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Article

Development of a Preoperative Mortality Risk Prediction Model and Scoring System for Patients With Acute Type A Aortic Dissection: A Retrospective Cohort Study

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Abstract

Background: Acute Stanford type A aortic dissection (ATAAD) remains a catastrophic cardiovascular emergency with substantial preoperative mortality and limited risk-stratification tools. To develop and validate a parsimonious bedside score to quantify individual risk of preoperative death in ATAAD. **Methods:** Consecutive ATAAD patients (≥18 years) presenting to a tertiary center in northwest China from March 2012 to April 2022 were enrolled. Baseline variables (n = 68) were first screened by the Least Absolute Shrinkage and Selection Operator (LASSO) regression in the entire cohort. Patients were then randomly split 7:3 into training (n = 404) and validation (n = 174) cohorts. Multivariable logistic regression was used to identify independent determinants of preoperative mortality. Model discrimination (area under the curve, AUC), calibration (Hosmer–Lemeshow), and clinical utility (decision curve analysis) were assessed. A Framingham-style integer score was constructed and stratified into low- (0–2), medium- (3–4), and high-risk (5–7) tiers. **Results:** Preoperative mortality occurred in 102 of 578 patients (17.7%). Six variables emerged as independent predictors: coma (odds ratio (OR) 5.93, 95% confidence interval (CI) 1.29–27.26), intimal tear in the ascending aorta (OR 6.28, 95% CI 2.16–18.32), intimal tear at the aortic root (OR 10.74, 95% CI 3.28–35.21), lactate >1.6 mmol/L (OR 2.43, 95% CI 1.32–4.46), hemoglobin <115 g/L (OR 2.61, 95% CI 1.15–5.94), and creatinine >115 µmol/L (OR 2.61, 95% CI 1.41–4.83). The model showed strong discrimination (AUC: 0.78 (training), 0.76 (validation)) and good calibration. In the training set, it rose from low risk (5.59%) to medium risk (27.18%) to high risk (63.16%). In the validation set, score-stratified mortality increased from low risk (4.71%) to medium risk (21.79%) to high risk (27.27%). **Conclusions:** The six-item ATAAD preoperative mortality score enables rapid, reproducible bedside risk assessment, facilitating early triage, resource allocation, and personalized perioperative management. Therefore, prospective, multicenter validation is warranted.

Keywords: acute Stanford type A aortic dissection; preoperative mortality; risk prediction model; scoring system

1. Introduction

Aortic dissection (AD) is a life-threatening vascular emergency characterized by an intimal tear in the aortic wall. When the ascending aorta is involved, it is classified as Stanford type A aortic dissection (TAAD). Acute TAAD (ATAAD), defined as symptom onset within 14 days, accounts for approximately 58–62% of all AD cases [1,2]. ATAAD has an annual incidence of approximately 3 per 100,000 population [3], population-based registry data from Japan indicate a higher incidence than has been reported in European and North American cohorts [4]. In China, approximately 20,000 new ATAAD cases occur annually, with a disproportionately higher prevalence in the northwestern regions [5,6].

ATAAD typically presents with abrupt and severe chest or back pain. Sympathetic activation secondary to pain may further elevate blood pressure and heart rate, increasing the risk of intimal rupture [7]. However, 5–17% of patients present with atypical symptoms, including syncope, abdominal pain, or painless presentations, contributing to misdiagnosis rates as high as 45.3% [8,9,10]. Without timely intervention, ATAAD may rapidly progress to aortic rupture, hemorrhagic shock, and multiorgan failure [11].

The pathophysiology of ATAAD is multifactorial, with hypertension, smoking, bicuspid aortic valve, connective tissue disorders, and atherosclerosis being established risk factors [12,13]. Persistent hypertension promotes medial degeneration [14], while admission pulse pressure has been reported to show a J-shaped association with in-hospital mortality [15]. Metabolic perturbations and systemic inflammation have also been implicated, with biomarkers such as interleukin-6 and C-reactive protein being independent predictors of mortality [13,16,17].

Emergency surgical repair remains the cornerstone of ATAAD management [18,19]. In untreated patients, mortality increases by approximately 1–2% per hour within the first 24 hours, reaching 50% at 48 hours and 70% at one week [19,20]. Given this fulminant course, validated tools for preoperative risk stratification are urgently needed.

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The aims of this study were therefore to: (1) identify independent determinants of preoperative mortality in ATAAD, and (2) develop and internally validate a parsimonious, bedside-applicable risk prediction model using data from consecutive patients admitted to the First Affiliated Hospital of Xinjiang Medical University.

2. Materials and Methods

2.1 Study Design and Study Participants

All patients in this study were diagnosed with ATAAD and admitted for treatment at the First Affiliated Hospital of Xinjiang Medical University between March 2012 and April 2022. Diagnosis was established according to the standard codes of the International Classification of Diseases, Tenth Revision (ICD-10). Ethics approval for this retrospective cohort study was obtained from the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University (Approval No. K202310-01), and the study was conducted in accordance with the principles of the Declaration of Helsinki. Data were collected solely through review of existing hospital medical records, with no direct patient contact or intervention. Consequently, the requirement for informed consent was waived by the Ethics Committee of the First Affiliated Hospital of Xinjiang Medical University. Inclusion criteria were as follows: (1) age ≥18 years; and (2) a definitive diagnosis of TAAD, with time from onset to hospital visit ≤14 days. Exclusion criteria were: (1) patients with conditions potentially impacting study variables, such as disseminated malignancies, autoimmune disorders, or hematological diseases; (2) AD caused by trauma or medical intervention; and (3) cases missing >10% of required data. Application of these rigorous inclusion and exclusion criteria served to ensure homogeneity of the study population and to minimize confounding influences on the study outcomes. The patient screening process is shown in **Supplementary Fig. 1**.

2.2 Data Collection

Clinical data for hospitalized ATAAD patients were collected via the hospital information system (HIS). For laboratory investigations with multiple measurements, the initial test result was used for analysis. A total of 68 variables were included, comprising: (1) baseline characteristics: sex, age (years), history of smoking and alcohol consumption, and smoking index (number of cigarettes smoked per day × number of years smoked); (2) comorbidities: presence of hypertension, diabetes mellitus, coronary atherosclerotic heart disease, and adequacy of blood pressure control; (3) physical examination findings: body temperature (°C), heart rate (beats/min), pulse pressure (mmHg), respiratory rate (breaths/min), presence of chest pain, chest tightness, syncope, coma, limb ischemic symptoms, diminished pulses, atypical symptoms (fatigue, headache, dizziness, pharyngeal discomfort), acute renal failure, and shock; (4) laboratory and clinical parameters: hematological tests, including white blood cell count (10^9/L), lymphocyte count (10^9/L), neutrophil count (10^9/L), neutrophil percentage (%), platelet count (10^9/L), hemoglobin (g/L); inflammatory markers, including C-reactive protein (mg/L), interleukin-6 (pg/mL), procalcitonin (ng/mL); liver function tests, including alanine transaminase (U/L), aspartate transaminase (U/L), direct bilirubin (μmol/L), indirect bilirubin (μmol/L); renal function tests, including creatinine (μmol/L), urea (mmol/L), uric acid (μmol/L); lipid profile, including total cholesterol (mmol/L), triglycerides (mmol/L), high-density lipoprotein cholesterol (mmol/L), low-density lipoprotein cholesterol (mmol/L); blood gas analysis, including pH, partial pressure of oxygen (mmHg), partial pressure of carbon dioxide (mmHg), oxygen saturation (%), lactate (mmol/L), oxygenation index (mmHg); coagulation parameters, including prothrombin time (s), activated partial thromboplastin time (s), international normalized ratio (INR), D-dimer (ng/mL), fibrinogen (g/L); cardiac function markers, including troponin (μg/L), N-terminal pro-brain natriuretic peptide (NT-proBNP, ng/L), left ventricular ejection fraction (%), left ventricular fractional shortening, sinus diameter (mm), ascending aortic diameter (mm), presence of aortic regurgitation, pericardial effusion, or bicuspid aortic valve; (5) other variables, including DeBakey classification, site of the intimal tear (as determined by computed tomography angiography (CTA) at admission and classified as aortic arch, ascending aorta, aortic root, or retrograde dissection), Modified Early Warning Score (MEWS), Numerical Rating Scale (NRS), Acute Physiology and Chronic Health Evaluation II (APACHE II) score, European System for Cardiac Operative Risk Evaluation (EuroSCORE), and occurrence of preoperative mortality. The rigorous inclusion of these variables was intended to ensure comprehensive assessment and to minimize bias in the evaluation of patient outcomes.

2.3 Study Outcome Events

Preoperative mortality was defined as in-hospital death occurring between hospital admission and the scheduled surgical procedure for ATAAD. Information on preoperative mortality was obtained from the HIS.

2.4 Statistical Methods

Statistical analyses were performed using SPSS 22.0 (SPSS Inc., Chicago, IL, USA) and R 4.4.2 (R Foundation for Statistical Computing, Vienna, Austria). For binary classification modelling, preoperative death was defined as the positive class. Continuous variables were assessed for normality using the Kolmogorov-Smirnov test. Normally distributed data were presented as the mean ± standard deviation and compared using the independent-samples *t*-test, while non-normally distributed data were expressed as the median (interquartile range) and analyzed using the Mann-Whitney U test. Categorical variables were summarized as frequencies (percentages) and compared using the chi-

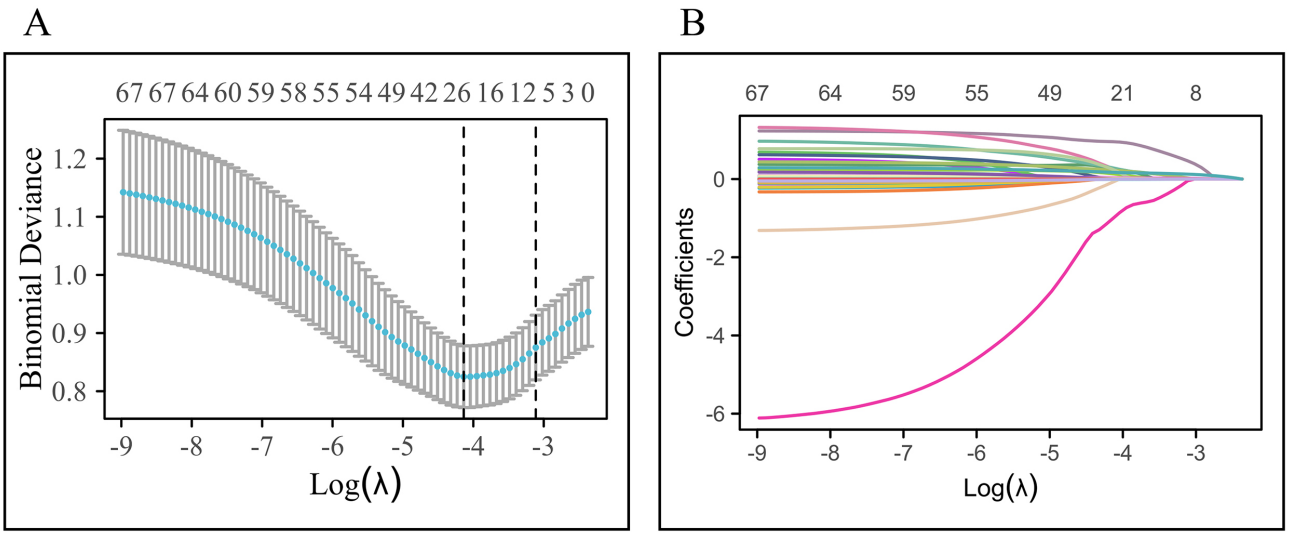


Fig. 1. Variable trajectory and selection process in LASSO regression. (A) Variable screening plot. (B) Coefficient trajectory plot. LASSO, Least Absolute Shrinkage and Selection Operator.

square test or Fisher's exact test, as appropriate. All tests were two-tailed, with $p < 0.05$ considered to represent statistical significance.

Missing data were addressed at two levels. At the variable level, variables with >25% missing values were excluded from the analysis. At the patient level, and consistent with exclusion criterion (3) described in "Study design and study participants", 10 patients (1.6% of the cohort) with >10% missing data across the recorded study variables were excluded from the cohort. For the remaining variables, the missing-data pattern was verified to follow the Missing At Random mechanism. Missing values were imputed using a stratified K-nearest-neighbour approach (K = 5), with stratification by preoperative survival status. Five imputed datasets were generated and combined for subsequent analyses. The relative increase in variance (RIV) was <0.3 for all imputed variables, confirming that the imputation procedure did not introduce significant bias.

We first performed variable selection using least absolute shrinkage and selection operator (LASSO) regression. This identifies key predictors by applying an L1-norm penalty that shrinks coefficients of non-informative variables to exactly zero. LASSO was chosen over traditional stepwise selection because, with 68 candidate variables and 102 events, stepwise methods are prone to overfitting and produce unstable variable subsets, whereas LASSO directly yields a parsimonious and reproducible set of predictors. The optimal lambda (lambda.min) was determined by ten-fold cross-validation, and variables with non-zero coefficients were retained as core predictors of preoperative mortality. The generalized variance inflation factor (GVIF) was calculated for each retained variable, and variables with $GVIF^{(1/(2\times Df))} > 2$ were excluded to ensure model stability. Patients were then randomly divided at a 7:3 ratio into a training cohort and a validation cohort. As this was a single-center study, this constituted internal split-sample validation.

Variables selected by LASSO were subsequently entered into univariate logistic regression analyses. Those with p-values < 0.05 in univariate analysis were included in the multivariable logistic regression model, with bidirectional stepwise selection to optimize the final model. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs), and a forest plot was generated to visualize the association between each predictor and preoperative mortality.

Model performance was evaluated by receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC) calculated to assess discriminative ability. Calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test and visualized with calibration curves. Clinical utility was evaluated using decision curve analysis (DCA). A nomogram was constructed to visualize the contribution of each predictor.

Finally, a Framingham-style risk scoring system [21] was developed based on the multivariable regression coefficients. Integer weights were assigned to each predictor, and patients were stratified into low-risk (0–2 points), medium-risk (3–4 points), and high-risk (5–7 points) categories. The association between risk category and preoperative mortality was assessed using logistic regression, and mortality rates across risk strata were visualized using stacked bar charts.

3. Results

3.1 LASSO Regression for Variable Selection

A total of 24 key variables were selected from the initial 68 variables in ATAAD patients using LASSO regres-

Table 1. Evaluation of the model for preoperative death in ATAAD patients.

Evaluation direction	Evaluation content	Training set		Validation set	
		Statistics	*p*	Statistics	*p*
Model verification	Likelihood ratio test	χ^2: 83.8870	<0.0010	χ^2: 24.2510	<0.0010
Discrimination assessment	C-index	0.813 (0.7660–0.8600)	<0.0010	0.7690 (0.6520–0.8860)	<0.0010
Calibration assessment	Goodness-of-fit test	χ^2: 7.9389	0.4395	χ^2: 5.8106	0.6684

sion. These comprised: presence of syncope, coma, limb ischemia symptoms, diminished pulses, acute renal failure; type of intimal tear; pericardial effusion; bicuspid aortic valve; heart rate; pulse pressure; pH; partial pressure of oxygen; lactate; hemoglobin; neutrophil count; interleukin-6; urea; creatinine; plasma prothrombin time; international normalized ratio; cholesterol; NT-proBNP; APACHE II score; and EuroSCORE. The variable selection trajectory and process of LASSO regression are outlined in Fig. 1.

3.2 Comparison Between Training and Validation Cohorts in the Preoperative Mortality Group

A total of 578 ATAAD patients were randomly allocated at a 7:3 ratio to a training cohort (n = 404) and an internal validation cohort (n = 174). The overall preoperative mortality rate was 17.65% (102/578; 19.31% in the training cohort and 13.79% in the validation cohort). No significant differences were observed between the training and validation cohorts before variable coding (**Supplementary Table 1**). After variable coding (**Supplementary Table 2**), no significant differences were found between the training and validation cohorts (**Supplementary Table 3**).

3.3 Comparison of Characteristics Between the Survival Group and the Preoperative Mortality Group in the Training Cohort

In the training cohort, the preoperative mortality group was characterized by higher median levels of lactate, neutrophil count, interleukin-6, urea, and creatinine, as well as lower pulse pressure and pH, and a greater proportion of patients with syncope, acute renal failure, and intimal tears at the aortic root (all $p < 0.05$). No significant differences were observed for the remaining variables. Full comparisons are presented in **Supplementary Tables 4,5**.

3.4 Univariate and Multivariate Logistic Regression Analyses of Preoperative Mortality in the Training Cohort

Univariate and multivariate logistic regression analyses identified six independent predictors of preoperative mortality (Fig. 2, **Supplementary Tables 6,7**): coma (OR = 5.93, 95% CI: 1.29–27.26, $p = 0.022$), intimal tear in the ascending aorta (OR = 6.28, 95% CI: 2.16–18.32, $p < 0.001$), intimal tear at the aortic root (OR = 10.74, 95% CI: 3.28–35.21, $p < 0.001$), lactate >1.6 mmol/L (OR = 2.43, 95% CI: 1.32–4.46, $p = 0.004$), hemoglobin <115 g/L (OR = 2.61, 95% CI: 1.15–5.94, $p = 0.022$), and creatinine >115 µmol/L (OR = 2.61, 95% CI: 1.41–4.83, $p = 0.002$). No significant multicollinearity was detected (all $GVIF^{(1/(2\times Df))} < 2$; **Supplementary Table 8**).

3.5 Evaluation and Performance of the Preoperative Mortality Prediction Model

The model demonstrated good discriminatory ability, with an AUC of 0.78 (95% CI: 0.73–0.83) in the training cohort and 0.76 (95% CI: 0.65–0.86) in the internal validation cohort (Fig. 3A). The C-index was 0.813 (95% CI: 0.766–0.860) in the training cohort and 0.769 (95% CI: 0.652–0.886) in the validation cohort, and likelihood ratio testing confirmed statistical significance in both cohorts ($p < 0.001$ and $p < 0.001$, respectively; Table 1). Calibration was satisfactory, with goodness-of-fit *p*-values of 0.440 and 0.668 for the training and validation cohorts, respectively (Fig. 3E,F and Table 1). Decision curve analysis indicated net clinical benefit across a probability threshold of 0.1–1.0 in the training cohort and 0.1–0.4 in the validation cohort (Fig. 3C,D). At the cut-off of 0.15, sensitivity was 0.85 in the training cohort and 0.75 in the validation cohort, with corresponding specificity of 0.58 and 0.60. The negative predictive value was 0.94 in both cohorts, whereas the positive predictive value was 0.33 and 0.23, respectively (Table 2). A nomogram incorporating all six predictors was constructed for bedside application, with intimal tear site contributing the greatest weight (Fig. 3B).

3.6 Scoring System and Risk Assessment for Preoperative Mortality in Patients With ATAAD

In accordance with the Framingham methodology, a scoring system was developed to predict preoperative mortality in patients with ATAAD. One point was assigned for lactate >1.6 mmol/L, one point for hemoglobin <115 g/L, and one point for creatinine >115 µmol/L. Two points were assigned for coma, two points for an intimal tear located in the ascending aorta, and three points for an intimal tear at the aortic root. The detailed scoring scheme is provided in **Supplementary Table 9**. Scores of 0–2 denoted low risk, 3–4 denoted medium risk, and 5–7 denoted high risk. A percentage bar chart was constructed according to patient scores, showing that mortality increased and survival decreased with rising scores. ORs for the medium- and high-risk groups in the training set were 6.31 and 28.97, respectively, while in the validation set they were 5.64 and 7.59, respectively. This indicates that OR values increased progressively with higher risk strata. A summary of the statistical analysis for risk scores in the training and validation

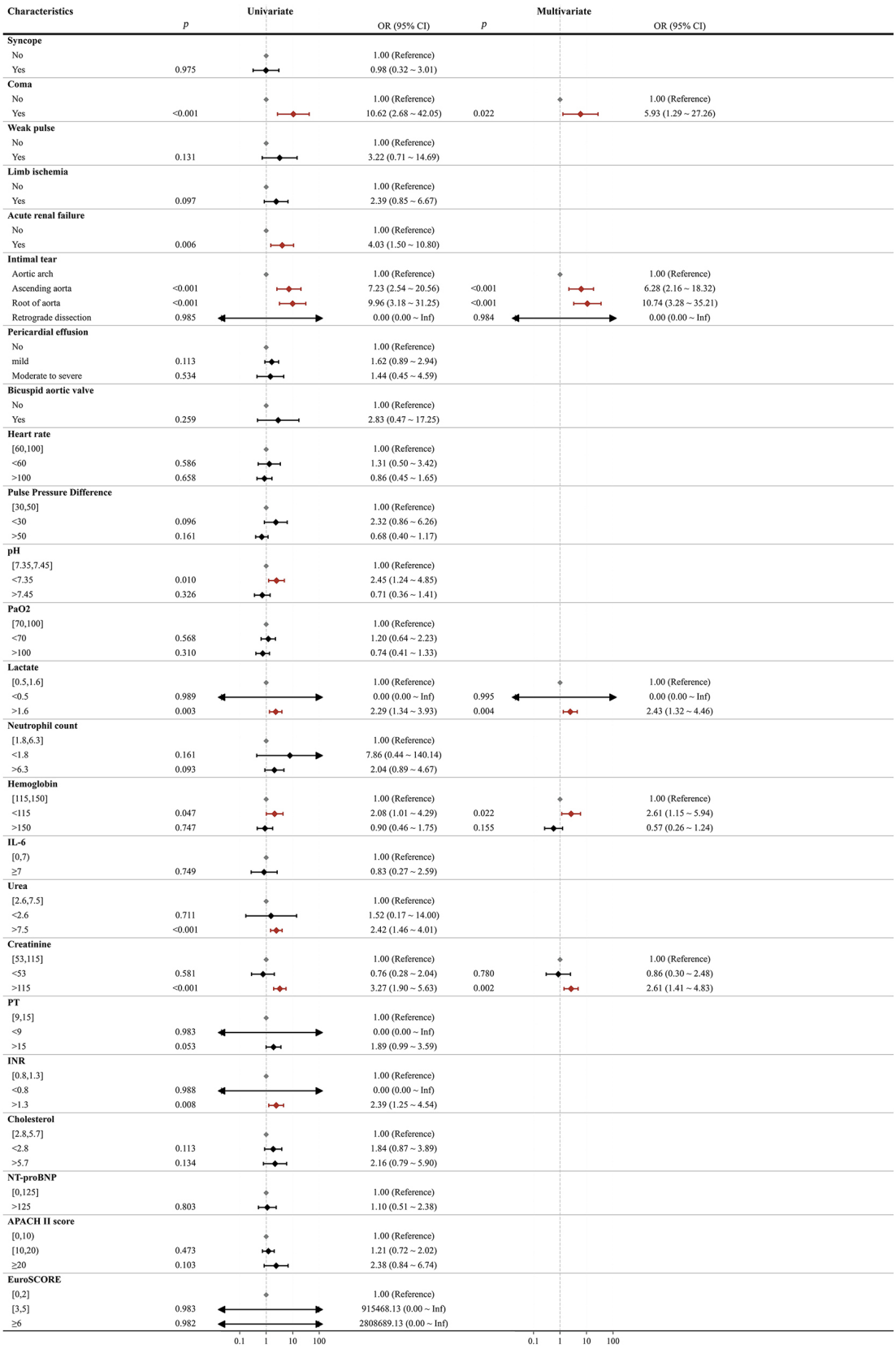


Fig. 2. Forest plot of the preoperative mortality risk in ATAAD patients. ATAAD, acute stanford type A aortic dissection; OR, odds ratio; CI, confidence interval; PT, prothrombin time; INR, international normalized ratio; NT-proBNP, N-terminal pro-brain natriuretic peptide; APACHE II, Acute Physiology and Chronic Health Evaluation II; EuroSCORE, European System for Cardiac Operative Risk Evaluation; Arrows indicate OR values beyond the plotted horizontal axis range (too large or too small to be fully displayed).

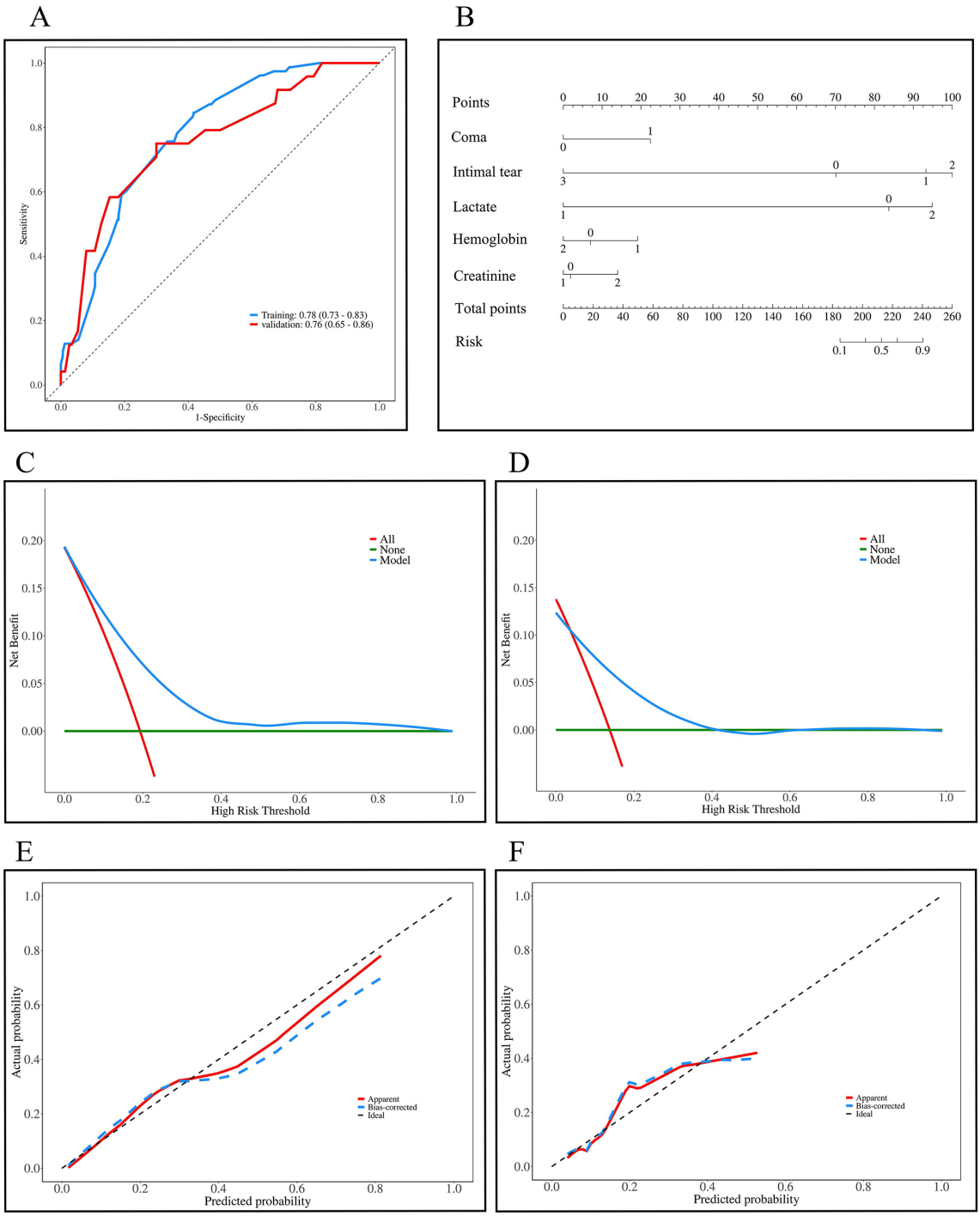


Fig. 3. Evaluation of model performance. (A) ROC curve. (B) Nomogram. (C) DCA curve of training set. (D) DCA curve of validation set. (E) Training set calibration curve. (F) Validation set calibration curve. ROC, receiver operating characteristic; DCA, decision curve analysis.

Table 2. Confusion matrix results for training and validation sets.

Data	AUC (95% CI)	Accuracy (95% CI)	Specificity (95% CI)	Sensitivity (95% CI)	NPV (95% CI)	PPV (95% CI)	Cut-off
Training set	0.78 (0.73–0.83)	0.63 (0.58–0.68)	0.58 (0.53–0.64)	0.85 (0.77–0.93)	0.94 (0.91–0.97)	0.33 (0.26–0.39)	0.15
Validation set	0.76 (0.65–0.86)	0.62 (0.54–0.69)	0.60 (0.52–0.68)	0.75 (0.58–0.92)	0.94 (0.89–0.99)	0.23 (0.14–0.32)	0.15

Notes: PPV, positive predictive value; NPV, negative predictive value; AUC, area under the curve.

Table 3. Logistic regression results according to different risk levels.

Grade	Training set					Validation set				
	β	SE	Z	*p*	OR (95% CI)	β	SE	Z	*p*	OR (95% CI)
0–2 points (low risk)					1.00 (reference)					1.00 (reference)
3–4 points (medium risk)	1.84	0.36	5.10	<0.001	6.31 (3.11–12.81)	1.73	0.58	2.98	0.003	5.64 (1.81–17.62)
5–7 points (high risk)	3.37	0.58	5.84	<0.001	28.97 (9.36–89.64)	2.03	0.85	2.39	0.017	7.59 (1.44–40.09)

cohorts is shown in **Supplementary Table 10**. Logistic regression results are presented in Table 3, and the percentage bar chart is depicted in Fig. 4.

4. Discussion

In this large, single-center retrospective cohort study, we developed and internally validated a parsimonious preoperative risk prediction model and bedside scoring system for patients presenting with ATAAD. Using LASSO regression and multivariable logistic modeling, we identified six independent predictors of preoperative mortality: coma, intimal tear site in the ascending aorta or aortic root, hyperlactatemia (>1.6 mmol/L), anemia (hemoglobin <115 g/L), and elevated serum creatinine (>115 μmol/L). These variables were integrated into a Framingham-style integer-based scoring system, which demonstrated strong discriminative performance (AUC 0.78 in training, 0.76 in validation) and good calibration. In the development cohort, observed mortality increased stepwise across the three risk categories (low: 5.59%; medium: 27.18%; high: 63.16%). In the internal validation cohort, the gradient was preserved between the low- and medium-risk groups (4.71% versus 21.79%) but attenuated in the high-risk category (27.27%), likely reflecting the small number of patients in the highest score stratum. This attenuation underscores the need for external validation prior to clinical implementation. Furthermore, the score should be interpreted with caution, particularly at the upper end of the risk spectrum, where the mortality estimate may be less stable.

4.1 Coma as a Marker of Cerebral Malperfusion

Coma emerged as the strongest clinical predictor of preoperative death, conferring a nearly six-fold increase in mortality risk. This finding aligns with prior reports highlighting neurological compromise as a key determinant of adverse outcomes in ATAAD [22,23,24]. The pathophysiology of ATAAD is multifactorial: extension of dissection into the aortic arch may compromise cerebral perfusion via compression or occlusion of the brachiocephalic vessels. Alternatively, acute tamponade or severe aortic regurgitation may precipitate global hypoperfusion and cerebral hypoxia. Furthermore, hemorrhagic shock secondary to adventitial rupture may result in rapid neurological deterioration. Notably, patients presenting with coma are often misdiagnosed with acute stroke, leading to delayed surgical intervention and further increasing the mortality risk [23]. These data emphasize the need for heightened clinical vigilance and prompt imaging in comatose patients with suspected ATAAD.

4.2 Impact of Intimal Tear Site

Our results demonstrate that the intimal tear site is a critical anatomical determinant of preoperative mortality. Tears involving the ascending aorta—particularly the aortic root—were associated with markedly increased risk. Root-level tears may compromise coronary ostial flow, precipitating acute myocardial malperfusion, cardiogenic shock, or malignant arrhythmias [25,26,27]. Moreover, their proximity to the aortic valve apparatus frequently results in acute regurgitation and left ventricular overload. Ascending aortic tears, especially those adjacent to the pericardial reflection, are prone to triggering rapid tamponade, a well-established mechanism of hemodynamic collapse and preoperative death [27,28,29]. Conversely, these findings suggest that retrograde type A dissection—in which the primary intimal tear originates in the descending aorta and extends retrogradely into the ascending aorta—is associated with lower preoperative mortality. In such cases, the entry tear is anatomically distant from the coronary ostia, the aortic valve apparatus, and the pericardial reflection, thus potentially reducing the risk of acute coronary malperfusion, severe aortic regurgitation, and cardiac tamponade. These findings support the integration of tear site into preoperative risk assessment. In particular, patients with proximal intimal disruption face the highest risk of preoperative death. While emergent surgery is mandatory for all ATAAD patients irrespective of risk profile, identifying this subgroup may help to prioritize the most time-sensitive cases and justify expedited protocols. These include rapid imaging, early mobilization for anesthesia, and direct transfer to the operating theatre, thereby minimizing pre-operative delay. Of note, pericardial effusion and coronary signs did not emerge as independent predictors in our LASSO-based variable selection, likely because both are downstream manifestations that are closely correlated with proximal intimal tear location. The tear site variable, which encompasses broader anatomical and hemodynamic information, may have subsumed the predictive signal carried by these individual findings. Additionally, coronary signs such as ST-segment changes were not systematically documented in our dataset as specific indicators of coronary ostial involvement, which may have further limited their ability to emerge as independent predictors.

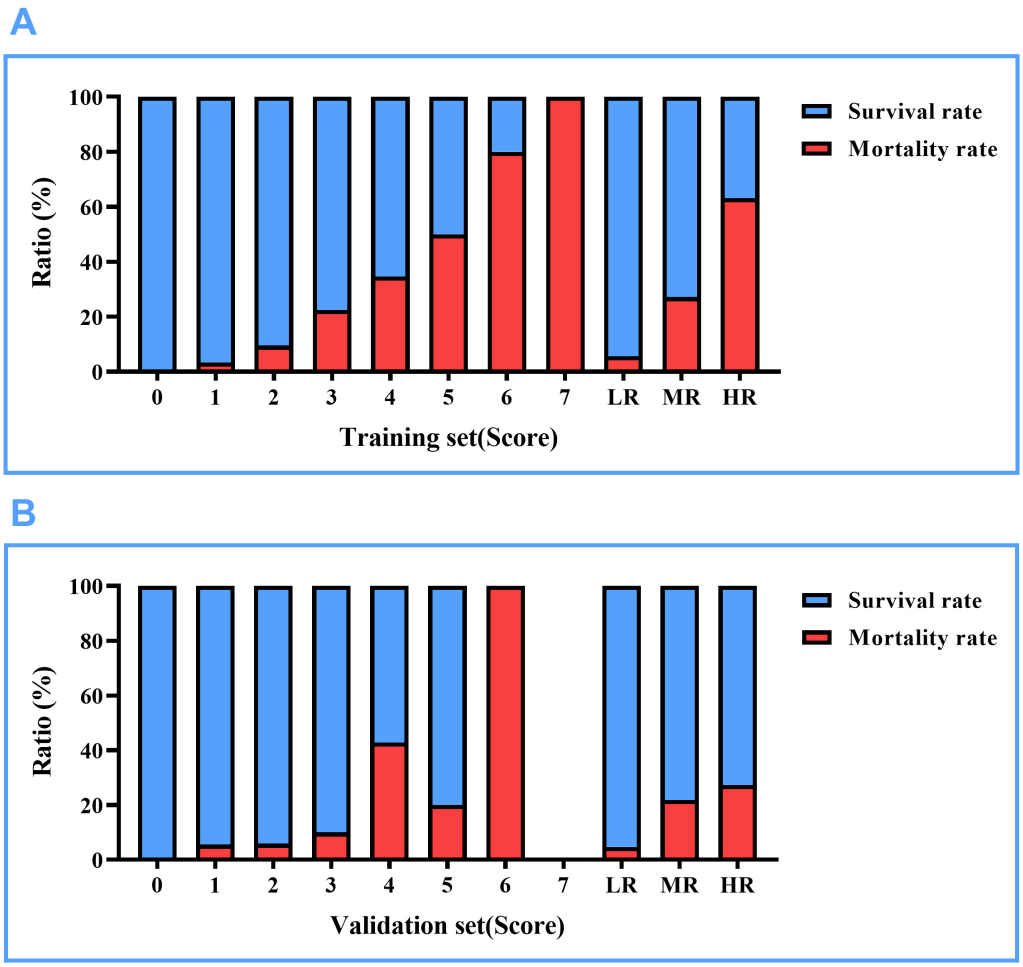


Fig. 4. Bar chart of preoperative mortality rates. Notes: (A) and (B) show results for the training and validation sets, respectively. LR, low risk; MR, medium risk; HR, high risk.

4.3 Hyperlactatemia: A Marker of Systemic Hypoperfusion

Elevated lactate (>1.6 mmol/L) was independently associated with preoperative mortality, consistent with prior studies implicating hyperlactatemia as a marker of systemic malperfusion and metabolic derangement [30,31,32]. In ATAAD, elevated lactate levels may reflect tissue hypoxia secondary to reduced cardiac output, impaired organ perfusion, or anaerobic metabolism due to malperfusion syndromes. Beyond its role as a prognostic biomarker, lactate may also directly contribute to myocardial dysfunction via intracellular acidosis and impaired calcium handling [33,34]. Its widespread availability and rapid turnaround time make lactate a valuable bedside tool for risk stratification in the emergency setting. From a triage perspective, markedly elevated lactate in a patient with a proximal intimal tear should raise concern for concurrent mesenteric malperfusion. In such cases, further imaging with CT angiography of the visceral vessels and urgent multidisciplinary assessment are warranted. Several expert centers advocate an endovascular fenestration-first or visceral stenting strategy before central aortic repair in this subgroup, with the aim of restoring mesenteric perfusion and reducing the cumulative ischemic burden of prolonged cardiopulmonary bypass [35,36,37]. While the optimal revascularization sequence remains a subject of ongoing debate, our findings—together with established evidence linking hyperlactatemia to visceral malperfusion—support the integration of lactate into early, anatomy-informed triage decisions.

4.4 Anemia and Renal Dysfunction as Indicators of Disease Severity

Both anemia (hemoglobin <115 g/L) and elevated serum creatinine (>115 μmol/L) were independently associated with preoperative death. Anemia may result from hemorrhage, hemodilution, or systemic inflammation, and has previously been linked to adverse outcomes following aortic surgery [38]. Similarly, elevated creatinine likely reflects renal malperfusion, a common complication of ATAAD due to involvement of the renal arteries or systemic hypoperfusion [39,40,41,42]. Elevated creatinine is a readily accessible biochemical marker that serves as an early warning sign of organ malperfusion and may guide timely

intervention. Together, these findings underscore the importance of integrating routine laboratory parameters into preoperative risk assessment.

4.5 Integration Into Clinical Practice: A Proposed Triage Framework

The derived model demonstrated robust discrimination and calibration across both training and validation cohorts. The risk-stratified preoperative mortality rates differed between the training and validation sets. In the training cohort, the mortality was 5.59% for the low-risk group, 27.18% for the intermediate-risk group, and 63.16% for the high-risk group. In the validation cohort, the mortality rates of low-, intermediate-, and high-risk strata were 4.71%, 21.79%, and 27.27%, respectively.

We emphasize that the score is not intended to determine whether a patient undergoes surgery, and emergent repair remains mandatory for all ATAAD patients, regardless of risk category. Rather, its principal value lies in surgical prioritization in high-volume centers and resource-constrained settings, where several AD patients may present simultaneously, and a rapid, objective ranking is needed to allocate operating theatre capacity, anesthesia teams, and blood products. In such settings, high-risk patients (observed mortality 63.16%) should be prioritized for immediate, parallel activation of multiple resources, with aggressive minimization of each component of pre-operative delay. Specifically, the presence of coma should prompt urgent CT angiography with intracranial vessel evaluation to exclude concurrent cerebral infarction, while avoiding any delay due to diagnostic uncertainty between ATAAD-related coma and primary stroke. A proximal intimal tear (ascending aorta or root) should trigger direct transfer to the operating theatre without intermediate stops, with simultaneous mobilization of the surgical and anesthesia teams. Markedly elevated lactate (>1.6 mmol/L) should prompt assessment for mesenteric malperfusion, including CT angiography of the visceral vessels, as well as multidisciplinary discussion (where appropriate) regarding the potential role of endovascular fenestration before central aortic repair. Concomitant anemia or renal dysfunction should lower the threshold for early blood product resuscitation and invasive hemodynamic monitoring. Medium-risk patients (observed mortality 27.18%) warrant a heightened level of vigilance, including early notification of the cardiac anesthesia team, preparation for potential hemodynamic support, and cross-matching of blood products before the patient reaches the operating theatre. Low-risk patients (observed mortality 5.59%) should proceed with standard expedited surgical preparation, recognizing that even this subgroup carries a non-trivial preoperative mortality risk.

Importantly, although the negative predictive value of the model was high (0.94; Table 2), caution is still warranted, since a classification of "low-risk" by this score does not imply the patient is at negligible risk of preoperative death. Even within the low-risk category, the observed mortality was 5.59%, which is clinically consequential in this universally lethal condition. Therefore, under no circumstances should a low-risk score be used to defer, delay, or rule out emergent surgical repair. The risk categories are intended solely to guide the intensity and urgency of preoperative preparation, not to determine whether a patient proceeds to surgery. All ATAAD patients require emergent surgical intervention. In centers with limited operating theatre capacity or simultaneous admissions, the score may provide an evidence-based framework for surgical prioritization.

Unlike existing risk scores that often require complex computations or specialized imaging, our model relies on six variables that can be obtained within minutes of presentation: neurological status via physical examination, intimal tear site via CT angiography (which is routinely performed for diagnostic confirmation), and three standard laboratory parameters (lactate, hemoglobin, creatinine). This facilitates rapid bedside application in both tertiary referral centers and lower-resource settings, where timely risk stratification can inform perioperative planning, resource allocation, and inter-hospital transfer decisions.

4.6 Comparison With Existing Risk Models

Several established risk stratification tools exist for AD and cardiac surgery, but none directly addresses preoperative mortality in ATAAD. The GERAADA score predicts 30-day postoperative mortality and relies on variables unavailable at initial emergency assessment [43]. EuroSCORE II, derived from a heterogeneous cardiac surgery population, does not capture the hyperacute ATAAD cascade and overpredicts mortality in this cohort [44]. IRAD-based predictors identify individual in-hospital mortality risk factors, but these have not been consolidated into a point-of-care scoring system calibrated for the preoperative window [45,46]. Our model differs in that it specifically targets preoperative death. This covers a clinically meaningful gap, since such patients are excluded from surgical registries yet constitute approximately 4–11% of ATAAD presentations [47]. Furthermore, our model relies on six immediately available bedside variables to guide the urgency of pre-operative preparation rather than surgical candidacy.

4.7 Strengths and Limitations

This study presents the first parsimonious bedside score for pre-operative mortality in ATAAD, derived via LASSO and internal validation using six immediately available variables that yield a steep risk gradient (5.59% → 27.18% → 63.16%) in training set. The single-center, retrospective design carries potential selection bias, and the absence of external validation in a geographically distinct cohort limits generalizability to international populations. In addition, the exclusion of patients with extensive comor-

bidities or missing data also limits generalizability. CT imaging details, including the distinction between root-level tears with and without coronary ostial involvement, were not systematically documented and therefore could not be incorporated into the analysis. Given the significant impact of acute coronary malperfusion on preoperative outcomes in ATAAD, further studies that integrate this distinction are warranted. Finally, the endpoint was restricted to in-hospital pre-operative death. The relatively small number of events (n = 102) tempers precision, necessitating multicenter prospective validation.

5. Conclusions

We developed and validated a simple, six-item preoperative mortality risk score for ATAAD. This score incorporates clinical and biochemical variables that are readily available at initial presentation. Our model demonstrates strong discriminative performance and clinical utility, offering a practical tool for bedside risk stratification. Early identification of high-risk patients may facilitate timely intervention, optimize perioperative management, and ultimately improve survival in this critically ill population.

Availability of Data and Materials

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

Author Contributions

JG: Methodology, Data collection, Formal analysis, Data Curation, Writing—Original Draft, Writing—Review & Editing. JYM: Data collection, Writing—Review & Editing. XYY: Conceptualization, Supervision, Writing—Original Draft, Writing—Review & Editing. All authors read and approved the final manuscript. All authors agree to be 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.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the First Affiliated Hospital of Xinjiang Medical University (Approval No. K202310-01; Date: 2023-10-11). Informed consent was waived by the Institutional Review Board of the Xinjiang Medical University Medical Ethics Committee due to the retrospective nature of the study, in accordance with national regulations.

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Conflicts of Interest

The authors declare no conflicts of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/HSF54732.

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