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ORIGINAL RESEARCH

Analysis of early in-hospital mortality risk factors in patients with Stanford type A aortic dissection involving coronary arteries after surgery

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Abstract

Background: To identify the risk factors associated with early in-hospital mortality in patients with Stanford Type A Aortic Dissection (ATAAD) involving the coronary arteries after surgical intervention. **Methods**: We retrospectively analyzed data from 74 patients diagnosed with ATAAD involving coronary arteries treated at our hospital between August 2018 and March 2024. They were categorized into two groups: a survival group (n = 30) and a mortality group (n = 44) based on their outcomes at discharge. Multivariate logistic regression analysis was performed to identify independent risk factors for early in-hospital mortality, based on which a diagnostic model was developed. **Results**: Independent risk factors for early in-hospital mortality included ascending aortic diameter (in mm), duration of ventilator use (≥96 hours), postoperative aspartate aminotransferase (AST) levels (in U/L), high-sensitivity troponin levels >2000 ng/L and the occurrence of malignant arrhythmia. The diagnostic model based on these factors exhibited excellent predictive performance, with an area under the curve (AUC) of 0.945, sensitivity of 88.6% and specificity of 93.3%. Additionally, the calibration plot demonstrated high consistency between predicted and observed outcomes. **Conclusions**: The proposed diagnostic model, using five key factors: ascending aortic diameter, duration of ventilator use, postoperative AST levels, high-sensitivity troponin levels and malignant arrhythmia, could be promising for predicting early in-hospital mortality in patients with ATAAD involving coronary arteries after surgery.

Keywords

Stanford type A aortic dissection; Involving coronary arteries; In-hospital mortality; Risk factors

1. Introduction

Acute Stanford Type A Aortic Dissection (ATAAD) is a highly life-threatening cardiovascular condition characterized by its sudden onset and rapid progression [1]. Despite significant advancements in surgical techniques and perioperative management, the in-hospital mortality rate after surgery remains alarmingly high, ranging from 10% to 20%, which continues to adversely impact patient prognosis. A major complication of ATAAD is coronary artery involvement, which is associated with irreversible myocardial damage [2] and occurs in approximately 10%–20% of cases [3, 4]. It is also considered an independent risk factor for postoperative mortality, with the in-hospital mortality rate for these patients reaching 41.8% [5].

The complexity of ATAAD increases when coronary arteries are involved, as it necessitates not only urgent management of the aortic dissection but also prompt restoration of coronary blood flow, thereby presenting a significant challenge for cardiac surgeons. Although medical technologies have advanced—particularly in local coronary artery repair and coronary artery bypass grafting (CABG) techniques—improvements in patient survival post-surgery have been modest. Nevertheless, the risk of early postoperative mortality remains considerable [6]. Previous studies have highlighted several factors, including age, preoperative cardiac function, surgical duration, cardiopulmonary bypass (CPB) time and postoperative renal dysfunction, which may influence the postoperative prognosis of ATAAD patients [7, 8]. However, there remains a lack of research specifically addressing the clinical outcomes of patients with ATAAD complicated by coronary artery involvement.

Therefore, this study aims to investigate the clinical characteristics and potential risk factors for early postoperative mortality in this high-risk subgroup to provide a scientific basis for clinical decision-making, optimize therapeutic strategies, and ultimately reduce the incidence of early postoperative

mortality.

2. Materials and methods

2.1 General information

Between August 2018 and March 2024, a total of 435 patients diagnosed with ATAAD underwent surgical treatment at our institution. Among these, 74 cases (17.01%) involved coronary artery involvement. Clinical data from these 74 patients were retrospectively collected and divided into two groups based on their outcomes at discharge: a survival group (n = 30) and a mortality group (n = 44). The study inclusion criteria were: (a) a clinically and radiologically confirmed diagnosis of ATAAD, meeting the diagnostic criteria for aortic dissection as outlined in "Aortic Dissection" [9]; (b) documented coronary artery involvement, characterized by ST-segment elevation $\geq$0.1 mV, ischemic changes such as ST segment depression or T-wave inversion on electrocardiogram (ECG), and elevated biomarkers such as creatine kinase-myocardial band (CK-MB) or high-sensitivity troponin T (hs-cTnT) [10]; (c) patients who underwent surgical treatment specifically for ATAAD; (d) availability of complete clinical data for analysis; and (e) patients aged 18 years or older.

Patients were excluded from the study if they met any of the following criteria: (a) a diagnosis other than ATAAD or those who did not undergo surgical treatment; (b) concurrent severe cardiovascular conditions, such as heart failure, that could confound the study outcomes; (c) incomplete follow-up data during the hospital stay; or (d) patients who died before undergoing surgery.

2.2 Treatment methods

Surgical Treatment: Before surgery, all patients underwent comprehensive preoperative assessments, including digital subtraction angiography (DSA) or computed tomography angiography (CTA) of the aorta, electrocardiography, and routine laboratory tests to determine the location of the dissection entry, identify the involvement of vital branch vessels, assess valve regurgitation and evaluate ventricular wall motion. After assessing coronary artery pathology, surgical interventions were tailored according to the extent of dissection involving the ascending aorta, aortic arch, aortic valve and coronary arteries. The following coronary artery surgical strategies were employed, based on the degree of dissection involvement, with values ranging from 1 to 6 corresponding to "coronary artery surgical strategy" in this study:

a. Coronary ostium involvement (right coronary artery (RCA) commonly affected): In cases where the dissection involved only the coronary ostium, typically with the RCA affected, careful reinforcement of the coronary ostium was performed. A double-layer Polytetrafluoroethylene felt strip was placed externally around the ostium, and continuous 5-0 polypropylene sutures were used to secure the dissected layers, effectively preventing bleeding and ensuring hemostasis. No coronary reimplantation was required, and the proximal aorta was then anastomosed end-to-end with the artificial graft.

b. Inner coronary artery ostium involvement with normal distal segment: When the dissection affected the inner portion of the coronary ostium, but the distal coronary segment remained normal, the dissected intima was excised to normal tissue. The left and right coronary ostia were directly anastomosed to the wall of the artificial graft. Meticulous hemostasis was achieved, and the proximal aorta was subsequently treated using either the Bentall or David procedure.

c. Dissecting aneurysm involving the coronary artery: For cases where the dissection involved the coronary artery, forming a dissecting aneurysm but with intact and unruptured intima, a buttoned coronary ostium repair was performed. This was followed by anastomosis of the coronary ostium to the side wall of the artificial grafts.

d. Coronary artery stenosis: For patients with preoperative coronary artery stenosis, CABG was the preferred approach. The saphenous vein grafts were primarily used for CABG, with the option to select the left internal mammary artery if the left subclavian artery was not involved and suitable conditions permitted.

e. Thrombus in the proximal end of the dissection extending to the coronary artery: In cases where a thrombus was present at the proximal end of the dissection and extended into the coronary artery, the thrombus was carefully removed. The coronary ostium was then probed with a 1.5 mm coronary artery dilator to confirm patency. Intraoperative Transesophageal Echocardiography (TEE) and coronary flow measurement using a Doppler flow probe were employed to assess distal perfusion. If patency was found to be inadequate, CABG was performed.

f. Complete avulsion of the coronary artery ostium intima: In cases of complete avulsion of the coronary artery ostium intima, leading to proximal flow occlusion, CABG was performed, and the closed coronary ostium was ligated.

Intensive Care Unit (ICU) Management: During the perioperative period, effective sedation and analgesia were administered to maintain a target systolic blood pressure between 100 and 120 mmHg and a heart rate below 60 beats per minute. Standardized hemodynamic monitoring was performed using a Swan-Ganz catheter (Edwards Lifesciences), which allowed continuous assessment of cardiac index, pulmonary artery wedge pressure, and systemic vascular resistance. These parameters were recorded hourly for the first 48 hours postoperatively to closely monitor the patient's hemodynamic status. The postoperative management focused on infection prevention and secondary prevention of coronary heart disease. Antiplatelet therapy strategies were individualized based on treatment protocols to carefully balance the therapeutic benefits and potential risks. Symptomatic and supportive care was provided as needed to stabilize the patient. In addition, all patients underwent daily transthoracic or TEE to evaluate ventricular function, valvular integrity and coronary perfusion. This comprehensive monitoring protocol enabled precise identification of the cause of mortality, such as heart failure versus systemic complications, by integrating real-time hemodynamic data with structural and functional cardiac imaging.

2.3 Observation indicators

The primary endpoint of this study was in-hospital mortality from any cause in patients with ATAAD involving coronary arteries following surgery. In addition to mortality data, specific causes of death were recorded to identify early postoperative risk factors for mortality in this patient cohort.

The baseline characteristics of the patients were comprehensively recorded, including age, sex, history of coronary artery disease, diabetes, hypertension, chronic kidney disease, hyperlipidemia and smoking (specifically smoking more than 20 cigarettes per day). Based on previous research indicating that smoking alone is not a highly significant risk factor for in-hospital mortality, we further refined this indicator, considering smoking >20 cigarettes per day as a relevant risk factor for this study.

Preoperative assessments included measurement of D-dimer (D-D), troponin T (TNT), total bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), high-sensitivity C-reactive protein, and the complete blood count (white blood cell count, platelet count and neutrophil percentage). Other recorded data included fibrinogen levels, the presence of pericardial effusion (including pericardial tamponade), and detailed assessments of the aorta and coronary arteries. This included the ascending aortic diameter, the location and size of the entry tear, and the involvement of critical vascular structures, such as the coronary arteries, innominate artery, left common carotid artery, left subclavian artery, renal artery and the presence of aortic insufficiency. Importantly, the "pericardial effusion" indicator was recorded as a binary variable (presence or absence), without further classification based on effusion volume or progression to cardiac tamponade. This limitation may influence the interpretation of myocardial injury markers, such as TNT and myocardial enzymes, in understanding the mechanisms contributing to early mortality.

The intraoperative data comprised key surgical and procedural parameters such as CPB time, aortic cross-clamp time, deep hypothermic circulatory arrest (DHCA) duration, total operative time and blood transfusion volume. The surgical strategy for coronary artery management was also documented. Deep hypothermic circulatory arrest was defined by a core body temperature maintained between 18 °C and 22 °C (measured via nasopharyngeal and bladder probes) during circulatory arrest, achieved through active cooling via CPB. DHCA was universally applied to all ATAAD patients requiring aortic arch reconstruction to protect vital organs from ischemic injury.

Postoperative management and complications were also recorded, which included the 24-hour postoperative drainage volume, total blood transfusion volume, need for re-thoracotomy, and the duration of mechanical ventilation (≥96 hours as the threshold). Postoperative complications, such as the occurrence of infection, acute kidney injury (AKI) stage 3, and various biochemical markers of myocardial injury, including postoperative AST, ALT, creatine kinase (CK), creatine kinase-MB (CK-MB) and myoglobin (>3000 ng/mL), were also assessed. High-sensitivity troponin (>2000 ng/L) levels and the occurrence of malignant arrhythmias (ventricular fibrillation or persistent ventricular tachycardia) were also monitored as part of the comprehensive postoperative evaluation.

2.4 Determination method of biomarker critical value

High-sensitivity Troponin (≥2000 ng/L): The reference threshold for diagnosing acute myocardial infarction is based on the 99th percentile, as defined by the European Society of Cardiology (ESC) guidelines, and this threshold is also supported by previous research on myocardial injury following cardiac surgery. Additionally, our center's prior cohort study demonstrated a significant predictive value for postoperative mortality when this threshold was used, with an area under the receiver operating characteristic (ROC) curve (AUC) of 0.78.

AST (≥1000 U/L), ALT (≥2000 U/L): These thresholds align with the definition of acute liver injury, as described by the ratio of AST to ALT exceeding 10 times the upper limit of normal (ULN). The thresholds also reflect the upper limit of the laboratory's linear detection range, with AST ≥1000 U/L representing the endpoint of this range. This approach is consistent with previous studies on liver dysfunction following cardiac surgery.

Myoglobin (≥3000 ng/mL): A myoglobin level greater than 3000 ng/mL (P75 threshold) was selected as a risk stratification threshold based on consensus guidelines for diagnosing cardiac surgery-related rhabdomyolysis. This value also represents the peak myoglobin level within 24 hours post-surgery, where the median value for this cohort was 2850 ng/mL.

2.5 Statistical analysis

All data were statistically analyzed using SPSS version 22.0 (IBM, Armonk, NY, USA) or R version 4.1.2 software. Continuous variables with a normal distribution were expressed as the mean ± standard deviation (SD) and compared using the Student's *t*-test. For continuous variables that were not normally distributed, data were presented as the median (interquartile range, IQR) and compared using the Mann-Whitney U test. Categorical variables are reported as counts (percentages) and analyzed using either the Pearson χ^2 test or Fisher's exact test, depending on the expected frequency of the categories. Univariate logistic regression analysis was performed to identify potential risk factors for in-hospital mortality. Variables with a p-value < 0.10 in the univariate analysis were included in the subsequent multivariate logistic regression model. Binary logistic regression was then applied to all selected variables to establish a risk prediction model. Statistical significance was set at $p < 0.05$.

For clinical application, a nomogram was constructed to provide a visual representation of the prediction model for approximate risk assessment. Additionally, an online risk prediction calculator was developed to allow for more precise calculations. The discriminative ability of the prediction model was evaluated using the C-statistic, which represents the AUC along with its 95% confidence interval (CI). A C-statistic value of 0.5 indicates no discrimination ability, whereas a value of 1.0 reflects perfect discrimination.

The calibration of the prediction model was assessed using

a calibration curve and the Brier score. A Brier score of 0 indicates perfect calibration, while a score of 1 represents the worst possible calibration.

3. Results

3.1 Comparison of general information

In the baseline data, a significantly higher proportion of smokers (defined as consuming more than 20 cigarettes daily) was observed in the deceased group compared to the survival group ($p < 0.05$). Regarding preoperative data, the deceased group exhibited a significantly smaller ascending aortic diameter compared to the survived group ($p < 0.05$). In terms of intraoperative data, the score for the coronary artery surgical procedure was significantly higher in the deceased group than in the survived group ($p < 0.05$).

In the postoperative data, several parameters were significantly worse in the deceased group. These included a longer duration of ventilator use, a higher proportion of patients with postoperative AKI stage 3, as well as significantly higher myoglobin levels, high-sensitivity troponin levels, and postoperative AST and ALT levels. Furthermore, the deceased group had a significantly higher proportion of patients with malignant arrhythmias compared to the survived group (all $p < 0.05$). No significant differences were observed between the two groups for the remaining variables ($p > 0.05$). For a detailed comparison of these findings, refer to Table 1.

3.2 Binary logistic regression analysis

To identify independent risk factors for early postoperative in-hospital mortality, significant variables from the general data were included in a binary logistic regression analysis. The dependent variable was group status, with survival coded as 1 and death as 2. Among the independent variables, the actual values of ascending aortic diameter and coronary artery surgical strategy were used. Binary indicators included smoking (defined as consuming more than 20 cigarettes daily), ventilator use duration ($\geq$96 hours), postoperative AKI stage 3, postoperative AST ($\geq$1000 U/L), postoperative ALT ($\geq$2000 U/L), myoglobin (>3000 ng/mL), high-sensitivity troponin (>2000 ng/L) and malignant arrhythmias. These variables were assigned a value of 1 if present and 0 if absent.

The regression analysis identified ascending aortic diameter (mm), ventilator use duration ($\geq$96 hours), postoperative AST (U/L), high-sensitivity troponin (>2000 ng/L), and malignant arrhythmias as independent risk factors for early postoperative in-hospital mortality in patients with ATAAD involving the coronary arteries (Table 2).

The independent risk factors with a variance inflation factor (VIF) <5 were included in the final regression model. The model was adjusted for clinically relevant confounders, including age, preoperative ejection fraction and CPB time. Based on biological plausibility and the predictive value of key factors, adjustments were made for variables with changes exceeding 10%. The logistic regression prediction model was constructed and fitted, as presented in Table 3. The Hosmer-Lemeshow test ($\chi^2 = 7.2$, $p = 0.41$) confirmed that the model demonstrated adequate calibration and good overall fit. The final logistic regression equation for predicting the probability of early postoperative mortality in patients with ATAAD involving the coronary artery was:

$$P = \frac{1}{1 + e^{-(1.948 - 0.094x_1 + 2.558x_2 + 1.851x_3 + 2.578x_4 + 1.889x_5)}}$$

3.3 Establishment of a nomogram

A nomogram was developed to visualize the predictive model for early postoperative in-hospital mortality using the "rms" package in R software. The independent risk factors identified in the logistic regression analysis, including ascending aortic diameter (mm), ventilator use duration ($\geq$96 hours), postoperative AST (U/L), high-sensitivity troponin (>2000 ng/L) and malignant arrhythmias, were incorporated into the model. The resulting nomogram provides an intuitive tool for estimating the probability of early postoperative mortality, as illustrated in Fig. 1.

3.4 Validation and evaluation of the prediction model

The predictive performance of the model was assessed using ROC curve analysis. The model demonstrated strong discriminative ability, with an AUC of 0.945, a sensitivity of 88.6% and a specificity of 93.3%. These findings indicate that the model could be a reliable tool for distinguishing between survival and mortality outcomes in patients with ATAAD involving the coronary arteries (Table 4 and Fig. 2).

The calibration plot was used to visually evaluate the agreement between the predicted and actual probabilities of early postoperative mortality. As shown in Fig. 3, the Apparent Line is closely aligned with the Ideal Line, particularly within the critical probability intervals, indicating that the model provides an accurate estimation of actual probabilities in most cases. To further validate the model's stability and accuracy, a Bias-Corrected Line was introduced, which demonstrated a trend closer to the Ideal Line after accounting for potential overfitting, which further confirmed the model's calibration ability and strong goodness of fit. The results of the calibration plot indicate that the predictive model not only exhibits high calibration accuracy but also maintains a strong agreement between predicted and observed outcomes, reinforcing its predictive reliability and robustness.

4. Discussion

Early postoperative in-hospital mortality remains a significant concern in patients with ATAAD involving coronary arteries, and identifying risk factors predictive of postoperative mortality is essential for optimizing clinical management and improving outcomes. This study demonstrated that ascending aortic diameter (mm), ventilator use duration ($\geq$96 hours), postoperative AST (U/L), high-sensitivity troponin (>2000 ng/L), and malignant arrhythmias are independent risk factors for early postoperative in-hospital mortality in this patient population. The logistic regression model incorporating these variables achieved an AUC of 0.945, with a sensitivity of

TABLE 1. Comparison of general information between the two groups.

Variables	Survival group (n = 30)	Death group (n = 44)	t/χ^2	p
Age (yr)	47.73 ± 12.60	52.66 ± 12.16	1.686	0.096
Gender (male)	24	40	1.816	0.178
History of coronary heart disease	2	4	0.141	0.708
Diabetes	0	2	1.402	0.236
Hypertension	16	30	1.672	0.196
Chronic kidney disease	3	5	0.034	0.853
Hyperlipidemia	3	6	0.221	0.638
Marfan syndrome	8	10	0.150	0.698
Smoking	18	38	6.735	0.009
D-dimer (mg/L)	2.84 ± 1.41	2.57 ± 1.36	0.812	0.419
TNT (μg/L)	100.03 ± 105.75	110.39 ± 158.12	0.314	0.755
Total bilirubin level (μmol/L)	15.63 ± 8.35	13.34 ± 7.38	1.243	0.218
ALT (U/L)	43.47 ± 45.94	28.82 ± 14.63	1.689	0.101
AST (U/L)	47.13 ± 74.06	38.14 ± 39.87	0.676	0.501
High-sensitivity C-reactive protein (mg/L)	10.32 ± 1.60	9.94 ± 1.83	0.938	0.351
White blood cell count ($\times 10^9$)	15.44 ± 5.81	15.71 ± 6.42	0.186	0.853
Platelet count ($\times 10^9$)	102.53 ± 20.81	107.35 ± 23.14	0.916	0.363
Neutrophil percentage (%)	78.89 ± 6.79	79.34 ± 13.25	0.190	0.850
Fibrinogen (g/L)	2.56 ± 0.90	2.11 ± 1.21	1.724	0.089
Presence of pericardial effusion	10	8	2.225	0.136
Diameter of ascending aorta (mm)	58.20 ± 9.38	47.64 ± 9.47	4.730	<0.001
CTA reveals coronary artery involvement	10	12	0.314	0.575
UCG indicates abnormal wall movement	8	12	0.003	0.954
Electrocardiogram ischemia STT	16	25	0.088	0.767
Pathological Q wave	3	6	0.221	0.638
Involvement of innominate artery	18	28	0.100	0.751
Involvement of left common carotid artery	14	24	0.443	0.506
Involvement of left subclavian artery	12	24	1.511	0.219
Involvement of renal artery	12	19	0.074	0.785
Aortic regurgitation	20	28	0.072	0.789
Cardiopulmonary bypass time (min)	322.63 ± 86.07	351.84 ± 78.88	1.507	0.136
Aortic cross-clamp time	197.10 ± 47.64	214.55 ± 65.94	1.321	0.191
Deep hypothermic circulatory arrest time	21.83 ± 2.67	22.93 ± 5.90	1.084	0.282
Total operative time (min)	598.27 ± 91.85	641.75 ± 106.18	1.825	0.072
Blood transfusion volume (mL)	3948.50 ± 1851.12	3654.62 ± 1443.92	0.766	0.446
coronary artery surgical strategy	2.67 ± 1.37	3.45 ± 1.50	2.293	0.025
Postoperative 24-hour drainage volume (mL)	1842.77 ± 1281.58	1852.77 ± 1069.16	0.036	0.971
Postoperative 24-hour blood transfusion volume	1362.43 ± 959.51	1881.86 ± 1443.43	1.859	0.067
Re-exploration of thorax	4	8	0.309	0.579
Duration of ventilator use (≥96 h)	7	26	9.231	0.002
Postoperative infection	12	18	0.006	0.938
Postoperative AKI stage 3	19	37	4.175	0.041

TABLE 1. Continued.

Variables	Survival group (n = 30)	Death group (n = 44)	t/χ^2	p
Postoperative AST (U/L)	6	29	15.082	<0.001
Postoperative ALT (U/L)	10	26	4.737	0.030
Postoperative creatine kinase (CK) (U/L)	4203.63 ± 4469.46	7010.18 ± 18132.27	0.829	0.410
Postoperative creatine kinase-MB (CK-MB) (U/L)	145.43 ± 136.71	220.59 ± 203.37	1.768	0.081
Myoglobin (>3000 ng/mL)	10	25	3.947	0.047
High-sensitivity troponin (>2000 ng/L)	4	25	14.154	<0.001
Malignant arrhythmia	7	30	14.352	<0.001

Note: Different surgical strategies for the "coronary artery surgical strategy" were assigned values ranging from 1 to 6. Details regarding the specific assignments are provided in the Methods section. TNT: troponin T; ALT: alanine aminotransferase; AST: aspartate aminotransferase; CTA: computed tomography angiography; AKI: acute kidney injury; CK: creatine kinase; UCG: echocardiography; STT: ST-T segment changes on electrocardiogram; CK-MB: creatine kinase-myocardial band.

TABLE 2. Univariate logistic regression analysis of early postoperative in-hospital mortality risk.

Variables	β	SE	Wald χ^2	p	OR	95% CI
Smoking (>20 cigarettes/d)	1.447	1.140	1.612	0.204	4.252	0.455–39.722
Diameter of ascending aorta (mm)	−0.087	0.041	4.496	0.034	0.916	0.845–0.993
coronary artery surgical strategy	0.024	0.328	0.005	0.942	1.024	0.538–1.950
Duration of ventilator use (≥96 h)	2.833	1.104	6.585	0.010	17.003	1.953–148.045
Postoperative AKI stage 3	0.154	1.077	0.021	0.886	1.167	0.141–9.637
Postoperative AST (U/L)	1.899	0.936	4.117	0.042	6.682	1.067–41.850
Postoperative ALT (U/L)	−1.017	1.080	0.886	0.347	0.362	0.044–3.006
Myoglobin (>3000 ng/mL)	1.385	0.995	1.937	0.164	3.995	0.568–28.092
High-sensitivity troponin (>2000 ng/L)	2.966	1.246	5.662	0.017	19.409	1.687–223.324
Malignant arrhythmia	2.170	1.033	4.411	0.036	8.755	1.156–66.309

AKI: acute kidney injury; AST: aspartate aminotransferase; ALT: alanine aminotransferase; CI: confidence interval; SE: standard error; OR: odds ratio.

TABLE 3. Multivariate logistic regression analysis of early postoperative in-hospital mortality risk.

Variables	β	SE	Wald χ^2	p	OR	95% CI
Diameter of ascending aorta (mm)	−0.094	0.040	5.540	0.019	0.910	0.842–0.984
Duration of ventilator use (≥96 h)	2.558	0.915	7.823	0.005	12.913	2.150–77.552
Postoperative AST (U/L)	1.851	0.840	4.854	0.028	6.365	1.227–33.028
High-sensitivity troponin (>2000 ng/L)	2.578	0.976	6.976	0.008	13.172	1.944–89.223
Malignant arrhythmia	1.889	0.877	4.641	0.031	6.612	1.186–36.870
constant	1.948	2.259	0.744	0.388	7.016	

AST: aspartate aminotransferase; CI: confidence interval; SE: standard error; OR: odds ratio.

88.6% and a specificity of 93.3%, indicating strong discriminative ability and predictive accuracy.

The incidence of coronary artery involvement in ATAAD patients in this study was 17.01%, consistent with previous reports. For instance, Wang *et al.* [3] reported a 14.9% incidence, and Bossone *et al.* [4] reported an incidence of 20%, supporting the reliability of these findings. Yuichi Saito *et al.* [11] further characterized coronary artery involvement in ATAAD, reporting that 52.3% of affected patients had RCA involvement, while 47.7% had left coronary artery (LCA) involvement. Notably, the postoperative in-hospital mortality rate in their study was 42.3%, with significantly higher mortality in patients with LCA involvement compared to RCA involvement (54.3% *vs.* 31.3%, $p < 0.001$). In contrast, Chang *et al.* [12] analyzed 267 patients and reported a one-year postoperative mortality rate ranging from 11.1% to 20.8%. The in-hospital mortality rate in the present study was 59.46%, closely aligning with the findings of Yuichi Saito . The lower mortality rate reported by Chang *et al.* [12] may be attributable to differences in baseline characteristics, treatment protocols

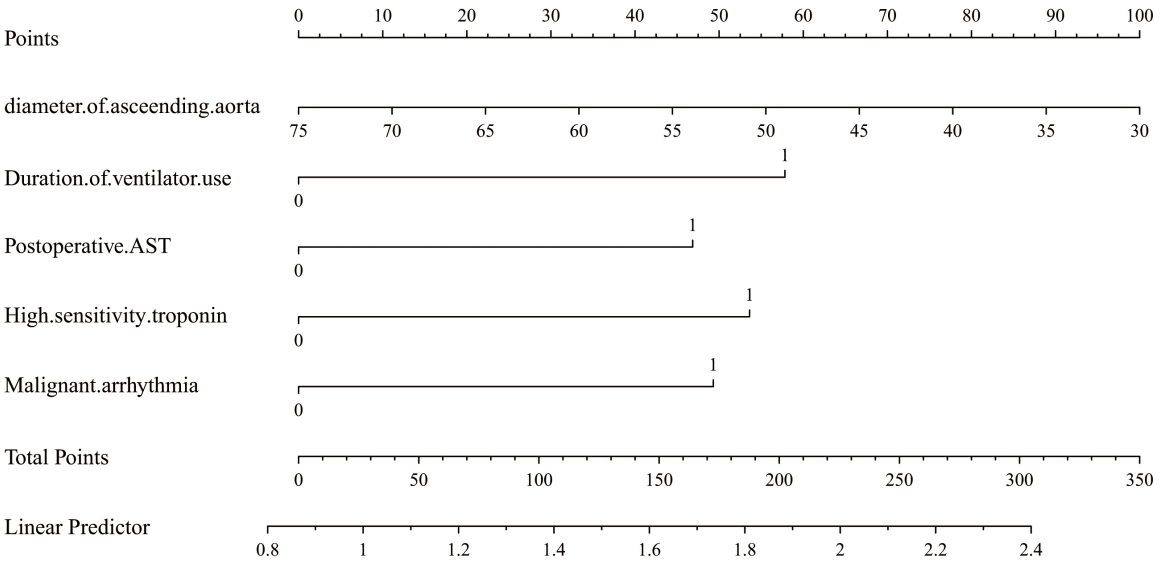

FIGURE 1. Nomogram for predicting in-hospital mortality of patients. AST: aspartate aminotransferase.

TABLE 4. Diagnostic value of independent risk factors.

Variables	Cut-off	AUC	SE	*p*	95% CI	Sensitivity	Specificity
Diameter of ascending aorta (mm)	49.500	0.804	0.052	<0.001	0.703–0.905	0.727	0.800
Duration of ventilator use (≥96 h)	0.500	0.679	0.063	0.009	0.555–0.803	0.591	0.767
Postoperative AST (U/L)	0.500	0.730	0.060	0.001	0.612–0.848	0.659	0.800
High-sensitivity troponin (>2000 ng/L)	0.500	0.717	0.060	0.002	0.600–0.835	0.568	0.867
Malignant arrhythmia	0.500	0.724	0.061	0.001	0.605–0.844	0.682	0.767
Model prediction probability	0.549	0.945	0.026	<0.001	0.894–0.995	0.886	0.933

AST: aspartate aminotransferase; AUC: area under the curve; CI: confidence interval; SE: standard error.

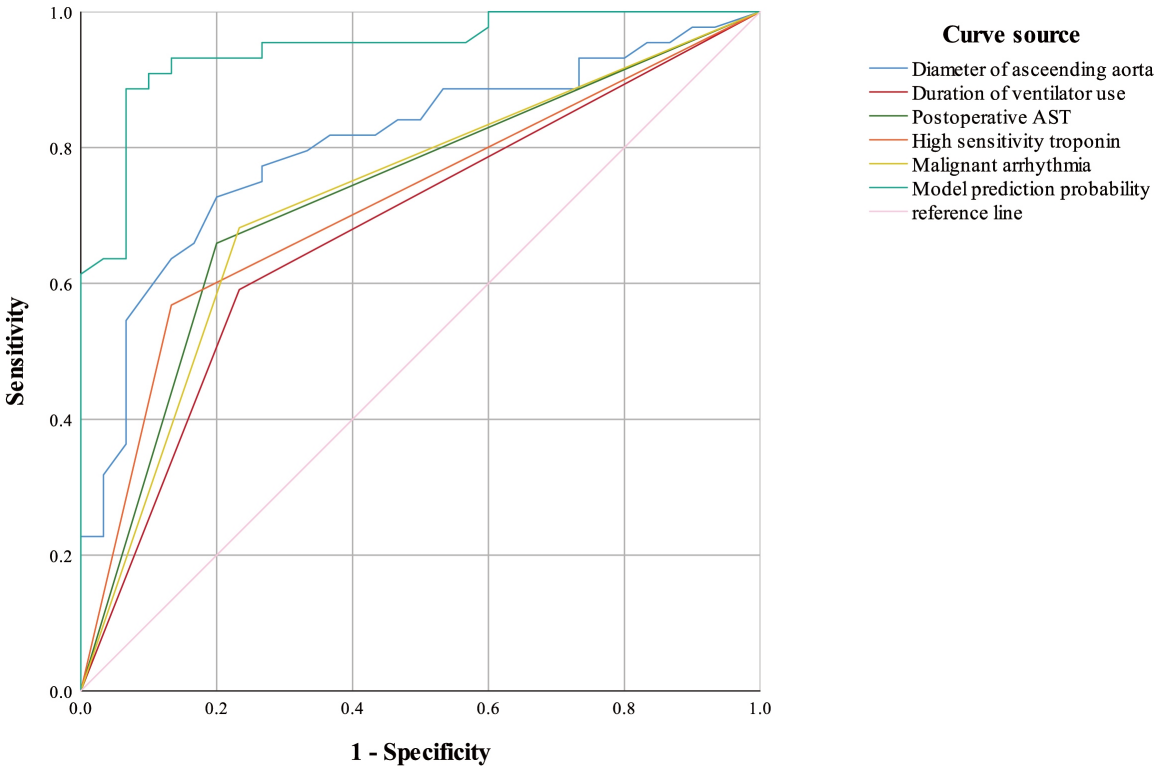

FIGURE 2. ROC Curves of independent risk factors. AST: aspartate aminotransferase.

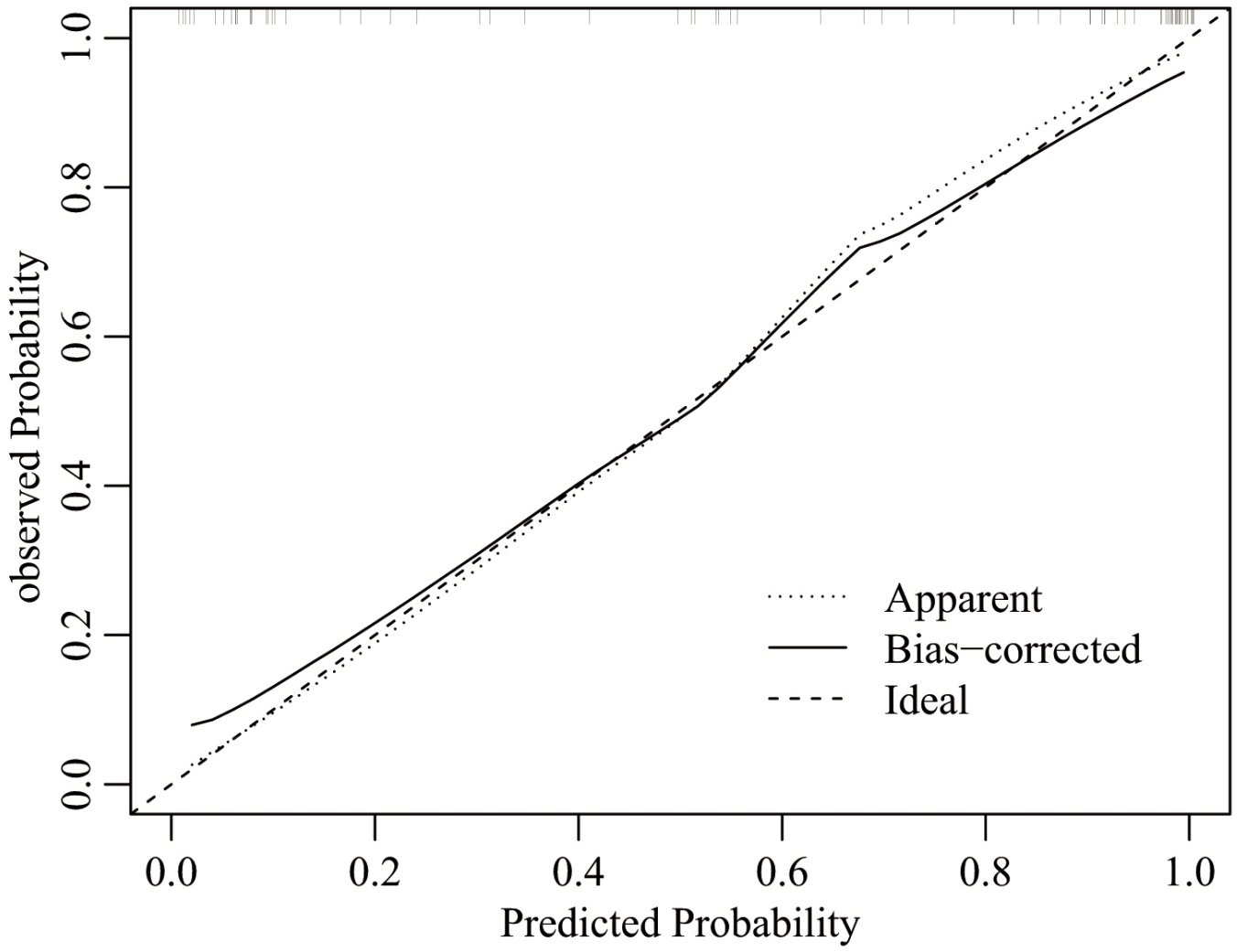

FIGURE 3. Calibration curve of the prediction model.

or follow-up duration. Additionally, malperfusion syndromes (mesenteric: 15%, cerebral: 12%) were not adjusted for in this study, which may have contributed to excess mortality. A meta-analysis of 23 studies involving 5510 patients identified age, male sex, shock, malperfusion and cardiac tamponade as key risk factors for early postoperative mortality in ATAAD patients [13]. However, the present study identified ascending aortic diameter, ventilator use duration, postoperative AST, high-sensitivity troponin, and malignant arrhythmias as significant predictors in ATAAD patients with coronary artery involvement. These findings suggest that the risk profile of this subgroup differs from that of the broader ATAAD population, highlighting the importance of tailored risk assessment and management strategies in this high-risk group.

Our findings indicate that ascending aortic diameter is an independent predictor of early postoperative mortality in patients with ATAAD involving coronary arteries (aOR = 1.32 per mm, 95% CI: 1.12–1.56), aligning with its well-established role in aortic dissection risk stratification [14]. Prior studies suggest that combining aortic diameter with geometric parameters, such as arch angle and curvature, may enhance predictive accuracy for dissection risk [15, 16].

The diameter of the ascending aorta is a key anatomical parameter in cardiovascular risk assessment, and differences in diameter and length may help differentiate patients with type A aortic dissection, normal controls, and those with thoracic aortic aneurysms without dissection [17]. Prior studies suggest that integrating geometric parameters with diameter measurements improves risk prediction [15, 16]; however, technical limitations in preoperative imaging may have influenced our findings, as 25% of patients underwent non-gated CTA, potentially limiting the detection of subtle anatomical correlations. Despite these limitations, our results indicate that ascending aortic diameter is a significant risk factor not only for the occurrence of aortic dissection but also for its progression and prognosis in patients with ATAAD involving coronary arteries.

Wu *et al.* [18] reported a negative correlation between ascending aortic diameter and long-term prognosis, suggesting that even smaller diameters may predispose patients to dissection with poor outcomes. In cases where ATAAD involves the coronary arteries, the primary blood supply to the heart, changes in aortic diameter may be more closely associated with myocardial ischemia or infarction, further influencing early mortality risk. However, these findings should be interpreted with caution. As Elefteriades *et al.* [19] highlighted, aortic size alone is a poor predictor of dissection risk in normoten-

sive patients. Future studies incorporating aortic geometry parameters, such as curvature and arch angle, alongside wall stress modeling, are necessary to further validate the prognostic significance of ascending aortic diameter in this patient population.

AST is an enzyme primarily found in myocardial cells, hepatocytes, skeletal muscle cells and the kidneys, playing a key role in amino acid metabolism within cells [20]. When tissue damage occurs, AST is released into the bloodstream, resulting in an elevated serum concentration [21]. Due to this property, AST serves as a biochemical marker for assessing tissue injury and is commonly used to evaluate liver function [22]. High-sensitivity troponin (hs-cTn) is a highly sensitive and specific biomarker widely used in the diagnosis of acute myocardial infarction and myocardial injury [23]. Compared to traditional myocardial enzymes, hs-cTn has greater sensitivity and specificity, allowing for the earlier detection of subtle myocardial damage [24]. The findings of this study indicate that postoperative AST levels (U/L) and hs-cTn levels $>$2000 ng/L are significant risk factors for early mortality in patients with ATAAD involving coronary arteries, underscoring the role of both hepatic and myocardial injury in postoperative outcomes. Previous studies have demonstrated a strong association between AST elevation and postoperative liver dysfunction in ATAAD patients [25–27]. Following surgery for ATAAD involving coronary arteries, multiple factors, including surgical trauma, ischemia-reperfusion injury and drug-induced hepatotoxicity, can lead to increased AST levels. Elevated AST may reflect severe liver injury, which in turn impairs metabolic and detoxification functions, thereby increasing the risk of postoperative complications and mortality. Similarly, hs-cTn has been identified as a predictor of aortic dissection with strong prognostic value, with studies suggesting its involvement in macrophage-driven inflammation [28]. Furthermore, hs-cTn has been associated with long-term mortality in ATAAD patients [29].

While elevated postoperative AST and hs-cTn levels were strong predictors of early mortality in this study, their interpretation requires careful consideration. For instance, for hs-cTn $>$2000 ng/L, although this elevation indicates myocardial injury, its underlying cause may extend beyond direct coronary artery involvement. Alternative mechanisms include ischemia due to cardiac tamponade, which was observed in 27% of patients in this study, or systemic inflammation resulting from prolonged CPB. In addition, for AST levels $\geq$1000 U/L, rather than reflecting isolated liver injury, this elevation is more likely attributable to hepatic congestion due to right heart strain and skeletal muscle breakdown secondary to prolonged immobilization. Given these findings, hs-cTn and AST should not be viewed as markers of isolated myocardial or hepatic injury but rather as indicators of systemic physiological derangement.

Tissue damage and functional decline are key contributors to early postoperative mortality in patients with ATAAD involving coronary arteries. Herein, we identified ventilator use duration ($\geq$96 hours) and malignant arrhythmias as critical risk factors for early mortality in this patient population. ATAAD involving coronary arteries leads to extensive vascular damage, which not only compromises cardiac blood supply but also contributes to systemic hemodynamic instability, potentially resulting in multiple organ dysfunction syndrome (MODS) [30]. Given the heart's central role in systemic circulation, insufficient myocardial perfusion exacerbates circulatory disturbances, creating a vicious cycle that further deteriorates organ function and survival outcomes. Coronary artery involvement is a key indicator of poor prognosis in ATAAD patients, as it directly reduces myocardial oxygen and nutrient supply, leading to ischemia, necrosis and potentially heart failure [31]. A rapid decline in cardiac function not only decreases effective cardiac output but also increases myocardial workload, predisposing patients to malignant arrhythmias, such as ventricular tachycardia and ventricular fibrillation, which are among the leading causes of early mortality [32]. Prolonged ventilator use often reflects postoperative respiratory dysfunction or the presence of severe complications, while malignant arrhythmias indicate significant cardiac electrical instability that may result in sudden cardiac arrest and circulatory collapse. Both factors contribute to systemic organ ischemia and hypoxia, exacerbating physiological stress and metabolic dysfunction. The combined presence of prolonged ventilator dependence and malignant arrhythmias represents significant cardiac and systemic compromise, markedly increasing the risk of early postoperative mortality in patients with ATAAD involving coronary arteries.

This study has several limitations. First, key variables such as the severity of cardiac tamponade and intraoperative hemodynamic fluctuations (*e.g.*, nadir Mean Arterial Pressure (MAP) $<$50 mmHg) were not analyzed due to inconsistent documentation, which may have affected the comprehensive assessment of hemodynamic instability. Second, the single-center study design limits the generalizability of the findings, particularly in relation to institution-specific surgical protocols, such as the uniform use of DHCA. Third, coronary artery involvement was categorized as a binary variable (present/absent), without considering the impact of collateral circulation or the completeness of revascularization, which may have influenced the prognostic implications of coronary involvement in ATAAD.

5. Conclusions

This study identifies ascending aortic diameter, prolonged ventilator use, elevated AST and hs-cTn levels, and malignant arrhythmias as potential predictors of early postoperative mortality in patients with ATAAD involving coronary arteries. While these markers may contribute to risk stratification, their predictive validity requires external validation in multicenter cohorts with standardized coronary assessment protocols. Future predictive models could incorporate tear location, laterality of coronary involvement and malperfusion syndromes to improve clinical applicability. These findings should be interpreted as hypothesis-generating rather than definitive, emphasizing the need for individualized risk assessment in clinical decision-making.

AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be

obtained from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

QLL, YQQ—designed the study and carried them out; prepared the manuscript for publication and reviewed the draft of the manuscript. QLL, YQQ, ZYZ, TY, HL, YC, HKL—supervised the data collection; analyzed the data; interpreted the data. All authors have read and approved the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of Zhongshan City People's Hospital (Approval no. KY2024-130). Written informed consents was obtained from a legally authorized representative for anonymized patient information to be published in this article.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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