mg/L)	0.024	0.012	0.051	1.024	1.000–1.049
WBC (10 ⁹ /L)	0.140	0.067	0.037	1.150	1.008–1.312
Hemoglobin (g/L)	−0.020	0.014	0.156	0.980	0.953–1.008
Random blood glucose (mmol/L)	0.077	0.059	0.187	1.080	0.963–1.212
cTnl (ng/ml)	0.026	0.017	0.123	1.026	0.993–1.060
LDL (mmol/L)	0.394	0.271	0.146	1.483	0.872–2.522
Blood urea nitrogen (mmol/L)	−0.001	0.108	0.995	0.999	0.809–1.234
Creatinine (μmol/L)	−0.005	0.005	0.343	0.995	0.986–1.005
Uric acid (μmol/L)	0.002	0.002	0.306	1.002	0.998–1.007
Lactate dehydrogenase (U/L)	0.002	0.001	0.036	1.002	1.000–1.004
Fibrinogen (g/L)	−0.846	0.256	0.001	0.429	0.260–0.709
A/G ratio	−3.066	1.062	0.004	0.047	0.006–0.374

Construction of nomogram

A nomogram was constructed based on risk factors identified by multivariate logistic analysis in the training set (Fig. 4). Scores for the seven variables (age, SBP, HR, WBC, LDH, FIB and A/G ratio) were summed to yield a total score, from which the predictive probability of in-hospital mortality in AMI patients with LAD occlusion was derived. For example, an AMI patient with HR 79 b.p.m, FIB 2.99g/L, WBC 12.2*10⁹/L, SBP 152mmHg, age 65, A/G ratio 1.2, and LDH 277U/L achieved a total nomogram score of 266, corresponding to an in-hospital mortality of 0.171.

Evaluation of nomogram

The ROC curve for in-hospital mortality in AMI patients with LAD occlusion was plotted to assess the accuracy of the nomogram (Fig. 5A). In the validation set, the area under the curve (AUC) of the

nomogram was 0.953 (95%CI: 0.918–0.989), suggesting high accuracy and favorable discrimination of the nomogram. Calibration of the nomogram was assessed using the Hosmer-Lemeshow test in the validation set ($\chi^2=12.454$, $p=0.132$). The calibration curve was further plotted and the apparent and bias-corrected curves closely approximated the ideal curve (Fig. 5B), suggesting satisfactory calibration performance of the prediction model. The DCA curve was plotted in the validation set to assess the clinical utility of the nomogram (Fig. 5C), and the results demonstrated its favorable clinical utility for predicting in-hospital mortality in AMI patients with LAD occlusion.

Discussion

- Patients with acute myocardial infarction frequently exhibit a poor prognosis, with an overall in-hospital mortality rate of 14.7%[10]. As a visualized, intuitive, simple and widely applied prediction model, the nomogram can assess patient prognosis and facilitate clinical decision-making[11]. Therefore, identifying risk factors for in-hospital mortality in AMI patients and developing a nomogram for predicting this outcome are essential for personalized clinical decision-making, and may assist clinicians in assessing patient prognosis.
- In this study, we constructed and validated a novel nomogram incorporating seven clinical parameters (HR, FIB, WBC, SBP, Age, A/G ratio, LDH) to assess the risk of in-hospital mortality in AMI patients with LAD occlusion. The nomogram achieved high accuracy and favorable discrimination, with an AUC value of 0.953 (95%CI: 0.918–0.989). The calibration curve showed satisfactory calibration performance, and the DCA curve revealed its good clinical practicality.
- Several studies have demonstrated that a lower left ventricular ejection fraction (LVEF) is an independent risk factor for increased mortality after acute myocardial infarction[12–14]. In this study, we found that LVEF serves as an independent predictor for assessing the prognosis of AMI patients with LAD occlusion. In the training set, OR = 0.777 (95% CI: 0.728–0.830) and AUC = 0.971 (95% CI: 0.954–0.989); in the validation set, OR = 0.766 (95%CI: 0.687–0.854) and AUC = 0.975 (95% CI: 0.946–1.000). Therefore, we excluded LVEF in the process of screening for risk factors. Cardiac troponin I (cTnI) was excluded from the risk factors screened by multivariate logistic analysis in this study. We speculate that the possible reason is that serum cTnI levels were normal in some death AMI patients in clinical practice.
- Elevated heart rate following acute myocardial infarction increases myocardial oxygen consumption and worsens myocardial ischemia, which in turn raises the risk of malignant arrhythmia, acute heart failure and cardiac rupture. Previous studies have found that a higher heart rate after AMI is significantly associated with in-hospital or 3-year mortality in AMI patients[15–17]. Differing from prior studies investigating the general AMI population, this study further confirmed that heart rate is an independent risk factor for in-hospital mortality in AMI patients with LAD occlusion. This finding suggests that targeted heart-rate monitoring and early individualized heart-rate control strategies are particularly critical for AMI patients with LAD occlusion in clinical practice, and these measures may help reduce in-hospital mortality and improve the prognosis of AMI patients.

- Fibrinogen is a critical component of the coagulation cascade, and exerts profound effects on blood viscosity and platelet aggregation[18]. Prior studies have indicated that fibrinogen contributes substantially to atherosclerotic plaque progression by modulating endothelial cell function[19], and serves as prospective factor of cardiovascular events risk[20]. Moreover, the fibrinogen to HDL-cholesterol ratio has been verified as an independent indicator of all-cause and cardiovascular mortality in AMI patients in previous research[21]. However, the prognostic value of single serum fibrinogen in AMI patients with LAD occlusion remains poorly elucidated. In the present study, we identified the serum fibrinogen as a risk factor for in-hospital mortality in this specific patient cohort. Compared with complex composite biomarkers, single-marker fibrinogen testing is simple, cost-effective, and rapidly available in clinical practice. Our findings add to the predictive value of fibrinogen in subgroup AMI patients, suggesting that early serum fibrinogen measurement may help clinicians rapidly stratify mortality risk of LAD-occluded AMI patients and inform individualized early intervention.
- White blood cells, a key component in the inflammatory cascade, plays an important role in the initiation and progression of atherosclerosis as well as acute myocardial infarction. Leukocytes are transported to systemic vessels throughout bloodstream, where they not only permeate the endothelial layer and trigger rupture of atherosclerosis plaque, but also intensify the inflammatory cascade following acute myocardial infarction[22, 23]. Some studies have confirmed the prognostic value of WBC for in-hospital mortality in AMI patients[24, 25]. Consistent with prior studies, WBC was identified as a risk factor for in-hospital mortality in AMI patients with LAD occlusion. Notably, most previous studies only validated the predictive effect of WBC on the prognosis of overall AMI patients, without performing subgroup analysis according to culprit vessel types. This study further refined the research population and confirmed the unique prognostic value of WBC in AMI patients with LAD occlusion, expanding the application scope of inflammatory markers for AMI risk stratification.

A protective effect was found between systolic blood pressure (SBP) and in-hospital mortality in AMI patients with LAD occlusion. After adjusting for confounding factors, SBP was identified as a protective factor for in-hospital mortality (OR = 0.961, 95% CI:0.939–0.985, $p = 0.001$), indicating that each 1-mmHg increment in SBP was associated with a 3.9% reduction in the risk of in-hospital mortality. The favorable prognostic value of elevated SBP in this specific population can be explained by underlying pathophysiological characteristics of LAD occlusion. As the primary coronary artery supplying the left ventricular anterior wall and interventricular septum, LAD occlusion is prone to cause severe left ventricular systolic dysfunction and cardiac hypoperfusion. A relatively higher SBP can maintain effective coronary perfusion pressure and systemic organ blood supply, alleviate myocardial ischemia and hypoperfusion-related secondary organ injury, and ultimately reduce the risk of in-hospital mortality. Consistent with this study, a prior study has reported that AMI patients maintaining a SBP between 90 and 130 mmHg exhibit a better long-term prognosis[26]. In contrast, AMI patients with persistently low SBP have an increased risk of in-hospital mortality[27]. In summary, maintaining normal SBP is very important for AMI patients in clinical practice.

- The relationship between age and the prognosis of AMI patients has been widely discussed. Previous studies have identified advanced age as an independent risk factor for poor prognosis in AMI patients[28, 29]. Another study reported that age ≥ 75 years was identified as a predictor of in-hospital cardiac arrest in patients with ST-segment elevation myocardial infarction[30]. In the present study, we identified age ≥ 69 years as a risk factor for in-hospital mortality in AMI patients with LAD occlusion. We speculate that this difference may be partly explained by the younger onset of acute myocardial infarction in clinical practice. From a clinical perspective, close monitoring should be emphasized for LAD-occluded AMI patients aged over 69 years to prevent poor outcomes.

As a major plasma protein in human physiology, serum albumin performs numerous physiological functions, including maintenance of colloid osmotic pressure, substance transportation, as well as antioxidant and anti-inflammatory activities[31]. Serum globulin, which is mainly synthesized by immune cells and the liver, performs physiological functions including immune defence, binding and transporting various substances, and participating in inflammatory responses[32]. Previous studies have demonstrated that either serum albumin or globulin may serve as a prognostic marker for AMI patients[33–35]. However, the prognostic value of the albumin-to-globulin ratio (A/G ratio) in AMI patients with LAD occlusion remains unclear. In this study, we found that the A/G ratio is a risk factor for in-hospital mortality in this specific patient population. Compared with individual albumin or globulin indices, the A/G ratio overcomes the limitations of single-index assessment and provides a more comprehensive reflection of the overall physiological status in AMI patients. As a routine, cost-effective and readily accessible serological marker in clinical settings, the A/G ratio possesses considerable clinical value for early risk stratification, timely intervention and prognostic evaluation in AMI patients with LAD occlusion.

Lactate dehydrogenase (LDH), an important enzyme involved in energy metabolism, is widely expressed in blood cells, hepatocytes and cardiomyocytes and is released upon tissue injury and inflammatory responses. Prior studies have reported that serum LDH seems to be a promising predictor of cardiac insufficiency or left ventricular remodeling in patients with acute myocardial infarction[36, 37]. Consistent with previous studies, we identified serum LDH as a risk factor for in-hospital mortality in AMI patients with LAD occlusion. As a retrospective observational study, this work only indicates an association rather than a causal relationship between serum LDH and in-hospital mortality. Collectively, we speculate that elevated serum LDH levels carry important clinical implications for risk stratification in patients with severe AMI, and may serve as an auxiliary laboratory marker when integrated with cTnI, B-type natriuretic peptide and other clinical variables.

This study possesses several strengths. First, we targeted a specific subgroup of AMI patients with LAD occlusion, enhancing the relevance and clinical significance of the findings. Second, we employed LASSO regression and random forest algorithms to screen potential risk factors, minimizing the risk of overlooking important variables. Third, this is a novel nomogram for predicting in-hospital mortality in AMI patients with LAD occlusion, offering a visually intuitive, individualized, comprehensive, and

quantitative approach to risk assessment. However, several limitations of this study should be acknowledged. First, only internal validation was performed, and external validation using multicenter cohorts was absent. Accordingly, the generalizability of this nomogram across different clinical settings remains unclear. Second, this study only focused on in-hospital mortality; long-term prognostic outcomes following discharge were not analyzed, which limits the evidence supporting long-term clinical management. Third, the retrospective study design inevitably introduced potential selection bias and residual confounding factors, which could affect the stability and reliability of our results.

In conclusion, a well-performing nomogram was successfully constructed for predicting in-hospital mortality in AMI patients with LAD occlusion and was internally validated with high predictive accuracy and clinical practicability. The nomogram effectively assists clinicians in conducting risk stratification and implementing early interventions for AMI patients with LAD occlusion.

Abbreviations

AMI Acute myocardial infarction

LM Left main artery

LAD Left anterior descending artery

LCX Left circumflex artery

RCA Right coronary artery

PCI Percutaneous coronary intervention

CABG Coronary artery bypass grafting

SBP Systolic blood pressure

DBP Diastolic blood pressure

HR Heart rate

CRP C-reactive protein

WBC White blood cells

cTnI Cardiac troponin I

CKMB Creatine kinase MB

HDL High-density lipoprotein

LDL Low-density lipoprotein

CO₂CP Carbon dioxide combining power

ALT Alanine aminotransferase

AST Aspartate aminotransferase

BUN Blood urea nitrogen

LDH Lactate dehydrogenase

FIB Fibrinogen

ROC Receiver operating characteristic

DCA Decision curve analysis

AUC Area under the curve

Declarations

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Authors' contributions

Chuan-De Zou: Writing—original draft, Visualization, Data curation, Conceptualization. **Lian-Lian Fang:** Writing—review & editing, Methodology, Data curation. **Dong-Lin Wang:** Methodology, Data curation. **Qian Tang:** Software, Formal analysis, Data curation. **He-Ping Zuo:** Writing—original draft, Supervision, Data curation.

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Data availability

The datasets used and analyzed in this study are available from the corresponding author upon reasonable request.

Clinical trial number

Not applicable

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Second Affiliated Hospital of Anhui Medical University, and informed consent was waived owing to its retrospective design.

Consent for publication

Not applicable

Competing interest

The authors declare no conflicts of interest.

Author details

^a Department of Emergency Internal Medicine, The Second Affiliated Hospital of Anhui Medical University, Hefei, China

^b Department of Emergency Internal Medicine, The First Affiliated Hospital of Anhui Medical University, Hefei, China

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Figures

Figure1

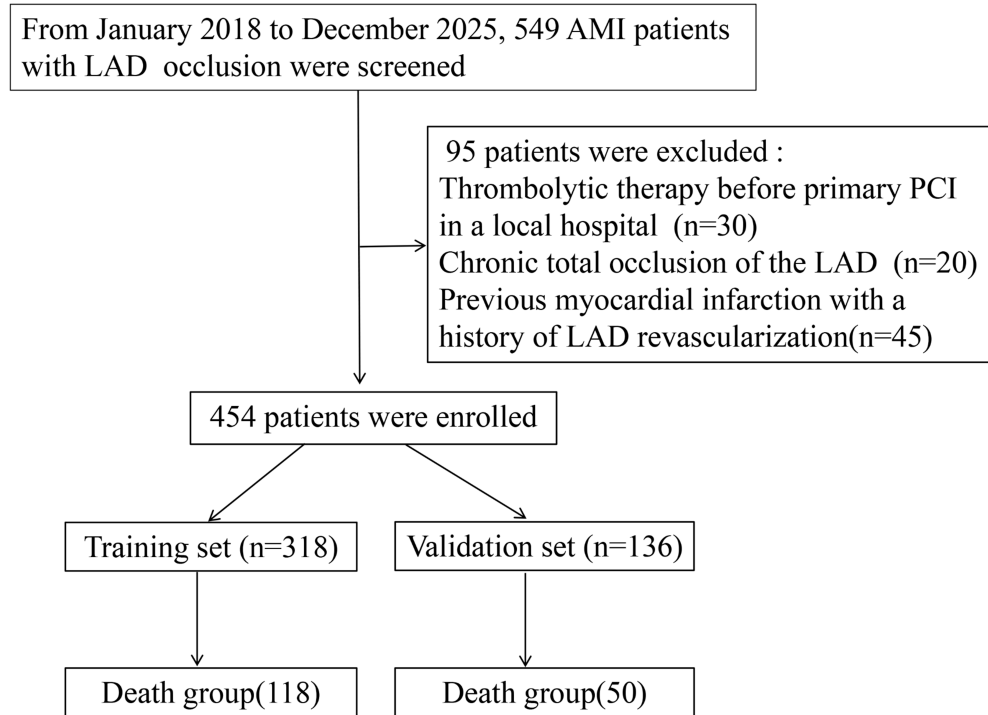


Figure 1

Flowchart for participants screening.

Figure2

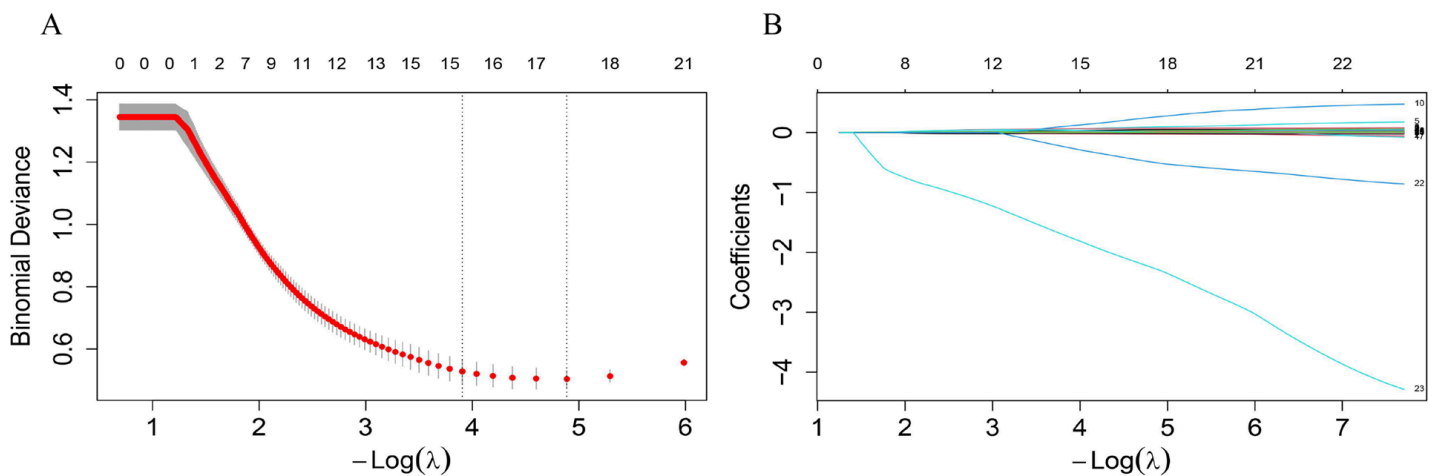


Figure 2

Variable selection using LASSO analysis. (A) Selection of tuning parameter (λ) using 3-fold cross-validation. (B) LASSO coefficient profiles of variables.

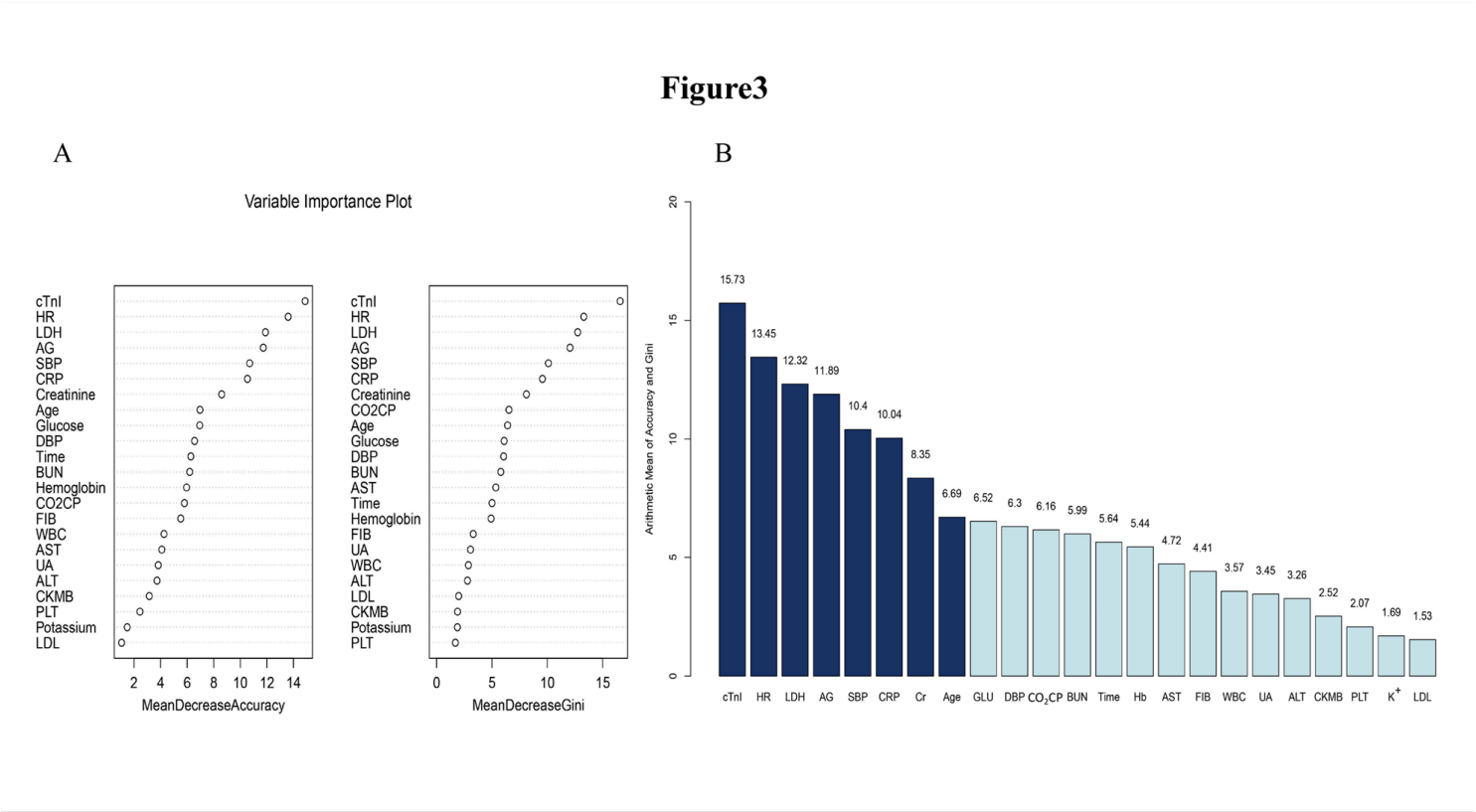


Figure 3

Results of the random forest model. (A) Mean Decrease Accuracy and Mean Decrease Gini Index for all variables. (B) Arithmetic mean of Mean Decrease Accuracy and Mean Decrease Gini Index across all variables.

Figure4

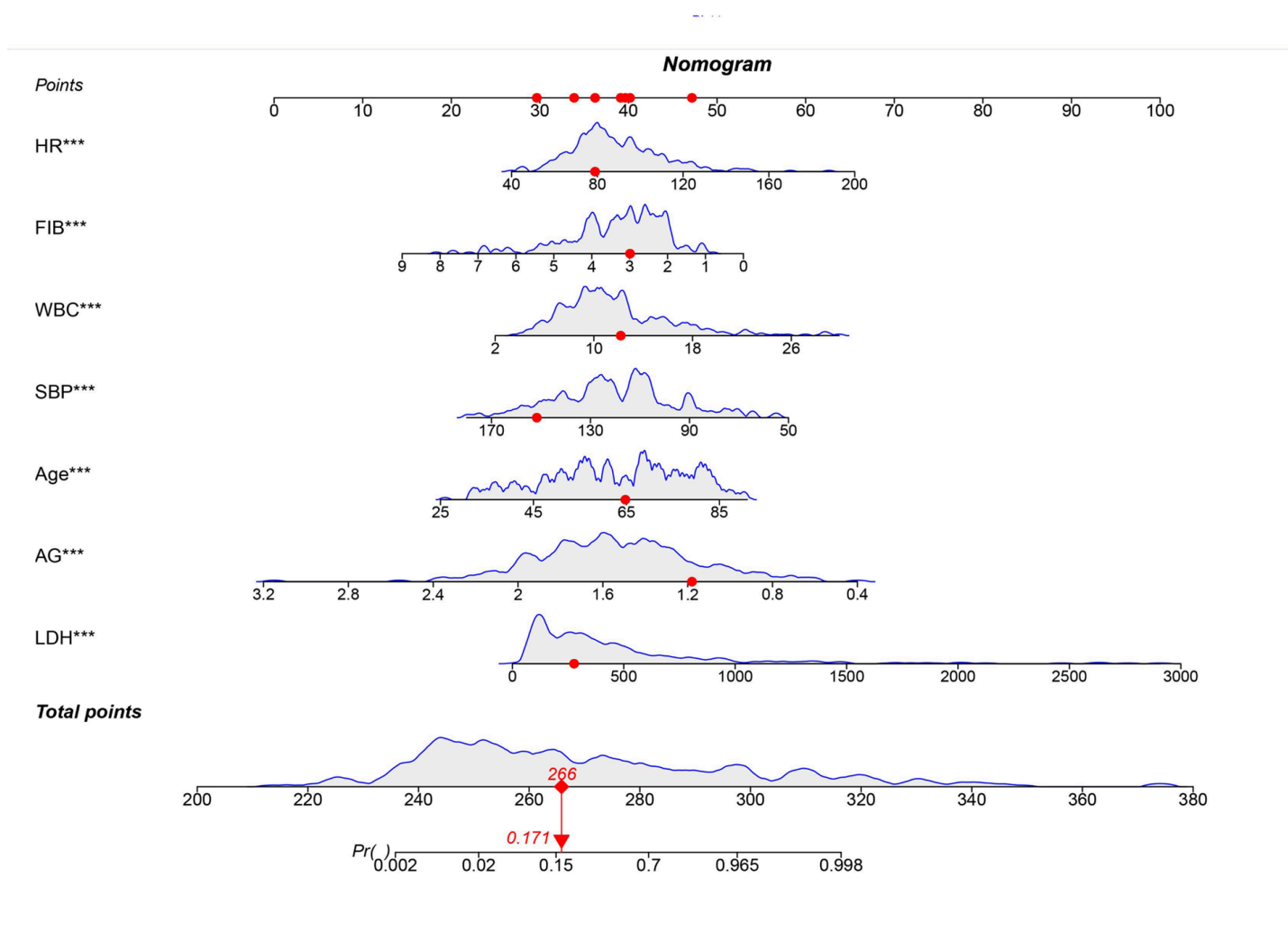


Figure 4

Nomogram for predict in-hospital mortality in AMI patients with LAD occlusion.

Figure5

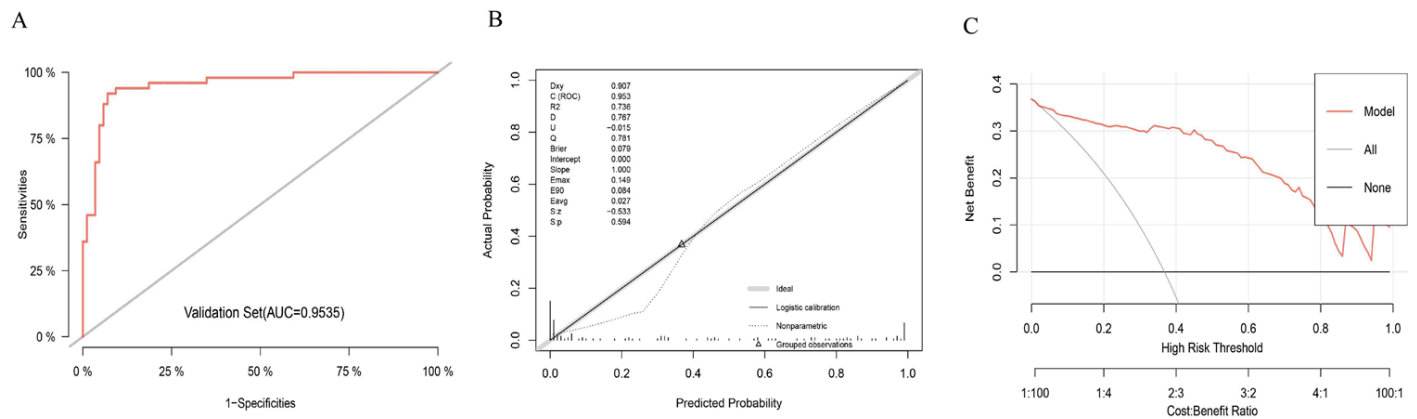


Figure 5

Evaluation of the nomogram. Receiver operating characteristic curve (A), calibration curve (B), and decision curve (C) for the nomogram.