


BMJ Open Development of a nomogram for the prediction of in-hospital mortality in patients with acute ST-elevation myocardial infarction after primary percutaneous coronary intervention: a multicentre, retrospective, observational study in Hebei province, China

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ABSTRACT

Objectives To establish a clinical prognostic nomogram for predicting in-hospital mortality after primary percutaneous coronary intervention (PCI) among patients with ST-elevation myocardial infarction (STEMI).

Design Retrospective, multicentre, observational study.

Setting Thirty-nine hospitals in Hebei province.

Participants Patients with STEMI who underwent PCI from January 2018 to December 2019.

Interventions A multivariable logistic regression model was used to identify the factors associated with in-hospital mortality, and a nomogram was established using these factors. The performance of the nomogram was evaluated by the discrimination, calibration and clinical usefulness.

Primary and secondary outcome measures The outcome was the factors associated with in-hospital mortality.

Results This study included 855 patients, among whom 223 died in hospital. Age, body mass index, systolic pressure on admission, haemoglobin, random blood glucose on admission, ejection fraction after PCI, use aspirin before admission, long lesions, thrombolysis in myocardial infarction flow grade and neutrophils/lymphocytes ratio were independently associated with in-hospital mortality (all $p < 0.05$). In the training set, the nomogram showed a C-index of 0.947, goodness-of-fit of 0.683 and area under the receiver operating characteristic curve (AUC) of 0.947 (95% CI 0.927 to 0.967). In the testing set, the C-index was 0.891, goodness-of-fit was 0.462 and AUC was 0.891 (95% CI 0.844 to 0.939). The results indicate that the nomogram had good discrimination and good prediction accuracy and could achieve a good net benefit.

Conclusions A nomogram to predict in-hospital mortality in patients with STEMI after PCI was developed and validated in Hebei, China and showed a satisfactory performance. Prospective studies will be necessary to confirm the performance and clinical applicability and practicality of the nomogram.

Strengths and limitations of this study

- This is a multicentre study, included 39 tertiary centres and 855 patients, including 223 (26.1%) patients who died in the hospital.
- The data were obtained retrospectively, and some patients died during the percutaneous coronary intervention, which may have led to some missing information.
- Prospective studies will be necessary to confirm the performance and clinical applicability and practicality of the nomogram.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI), a type of coronary artery disease (CAD), is a common clinical emergency and critical illness.¹ STEMI is most often caused by plaque rupture of an atherosclerotic lesion in the affected (culprit) coronary artery followed by total occlusion of the vessel lumen with a thrombus.^{2,3} Common risk factors for STEMI are tobacco abuse, dyslipidaemias, hypertension, diabetes mellitus and a family history of CAD.⁴ In recent years, with well-established diagnosis and treatment guidelines, continuous standardisation of the treatment of STEMI, increasing evidence of determinants of patient prognosis and development of emerging technologies, there has been a considerable reduction in STEMI mortality; still, mortality seems to have plateaued.³

Primary percutaneous coronary intervention (PCI) has become the preferred reperfusion strategy in patients with STEMI according

to the current clinical guidelines for STEMI in the USA and Europe.^{5 6} Nevertheless, even if such patients receive timely PCI and/or appropriate antiplatelet drugs, the prognosis is still unsatisfying, and a substantial number of STEMI patients still die in-hospital after PCI (about 6%).^{3 7 8} Therefore, there is still room for improving the short-term outcomes of these patients on top of a timely PCI.

Various studies examined the risk factors of short and long-term mortality of STEMI patients after PCI.^{9–11} Guidelines encourage the use of clinical scores such as the thrombolysis in myocardial infarction (TIMI) or The Global Registry of Acute Coronary Events for STEMI to assess early and long-term risk.^{5 6 12} Several biomarkers have been reported to confer independent prognostic information after STEMI, including Cardiac Troponin, brain natriuretic peptide (BNP), amino-terminal pro-BNP, and D-dimer.^{13–16} Unfortunately, these studies often exclude patients with advanced age, liver or kidney dysfunction, and other comorbidities and complications. The generalisability of those studies is limited, and it is difficult to summarise and reflect the real-world treatment situation comprehensively.

Therefore, the objective of this study was to develop a clinical nomogram for predicting in-hospital mortality of patients with STEMI after PCI. The results could provide clinical guidance and improve the outcome of STEMI patients.

PATIENTS AND METHODS

Study design and patients

This multicentre, retrospective, observational study included STEMI patients treated with PCI at 39 PCI hospitals in Hebei province from January 2018 to December 2019. The cohort was divided into a training set and a time-independent validation set. The training set refers to the use of modelled data to verify the predictive effect of the model, while test set is to use another group of patients' data (namely external data) to verify the prediction accuracy of the model. The training set patients enrolled from January 2018 to December 2018 and the testing set patients enrolled from January 2019 to December 2019.

All patients met the diagnostic criteria of acute STEMI based on their symptoms and/or ECG, myocardial damage markers and other test results and underwent primary PCI according to the 2017 ESC guidelines for the management of STEMI,⁵ namely with persistent chest discomfort or other symptoms suggestive of ischaemia and ST-segment elevation in at least two contiguous leads. Patients with non-STEMI or unstable angina or STEMI patients who did not undergo PCI were excluded. Patients who were readmitted to the hospital for revascularisation of non-culprit vessel were also excluded. The treatment strategy after PCI of surviving patients is determined by the doctor in charge in accordance with relevant guidelines.

The study was conducted according to the tenets of the Declaration of Helsinki for Medical Research Involving Human Subjects and Good Clinical Practice.

Patient and public involvement

Patients or the public were not involved in the design or reporting or dissemination plans of our research as this study is a retrospective, observational study.

Definitions

Long lesions was defined as the stenosis that has as $\geq 50\%$ reduction and more than 20 mm in luminal diameter.¹⁷

Residual stenosis was defined as $> 30\%$ residual stenosis of the target lesion after PCI.

Bleeding was defined as a composite of major bleeding according to Bleeding Academic Research Consortium Definition for Bleeding type 3 or 5, but was not related to coronary-artery bypass grafting.¹⁸

Major adverse cardiovascular event (MACE) refers to a combined or composite clinical endpoint that is used for outcome evaluations in clinical trials for cardiovascular research.

Acute coronary syndrome (ACS) is a term used to describe a range of conditions associated with sudden, reduced blood flow to the heart.

Data collection

Demographics (age, sex, and body mass index (BMI)), medical history (hypertension, diabetes mellitus, atrial fibrillation (AF), hyperlipidaemia and family history of CAD, stroke, renal failure and peripheral artery disease), angiographic characteristics and information of cardiac procedures (disease condition, TIMI flow grade, number of stents, use of intra-aortic balloon pump (IABP), use of temporary pacemaker, use of ventilator and whether there was no-reflow, coronary perforation and cardiac arrest), medications on admission (antiplatelet agents, β -blockers, nitrate, ACE inhibitors (ACEI), angiotensin receptor blockers (ARB) and statin), biochemical markers (neutrophils/lymphocytes, N/L ratio), haematocrit, haemoglobin (HGB), platelets (PLT) and random blood glucose on admission) and left ventricular ejection fraction (LVEF) after PCI were extracted from the medical charts. All treatments were according to the current guidelines.

Nomogram construction

Demographics, medical history, vital signs before and after PCI, and auxiliary examinations were evaluated using univariable logistic regression. Variables with $p < 0.05$ in the univariable logistic analyses were included for multivariable logistic analysis and nomogram construction. Receiver operator characteristic (ROC) curve analysis was used to quantify the prediction performance of the nomogram. A calibration curve was used to evaluate the calibration of the nomogram, and its goodness-of-fit was assessed using the Hosmer-Lemeshow test. Finally, the clinical usefulness of the nomogram was accessed using a decision curve analysis (DCA).

Statistical analysis

Statistical analyses were performed using R V.4.0.3 (R Foundation for Statistical Computing) with RStudio (V.1.3.959; RStudio, Auckland, New Zealand). R packages used in this study were rms, reader, tableone, pROC, ResourceSelection and rmda. The predictive accuracy of the nomogram was measured using the C-statistic (Bootstrap method, 1000 times). Calibration was evaluated using the Hosmer-Lemeshow statistic. Categorical variables were presented as frequencies with percentages, normally distributed continuous variables as means±SD, and other data as medians with IQRs. Categorical variables were compared using the χ^2 test or Fisher's test if the expected cell count was <5. Student's t-test was used to compare normally distributed continuous variables. Otherwise, the Mann-Whitney U test was used. The significance level was set at 0.05, and two-sided tests were used.

RESULTS

Characteristics of the patients

The whole study population consisted of 855 patients diagnosed with STEMI and who underwent PCI, including 396 in the training set (132 (33.3%) dead patients and, 264 (66.7%) survivors) and 459 (91 (19.8%) dead patients, 368 (80.2%) survivors) in the test set (figure 1). The clinical characteristics, including demographic, medical history, angiographic characteristics and information of cardiac procedures, medications and biochemical markers, are summarised in online supplemental table 1. The clinical characteristics selected as predictors for the nomogram are summarised in table 1. The patients who died in the hospital were older (69.8±10.2 vs 60.2±12.6 years, $p<0.01$), more likely to be women (32.7% vs 21.5%, $p<0.01$) and more had complications like hypertension, AF and hyperlipidaemia. The hospital stay was 8.51±5.11 days in the training set and 8.32±4.70 days in the test set.

Nomogram construction

According to the multivariable logistic analysis, 10 variables meet the threshold of $p<0.05$. Age (OR 1.069, 95% CI 1.048 to 1.092, $p=0.049$), BMI (OR 0.55, 95% CI 0.31 to 0.87, $p=0.019$), SBP on admission (OR 0.92, 95% CI 0.86 to 0.97, $p=0.009$), HGB (OR 0.85, 95% CI 0.73 to 0.97, $p=0.017$), random blood glucose on admission (OR 1.53, 95% CI 1.13 to 2.21, $p=0.011$), EF after PCI (OR 0.89, 95% CI 0.80 to 0.97, $p=0.015$), aspirin (OR 0.001, 95% CI 0.009 to 0.04, $p=0.001$), N/L ratio (OR 1.34, 95% CI 1.12 to 1.69, $p=0.004$), long lesions (OR 2.00, 95% CI 1.310 to 3.084, $p<0.001$) and TIMI flow grade (OR 2.15, 95% CI 1.242 to 3.900, $p=0.008$) were independently associated with in-hospital mortality after PCI of STEMI (table 2). The nomogram is shown in figure 2. The formula for calculating the total point of the nomogram is showed below:

$$\text{Score} = 15.5628 + 0.0320 \times \text{age} - 0.2991 \times \text{BMI} - 0.0184 \times \text{SBP} - 0.0331 \times \text{HGB} + 0.3663 \times \text{random blood}$$

$$\text{glucose on admission} - 0.1188 \times \text{LVEF} \text{ after PCI} - 4.7705 \times \text{aspirin} + 0.0521 \times \text{N/L ratio} - 2.4688 \times \text{long lesions} + 5.1018 \times \text{TIMI flow grade}.$$

Evaluation of the nomogram

In the training set, the C-index was 0.947, indicating that the prediction model was valuable in clinical practice (figure 3A). The value of goodness-of-fit was 0.683, indicating a good prediction accuracy. The ROC curve is shown in figure 4A (area under the curve, AUC=0.947, 95% CI 0.927 to 0.967). Figure 5A shows the DCA curve for the training set, indicating that the nomogram had a high overall net benefit in predicting in-hospital mortality after PCI treatment.

In the testing set, the C-index was 0.891. Figure 3B shows the calibration curve, and the value of goodness-of-fit was 0.462. The ROC curve is shown in figure 4B (AUC 0.891, 95% CI 0.844 to 0.939). The DCA curve is shown in figure 5B. The results of the testing set indicate that the nomogram had good discrimination and good prediction accuracy which could achieve a good net benefit.

DISCUSSION

In this study, a relatively accurate clinical nomogram was constructed, which demonstrated adequate discrimination and calibration power to provide an individualised estimation for the in-hospital mortality in STEMI patients after PCI. For the construction of the nomogram, 10 significant predictors were screened by multivariable logistic analysis.

In our study, age was an independent risk factor of STEMI patients, in accordance with other analyses of STEMI patients and underlining the high-risk profile of elderly patients, as they usually present with more risk factors and comorbidities than younger patients,^{19 20} such as the higher prevalence of renal insufficiency, lower LVEF. High mortality in the older patients might also result from end-organ dysfunction, competing risks might also offset the benefits from reperfusion, such that successful outcomes are more dependent on overall health issues. Therefore, for older patients, some authors have also questioned the benefit of reperfusion therapy.²¹

For previous view, obesity increases insulin resistance, worsens plasma lipid profiles and increases arterial blood pressure, which has adverse effects on patients with CAD through the indirect effects of other risk factors (such as hypertension, impaired glucose tolerance and hyperinsulinaemia).²² Therefore, obese patients demonstrate greater adverse left ventricle (LV) remodelling and more impaired LV deformation after STEMI compared with those similar infarct characteristics but normal BMI.^{23 24} Interestingly, some studies have shown the so-called 'obesity paradox', whereby obesity is related to better clinical outcomes,^{22 25–27} consistent with this study. Fukuoka *et al*²⁸ reported that this phenomenon is only observed in elderly patients, not in younger patients, so the influence of BMI on risk factors for death might vary

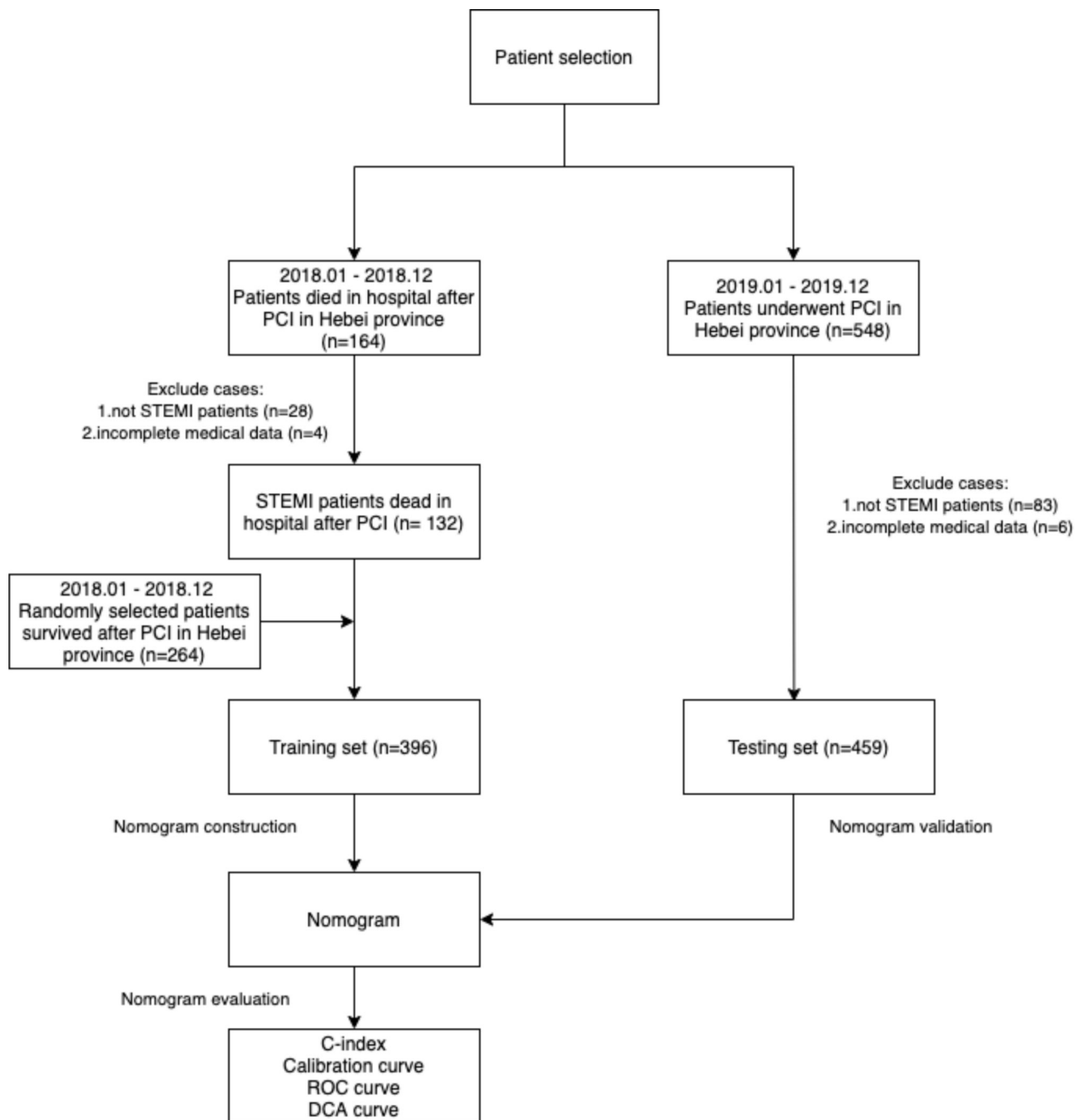


Figure 1 Flow chart illustrating the process of patient selection. DCA, decision curve analysis; PCI, percutaneous coronary intervention; ROC, receiver operator characteristic; STEMI, ST-elevation myocardial infarction.

with age. Nevertheless, obesity is currently recognised as a risk factor for the long-term prognosis of patients with CAD, and it is worth recommending maintaining BMI at a normal level.²⁸

Acute stress has been shown to regulate the immune response of lymphocytes and reduce the number of peripheral blood lymphocytes. The smaller the value, the higher the body's stress level. Therefore, the N/L ratio, an index for systemic inflammatory status, usually increases after STEMI.^{29–31} Pan *et al*³² demonstrated the independent association between increased N/L ratio and short-term mortality in STEMI patients after PCI.

The predictive value of the N/L ratio may be based on the following reasons. Stimulated neutrophils release superoxide radicals, proteolytic enzymes, and arachidonic acid metabolites that increase the infarct size and lead to cardiac electrical instability by damaging endothelial cells, activating coagulation cascade, aggregation of leukocytic cells and plugging the microarteries.³³ These actions will participate in the extension of the areas of myocardial infarction, impaired epicardial and microvascular perfusion, no-reflow/slow flow during PCI, decreased LVEF and postinfarction death.^{34–36}

Table 1 Clinical characteristics of the patients selected as predictors for the nomogram

Variables	Training set			Testing set			P value	P value
	All			All				
	(n=396)	Survival (n=264)	In-hospital mortality (n=132)	(n=459)	Survival (n=368)	In-hospital mortality (n=91)		
Age (years) (mean±SD)	63.3±12.7	60.3±12.9	69.3±9.8	62.1±12.8	59.8±12.4	70.2±11.3	<0.001	<0.001
BMI (kg/m ²)	25.8 (24.6, 26.1)	26.0 (25.3, 26.5)	24.9 (24.4, 25.5)	25.4 (23.4, 27.3)	25.5±3.0	25.3 (23.4, 27.5)	<0.001	0.047
SBP on admission (median (IQR))	128 (110–146)	133 (114–149)	118 (100–140)	125 (110–140)	129±25	121 (107–135)	<0.001	0.009
Long lesions (n (%))	245 (61.9)	178 (67.4)	67 (50.8)	194 (42.3)	131 (35.6)	63 (69.2)	0.002	<0.001
TIMI flow grade 0–1 before PCI (n (%))	311 (78.5)	197 (74.6)	114 (86.4)	339 (73.9)	274 (74.5)	65 (71.4)	0.011	0.556
N/L ratio (median (IQR))	5.47 (2.82–10.00)	4.70 (2.68–7.87)	8.54 (3.19–11.46)	6.15 (3.48–9.52)	5.08 (3.65–9.46)	9.1 (3.81–12.51)	<0.001	<0.001
HGB, g/L (median (IQR))	137.0 (126.0–269.0)	142.0 (129.0–155.0)	129.0 (119.0–137.3)	137.2±19.8	138.5±19.1	131.9±21.5	<0.001	0.004
Random blood glucose on admission, mmol/L (median (IQR))	6.84 (5.47–9.92)	5.95 (5.02–7.44)	9.81 (7.96–11.04)	6.73 (5.27–10.10)	6.12 (5.10–8.10)	10.96 (8.40–11.78)	<0.001	<0.001
EF after PCI (median (IQR))	51.0 (43.0–58.0)	54.0 (47.8–59.0)	43.0 (38.0–48.5)	55 (46–60)	56 (51–61)	45 (37–53)	<0.001	<0.001
Use Aspirin on admission (n (%))	379 (95.7)	262 (99.2)	117 (88.6)	404 (88.0)	332 (90.2)	72 (79.1)	<0.001	0.004

BMI, body mass index; EF, ejection fraction; HGB, haemoglobin; PCI, percutaneous coronary intervention; N/L ratio, neutrophils/lymphocytes ratio; SBP, systolic blood pressure; TIMI, thrombolysis in myocardial infarction.

Table 2 Variables selected as predictors for the nomogram according to the multivariable logistic analysis

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	P value	OR	95% CI	P value
Age	1.07	1.05 to 1.09	<0.001	1.07	1.05 to 1.09	0.049
BMI	0.79	0.70 to 0.87	<0.001	0.55	0.31 to 0.87	0.019
SBP on admission	0.98	0.97 to 0.99	<0.001	0.92	0.86 to 0.97	0.009
HGB	0.97	0.95 to 0.98	<0.001	0.85	0.73 to 0.97	0.017
Random blood glucose on admission	1.38	1.27 to 1.51	<0.001	1.53	1.13 to 2.21	0.011
EF after PCI	0.91	0.88 to 0.93	<0.001	0.89	0.80 to 0.97	0.015
Use aspirin before admission	0.06	0.01 to 0.22	<0.001	0.01	0.009 to 0.04	0.001
N/L ratio	1.08	1.04 to 1.12	<0.001	1.34	1.12 to 1.69	0.004
Long lesions	0.50	0.32 to 0.76	0.001	2.00	1.31 to 3.08	<0.001
TIMI flow grade 0–1 before PCI	2.15	1.24 to 3.90	<0.001	2.15	1.24 to 3.90	0.008

BMI, body mass index; EF, ejection fraction; HGB, haemoglobin; PCI, percutaneous coronary intervention; N/L ratio, neutrophils/lymphocytes ratio; SBP, systolic blood pressure; TIMI, thrombolysis in myocardial infarction.

The acute phase of STEMI leads to insulin resistance, glucose intolerance and hyperglycaemic. The elevated levels of cytokines, growth hormone, glucagon and cortisol result in increased hepatic glucose production. Hepatic glycogenolysis is further enhanced by catecholamines that also inhibit glycogenesis and stimulate the release of free fatty acids (FFAs). High concentrations of FFAs will increase myocardial oxygen requirement, reduce myocardial activity and contractility, impair calcium homeostasis and increase the production of free radicals, leading to an increased risk of myocardial damage and arrhythmias.^{37–40} Thus, acute hyperglycaemic might contribute to a poor outcome. Previous studies reported that higher admission glucose was strongly correlated with larger infarct size, lower LVEF, and increased mortality risk in patients with and without diabetes.^{41–42} Exercise training, dietary modifications, and intervention in the hospital, such as tight glycaemic control during early PCI or at least within 24 hours after STEMI might reduce the mortality risk in such patients.^{43–44}

Lower admission HGB was associated with higher in-hospital mortality when analysed as a continuous variable (OR 0.966, 95% CI 0.954 to 0.978). In the study from Shacham *et al*,⁴⁵ they revealed the longer total ischaemic time, namely an ongoing inflammatory process, the lower admission HGB levels. HGB levels and inflammation are closely related. In patients with STEMI, inflammation block occurs, that is, an abundance of hepcidin leads to poor uptake of iron from the gastrointestinal tract, iron sequestration in macrophages, little iron recycling to the erythron for red-cell production and microcytic anaemia, which can cause a lower HGB level.⁴⁶

Because of the important role of PLT in thrombus formation, this study showed that prior aspirin use could reduce in-hospital mortality of STEMI patients after PCI, as supported by earlier clinical trials.^{47–48} Weidmann *et al*⁴⁸ provided evidence suggesting that pre-existing treatment with aspirin favourably affected the clinical presentation,

infarct size and degree of inflammation of patients with STEMI. Yonetsu *et al*⁴⁹ reported that aspirin inhibits PLT aggregation and therefore reduces the probability of an occluding clot on top of a ruptured plaque and, conversely, the occurrence of STEMI.

Previous studies indicated that lesion length is associated with long-term adverse events after PCI and is an important risk factor for restenosis and stent thrombosis.^{50–52} A longer lesion, with its greater plaque burden, is conceived to provide a major source of smooth muscle cells that will then proliferate to form neointima. Atherosclerotic plaques have often been found to demonstrate an increased expression of isoforms characteristic of activated smooth muscle cells that are not present in normal vasculature.⁵³ Still, there are few studies on lesion length and in-hospital mortality, and further studies are still necessary. Preprocedural reperfusion might have a prognostic value.⁵⁴ A strong relationship exists between preprocedural TIMI flow grade and infarct size and predischage LVEF.⁵⁵ SBP is a critical factor, and hypotension was associated with a decrease in survival.⁵⁶

In our multivariate analysis, the higher Killip Class is not a predictor of in-hospital mortality in STEMI patients. However, in a recent work from Del Buono *et al*,⁵⁷ it was proved that a higher Killip Class is an independent risk factor for MACE events and in-hospital mortality in patients with anterior myocardial infarction. This is the first study including only patients with STEMI in the anterior location and excluding patients with history of cardiovascular diseases in order to reduce the heterogeneity of the population enrolled. This may be one of the reasons for the inconsistency of the two studies. Nevertheless, Killip classification is a simple and convenient clinical tool that can quickly stratify the risk of ACS patients and is likely to become an independent predictor of long-term follow-up results again.

The nomogram is a simple and intuitive representation of the mathematical model.⁵⁸ In addition, to be of

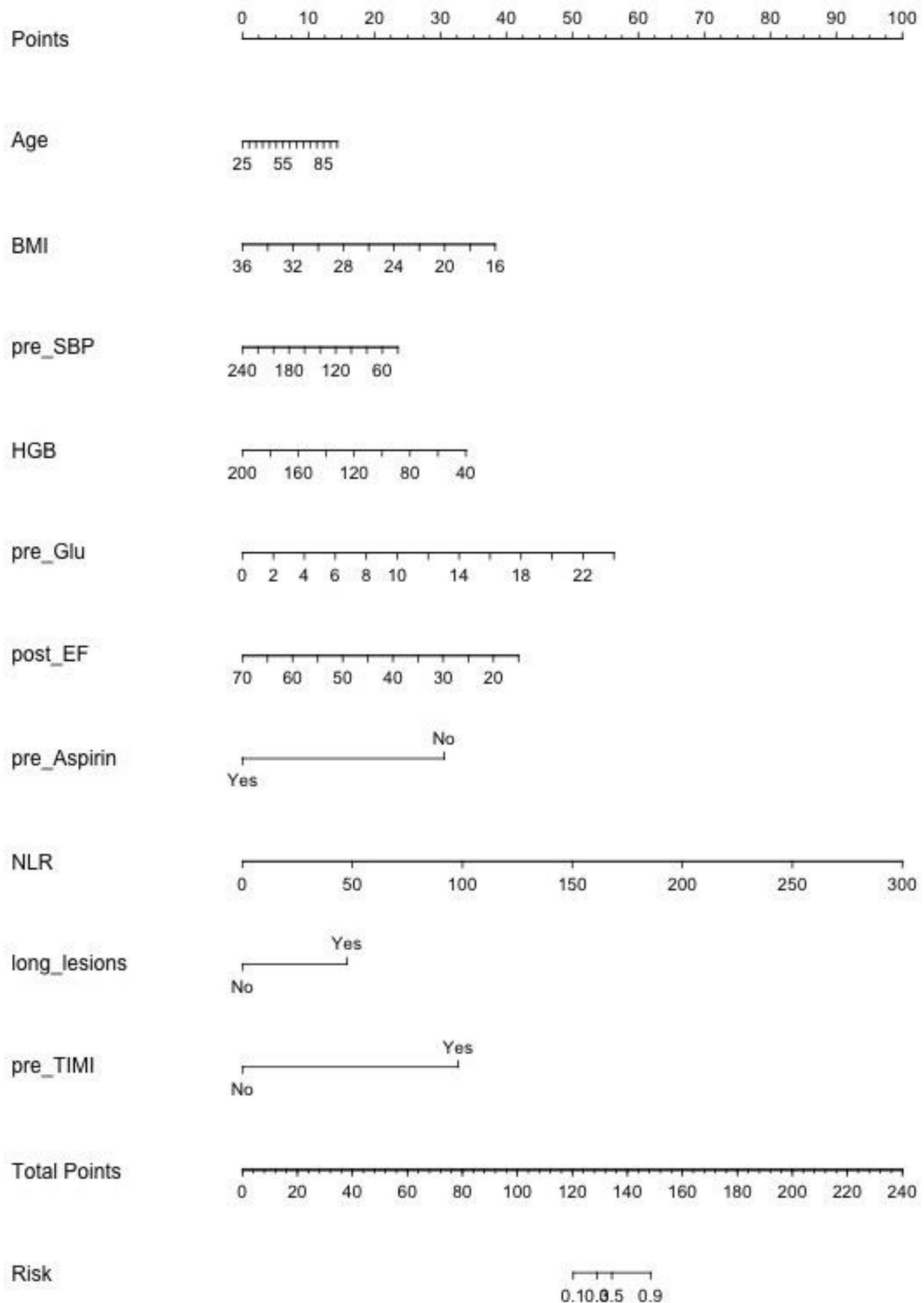


Figure 2 The nomogram for the prediction of in-hospital mortality in patients with acute ST-elevation myocardial infarction after primary PCI. BMI, body mass index; EF, ejection fraction; HGB, haemoglobin; N/LR, neutrophils/lymphocytes ratio; PCI, percutaneous coronary intervention; SBP, systolic blood pressure; TIMI, thrombolysis in myocardial infarction.

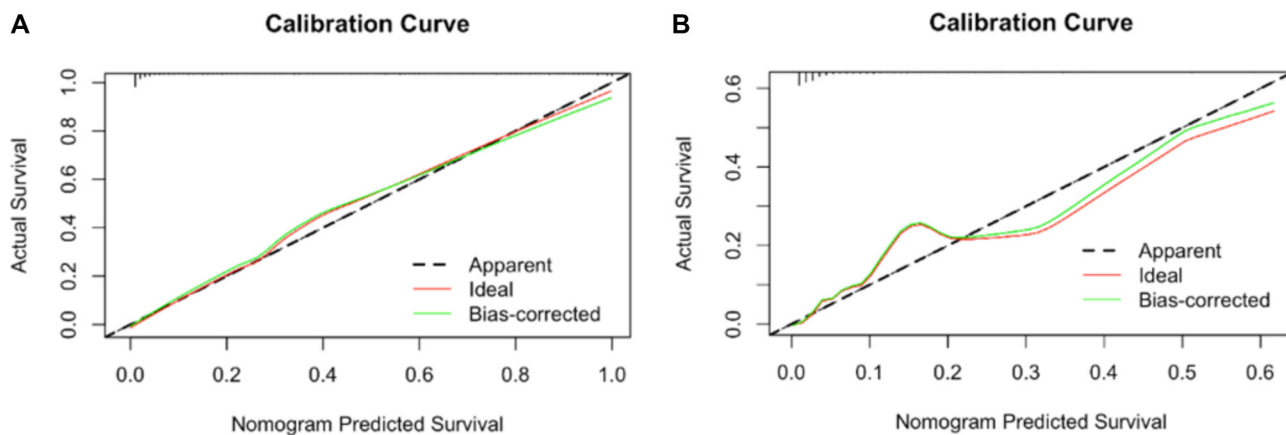


Figure 3 The calibration curves of the nomogram for the training set (A) and the testing set (B).

clinical usefulness in a routine setting, the nomogram must contain variables assessed in the routine clinical setting, which is the case with the nomogram developed here. It can simplify the statistical prediction model to the numerical probability of disease recurrence or death. The identification and stratification of patients becomes a simple tool with many advantages. The most prominent advantage is that it can predict individualised risks based on patient and disease characteristics. Second, it is easy to use and can help doctors develop individualised treatment plans. However, although the current clinical use of nomograms has increased, there are limited data on patient satisfaction or quality of life after it assists in medical decision making. In addition, although nomograms are widely used clinically, they are rarely evaluated prospectively to determine whether their use actually improves the prognosis of patients.^{59 60} Therefore, it remains to be explored how this risk model can be better applied to the clinic. The results indicate that the nomogram had good discrimination, well prediction accuracy and could achieve satisfactory net benefit. Another nomogram based on other variables (left main CAD, grading of thrombus, TIMI classification, slow flow, use of IABP, use of β -blocker, use of ACEI/ARB, symptom-to-door time,

symptom-to-balloon time, syntax score, LVEF, and CK-MB peak) also showed a high AUC for in-hospital mortality of patients with STEMI after PCI.⁶¹ Three main reasons fame justify the different predictors we found in our study: different research methods, the hospitals and time nodes that included patients are different and different statistical methods. Nevertheless, we are planning to combine the two parts of patients to get a more accurate risk model of in-hospital mortality.

Some study limitations should be mentioned: (1) This study has limitations that are inherent to retrospective observational studies. Many hospitals and doctors involved, which can lead to some missing information, such as liver enzymes, more information regarding the PCI procedure and other inflammatory index; (2) As the ischaemic time is shortened as much as possible, patients whose symptoms and/or ECG can be diagnosed are directly treated with PCI. Therefore, other potential risk factors in our study, such as LVEF before PCI, could not be included in the analyses. And some patients died during the PCI, resulting in the lack of postoperative treatment information. Further prospective studies are still necessary to confirm the performance of the clinical

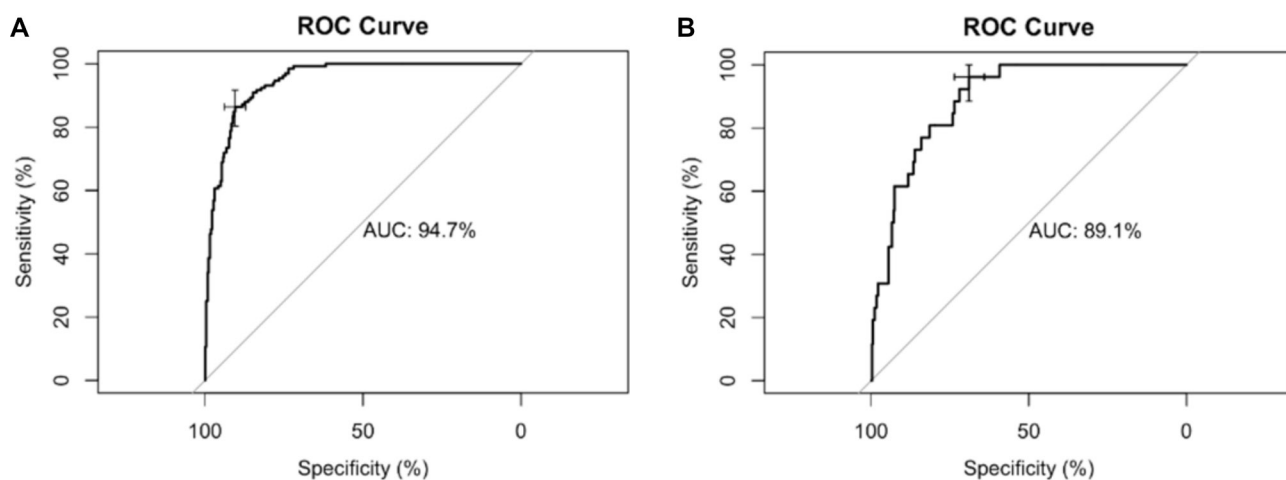


Figure 4 The received operating characteristics (ROC) curves of the nomogram for the training set (A) and the testing set (B). AUC, area under the curve.

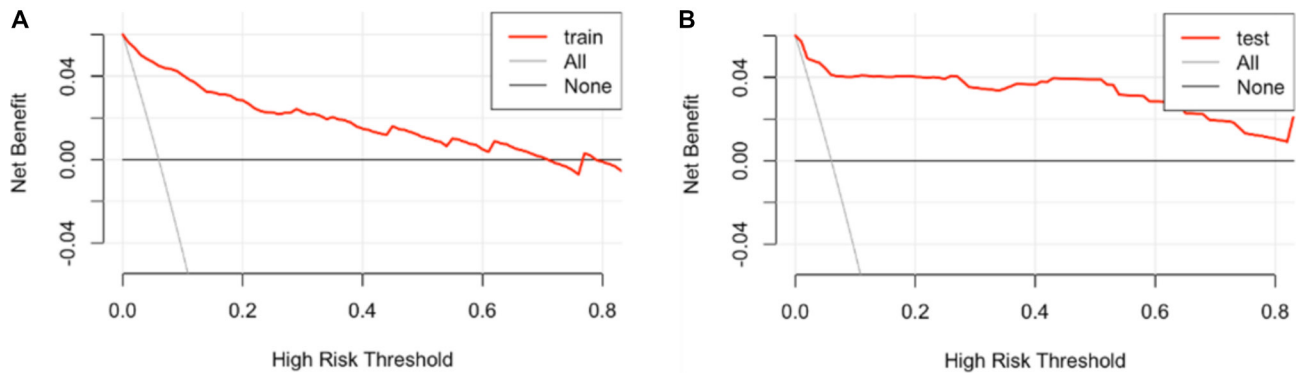


Figure 5 The decision curve analysis for the risk model for the training set (A) and the testing set (B).

applicability in future investigations and verify the practicality in ICU.

In conclusion, a nomogram to predict in-hospital mortality in patients with STEMI after PCI was developed and validated in Hebei, China. The nomogram showed a satisfactory performance, with a C-index of 0.948. Thus, this nomogram might be a precisely individualised predictive tool for prognosis. However, additional studies are needed to confirm the performance and clinical applicability and practicality of the nomogram.

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Competing interests None declared.

Patient consent for publication Consent obtained from next of kin.

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