Original Article

A nomogram for predicting in-hospital mortality in acute type A aortic dissection patients

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Background: Although there are several biomarkers for identifying in-hospital mortality in acute type A aortic dissection (AAD), timely as well as perfect prediction in-hospital mortality is still not attained. Herein, we intend to develop as well to validate an in-hospital mortality risk independent predictive nomogram for AAD patients.

Methods: From January 2014 to December 2018, 703 individuals with AAD were involved in this study. They were indiscriminately categorized into training (n=520) and validation (n=183) sets. The univariate and multivariate analyses were used to screen in-hospital mortality predictors from the entire training set data. The predictors were used to establish a nomogram which was confirmed via internal as well as external authentication. This validation included discriminative capacity defined by the receiver operating characteristic (ROC) curve area under the curve (AUC) and the predictive precision via calibration curves.

Results: There was 33.43% in-hospital mortality overall incidence. The uric acid, D-dimer, C-reactive protein and management were individually related to in-hospital mortality as per multivariate logistic regression. On the basis of four variables with internal of AUC 0.901 and external validation of AUC 0.903, a nomogram was established. Calibration plots showed that the predicted and actual in-hospital mortality probabilities were fitted well on both internal and external validation.

Conclusions: This recommended nomogram can calculate the specific possibility of in-hospital mortality with good precision, high discrimination, and probable clinical application in AAD patients.

Keywords: Acute aortic dissection; nomogram; in-hospital mortality

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Introduction

Acute type A aortic dissection (AAD), a fatal aortic sickness has a high death rate and morbidity which demands prompt examination and treatment (1,2). From 1% to 2% of patients with AAD die per hour for the first 24–48 hours (3). Unfortunately, there is still a lack of effective methods to accurately predict the short-term results of these patients. Recently, there has been a countless pursuit to ascertain the severity outcome of AAD, but, their efficacy in predicting in-hospital mortality remains controversial (4,5). Presently, in spite of extensive appreciation of the significance of in-hospital mortality, there is a lack of a perfect predictive method to identify in-hospital mortality in time to induce prompt intervention. A nomogram can offer, convenient form, precise risk estimate of patient individual clinical results grounded on major factors (6,7). Currently, to identify several disease diagnosis and

management strategies, nomograms have been suggested (8,9). The aim of this research is to define in-hospital mortality risk potential predictors and to build its predictive nomogram prototype, that will be important in detecting high-risk patients and subsequently guide them through psychotherapy and choosing the proper therapy, upon AAD.

Methods

Study design and settings

This is a retrospective observational study design. Medical records of AAD patients admitted to the Second Xiangya Hospital of Central South University from January 2014 to December 2018 were investigated. It was appraised and ratified by the hospital ethics committee, and as a retrospective study, the informed consent was waived.

Stanford standards were used to categorize AAD form and the diagnosis was established via computed tomography angiography (CTA) or magnetic resonance angiography (MRA) (10). AAD patients with a time interval of ≤14 days from the onset of symptoms to hospital admission were included in the present study. Exclusion criteria included: (I) diagnosis with pregnancy; (II) presence of intramural hematoma; (II) presence of symptoms for more than 14 days.

Collection of data

Each patient's clinical and laboratory information was acquired through electronic medical registers. Elementary clinical information comprised date of birth, sex, body mass index (BMI), the presence of hypertension, diabetes, stroke, atherosclerosis, Marfan syndrome, chronic renal insufficiency (CRI), smoking, symptom, blood pressure, pulse pressure, and management.

Laboratory variables included white blood cell (WBC), neutrophil ratio (N%), platelet (PLT), hemoglobin (Hb), alanine transaminase (ALT), aspartate aminotransferase (AST), albumin (ALB), total bilirubin (TB), direct bilirubin (DB), creatinine (Cr), blood urea nitrogen (BUN), uric acid (UA), estimated glomerular filtration rate (eGFR), creatine kinase (CK), creatine kinase-MB (CK-MB), troponin T (TNT), B-type natriuretic peptide (BNP), D-dimer (DDR), and C-reactive protein (CRP). Venous blood was drawn from all patients after admission. Plasma was obtained after rapid centrifugation and was immediately stored at –20 ℃ for further analysis. Serum biomarkers were determined using commercial kits and an automated biochemical analyzer. Once admitted all the above were analyzed the main laboratory of the Second Xiangya Hospital of Central South University.

Clinical endpoint

The clinical study was terminated upon death in the hospital.

Statistical analysis

Statistical results were presented as the mean ± standard deviation for normal data whereas non-normal data, interquartile range (IQR) and median were used. The categorical variables were presented as percentages and numbers. The non-normally distributed continuous variables Wilcoxon Mann-Whitney tests in addition to unpaired Student *t*-tests for normally distributed continuous variables were utilized to establish the correlations among the survivor and the non-survivor groups. Fisher's exact/Chi-squared tests were utilized to evaluate clustered variables.

The initial dataset was indiscriminately divided into training and validating set at a 3:1 ratio and was utilized in producing and authenticating the model, respectively. To identify the in-hospital death risk features among AAD victims, the study was carried out using multivariate regression in the training set. In-hospital mortality was used as reaction variable adopting a binomial distribution, and covariates with lesser than 0.05 P values were incorporated in the model.

An in-hospital mortality AAD patients' predictive nomogram was established from clinical significant variables. The validation involved two events: internal and external authentication. To begin with, internal validation was completed through a bootstrap process using 500 resamples. The discrimination capacity was studied via the receiver operating characteristic (ROC) curve area under the curve (AUC). In addition, a calibration curve was drawn to quantify the consistency of nomogram predicted chances and the experienced in-hospital mortality frequency. Secondly, external authentication was executed in the validation group and the nomogram employed for individual patient validation. To evaluate the nomogram prototype discriminative achievements and predictive precision, AUC and calibration plot was made. It was found out that the P values were all 2-sided and P<0.05

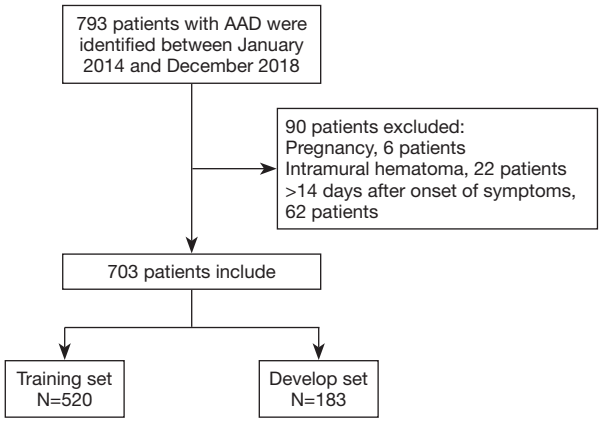

Figure 1 Flow chart of patient enrollment.

was statistically significant. R (http://www.R-project.org) and Empowerstates (http://www.empowerstates.com, X&Y Solution, Inc, Boston, MA, USA) software were used to carry out all the statistical analyses.

Results

Study population and clinical characteristics of the study cohort

Seven hundred and ninety-three patients were identified with a diagnosis of AAD. Among them, 6 patients were expectant, 22 patients were diagnosed intramural hematoma, and 62 patients got admitted after 24 hours of symptoms origin of were excluded from the analysis. Here, a cohort of 703 AAD victims was incorporated, of which three-quarters were indiscriminately allocated to the training set (n=520) to generate a predictive nomogram model, while the 183 left were apportioned to the validation set to assess the model success (*Figure 1*). There was no significant variation in most elementary clinical information and laboratory variables among the training and validation groups (*Table 1*). The AAD in-hospital mortality between the two groups was similar, 175 (33.65%) in the training set and 60 (32.79%) in the validation set, respectively.

Independent predictors of in-hospital mortality for AAD victims

Univariate analysis indicated that age, SBP, DBP, pulse pressure, WBC, N%, PLT, ALT, AST, ALB, Cr, BUN, UA, eGFR, CK, CK-MB, TNT, BNP, DDR, CRP, atherosclerosis, CRI, and management were associated with in-hospital mortality for the training set as shown in *Table 2*. The above were incorporated in multivariate regression analysis to monitor the important in-hospital mortality predictors. This analysis outcome disclosed four self-sufficient predictor variables including UA, DDR, CRP, and management for in-hospital mortality AAD victims (*Table 3*).

Development of in-hospital mortality predictive nomogram for AAD patients

The predictive nomogram (*Figure 2*), was developed from the four independent in-hospital mortality predictors. Individually, subsets in these variables were allocated a count on the point set of scales. Once we added up the cumulative score and tracing on the overall point scale, we plotted a straight line down to obtain in-hospital mortality predicted probability. A higher score of overall points revealed a better in-hospital mortality chance.

Internal validation of the predictive nomogram

Upon exposure to internal authentication, the predictive nomogram validated a perfect discriminative capacity of AUC 0.901 (95% CI: 0.869–0.933) (*Figure 3A*). The standard curve disclosed that predicted nomogram possibilities of in-hospital sensibly estimated actual probabilities (*Figure 3B*).

External validation of the predictive nomogram

External authentication was achieved by comparing the predictive nomogram and individual actual possibility in the authentication group. For validation group, predictive nomogram AUC as 0.903 (95% CI: 0.858–0.967) (*Figure 4A*). The standardization plot revealed that there was perfect synchronization among predicted and real possibilities (*Figure 4B*).

Clinical application of the nomogram

Meanwhile, we chose two AAD victims as specimens for the nomogram presentation. The levels of UA, DDR, and CRP in the first patient were 926.1 μmol/L (84 points), 3.3 μg/mL (2 points), 122 mg/L (18 points), respectively. His medical treatment at 100 points. There were 204 estimated points nomogram count and above 0.9 probability of in-hospital mortality. The patient had a high chance of achieving in-hospital mortality. In fact, he acquired

Table 1 Baseline characteristics of the patients in training set and validation set

Characteristics	Training set (n=520)	Validation set (n=183)	P value
Age (years)	50.10±11.58	51.55±10.62	0.138
BMI (kg/m^2)	25.36±4.55	24.79±3.81	0.132
SBP (mmHg)	144.09±31.80	138.95±28.65	0.054
DBP (mmHg)	78.77±19.86	75.03±17.78	0.085
Pulse pressure (mmHg)	65.33±21.34	63.92±21.27	0.443
White blood cell (×10^9/L)	12.31±4.09	11.83±4.42	0.182
Neutrophil ratio (%)	82.79±7.86	81.01±8.80	0.011
Platelet (×10^9/L)	185.23±81.61	189.89±88.18	0.516
Hemoglobin (g/L)	124.50±20.31	124.07±20.76	0.805
Alanine transaminase (μ/L)	22.70 (14.60–44.62)	20.30 (13.35–36.85)	0.699
Aspartate aminotransferase (μ/L)	23.30 (16.58–42.08)	21.70 (16.05–39.30)	0.767
Albumin (g/L)	35.91±4.50	35.97±4.58	0.889
Total bilirubin (μmol/L)	18.19±10.06	17.87±9.23	0.709
Direct bilirubin (μmol/L)	7.32±5.95	7.08±4.58	0.620
Creatinine (μmol/L)	84.85 (66.88–121.15)	83.40 (67.90–130.10)	0.833
Blood urea nitrogen (mmol/L)	8.22±7.33	8.05±4.55	0.780
Uric acid (μmol/L)	351.55±141.58	342.48±125.91	0.444
eGFR	88.90±42.32	82.07±38.31	0.055
Creatine Kinase (μ/L)	109.20 (64.12–242.00)	110.40 (58.65–241.70)	0.939
Creatine Kinase-MB (μ/L)	12.30 (6.15–18.83)	10.90 (2.30–16.80)	0.583
Troponin T (pg/mL)	10.35 (2.48–33.94)	9.23 (0.47–25.50)	0.272
B-type natriuretic peptide (pg/mL)	307.50 (118.00–832.25)	304.40 (132.50–819.50)	0.435
D-dimer (μg/mL)	4.03 (2.51–11.11)	3.72 (2.57–10.32)	0.632
C-reactive protein (mg/L)	54.75 (9.40–105.00)	31.70 (9.38–108.50)	0.426
Gender			0.187
Male	403 (77.50%)	133 (72.68%)	
Female	117 (22.50%)	50 (27.32%)	
Hypertension			0.792
No	165 (31.73%)	60 (32.79%)	
Yes	355 (68.27%)	123 (67.21%)	
Diabetes			0.530
No	505 (97.12%)	176 (96.17%)	
Yes	15 (2.88%)	7 (3.83%)	

Table 1 (*Continued*)

Table 1 (*Continued*)

Characteristics	Training set (n=520)	Validation set (n=183)	P value
Stroke			0.024
No	507 (97.50%)	172 (93.99%)	
Yes	13 (2.50%)	11 (6.01%)	
Atherosclerosis			0.395
No	482 (92.69%)	173 (94.54%)	
Yes	38 (7.31%)	10 (5.46%)	
Marfan syndrome			0.354
No	507 (97.50%)	176 (96.17%)	
Yes	13 (2.50%)	7 (3.83%)	
Chronic renal insufficiency			0.864
No	507 (97.50%)	178 (97.27%)	
Yes	13 (2.50%)	5 (2.73%)	
Smoking			0.806
No	373 (71.73%)	133 (72.68%)	
Yes	147 (28.27%)	50 (27.32%)	
Symptom			0.021
Chest pain	433 (83.27%)	148 (80.87%)	
Back pain	18 (3.46%)	4 (2.19%)	
Abdominal pain	23 (4.42%)	2 (1.09%)	
Syncope	12 (2.31%)	6 (3.28%)	
Other	34 (6.54%)	23 (12.57%)	
Management			0.787
Medical	159 (30.58%)	54 (29.51%)	
Surgical	361 (69.42%)	129 (70.49%)	
Mortality			0.831
Survivor	345 (66.35%)	123 (67.21%)	
Non-survivor	175 (33.65%)	60 (32.79%)	

in-hospital mortality in admission. The 2nd patient with surgical treatment (0 points). The levels of UA, DDR and CRP were 89.7 μmol/L (8 points), 9.24 μg/mL (6 points), 68.1 mg/L (27 points), respectively. The nomogram was 41 points, and there was <0.1 in-hospital mortality probability. This patient had low chances of achieving in-hospital mortality. Besides, he did not acquire in-hospital mortality.

Discussion

Timely diagnosis of in-hospital mortality high-risk patients can help to manage a disease, improve patient-clinician interaction, and facilitate the patient's prognosis. However, up to now, the timely or perfect diagnostic prototype for in-hospital mortality in AAD patients is lacking. The

Table 2 Univariate analyses of factors associated with in-hospital mortality in the training set

Characteristics	Survivor (n=345)	Non-survivor (n=175)	P value
Age (years)	49.32±11.17	51.64±12.25	0.031
BMI (kg/m^2)	25.27±4.25	25.52±5.10	0.553
SBP (mmHg)	148.83±28.96	134.75±35.00	<0.001
DBP (mmHg)	81.31±19.71	73.76±19.26	<0.001
Pulse pressure (mmHg)	67.52±18.95	60.99±24.89	<0.001
White blood cell (×10^9/L)	11.83±3.82	13.25±4.44	<0.001
Neutrophil ratio (%)	82.45±7.74	83.45±8.08	0.041
Platelet (×10^9/L)	190.52±82.58	174.80±78.86	0.038
Hemoglobin (g/L)	125.13±18.53	123.27±23.45	0.324
Alanine transaminase (μ/L)	20.80 (13.80–38.20)	25.00 (15.75–64.85)	<0.001
Aspartate aminotransferase (μ/L)	21.20 (15.80–33.90)	29.50 (18.55–65.05)	<0.001
Albumin (g/L)	36.29±4.57	35.17±4.27	0.007
Total bilirubin (μmol/L)	18.53±10.25	17.52±9.68	0.281
Direct bilirubin (μmol/L)	7.26±5.07	7.44±7.40	0.757
Creatinine (μmol/L)	81.20 (65.60–106.70)	101.00 (71.10–153.95)	0.002
Blood urea nitrogen (mmol/L)	7.40±4.28	9.83±10.97	<0.001
Uric acid (μmol/L)	332.51±124.80	389.08±163.89	<0.001
eGFR	93.92±39.01	78.98±46.73	<0.001
Creatine kinase (μ/L)	101.80 (60.00–216.00)	135.90 (72.65–335.22)	0.006
Creatine kinase-MB (μ/L)	11.90 (5.80–17.60)	13.30 (7.05–22.60)	0.004
Troponin T (pg/mL)	9.15 (0.55–25.00)	16.00 (3.63–66.06)	0.006
B-type natriuretic peptide (pg/mL)	280.00 (109.50–770.00)	340.00 (158.10–1,034.50)	0.048
D-dimer (μg/mL)	3.59 (2.14– 7.53)	5.74 (3.04–18.79)	<0.001
C-reactive protein (mg/L)	66.60 (12.80–114.00)	19.20 (6.94– 85.00)	<0.001
Gender			0.331
Male	263 (76.23%)	140 (80.00%)	
Female	82 (23.77%)	35 (20.00%)	
Hypertension			0.614
No	112 (32.46%)	53 (30.29%)	
Yes	233 (67.54%)	122 (69.71%)	
Diabetes			0.561
No	334 (96.81%)	171 (97.71%)	
Yes	11 (3.19%)	4 (2.29%)	

Table 2 (*Continued*)

Table 2 (*Continued*)

Characteristics	Survivor (n=345)	Non-survivor (n=175)	P value
Stroke			0.710
No	337 (97.68%)	170 (97.14%)	
Yes	8 (2.32%)	5 (2.86%)	
Atherosclerosis			0.027
No	326 (94.49%)	156 (89.14%)	
Yes	19 (5.51%)	19 (10.86%)	
Marfan syndrome			0.710
No	337 (97.68%)	170 (97.14%)	
Yes	8 (2.32%)	5 (2.86%)	
Chronic renal insufficiency			0.031
No	340 (98.55%)	167 (95.43%)	
Yes	5 (1.45%)	8 (4.57%)	
Smoking			0.923
No	247 (71.59%)	126 (72.00%)	
Yes	98 (28.41%)	49 (28.00%)	
Symptom			0.547
Chest pain	282 (81.74%)	151 (86.29%)	
Back pain	14 (4.06%)	4 (2.29%)	
Abdominal pain	17 (4.93%)	6 (3.43%)	
Syncope	7 (2.03%)	5 (2.86%)	
Other	25 (7.25%)	9 (5.14%)	
Management			<0.001
Medical	22 (6.38%)	137 (78.29%)	
Surgical	323 (93.62%)	38 (21.71%)	

importance of consistent available clinical variables for timely identification of in-hospital mortality has attracted scientific attention. Here, we targeted to develop as well as to authenticate a predictive nomogram for the in-hospital mortality risk in AAD patients.

To construct the nomogram, the in-hospital mortality independent predictive aspects in AAD patients were first identified. Through the multivariate logistic regression analysis, the in-hospital mortality independent predictors shown to be UA, DDR, CRP, and management. Similar to earlier studies (11,12), it was shown that medical treatment could contribute to the development of in-hospital mortality.

Evidently, UA, an end product of purine metabolism that is a resilient independent predictor for long-standing mortality in cardiac-related disorders (13,14). Previous studies had revealed that elevated levels of UA were correlated to a higher risk of in-hospital mortality for cardiovascular disease victims. Jiang *et al.* (15) found that serum uric acid is associated with aortic dissection in Chinese men. A meta-analysis provides evidence that SUA levels are significantly higher among patients with aortic dissection than those in controls. Elevated SUA levels may contribute to the pathogenesis of aortic dissection (16). Experimental and clinical studies have evidenced several mechanisms through which elevated UA level exerts deleterious effects on cardiovascular health

Table 3 Multivariable logistic regression for in-hospital mortality

Variable	OR	95% CI lower	95% CI upper	P value
Age	1.000	0.974	1.028	0.972
SBP (mmHg)	1.000	0.987	1.013	0.993
DBP (mmHg)	0.982	0.962	1.003	0.093
White blood cell ($\times10^9$/L)	1.036	0.958	1.122	0.376
Neutrophil ratio (%)	1.011	0.970	1.053	0.614
Platelet ($\times10^9$/L)	0.998	0.994	1.001	0.228
Alanine transaminase (μ/L)	1.001	0.999	1.003	0.235
Aspartate aminotransferase (μ/L)	0.999	0.998	1.001	0.440
Albumin (g/L)	0.949	0.895	1.006	0.079
Creatinine (μmol/L)	0.999	0.995	1.003	0.677
Blood urea nitrogen (mmol/L)	1.007	0.961	1.054	0.774
Uric acid (μmol/L)	1.003	1.001	1.005	0.003
Low (53.40–336.70)	Ref			
High (337.40–986.40)	1.935	1.102	3.397	0.022
eGFR	0.999	0.990	1.008	0.848
Creatine kinase (μ/L)	1.000	0.999	1.001	0.316
Creatine kinase-MB (μ/L)	0.999	0.988	1.010	0.807
Troponin T (pg/mL)	1.001	0.999	1.001	0.110
B-type natriuretic peptide (pg/mL)	1.000	0.999	1.000	0.103
D-dimer (μg/mL)	1.015	1.001	1.039	0.041
Low (0.01–3.89)	Ref			
High (3.91–40.00)	1.946	1.139	3.324	0.015
C-reactive protein (mg/L)	0.996	0.991	1.000	0.042
Low (1.00–45.70)	Ref			
High (47.10–305.00)	0.613	0.362	0.938	0.048
Atherosclerosis				
No	Ref			
Yes	0.942	0.328	2.706	0.912
Chronic renal insufficiency				
No	Ref			
Yes	5.665	0.416	77.120	0.193
Management				
Medical	Ref			
Surgical	0.014	0.008	0.025	<0.001

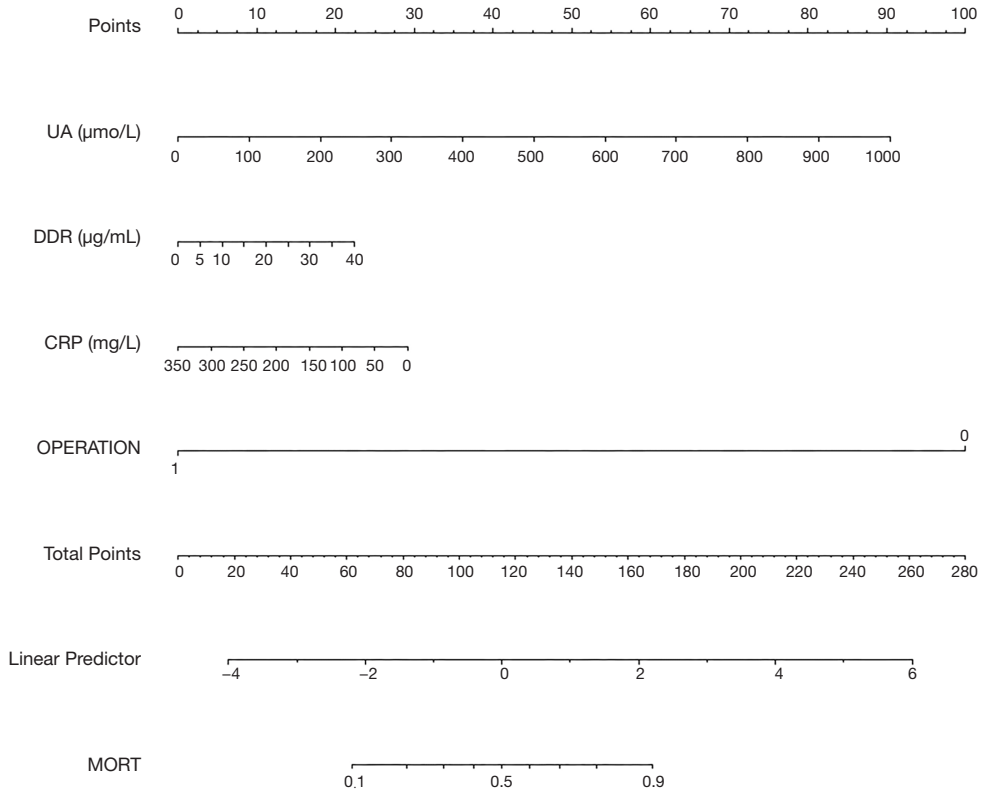

Figure 2 Nomogram model for predicting the risk of in-hospital in patients with AAD. AAD, acute type A aortic dissection; UA, uric acid; DDR, D-dimer; CRP, C-reactive protein; OPERATION, 0= medical, 1= surgical.

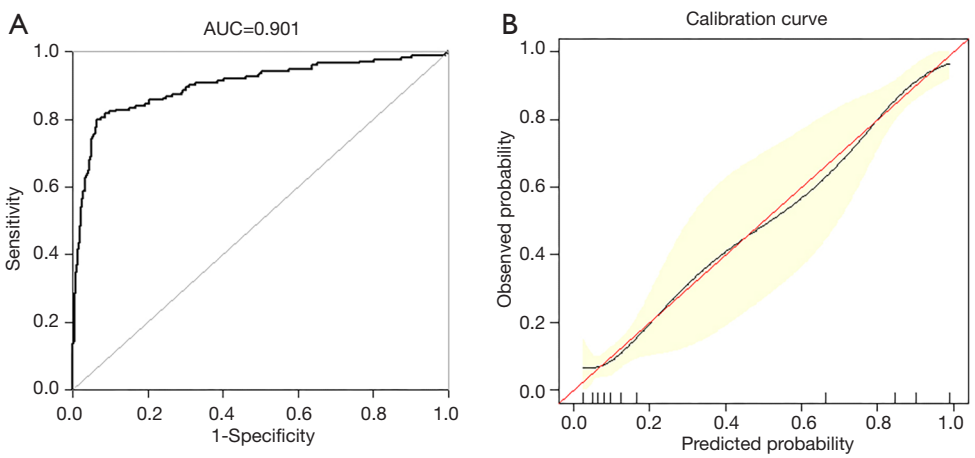

Figure 3 Internal validation of nomogram in the training set. (A) Discrimination: AUC of the ROC curve was 0.901 (95% CI, 0.869–0.933). (B) Nomogram calibration curve. The red line indicates perfect prediction by an ideal model. The black line depicts the model's performance. ROC, receiver operating characteristic; AUC, area under the curve.

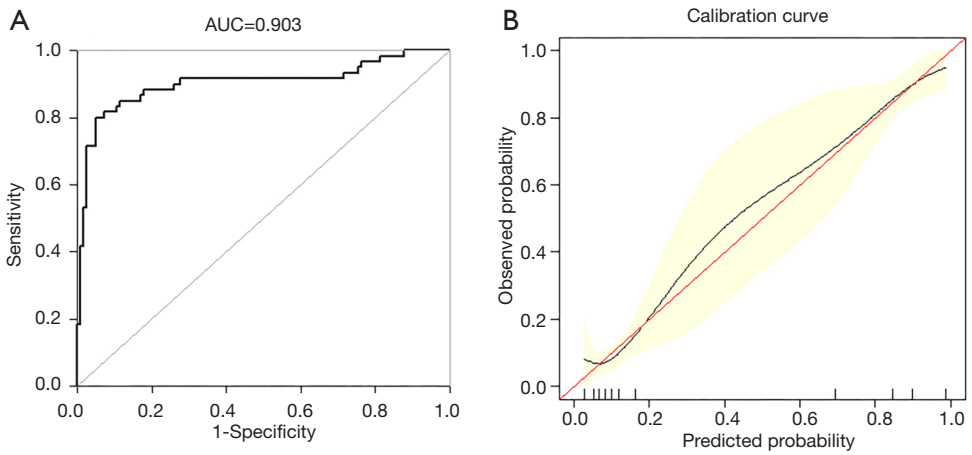

Figure 4 External validation of nomogram in the validation set. (A) Discrimination: AUC of the ROC curve was 0.903 (95% CI: 0.858–0.967). (B) Nomogram calibration curve. The red line indicates perfect prediction by an ideal model. The black line depicts the model's performance. ROC, receiver operating characteristic; AUC, area under the curve.

including increased oxidative stress, reduced availability of nitric oxide and endothelial dysfunction, promotion of local and systemic inflammation. Hong *et al.* (17) revealed hyperuricemia induces endothelial dysfunction via mitochondrial Na+/Ca2+ exchanger-mediated mitochondrial calcium overload. Esen *et al.* (18) revealed that uric acid as a marker of oxidative stress in dilatation of the ascending aorta. In the present research, for AAD patients in-hospital mortality, UA was of predictive significance.

In AAD patients, D-dimer, a cross-linked fibrin degradation yield, is raised (19). Recently, a number of studies estimated the D-dimer levels prognostic value of admitted AAD patients and stated that high levels of D-dimer were related to reduced short-range outcomes (19,20). Our previous study also found that elevated admitted based D-dimer levels can predict a higher risk of in-hospital mortality (21). Besides, when levels of serum D-dimer are high may be a manifestation of more severe and extensive tearing. As in earlier studies, this study maintained that there is a high predictive value of DDR for in-hospital mortality risk.

CRP, a well-known profound systemic inflammation marker, is able to stimulate the clotting system resulting in weakened oxidant defense hence endothelial dysfunction (22,23). AAD is related to inflammatory response, thrombosis, and oxidative pressure demonstrated via a major rise in inflammatory indicators, like CRP (24). AlMahameed *et al.* (25) suggested that patients with aortic dissection have the highest CRP levels at presentation. Elevated CRP independently predicted higher lasting mortality for AAD patients. Nevertheless, Sakakura *et al.* (26) found that primary levels of CRP weren't linked to adverse activities, peak CPR intensities were considerably associated with adverse activities. The highest levels of CRP were a superior indicator of primary levels of CRP in AAD type B victims risk stratification. Since it takes a few days to attain CRP peak, primary levels of CRP may not reveal entire aortic dissection severity. Okina *et al.* (27) also conclude that cardiovascular events cannot be predicted by a CRP value obtained at a single time point, recognition of the overall CRP pattern is far more important. It is interesting that in our research, CRP admission low levels were significantly predictive for AAD in-hospital mortality. These varying outcomes may be due to diverse participants or CRP levels were measured at admission rather than the peak value of monitoring.

According to us, this is the first predictive nomogram for in-hospital mortality risk in AAD patients. It is grounded in four achievable and cost-effective variables that allow ease in determining personalized in-hospital mortality risk evaluations for AAD patients. The training set nomogram prediction AUC was 0.901 and that for validation set was 0.903. It was revealed by the standardization plots that the predictive nomogram fitted perfectly to the actual in-hospital mortality likelihood on the internal as well as external authentication. These outcomes propose that the predictive nomogram possessed necessary discriminative

strength and worthy accuracy to predict in-hospital mortality in AAD patients.

Moreover, this study has a few limitations. Firstly, there was only one center in this study. However, even though the training and validation set came from the same institution, this is a tolerable process of developing and authenticating nomogram once the external cohort is non-existing. Nevertheless, the nomogram requires validation from other centers to check its uniqueness. Secondly, there were zero follow-up parameters for nomogram assimilated variables. Yet, the study's goal was to project in-hospital mortality risk from the admission clinical features to bring opportune intervention. Lastly, there was significant variation in a few variables among the training and validation groups. However, these patients were randomly distributed by R software between the two groups, which ensure that there are no significant differences in most variables like the outcome.

Conclusions

In summary, this proposed nomogram can determine specifically in-hospital mortality risk with clear accuracy, perfect discrimination, and probable clinical application in AAD patients.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The Ethics Committee of the Second Xiangya Hospital, Central South University (Changsha, China) approved this study and because of the study retrospective nature (No. 2018S052), informed consent was omitted.

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