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Article

Analysis of the Incidence, Risk Factors, and Prognosis of Early Acute Heart Failure After TEVAR for Complex Acute Type B Aortic Dissection

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Abstract

Background: Complex acute type B aortic dissection (ATBAD) is an aortic emergency associated with high mortality. Thoracic endovascular aortic repair (TEVAR) has become the treatment of choice for complex ATBAD. However, early acute heart failure (AHF) after TEVAR can significantly increase perioperative mortality risk, and systematic studies on its incidence, multidimensional risk factors, and independent impact on mid- and long-term prognosis remain insufficient. **Methods:** A total of 225 patients with complex ATBAD who underwent TEVAR at our hospital from January 2021 to January 2025 were prospectively enrolled. Patients were divided into an AHF group (n = 34) and a non-AHF group (n = 191) according to whether AHF occurred within 30 days postoperatively. Baseline data, imaging characteristics, laboratory parameters, and perioperative data were collected. Univariable and multivariable binary logistic regression analyses were used to identify independent risk factors for AHF. Receiver operating characteristic (ROC) curves were plotted to evaluate predictive performance, and internal validation was conducted using Bootstrap resampling (B = 1000); Kaplan-Meier analysis and Cox proportional hazards regression (Fine-Gray competing risks model) were used to assess the impact of AHF on 1-year all-cause mortality. **Results:** Among 225 patients, the incidence of AHF within 30 days after TEVAR was 15.1% (34/225). Multivariable logistic regression identified four independent risk factors: preoperative B-type natriuretic peptide (BNP) ≥100 pg/mL (odds ratio [OR] = 7.84, 95% confidence interval [CI] 3.12–19.68), concomitant coronary artery disease (CAD) (OR = 3.10, 95% CI 1.05–9.13), admission heart rate per 10 beats/min increase (OR = 1.72, 95% CI 1.28–2.31), and stent coverage length per 10 mm increase (OR = 1.14, 95% CI 1.04–1.25) (all $p < 0.05$). The combined prediction model constructed from these four factors achieved an area under the curve (AUC) of 0.897 (95% CI 0.842–0.952); Bootstrap-corrected AUC was 0.868 (95% CI 0.801–0.935); the AUC for preoperative BNP alone was 0.842 (optimal cutoff 98.5 pg/mL). The AHF group had significantly higher 30-day all-cause mortality (17.6% vs 2.6%, $p = 0.002$) and 30-day major adverse cardiac and cerebrovascular events (MACCE) rate (35.3% vs 9.4%, $p < 0.001$) than the non-AHF group. Kaplan-Meier analysis showed a lower 1-year cumulative survival rate in the AHF group (73.5%) compared with the non-AHF group (93.7%, Log-rank $p < 0.001$). After adjusting for confounders, AHF remained an independent risk factor for 1-year all-cause mortality (hazard ratio [HR] = 4.32, 95% CI 1.86–10.04, $p = 0.001$). **Conclusions:** The incidence of early AHF after TEVAR in complex ATBAD patients is 15.1%. Preoperative BNP ≥100 pg/mL, concomitant CAD, elevated admission heart rate, and longer stent coverage length are independent risk factors. AHF independently increases perioperative and 1-year all-cause mortality risk.

Keywords: acute type B aortic dissection; thoracic endovascular aortic repair; acute heart failure; risk factors; prognosis

1. Introduction

Acute type B aortic dissection (ATBAD) is an aortic emergency with an acute onset and rapid progression. Based on the presence or absence of malperfusion syndrome, persistent pain, refractory hypertension, rapid aortic expansion, or rupture, ATBAD is classified as complicated or uncomplicated [1]. For complicated ATBAD, in-hospital mortality can reach 10%–25% without timely and effective intervention [1,2]. In recent years, thoracic endovascular aortic repair (TEVAR) has become the treatment of choice for complicated ATBAD (Class I recommendation) due to its minimally invasive nature and repeatability, playing an important role in promoting false lumen thrombosis and preventing further aortic expansion [3]. However, it should be noted that for uncomplicated ATBAD, current guidelines still recommend optimal medical therapy (OMT) as the first choice [4].

Early cardiac complications after TEVAR should not be overlooked; acute heart failure (AHF) is one of the most harmful, significantly prolonging intensive care unit (ICU) stay and increasing perioperative mortality risk [5]. From a pathophysiological perspective, ATBAD patients often have myocardial hypertrophy and diastolic dysfunction secondary to long-term hypertension, with varying degrees of impaired cardiac compensatory reserve. The acute change in aortic wall compliance after stent deployment can lead to a sudden increase in left ventricular afterload; combined with excessive catecholamine and renin-angiotensin-

aldosterone system (RAAS) activation induced by surgical stress, this increases the risk of cardiac decompensation [6]. Meanwhile, when the dissection involves a wide range, true lumen compression, coronary ostial involvement, or pericardial hemorrhage can further aggravate myocardial injury [7].

However, clinical studies on early AHF after TEVAR have the following limitations: first, most studies are retrospective case analyses with inconsistent inclusion criteria and AHF diagnostic definitions, resulting in widely variable reported incidences (7%–22%) [8,9]; second, risk factor analyses are largely confined to a single dimension (preoperative biomarkers or intraoperative parameters), lacking multidimensional systematic assessments encompassing baseline characteristics, imaging morphology, laboratory parameters, and surgical parameters; third, although the impact of AHF on short-term mortality has been reported, its independent effect on mid- and long-term prognosis (especially 1-year all-cause mortality) has not been systematically evaluated [10].

Based on these research gaps, this study aimed to: determine the incidence of AHF within 30 days after TEVAR in complex ATBAD patients; systematically identify independent risk factors for AHF and construct a combined prediction model; and evaluate the independent impact of AHF on perioperative and 1-year prognosis, so as to provide evidence-based references for early identification of high-risk patients and formulation of precision perioperative management strategies.

2. Materials and Methods

2.1 Study Population

The protocol was reviewed and approved by the Medical Ethics Committee; all enrolled patients or their legal representatives signed informed consent forms, and the study was conducted in strict accordance with the principles of the Declaration of Helsinki. A consecutive series of 225 complex ATBAD patients who underwent TEVAR at our institution between January 2021 and January 2025 was enrolled.

Inclusion criteria: ① Age ≥18 years; ② Stanford type B aortic dissection confirmed by computed tomography angiography (CTA); ③ Time from symptom onset to surgery <14 days (acute phase); ④ Meeting diagnostic criteria for complex ATBAD, with TEVAR indication confirmed by the aortic multidisciplinary team (MDT); ⑤ Underwent TEVAR at our institution; ⑥ Complete clinical data with available postoperative follow-up data.

Definition of complex ATBAD: Patients with one or more of the following clinical features were defined as complex ATBAD [2,11]: ① Malperfusion syndrome, including renal malperfusion (acute kidney injury or anuria), mesenteric malperfusion (abdominal pain with elevated lactate), or lower extremity malperfusion (acute limb ischemia); ② Persistent or recurrent pain despite adequate analgesia and antihypertensive therapy; ③ Refractory hypertension that cannot be effectively controlled despite the combined intravenous use of three or more antihypertensive agents; ④ Rapid aortic expansion, defined as CTA showing an increase in maximum aortic diameter >5 mm over a short period or maximum diameter ≥40 mm with a progressive trend; ⑤ Signs of dissection rupture or impending rupture, with imaging showing hemothorax, mediastinal hemorrhage, or effusion. All enrolled patients met at least one of the above complex features, and the surgical indications were confirmed by an aortic MDT discussion.

Exclusion criteria: ① Previously confirmed heart failure [New York Heart Association (NYHA) Class ≥II] or history of heart failure; ② Concomitant Stanford type A aortic dissection or Marfan syndrome; ③ Prior aortic or cardiac surgery [coronary artery bypass grafting (CABG), valve surgery, etc.]; ④ Preoperative severe arrhythmia (e.g., sustained ventricular tachycardia, third-degree atrioventricular block); ⑤ Severe hepatic or renal insufficiency (preoperative serum creatinine >450 μmol/L or Child-Pugh Class C cirrhosis); ⑥ Concomitant malignancy or expected survival <6 months; ⑦ Pregnancy or lactation; ⑧ Withdrawal of informed consent or loss to follow-up.

2.2 Surgical Procedure

All patients underwent TEVAR under general anesthesia or local anesthesia combined with monitored anesthesia care. Preoperative CTA was used to accurately measure the proximal landing zone length, aortic diameter, and location of the primary entry tear; appropriately sized covered stent grafts were selected (covering the primary tear with proximal landing zone ≥15 mm). Stent deployment was performed via femoral artery access under digital subtraction angiography (DSA) guidance; left subclavian artery (LSA) reconstruction or chimney stent technique was applied as needed to maintain arch branch perfusion. Postdeployment angiography confirmed satisfactory stent position, endoleak status, and patency of major branch vessels.

2.3 Definition and Diagnosis of Early Postoperative AHF

Early postoperative AHF was defined as new-onset acute heart failure within 30 days after TEVAR. Diagnostic criteria were based on the 2021 European Society of Cardiology (ESC) Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure, requiring simultaneous fulfillment of the following conditions: ① Acutely onset symptoms and signs of heart failure (dyspnea, orthopnea, pulmonary rales, peripheral edema, etc.); ② Plasma B-type natriuretic peptide (BNP) >100 pg/mL or N-terminal pro-B-type natriuretic peptide (NT-proBNP) >300 pg/mL; ③ Echocardiography showing structural or functional cardiac abnormalities (reduced ejection fraction, diastolic dysfunction, or wall motion abnormality); ④ Exclusion of noncardiogenic causes (e.g., dilutional pulmonary edema from

large-volume fluid infusion, pulmonary infection, pneumothorax, etc.).

All suspected AHF cases were confirmed by cardiovascular physicians; when there was disagreement in diagnosis, two physicians of associate chief physician rank or above jointly reviewed the cases.

2.4 Data Collection

The following data were collected by specially trained research assistants using standardized electronic case report forms (CRF):

Baseline data: Age, sex, body mass index (BMI), history of hypertension, diabetes mellitus, CAD, cerebrovascular disease, chronic obstructive pulmonary disease (COPD), chronic kidney disease, smoking history.

Classification of complex dissection features: malperfusion syndrome, persistent pain, refractory hypertension, rapid aortic expansion, rupture or impending rupture.

Clinical features: Time from onset to surgery, admission systolic and diastolic blood pressure, heart rate, Numerical Rating Scale (NRS) pain score.

Imaging characteristics: Location of dissection entry tear, extent of dissection involvement (number of descending aorta segments involved), maximum aortic diameter, involvement of visceral arteries, false lumen/true lumen ratio.

Laboratory parameters: Preoperative white blood cell (WBC) count, neutrophil-to-lymphocyte ratio (NLR), platelet count, hemoglobin, serum creatinine, blood urea nitrogen (BUN), liver function tests [alanine aminotransferase (ALT), aspartate aminotransferase (AST)], fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), high-sensitivity C-reactive protein (hs-CRP), D-dimer, preoperative BNP or NT-proBNP.

Intraoperative data: Type of anesthesia, operation time, stent coverage length, proximal landing zone (arch branch involvement), whether LSA reconstruction was performed, intraoperative blood loss, intraoperative contrast volume, presence of immediate endoleak.

Postoperative data: Mechanical ventilation duration, postoperative hemodynamic parameters, perioperative medication use (β-blockers, RAAS inhibitors, and diuretics).

2.5 Follow-Up Protocol

All patients were followed up at outpatient visits or by telephone at 1, 3, 6, and 12 months postoperatively, and annually thereafter. Follow-up was terminated upon all-cause death or the administrative end date of the study (January 30, 2026), whichever came first. Follow-up included: symptom assessment (NYHA class), echocardiography (ejection fraction, wall motion), aortic CTA (standardly performed at 3 months, 12 months, and annually thereafter, or earlier if clinical symptoms changed), and BNP/NT-proBNP measurements.

The primary outcome was 30-day all-cause mortality. Secondary outcomes included: major adverse cardiac and cerebrovascular events (MACCE, including cardiovascular death, non-fatal myocardial infarction, stroke, and repeat revascularization), aortic-related adverse events (retrograde type A dissection, stent-related complications, reintervention), and 1-year all-cause mortality.

2.6 Group Assignment

Patients were divided into the AHF group and the non-AHF group according to whether AHF occurred within 30 days after TEVAR. Baseline characteristics, perioperative features, and outcome measures were compared between groups.

2.7 Statistical Methods

Statistical analyses were performed using SPSS 26.0 (IBM Corp., Armonk, NY, USA) and R 4.3.0 (R Foundation for Statistical Computing, Vienna, Austria). Descriptive statistics and between-group comparisons: Normally distributed continuous variables are expressed as mean ± standard deviation ($\bar{x} \pm s$) and compared using independent-samples *t*-tests; non-normally distributed continuous variables are expressed as median (interquartile range) [M (P_{25}, P_{75})] and compared using the Mann-Whitney U test. Categorical variables are expressed as frequency (percentage) [n (%)] and compared using the chi-squared test or Fisher's exact test (when expected cell frequency <5). Risk factor analysis: Only preoperative baseline variables (demographic data, comorbidities, complex feature classification, admission clinical parameters, imaging characteristics, preoperative laboratory parameters) and intraoperative variables (operation time, stent coverage length, intraoperative blood loss, contrast volume, immediate endoleak) were considered as candidate predictors. Postoperative variables (mechanical ventilation duration, peak postoperative blood pressure and heart rate, perioperative medication use) carry a risk of reverse causality and were included only in descriptive comparisons, not in regression analyses. Candidate variable selection was based on three principles: ① Variables associated with AHF or perioperative cardiac complications in aortic surgery; ② Key variables reflecting the pathophysiological features of ATBAD; ③ Surgery-related technical parameters. For highly collinear variable clusters (Spearman correlation coefficient >0.5 or variance inflation factor [VIF] >5), the variable with the strongest clinical representativeness or largest univariable effect size was retained. Specifically, among inflammatory markers (NLR, hs-CRP, D-dimer; r = 0.52–0.68), NLR was retained; among renal function markers (serum creatinine and BUN; r = 0.71), serum creatinine was retained. Admission systolic blood pressure and admission heart rate had a correlation coefficient of 0.46, which did not meet the threshold for strong collinearity (VIF both <5), and both were therefore included as candidates. Among imaging parameters,

maximum aortic diameter and false lumen/true lumen ratio had a correlation coefficient of 0.53; the false lumen/true lumen ratio with the larger univariable effect size was retained as the candidate. After this grouping and screening process, the number of candidate variables for the multivariable model was reduced from 21 to 14. Univariable logistic regression was performed first; variables with $p < 0.10$ and clinically important variables were included in the multivariable binary logistic regression model, and independent risk factors were selected using the backward stepwise method (likelihood ratio). Given that this study had 34 AHF events, based on the events-per-variable (EPV) ≥8 principle, the multivariable model should include no more than 4 variables; the collinearity grouping strategy described above was applied during modeling to reduce the number of candidate variables and lower the risk of overfitting. Results are expressed as odds ratio (OR) and 95% confidence interval (CI). Prediction model evaluation and internal validation: receiver operating characteristic (ROC) curves were plotted and area under the curve (AUC) was calculated to evaluate the predictive performance of independent risk factors; optimal cutoff values were determined by the maximum Youden index. Bootstrap resampling (B = 1000) was used to calculate the optimism-corrected C-statistic and calibration slope. Ten-fold cross-validation was performed as a sensitivity analysis to evaluate the internal validity of the combined prediction model. Survival analysis: Kaplan-Meier curves were plotted and Log-rank tests were used to compare survival differences between groups. Cox proportional hazards regression (Fine-Gray competing risks model adjusting for non-cardiovascular death) was used to analyze the impact of AHF on 1-year all-cause mortality; hazard ratio (HR) and 95% CI are reported after adjustment for baseline confounders. All statistical tests were two-sided; $p < 0.05$ was considered statistically significant.

3. Results

3.1 Comparison of Baseline Clinical Characteristics Between Groups

Among 225 patients, 34 developed new-onset AHF within 30 days after TEVAR, with an incidence of 15.1%; the remaining 191 (84.9%) did not develop AHF and were included in the non-AHF group.

The AHF group had significantly higher age (62.3 ± 10.5 vs 55.8 ± 11.2 years, $p = 0.001$), hypertension prevalence (94.1% vs 73.8%, $p = 0.011$), CAD prevalence (23.5% vs 8.9%, $p = 0.014$), admission systolic blood pressure (172.4 ± 26.3 vs 157.3 ± 23.8 mmHg, $p = 0.002$), and admission heart rate (90.5 ± 14.8 vs 79.2 ± 12.3 beats/min, $p < 0.001$) than the non-AHF group. There were no statistically significant differences between groups in sex distribution, BMI, diabetes mellitus, cerebrovascular disease, COPD, chronic kidney disease, smoking history, admission diastolic blood pressure, NRS pain score, or time from onset to surgery (all $p > 0.05$) (**Supplementary Table 1**).

3.2 Distribution of Complex Features in the Study Population

All 225 patients had complex ATBAD and had TEVAR indications confirmed by aortic MDT assessment. The distribution of complex features was as follows: malperfusion syndrome in 87 patients (38.7%), including renal malperfusion in 42 (18.7%), mesenteric malperfusion in 21 (9.3%), and lower extremity malperfusion in 38 (16.9%) (with overlap); persistent pain in 68 (30.2%); refractory hypertension in 53 (23.6%); rapid aortic expansion in 41 (18.2%); and rupture or impending rupture in 12 (5.3%). Eighty-two patients (36.4%) had two or more concurrent complex features. There were no statistically significant differences in the distribution of complex features between the two groups (all $p > 0.05$) (**Supplementary Table 2**).

3.3 Comparison of Imaging Characteristics Between Groups

The AHF group had significantly larger maximum aortic diameter (42.6 ± 8.3 vs 38.9 ± 7.1 mm, $p = 0.008$) and false lumen/true lumen ratio [2.8 (2.0, 3.8) vs 2.3 (1.7, 3.1), $p = 0.032$] than the non-AHF group. The proportion of visceral artery involvement was higher in the AHF group, approaching the threshold for statistical significance (41.2% vs 27.2%, $p = 0.099$). There were no significant differences between groups in entry tear location or number of descending aorta segments involved ($p > 0.05$) (**Supplementary Table 3**).

3.4 Comparison of Laboratory Parameters Between Groups

The AHF group had significantly higher WBC count, NLR, hs-CRP, D-dimer, and preoperative BNP levels than the non-AHF group (all $p < 0.05$). Hemoglobin was lower in the AHF group ($p = 0.024$). Serum creatinine, BUN, and fasting blood glucose also differed significantly between groups (all $p < 0.05$). There were no significant differences in platelet count, ALT, AST, or any lipid parameters (all $p > 0.05$) (**Supplementary Table 4**).

3.5 Comparison of Perioperative Data Between Groups

Intraoperative: The AHF group had significantly longer operation time [96.5 (75.0, 128.5) vs 82.0 (65.0, 108.0) min, $p = 0.016$], greater stent coverage length (186.3 ± 42.8 vs 162.5 ± 38.4 mm, $p = 0.003$), higher intraoperative blood loss [280 (180, 420) vs 200 (120, 320) mL, $p = 0.005$], and greater contrast volume (128.4 ± 38.6 vs 112.6 ± 34.2 mL, $p = 0.017$) than the non-AHF group. There were no significant differences in the proportion of proximal landing zone in Zone 2 or LSA reconstruction between groups ($p > 0.05$). Immediate endoleak was more frequent in the AHF group, approaching the threshold for statistical significance (23.5% vs 12.6%, $p = 0.095$) (**Supplementary Table 5**). Postoperative: The AHF group had significantly

longer mechanical ventilation duration, higher peak systolic blood pressure, and higher peak heart rate than the non-AHF group (all $p < 0.001$). Diuretic use differed significantly between groups ($p < 0.001$), while β-blocker and RAAS inhibitor use rates were similar between groups ($p > 0.05$) (**Supplementary Table 6**).

3.6 Risk Factor Analysis for AHF

In the collinearity assessment, Spearman correlation coefficients among the inflammatory markers NLR, hs-CRP, and D-dimer ranged from 0.52 to 0.68; NLR was retained as the representative variable for this cluster. The correlation coefficient between serum creatinine and BUN was 0.71; serum creatinine was retained. Admission systolic blood pressure and admission heart rate had a correlation coefficient of 0.46, which did not meet the threshold for strong collinearity (both VIF <5), and both were included as candidates. In the imaging parameter cluster, the correlation coefficient between maximum aortic diameter and false lumen/true lumen ratio was 0.53; the false lumen/true lumen ratio, which had the larger univariable effect size, was retained as the candidate. After this grouping and screening, the number of candidate variables actually entered into the multivariable model was reduced from 21 to 14.

Univariable logistic regression showed that age, hypertension, CAD, admission systolic blood pressure, admission heart rate, maximum aortic diameter, false lumen/true lumen ratio, visceral artery involvement, NLR, hemoglobin, serum creatinine, fasting blood glucose, preoperative BNP ≥100 pg/mL, operation time, stent coverage length, intraoperative blood loss, contrast volume, and immediate endoleak were associated with AHF occurrence ($p < 0.10$). Additionally, refractory hypertension had a between-group difference approaching statistical significance ($p = 0.082$) in **Supplementary Table 2** and was therefore included in the univariable analysis; however, its OR = 1.99 (95% CI 0.88–4.50, $p = 0.097$), and it did not enter the final multivariable model. These variables along with dia-

Table 1. Univariable and multivariable logistic regression analysis of risk factors for post-TEVAR AHF.

Variable	Univariable OR (95% CI)	*p*	Multivariable OR (95% CI)	*p*
Age (per 10-year increase)	1.51 (1.16–1.97)	0.003	—	—
Hypertension	5.67 (1.24–25.96)†	0.029	—	—
Diabetes mellitus	2.19 (0.91–5.27)	0.073	—	—
CAD	3.15 (1.24–8.03)	0.016	3.10 (1.05–9.13)	0.030
Refractory hypertension	1.99 (0.88–4.50)	0.097	—	—
Admission systolic BP (per 10 mmHg increase)	1.28 (1.10–1.48)	0.002	—	—
Admission heart rate (per 10 beats/min increase)	1.82 (1.40–2.37)	<0.001	1.72 (1.28–2.31)	<0.001
Maximum aortic diameter (per 1 mm increase)	1.07 (1.02–1.13)	0.009	—	—
Visceral artery involvement	1.87 (0.86–4.08)	0.099	—	—
False lumen/true lumen ratio	1.52 (1.04–2.22)	0.030	—	—
NLR (per 1 unit increase)	1.16 (1.06–1.27)	0.001	—	—
Hemoglobin (per 10 g/L decrease)	1.24 (1.03–1.49)	0.022	—	—
Serum creatinine (per 10 μmol/L increase)	1.08 (1.01–1.16)	0.031	—	—
Fasting blood glucose (per 1 mmol/L increase)	1.28 (1.04–1.57)	0.018	—	—
Preoperative BNP ≥100 pg/mL	11.24 (4.87–25.92)	<0.001	7.84 (3.12–19.68)	<0.001
Operation time (per 10 min increase)	1.14 (1.03–1.26)	0.009	—	—
Stent coverage length (per 10 mm increase)	1.16 (1.07–1.27)	<0.001	1.14 (1.04–1.25)	0.005
Intraoperative blood loss (per 100 mL increase)	1.22 (1.05–1.41)	0.009	—	—
Contrast volume (per 10 mL increase)	1.12 (1.02–1.23)	0.016	—	—
Immediate endoleak	2.15 (0.87–5.32)	0.097	—	—

Note: The multivariable model used the backward stepwise method (likelihood ratio), with 4 independent risk factors ultimately retained. Hosmer-Lemeshow goodness-of-fit test $p = 0.512$, indicating good model fit. "—" indicates the variable was not retained in the final model after backward stepwise selection. †The univariable OR for hypertension was calculated using the Firth penalized likelihood method (as only 2 patients in the AHF group had no hypertension, making standard maximum likelihood estimation unstable); the 95% CI is very wide and results are for reference only; this variable was not entered into the multivariable model. Preoperative BNP ≥100 pg/mL: 28 patients in the AHF group (82.4%) and 48 in the non-AHF group (25.1%) had BNP ≥100 pg/mL. Postoperative variables (mechanical ventilation duration, peak postoperative blood pressure/heart rate, medication use) were excluded from the univariable analysis to avoid reverse causality bias. Maximum aortic diameter was not entered as an independent candidate for the multivariable model due to collinearity with the false lumen/true lumen ratio (Spearman r = 0.53); the false lumen/true lumen ratio was also not retained in the final multivariable model after backward stepwise selection. CAD, coronary artery disease; NLR, neutrophil-to-lymphocyte ratio; BNP, B-type natriuretic peptide; TEVAR, thoracic endovascular aortic repair; AHF, acute heart failure; OR, odds ratio; CI, confidence interval; BP, blood pressure.

Table 2. ROC curve analysis of independent risk factors for predicting post-TEVAR AHF.

Predictor	AUC	95% CI	Optimal cutoff	Sensitivity (%)	Specificity (%)	Youden index
Preoperative BNP	0.842	0.770–0.914	98.5 pg/mL	82.4	74.3	0.567
Admission heart rate	0.724	0.622–0.826	85 beats/min	70.6	68.6	0.392
Stent coverage length	0.668	0.565–0.771	172 mm	64.7	65.4	0.301
Combined prediction model (apparent)	0.897	0.842–0.952	—	85.3	83.2	0.685
Combined prediction model (Bootstrap-corrected)	0.868	0.801–0.935	—	—	—	—

Note: CAD is a binary variable and is not included in cutoff analysis. The combined prediction model is based on the fitted probability from multivariable logistic regression. Bootstrap-corrected AUC was derived by calculating optimism via resampling (B = 1000) and then correcting the apparent AUC. BNP, B-type natriuretic peptide; AUC, area under the curve; ROC, receiver operating characteristic.

betes mellitus (clinically important) were entered into multivariable binary logistic regression (backward stepwise, likelihood ratio); after multicollinearity testing (VIF <5 for all included variables), four independent risk factors for AHF were identified: preoperative BNP ≥100 pg/mL (OR = 7.84, 95% CI 3.12–19.68, $p < 0.001$), CAD (OR = 3.10, 95% CI 1.05–9.13, $p = 0.030$), admission heart rate per 10 beats/min increase (OR = 1.72, 95% CI 1.28–2.31, $p < 0.001$), and stent coverage length per 10 mm increase (OR = 1.14, 95% CI 1.04–1.25, $p = 0.005$) (Table 1).

3.7 Predictive Performance of Independent Risk Factors

ROC curve analysis was performed for each of the four independent risk factors and their combined model. Preoperative BNP (optimal cutoff 98.5 pg/mL) had the highest AUC (0.842), followed by admission heart rate (optimal cutoff 85 beats/min, AUC = 0.724). The combined prediction model constructed from the four independent risk factors achieved an AUC of 0.897, higher than any single indicator (Table 2, Fig.1).

Internal validation results: Bootstrap resampling (B = 1000) showed an apparent AUC of 0.897, optimism of 0.029, and Bootstrap-corrected AUC of 0.868 (95% CI 0.801–0.935). The calibration slope was 0.87 (ideal value 1.0), indicating good model calibration. Ten-fold cross-validation AUC was 0.861, consistent with the Bootstrap results (Fig. 2).

3.8 Comparison of Outcomes Between Groups

The AHF group had significantly higher 30-day all-cause mortality (17.6% vs 2.6%, Fisher's exact test $p = 0.002$), 30-day MACCE rate (35.3% vs 9.4%, $p < 0.001$), and aortic-related adverse event rate (23.5% vs 8.4%, $p = 0.010$) than the non-AHF group. Kaplan-Meier analysis showed a 1-year cumulative survival rate of 73.5% in the AHF group, compared with 93.7% in the non-AHF group (Log-rank $\chi^2 = 18.432$, $p < 0.001$). After adjustment for baseline confounders, Cox proportional hazards regression (Fine-Gray competing risks model) showed that AHF was an independent risk factor for 1-year all-cause mortality (adjusted HR = 4.32, 95% CI 1.86–10.04, $p = 0.001$) (Table 3).

4. Discussion

This study enrolled 225 complex ATBAD patients and systematically analyzed the incidence of AHF within 30 days after TEVAR, its independent risk factors, and its impact on short- and long-term prognosis. All patients in this study had complex ATBAD with confirmed TEVAR indications; malperfusion syndrome and persistent pain were the most common complex features. The AHF incidence observed in this study is broadly consistent with previously reported rates [12,13], suggesting that this complication is not uncommon in clinical practice and warrants sufficient attention.

Regarding risk factors, preoperative BNP was the strongest predictor in this study; elevated preoperative BNP levels indicate pre-existing impairment of cardiac compensatory function, which, when combined with surgical stress and acute afterload increase, can heighten the risk of cardiac decompensation. This is consistent with findings from related studies in cardiac and vascular surgery [14]. Elevated admission heart rate was also an independent risk factor; this finding aligns with the pathophysiological mechanism of excessive sympathetic nervous system activation and increased myocardial oxygen consumption during the acute phase of aortic dissection [15], suggesting that preoperative aggressive heart rate control is of important clinical value in reducing AHF risk [14].

CAD as an independent risk factor may be mechanistically related to pre-existing decline in myocardial reserve function and coronary microvascular dysfunction [15]. In ATBAD patients with CAD, coronary perfusion is already at a critical level; hemodynamic fluctuations during surgery can further aggravate myocardial ischemia, thereby increasing the risk of acute cardiac dysfunction [16]. Stent coverage length was also identified as an independent risk factor; the greater the coverage extent, the more significant the change in aortic wall compliance and the greater the increase in left ventricular afterload [17]. It is noteworthy that the maximum aortic diameter was significantly associated with AHF in univariable analysis, but was not entered as an independent candidate into the multivariable model due to collinearity with the false lumen/true lumen ratio at the grouping stage. This does not negate the potential role of aortic dilation in AHF pathogenesis—increased diameter

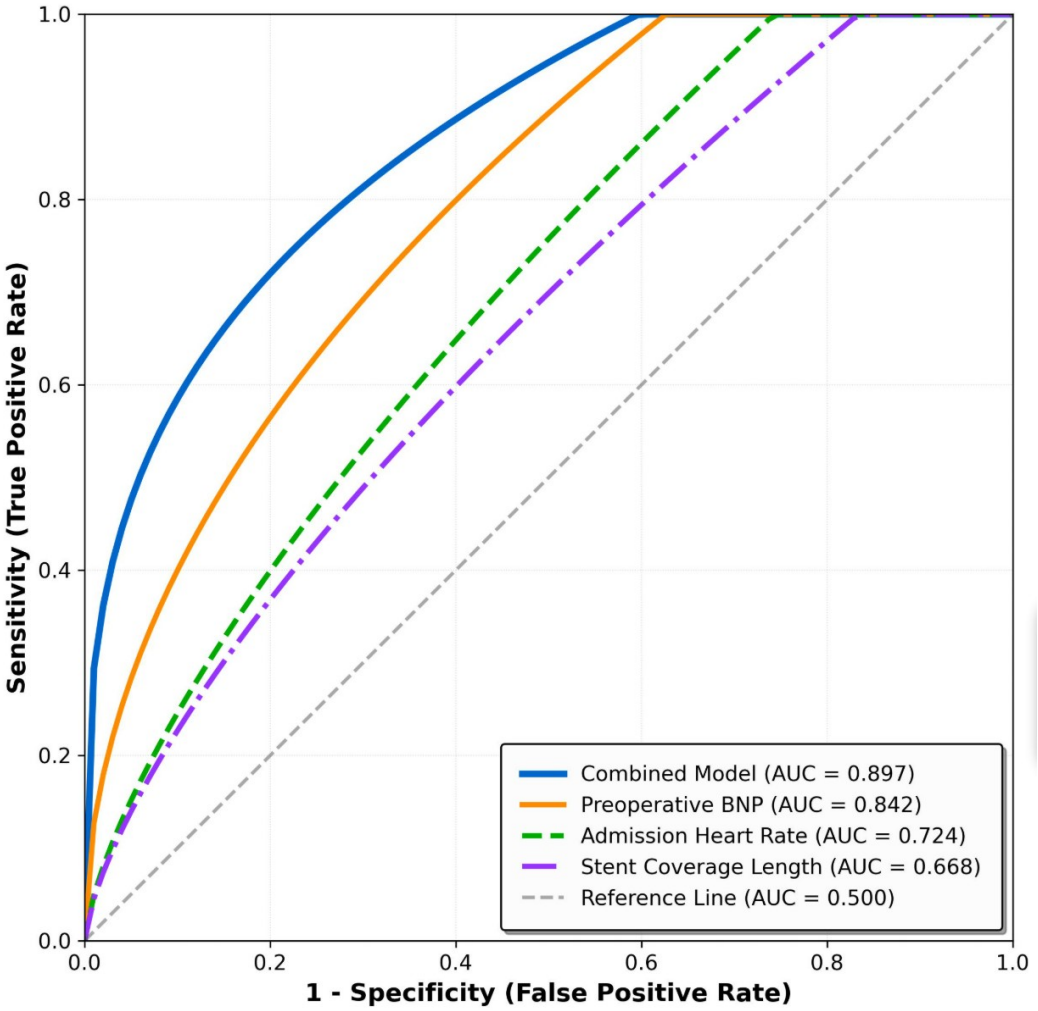

Fig. 1. ROC curves for predictors of post-TEVAR acute heart failure.

is often accompanied by more severe true lumen compression and abnormal cardiac preload [18]; future studies with larger samples can further explore its independent predictive value.

In terms of predictive performance, after Bootstrap internal validation, the discriminative ability of the combined prediction model showed some attenuation but remained generally good overall; the calibration slope indicated good model calibration. Cross-validation results further confirmed the robustness of the model's discriminative ability. These post-validation performance metrics are comparable to previously reported perioperative complication prediction models [19]. However, internal validation can only partially assess model generalizability, and this model still requires independent validation in external cohorts. Preoperative BNP alone demonstrated high discriminative ability; its optimal cutoff is close to the diagnostic threshold recommended by current guidelines [20] and can serve as an objective reference for rapid preoperative risk stratification.

Prognostic analysis showed that perioperative mortality risk was significantly elevated in AHF patients, with a markedly higher 30-day all-cause mortality compared with non-AHF patients; this adverse impact persisted throughout the 1-year follow-up period. After adjustment for baseline confounders, AHF remained an independent risk factor for 1-year all-cause mortality, consistent with the adverse effects of cardiac complications on long-term prognosis reported in studies of aortic disease [21]. AHF not only directly increases perioperative mortality risk but can also create a vicious cycle by prolonging mechanical ventilation duration and aggravating multi-organ dysfunction, further compromising long-term survival [22].

Of note, the perioperative mortality rate in the AHF group falls within the range of in-hospital mortality (10%–25%) for untreated ATBAD. This finding needs to be understood from the following perspectives: first, the 10%–25% mortality cited in the introduction was derived from overall ATBAD populations including all types, whereas this study enrolled only complex ATBAD patients, whose baseline risk is higher than that of the overall population

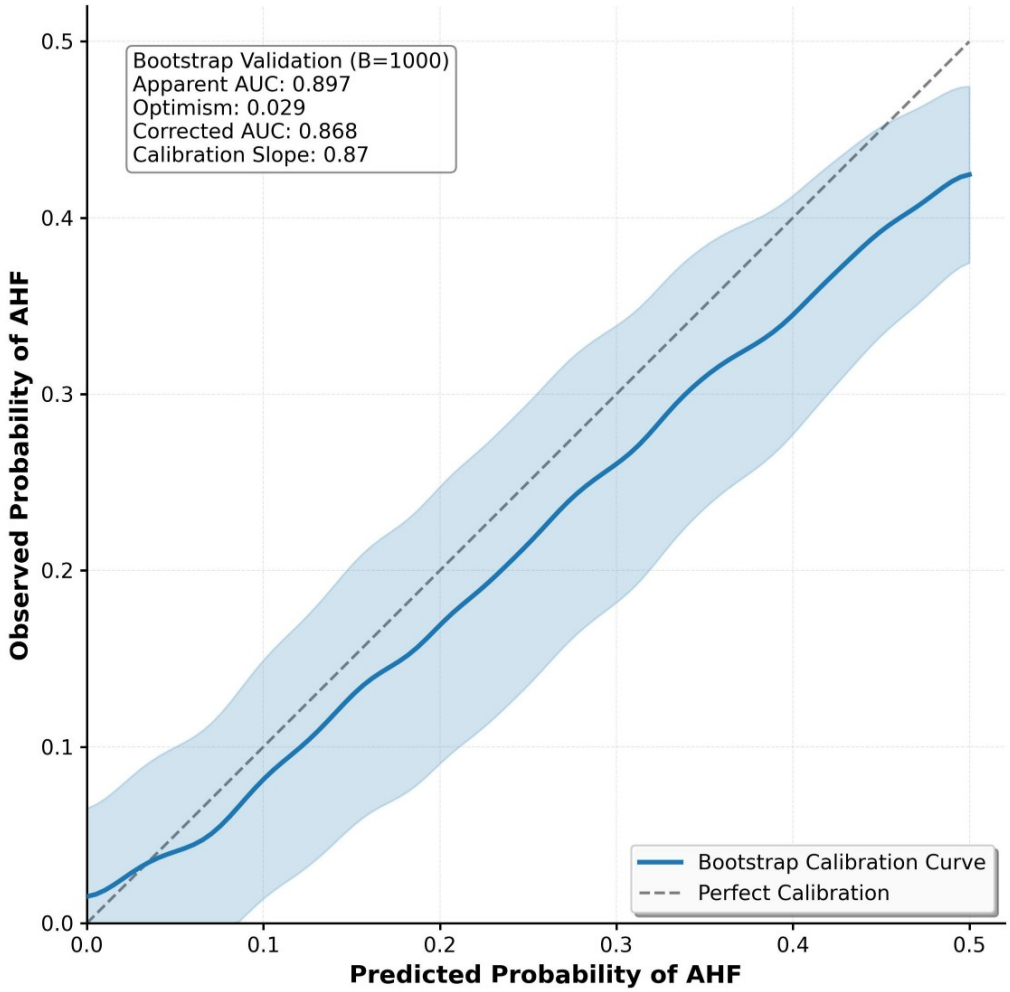

Fig. 2. Bootstrap calibration curve for the combined prediction model (B = 1000).

[23]; second, AHF group patients already had significant impairment of cardiac reserve preoperatively, a higher proportion of concomitant CAD, and elevated admission heart rate, representing a subgroup within complex ATBAD with particularly high cardiac risk [24]; third, although TEVAR effectively seals the entry tear and restores true lumen perfusion, perioperative hemodynamic fluctuations and sudden afterload increase can further burden an already vulnerable heart [25]. Therefore, the findings of this study should not be interpreted as TEVAR failing to reduce mortality in AHF patients; rather, they precisely indicate that AHF is a key marker for identifying the high-mortality subgroup after TEVAR—the low perioperative mortality rate in the non-AHF group fully confirms that TEVAR still provides significant benefit for the majority of complex ATBAD patients.

Clinical implications and perioperative management recommendations: The four independent risk factors identified in this study have direct clinical translational value and can guide perioperative management at the following levels. For preoperative risk stratification, preoperative BNP as the strongest predictor, should be recommended as a routine preoperative screening measure for all ATBAD patients planned for TEVAR. Preoperative BNP combined with admission heart rate can rapidly identify high-risk patients for AHF; for patients exceeding the cutoff values for both, preoperative echocardiography to assess diastolic and systolic function and initiation of multidisciplinary consultation are recommended. For preoperative optimization, patients with concomitant CAD should have preoperative assessment of coronary artery disease severity and myocardial ischemic burden, with optimization of anti-ischemic therapy as needed; for patients with elevated admission heart rate, short-acting β-blockers (e.g., esmolol) can be considered preoperatively to control heart rate to 60–80 beats/min, provided hemodynamic stability is not compromised. For intraoperative strategy, stent coverage length as a modifiable surgical factor suggests that operators should avoid excessively extending the coverage range while ensuring adequate entry tear sealing, to minimize excessive changes in aortic compliance. For postoperative monitoring, high-risk patients identified preoperatively should have extended intensive care, with close dynamic monitoring of BNP levels (recommended at 6 hours, 24 hours, and 72 hours postoper-

Table 3. Comparison of outcome measures between groups.

Outcome	AHF group (n = 34)	Non-AHF group (n = 191)	Test	*p*
Primary endpoint				
30-day all-cause death, n (%)	6 (17.6)	5 (2.6)	Fisher's exact	0.002
Secondary endpoint: 30-day MACCE				
Total MACCE, n (%)†	12 (35.3)	18 (9.4)	Fisher's exact	<0.001
Cardiovascular death, n (%)	4 (11.8)	3 (1.6)	Fisher's exact	0.010
Non-fatal myocardial infarction, n (%)	2 (5.9)	3 (1.6)	Fisher's exact	0.168
Stroke, n (%)	3 (8.8)	7 (3.7)	Fisher's exact	0.179
Repeat revascularization, n (%)	3 (8.8)	5 (2.6)	Fisher's exact	0.090
Secondary endpoint: aortic-related adverse events				
Total aortic-related adverse events, n (%)	8 (23.5)	16 (8.4)	Fisher's exact	0.010
Retrograde type A dissection, n (%)	2 (5.9)	2 (1.0)	Fisher's exact	0.093
Stent-related complications, n (%)	4 (11.8)	10 (5.2)	Fisher's exact	0.126
Reintervention, n (%)	5 (14.7)	7 (3.7)	Fisher's exact	0.009
Long-term outcome				
1-year cumulative all-cause death, n (%)‡	9 (26.5)	12 (6.3)	Fisher's exact	<0.001
1-year cumulative survival rate (%)	73.5	93.7	Log-rank χ^2=18.432	<0.001
Adjusted HR for AHF on 1-year mortality (95% CI)	4.32 (1.86–10.04)	—	Wald χ^2=12.364	0.001

Note: Categorical variables are expressed as n (%) and compared using Fisher's exact test. Adjusted HR was derived from Cox proportional hazards regression (Fine-Gray competing risks model), with adjustment for age, sex, hypertension, CAD, renal function (serum creatinine), and surgical parameters (stent coverage length, intraoperative blood loss). †Total MACCE represents the number of patients experiencing at least one MACCE event (deduplicated); patients experiencing multiple MACCE events are counted only once. ‡1-year cumulative all-cause deaths refer to cumulative all-cause deaths within 1 year postoperatively, including deaths within 30 days (AHF group: 6 cases; non-AHF group: 5 cases). MACCE, major adverse cardiac and cerebrovascular events; CAD, coronary artery disease; HR, hazard ratio.

atively) to enable early detection of AHF signs and timely initiation of treatment.

The following limitations should be acknowledged: First, this study is a single-center study with a relatively limited sample size, enrolling only complex ATBAD patients; conclusions should not be directly extrapolated to uncomplicated ATBAD populations. Second, there were individual differences in the timing of imaging follow-up among some patients, which may have introduced some bias in the identification of aortic-related adverse events. Third, echocardiographic diastolic function grading data were not systematically and comprehensively collected, limiting the in-depth analysis of AHF subtypes. Fourth, the EPV of the final model is 8.5 (34/4); although this satisfies the EPV ≥8 modeling requirement by controlling to 4 variables, it remains below the conventionally recommended EPV ≥10 threshold. Although Bootstrap internal validation showed acceptable post-correction discriminative ability, external cohort validation is still needed.

5. Conclusion

Early AHF after TEVAR is not uncommon in complex ATBAD patients. Elevated preoperative BNP (≥100 pg/mL), concomitant CAD, elevated admission heart rate, and longer stent coverage length are independent risk factors for AHF. The combined prediction model constructed from these factors still demonstrates good discriminative ability after internal validation, providing an objective basis for preoperative screening of high-risk patients. AHF significantly increases perioperative and 1-year all-cause mortality risk; clinicians should emphasize preoperative cardiac function assessment and BNP monitoring, and develop individualized perioperative management strategies for high-risk patients to improve overall prognosis.

Availability of Data and Materials

The datasets generated or analyzed during the current study are not publicly available due to patient privacy protection requirements in accordance with the institutional ethics protocol, but are available from the corresponding author on reasonable request.

Author Contributions

JFZ and LY conceived and designed the study. JFZ was responsible for project administration, patient enrollment, and overall supervision. RZZ and ZYZ performed data collection, imaging and laboratory parameter recording, and perioperative data compilation. DX contributed to patient follow-up coordination and outcome data verification. JFZ and RZZ jointly conducted the statistical analysis and interpreted the results. JFZ drafted the manuscript. LY, ZYZ, and DX critically revised the manuscript for important intellectual content. All authors contributed to editorial revisions of the manuscript, read and approved the final

version, and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Shanghai Ninth People's Hospital (Approval No.: 2021-KY-00067; Approval Date: January 8, 2021). The study was conducted in strict accordance with the principles of the Declaration of Helsinki. All enrolled patients or their legally authorized representatives provided written informed consent prior to participation.

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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/HSF51380.

References

[1] Isselbacher EM, Preventza O, Hamilton Black J, Augoustides JG, Beck AW, Bolen MA, et al. 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. Circulation. 2022; 146: e334–e482. https://doi.org/10.1161/CIR.0000000000001106

[2] Krebs JR, Filiberto AC, Fazzone B, Jacobs CR, Anderson EM, Shahid Z, et al. Outcomes of Patients with Acute Type B Aortic Dissection and High-Risk Features. Annals of Vascular Surgery. 2024; 106: 99–107. https://doi.org/10.1016/j.avsg.2024.01.012

[3] Spinelli D, Weaver FA, Azizzadeh A, Magee GA, Piffaretti G, Benedetto F, et al. Endovascular treatment of complicated versus uncomplicated acute type B aortic dissection. The Journal of Thoracic and Cardiovascular Surgery. 2023; 165: 4–13.e1. https://doi.org/10.1016/j.jtcvs.2021.01.027

[4] Mazzolai L, Teixido-Tura G, Lanzi S, Boc V, Bossone E, Brodmann M, et al. 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases. European Heart Journal. 2024; 45: 3538–3700. https://doi.org/10.1093/eurheartj/ehae179

[5] Harris KM, Nienaber CA, Peterson MD, Woznicki EM, Braverman AC, Trimarchi S, et al. Early Mortality in Type A Acute Aortic Dissection: Insights From the International Registry of Acute Aortic Dissection. JAMA Cardiology. 2022; 7: 1009–1015. https://doi.org/10.1001/jamacardio.2022.2718

[6] Stonko DP, Edwards J, Abdou H, Treffalls RN, Walker P, DeMartino RR, et al. Thoracic Endovascular Aortic RepairAcutely Augments Left Ventricular Biomechanics in An Animal Model: A Mechanism for Postoperative Heart Failure and Hypertension. Annals of Vascular Surgery. 2023; 97: 18–26. https://doi.org/10.1016/j.avsg.2023.04.007

[7] Kayali F, Jubouri M, Al-Tawil M, Tan SZCP, Williams IM, Mohammed I, et al. Coronary artery involvement in type A aortic dissection: Fate of the coronaries. Journal of Cardiac Surgery. 2022; 37: 5233–5242. https://doi.org/10.1111/jocs.16981

[8] Pedersen MW, Kragholm K, Oksjoki R, Møller JE, Gundlund A, Fosbøl E, et al. Characteristics and Outcomes in Patients With Acute Aortic Dissection: A Nationwide Registry Study. The Annals of Thoracic Surgery. 2023; 116: 1177–1184. https://doi.org/10.1016/j.athoracsur.2023.06.019

[9] Herajärvi J, Mustonen C, Kesävuori R, Raivio P, Biancari F, Jormalainen M, et al. Impact of high-risk features on outcome of acute type B aortic dissection. JTCVS Open. 2023; 13: 20–31. https://doi.org/10.1016/j.xjon.2023.01.005

[10] Cha E, Eidt JF, Vasquez J. Variability in the Treatment of High-Risk Type B Aortic Dissection at a Single Center. The American Journal of Cardiology. 2024; 230: 58–61. https://doi.org/10.1016/j.amjcard.2024.08.025

[11] MacGillivray TE, Gleason TG, Patel HJ, Aldea GS, Bavaria JE, Beaver TM, et al. The Society of Thoracic Surgeons/American Association for Thoracic Surgery clinical practice guidelines on the management of type B aortic dissection. The Journal of Thoracic and Cardiovascular Surgery. 2022; 163: 1231–1249. https://doi.org/10.1016/j.jtcvs.2021.11.091

[12] Ahmad W, Brunkwall J, Bunck AC, Dorweiler B, Mylonas S. Favorable Remodeling After TEVAR in Uncomplicated Acute and Subacute Type B Aortic Dissection in Comparison to Conservative Treatment: A Midterm Analysis. Journal of Endovascular Therapy : an Official Journal of the International Society of Endovascular Specialists. 2024; 31: 964–974. https://doi.org/10.1177/15266028231158971

[13] Patrick WL, Yarlagadda S, Bavaria JE, Kelly JJ, Kalva S, Grimm JC, et al. The Penn Classification System for Malperfusion in Acute Type A Dissection: A 25-Year Experience. The Annals of Thoracic Surgery. 2023; 115: 1109–1117. https://doi.org/10.1016/j.athoracsur.2022.10.028

[14] Comanici M, Nadarajah D, Katumalla E, Cyclewala S, Raja SG. Use of Preoperative Natriuretic Peptide in Predicting Mortality After Coronary Artery Bypass Grafting: A Systematic Review and Meta-analysis. Journal of Cardiothoracic and Vascular Anesthesia. 2023; 37: 1785–1792. https://doi.org/10.1053/j.jvca.2023.04.020

[15] Chen Z, Pan W, Cao J, Dai X, Lin W, Chen H, et al. Admission Heart Rate and Mortality in Critically Ill Patients with Acute Aortic Dissection. International Heart Journal. 2023; 64: 44–52. https://doi.org/10.1536/ihj.22-346

[16] Wang M, Fan R, Gu T, Zou C, Zhang Z, Liu Z, et al. Short-term outcomes of acute coronary involvement in type A aortic dissection without myocardial ischemia: a multiple center retrospective cohort study. Journal of Cardiothoracic Surgery. 2021; 16: 107. https://doi.org/10.1186/s13019-021-01469-z

[17] Mandigers TJ, Ramella A, Bissacco D, Domanin M, van Herwaarden JA, Heijmen R, et al. Thoracic Stent Graft Numerical Models To Virtually Simulate Thoracic Endovascular Aortic Repair: A Scoping Review. European Journal of Vascular

and Endovascular Surgery : the Official Journal of the European Society for Vascular Surgery. 2023; 66: 784–796. https://doi.org/10.1016/j.ejvs.2023.06.006

[18] Karaolanis GI, Antonopoulos CN, Charbonneau P, Georgakarakos E, Moris D, Scali S, et al. A systematic review and meta-analysis of stroke rates in patients undergoing thoracic endovascular aortic repair for descending thoracic aortic aneurysm and type B dissection. Journal of Vascular Surgery. 2022; 76: 292–301.e3. https://doi.org/10.1016/j.jvs.2022.02.031

[19] Wu Z, Li Y, Xu Z, Liu H, Liu K, Qiu P, et al. Prediction of preoperative in-hospital mortality rate in patients with acute aortic dissection by machine learning: a two-centre, retrospective cohort study. BMJ Open. 2023; 13: e066782. https://doi.org/10.1136/bmjopen-2022-066782

[20] McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. 2023 Focused Update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. European Heart Journal. 2023; 44: 3627–3639. https://doi.org/10.1093/eurheartj/ehad195

[21] Olsson KW, Mani K, Burdess A, Patterson S, Scali ST, Kölbel T, et al. Outcomes After Endovascular Aortic Intervention in Patients With Connective Tissue Disease. JAMA Surgery. 2023; 158: 832–839. https://doi.org/10.1001/jamasurg.2023.2128

[22] Kuzniar M, Wanhainen A, Tegler G, Hansen T, Mani K. Longitudinal Assessment of Inflammatory Activity in Acute Type B Aortic Dissection with Integrated Fluorodeoxyglucose Positron Emission Tomography/Magnetic Resonance Imaging. European Journal of Vascular and Endovascular Surgery : the Official Journal of the European Society for Vascular Surgery. 2023; 66: 323–331. https://doi.org/10.1016/j.ejvs.2023.05.039

[23] Desai ND, Wang GJ, Brinkman W, Coselli J, Taylor B, Patel H, et al. Outcomes of a Novel Single-Branched Aortic Stent Graft for Treatment of Type B Aortic Dissection. The Annals of Thoracic Surgery. 2025; 119: 826–834. https://doi.org/10.1016/j.athoracsur.2024.07.053

[24] Wang GQ, Qin YF, Shi ST, Zhang KW, Zhai ST, Li TX. Retrograde type A aortic dissection during or after thoracic endovascular aortic repair: a single center 16-year experience. Frontiers in Cardiovascular Medicine. 2023; 10: 1160142. https://doi.org/10.3389/fcvm.2023.1160142

[25] Schizas N, Nazou G, Samiotis I, Antonopoulos CN, Angouras DC. Is TEVAR an Effective Approach to Prevent Complications after Surgery for Aortic Dissection Type A? A Systematic Review. Healthcare (Basel, Switzerland). 2024; 12: 1263. https://doi.org/10.3390/healthcare12131263