The Heart Surgery Forum

Heart Surg. Forum **2026**; 29(3): 48130
https://doi.org/10.31083/HSF48130

Article

Analysis of the Incidence and Risk Factors for Postoperative Bloodstream Infection After Stanford Type A Aortic Dissection and Development of a Predictive Model

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Academic Editor: Castigliano M. Bhamidipati
Submitted: 10 November 2025 Revised: 9 January 2026 Accepted: 26 January 2026 Published: 24 March 2026

Abstract

Background: This study aimed to investigate the incidence and risk factors of postoperative bloodstream infections (BSIs) in patients with Stanford type A aortic dissection (SAAD) and to develop a reliable predictive model to provide a more comprehensive understanding of the characteristics of this complication. **Methods**: Clinical data from 257 patients who underwent surgical repair for SAAD at the Shandong Provincial Hospital Affiliated to Shandong First Medical University between January 2017 and July 2023 were retrospectively analyzed. Risk factors for postoperative BSIs were identified using univariate and multivariate logistic regression. A predictive model was constructed and validated based on the receiver operating characteristic (ROC) curve. **Results**: Based on a comprehensive analysis of 257 patients who underwent surgical repair for type A aortic dissection, this study identified an incidence of postoperative BSIs of 10.5%. Patients with BSIs experienced significantly worse outcomes, including prolonged intensive care unit (ICU) and overall hospital stays, and a higher incidence of complications such as liver failure, acute kidney injury, and cerebral infarction. In addition, postoperative BSI was associated with an increase in in-hospital mortality. Blood culture analysis revealed Gram-negative bacilli as the primary pathogens, with *Acinetobacter baumannii* and *Enterobacter cloacae* being the most prevalent, collectively accounting for 22.22% of all BSI cases. Multivariable analysis identified the following independent risk factors for postoperative BSI: preoperative C-reactive protein (odds ratios (OR) = 1.010, 95% confidence interval (CI) 1.002–1.019, p = 0.020), tracheostomy (OR = 9.186, 95% CI 2.463–34.266, p = 0.001), infectious pneumonia (OR = 32.872, 95% CI 4.186–258.174, p = 0.001), circulatory arrest time (OR = 1.048, 95% CI 1.004–1.093, p = 0.033), and age (OR = 1.055, 95% CI 1.010–1.103, p = 0.016). A predictive model constructed from these factors demonstrated strong discriminatory power, with an area under the ROC curve of 0.897. The model exhibited a sensitivity of 85.0% and a specificity of 90.0%, indicating good predictive accuracy within the study cohort. **Conclusion**: Postoperative bloodstream infection is a significant complication after surgical repair of Stanford type A aortic dissection, and is associated with worse clinical outcomes. A predictive model incorporating the independent risk factors of advanced age, elevated preoperative C-reactive protein, prolonged circulatory arrest time, tracheostomy, and infectious pneumonia aids in the early identification of high-risk patients. Future large-scale, multi-center studies are warranted to further validate and future refine these findings.

Keywords: Stanford type A aortic dissection; bloodstream infection; risk factors; predictive model

1. Introduction

Aortic dissection is a critical condition, with Stanford type A aortic dissection (SAAD) representing 60–70% of cases and carrying a particularly high mortality risk. International data show that without surgery, mortality exceeds 50% within 48 hours and reaches 70% within two weeks of onset [1,2]. Although surgical advances have substantially reduced postoperative mortality, early complications remain a major cause of in-hospital death [3,4]. Among these, bloodstream infection (BSI) is a serious postoperative complication characterized by an insidious onset and a diagnosis largely dependent on time-consuming blood cultures, which complicates early detection [5]. Without timely intervention, BSI may progress to shock and multi-organ failure, resulting in increased morbidity and mortality.

Given these challenges, identifying risk factors for BSI after SAAD surgery is clinically important for guiding treatment and improving outcomes. This study systematically examined the incidence and risk factors for BSI fol-

lowing SAAD surgery and developed a predictive model to identify high-risk patients. We analyzed clinical data from 257 SAAD patients who underwent conventional open surgery at the Shandong Provincial Hospital Affiliated to Shandong First Medical University between January 2017 and July 2023. Based on comprehensive statistical analyses, a predictive model was established to assess the individual risk of infection and to facilitate targeted preventive strategies. The findings provide valuable scientific insights for postoperative management and prevention of complications in SAAD patients.

2. Materials and Methods

2.1 Study Subjects

This study retrospectively identified patients with SAAD who underwent conventional open-heart surgery with cardiopulmonary bypass between January 2017 and July 2023 through the electronic medical record system of Shandong Provincial Hospital. SAAD diagnosis was based on the 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease [6]. All data—including medical history, surgical, anesthetic, and bypass records, nursing charts, imaging, and laboratory results—were derived from original sources to ensure authenticity and traceability. The requirement for informed consent was waived due to the retrospective nature of the study, which was conducted in compliance with Good Clinical Practice (GCP) and the Declaration of Helsinki.

2.2 Inclusion and Exclusion Criteria

2.2.1 Inclusion Criteria

(1) Patients with a definitive diagnosis of Stanford Type A aortic dissection, confirmed by preoperative imaging (echocardiography or CTA) and subsequent intraoperative findings.

(2) Patients who were assessed as surgical candidates by cardiovascular surgeons and subsequently underwent open aortic surgery with cardiopulmonary bypass.

(3) Patients with complete demographic and essential perioperative clinical data.

2.2.2 Exclusion Criteria

(1) Patients scheduled for aortic surgery who did not undergo cardiopulmonary bypass, including those who did not receive surgery or underwent only endovascular aortic repair.

(2) Those diagnosed with Stanford type B aortic dissection.

(3) Patients who died before surgery, during surgery, or within 72 hours postoperatively.

(4) Cases with missing essential research data.

2.2.3 Chart of Patient Selection Criteria

The following is a flow chart of patient selection criteria (Fig. 1).

2.3 Diagnostic Criteria for Bloodstream Infection

2.3.1 Bloodstream Infection (BSI)

The diagnosis of bloodstream infection was established in accordance with the Diagnostic Criteria for Hospital Infections (Trial) issued by the Ministry of Health of China in 2001, and aligns with relevant guidelines from international societies such as the Infectious Diseases Society of America (IDSA) [7]. Patients were required to meet one of the following sets of criteria [8]:

(1) Positive blood culture for a recognized pathogen, or repeated positivity for common skin commensals, with clinical symptoms not attributable to other infections.

(2) Positive blood culture with an identified portal of entry or metastatic infectious focus.

(3) Clinical signs of systemic infection (e.g., fever >38 °C or <36 °C, chills, hypotension) without other explainable foci.

2.3.2 Acute Renal Insufficiency

Acute renal insufficiency is defined as a postoperative serum creatinine level increased by more than 50% from the preoperative baseline level or an absolute increase exceeding 26.5 µmol/L [9].

2.3.3 Neurological Complications

Following surgery for aortic dissection, patients were closely monitored for level of consciousness and promptly assessed for focal neurological signs. The occurrence of neurological complications was comprehensively evaluated by integrating various imaging modalities—including transcranial Doppler, cranial CT, MRI, and electromyography—with consultations from neurology and neurosurgery specialists [10].

2.3.4 Liver Failure

Liver failure is defined as serum total bilirubin concentration exceeding 205 µmol/L, or the concurrent presence of bilirubin-enzyme dissociation [11].

2.3.5 Pulmonary Infection

Postoperative pulmonary infection was defined by the presence of cough with purulent sputum and moist rales on auscultation, plus at least one of the following criteria: fever; elevated white blood cell count or neutrophil percentage; inflammatory infiltrates on chest radiography; with confirmation requiring the identification of the same pathogen in two separate sputum cultures [12].

2.4 Data Collection for the Study Population

Clinical data for all enrolled patients were systematically collected from the electronic medical record system of

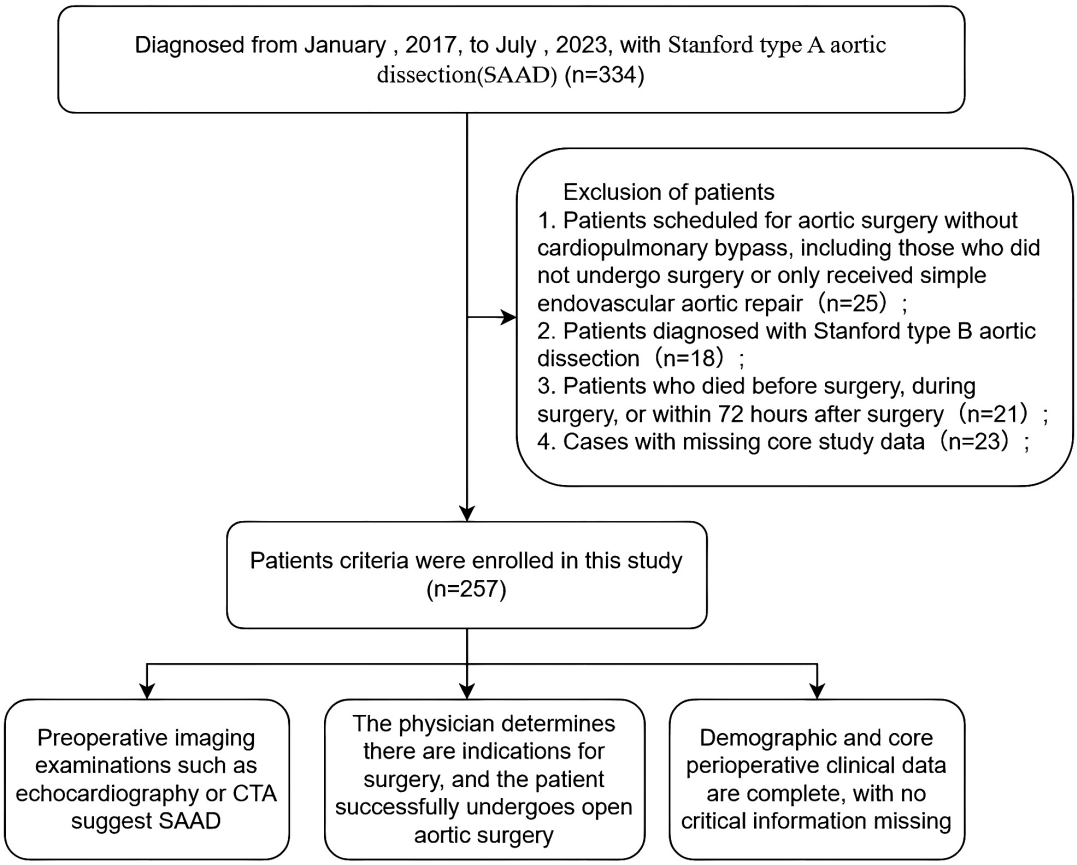

Fig. 1. Flowchart of study patient selection and decision-making.

Shandong Provincial Hospital Affiliated to Shandong First Medical University The collected variables encompassed baseline characteristics, preoperative status, intraoperative details, and postoperative course.

Baseline information included patient demographics (age, sex), health habits (smoking history, alcohol use), and relevant medical history (hypertension, diabetes, atherosclerosis, aortic aneurysm, aortic valve regurgitation, prior cardiac surgery, Marfan syndrome). Presenting symptoms such as chest pain, back pain, abdominal pain, or syncope were also documented.

Preoperative assessment included left ventricular ejection fraction (LVEF), preoperative malperfusion (including intestinal ischemia, acute kidney injury, coronary artery ischemia, and lower extremity ischemia), left ventricular hypertrophy, heart failure, pleural or pericardial effusion, and a panel of laboratory tests including hemoglobin, white blood cell count, neutrophil, lymphocyte, monocyte, and platelet counts, International Normalized Ratio (INR), C-reactive protein (CRP), serum creatinine, liver function markers (aspartate aminotransferase, alanine aminotransferase, albumin, prealbumin, albumin-to-globulin ratio, bilirubin fractions, alkaline phosphatase, gamma-glutamyl transferase), and cardiac injury biomarkers (high-sensitivity troponin T, myoglobin, creatine kinase-MB mass, D-dimer).

Intraoperative variables recorded were the emergency status of the surgery, key procedural times (total operative time, cardiopulmonary bypass time, aortic cross-clamp time, and circulatory arrest time), and the types and quantities of blood products transfused, including red blood cells, plasma, platelets, and cryoprecipitate.

Postoperative data collection focused on serial laboratory values (hemoglobin, platelet count, white blood cell and differential counts, CRP), the occurrence of specific complications (acute renal insufficiency, cerebral infarction, liver failure, pulmonary infection, pleural effusion requiring thoracentesis), and details of postoperative support. This included durations of various catheter placements (arterial, central venous, pericardial/mediastinal drainage, nasogastric), tracheal intubation and tracheostomy events, need for continuous renal replacement therapy (CRRT), re-operation details (exploratory thoracotomy, wound debridement, secondary cardiopulmonary bypass), transfusion volumes post-surgery, antibiotic administration (classes and duration), maximum body temperature, and ICU length of stay.

2.5 Statistical Analysis

Data analysis was performed using R software (version 4.5.1, Vienna, Vienna, Austria) and R Studio (version 2025.05.1+513, Boston, Massachusetts, United States),

Table 1. Basic characteristics of the included subjects.

	Whole cohort	Positive group	Negative group	*p*-value
	N = 257	N = 27	N = 230	
Aged ≥70 years	14 (5.45)	5 (18.52)	9 (3.91)	0.001
Aged <70 years	243 (94.55)	23 (85.19)	220 (95.65)	
Male	172 (66.93)	22 (81.48)	150 (65.22)	0.138
SBP (mmHg)	137.02 ± 24.34	149.89 ± 31.26	135.51 ± 23.01	0.012
DBP (mmHg)	76.18 ± 15.31	79.04 ± 16.99	75.85 ± 15.11	0.368
Smoking history	98 (38.12)	9 (33.33)	89 (38.70)	0.739
History of alcohol use	82 (31.90)	14 (51.85)	68 (29.56)	0.033
Hypertension	188 (73.15)	22 (81.48)	166 (72.17)	0.422
Diabetes mellitus	14 (5.45)	0 (0)	14 (6.09)	0.384
History of cardiac surgery	11 (4.28)	0 (0)	11 (4.78)	0.510
Marfan syndrome	13 (5.06)	0 (0)	13 (5.65)	0.422
Aortic aneurysm	56 (21.79)	3 (11.11)	53 (23.04)	0.240
Chest pain	197 (76.65)	24 (88.89)	173 (75.22)	0.178
Back pain	120 (46.69)	19 (70.37)	101 (43.91)	0.016
Abdominal pain	82 (31.90)	6 (22.22)	76 (33.04)	0.356
Syncope	14 (5.44)	0 (0)	14 (6.08)	0.384
Preoperative malperfusion state	41 (15.95)	10 (37.04)	31 (13.48)	0.004
Intestinal ischemia	12 (4.67)	4 (14.81)	8 (3.48)	0.080
Acute kidney injury	23 (8.95)	3 (11.11)	20 (8.70)	0.270
Coronary artery ischemia	15 (5.84)	4 (14.81)	11 (4.78)	0.310
Lower extremity ischemia	31 (12.06)	5 (18.52)	26 (11.30)	0.067

Footnotes:

The comparative analysis of baseline characteristics of the two groups of patients.

SBP, systolic blood pressure; DBP, diastolic blood pressure.

with a two-sided p-value < 0.05 considered statistically significant. After data import, descriptive statistics were applied according to variable types. Continuous variables following a normal distribution were expressed as Mean ± Standard Deviation and compared using the independent samples t-test; otherwise, they were reported as median with interquartile range (Q2 (Q1–Q3)) and compared with the Mann-Whitney U test. Normality was assessed using the Kolmogorov-Smirnov test. Categorical variables were summarized as frequencies and percentages and analyzed using the chi-square test.

To identify risk factors and develop a prediction model for postoperative BSI, patients were categorized into BSI-positive and BSI-negative groups. Binary logistic regression with Forward Likelihood Ratio (Forward LR) stepwise selection was used to identify independent risk factors. Model performance was evaluated using the Wald test, the Hosmer-Lemeshow goodness-of-fit test, and receiver operating characteristic (ROC) curve analysis, with results reported as the area under the curve (AUC). Effect estimates are presented as odds ratios (ORs) with 95% confidence intervals (CIs).

To internally validate the prediction model and estimate its optimism, we performed bootstrap resampling with 1000 repetitions. The optimism-corrected performance metrics, including the area under the ROC curve, were calculated.

All statistical test results in this article have been cross-validated by Stata software.

3. Results

Of the 257 patients included in the final analysis, 27 (10.5%) developed postoperative bloodstream infection (BSI). A comprehensive analysis was performed to characterize postoperative BSI, evaluating baseline admission characteristics, preoperative status, and intraoperative and postoperative indicators. Key findings are summarized below.

3.1 Admission and Baseline Characteristics

The cohort was predominantly male (172 patients, 66.9%), with a mean age of 50.81 ± 11.35 years. Mean admission systolic and diastolic blood pressures were 137.02 ± 24.34 mmHg and 76.18 ± 15.31 mmHg, respectively. A smoking history was reported in 38.1% of patients, and an alcohol consumption history in 73.1%. Comorbidities included hypertension (68.5%), diabetes mellitus (5.4%), prior cardiac surgery (4.2%), Marfan syndrome (5.0%), atherosclerosis (35.7%), and aortic aneurysm (21.7%).

Table 2. Preoperative profile of the included subjects.

	Whole cohort	Positive group	Negative group	*p*-value
	N = 257	N = 27	N = 230	
WBC (10^9/L)	10.54 ± 3.85	12.85 ± 5.27	10.27 ± 3.57	0.019
RBC (10^{12}/L)	4.19 ± 0.60	4.15 ± 0.74	4.19 ± 0.59	0.763
Hb (g/L)	128.34 ± 19.18	128.89 ± 22.51	128.28 ± 18.81	0.893
Plt (10^9/L)	191.49 ± 74.36	186.85 ± 61.51	192.03 ± 75.82	0.689
LYM (10^9/L)	1.22 ± 0.60	1.31 ± 0.67	1.21 ± 0.59	0.490
MONO (10^9/L)	0.73 ± 0.34	0.95 ± 0.42	0.70 ± 0.33	0.007
NE (10^9/L)	8.42 ± 3.51	10.58 ± 4.73	7.485 ± 4.775	0.015
CRP (mg/L)	45.35 ± 51.95	64.53 ± 69.83	43.09 ± 49.14	0.032
AST (U/L)	24.0 (18.0–49.0)	27.0 (22.5–53.0)	23.5 (22.5–53.0)	0.160
ALT (U/L)	22.0 (13.0–42.0)	26.0 (17.0–47.5)	21.0 (13.0–40.0)	0.130
GGT (U/L)	28.0 (17.0–51.0)	37.0 (17.0–74.5)	28.0 (17.0–48.0)	0.314
ALP (U/L)	71.0 (59.0–91.0)	73.0 (56.5–83.0)	71.0 (59.0–91.0)	0.930
Prealbumin	198.16 ± 70.07	220.84 ± 66.22	195.50 ± 70.16	0.070
Albumin	3.67 ± 4.54	37.07 ± 5.08	36.62 ± 4.48	0.665
A/G Ratio	1.45 ± 0.288	1.51 ± 0.306	1.44 ± 0.286	0.315
INR	1.13 (1.07–1.22)	1.11 (1.06–1.165)	1.13 (1.07–1.22)	0.198
Creatinine	82.4 (66.71–111.5)	107.1 (86.5–137.14)	80.15(66.05–107.83)	0.004
hs-cTnl	20.94 (9.83–140.9)	29.46 (10.93–194.65)	20.74 (9.54–137.35)	0.366
CK-MB	2.26 (1.12–7.2)	3.21 (1.37–19.42)	2.15 (1.11–6.61)	0.106
Myoglobin	51.21 (24.1–195.0)	124.0 (36.94–1128.0)	47.04 (23.57–141.88)	0.009
T-Bil	5.7 (3.3–10.8)	7.15 (4.27–14.11)	5.52 (3.20–10.63)	0.108
Direct bilirubin	14.6 (9.08–21.5)	15.1 (9.57–22.72)	14.57 (9.11–21.42)	0.761
Indirect bilirubin	20.1 (13.33–29.72)	20.88 (13.79–29.13)	20.1 (13.33–29.85)	0.924

Footnote:

Based on the preoperative characteristic data in Table 2, the laboratory test results of the positive group (N = 27) and the negative group (N = 230) were compared and analyzed.

WBC, White blood cell; RBC, Red blood cell; Hb, Hemoglobin; Plt, Platelet; LYM, Lymphocytic lymphoid cell; MONO, Monocytes; NE, Neutrophil granulocyte; CRP, C-reactive protein; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; GGT, Gamma-glutamyl transferase; ALP, Alkaline Phosphatase; A/G Ratio, Albumin-to-Globulin Ratio; INR, International Normalized Ratio; hs-cTnl, High-sensitivity cardiactroponin T; CK-MB, creatine kinase-myocardial band; T-Bil, Total bilirubin.

Comparative analysis revealed that patients who developed bloodstream infection were more likely to have higher preoperative systolic blood pressure ($p = 0.012$), a history of alcohol consumption ($p = 0.033$), and an age of 70 years or older ($p = 0.001$). Back pain was significantly more common in this group (70.37% vs. 43.91%, $p < 0.05$). No significant differences were observed in other presenting symptoms such as syncope, chest pain, or abdominal pain (Table 1).

3.2 Preoperative Profile

Laboratory findings indicated a proinflammatory and prothrombotic state in the BSI group, with significantly elevated levels of C-reactive protein, white blood cell counts, absolute monocyte counts, absolute neutrophil counts, and D-dimer (all $p < 0.05$). Increased preoperative creatinine and myoglobin levels were also observed (all $p < 0.05$), suggesting potential renal dysfunction and myocardial injury (Table 2).

3.3 Intraoperative Factors

In this study, several cardiac surgical procedures were identified and defined. They are: S stands for Sun's procedure; B is for Bentall procedure; C is for Cabrol procedure; D is for David procedure; W is for Wheat surgery; E is for total arch or half arch prosthesis replacement of the ascending aorta; F is for ascending aorta prosthesis replacement; G is for aortoplasty. In addition, we also defined hybrid procedures, such as B+S, representing Bentall + Sun's procedure. "Emergency surgery" is defined in this study as follows: after a patient is diagnosed with Stanford type A (or DeBakey type I, II) aortic dissection via imaging examination, the cardiovascular surgery team evaluates the case as the highest priority with an immediate life-threatening risk, which requires scheduling and performing the surgery within a few hours after admission.

When exploring the relationship between each surgical procedure and bloodstream infection, we found that the association between surgical procedure S and bloodstream

Table 3. Intraoperative factors of the included subjects.

	Whole cohort	Positive group	Negative group	*p*-value
	N = 257	N = 27	N = 230	
Emergency surgery	149 (57.98)	19 (70.37)	130 (56.52)	0.241
Aortic cross-clamp time (min)	113.98 ± 34.08	117.19 ± 31.86	113.60 ± 34.38	0.606
CPB time (min)	204.21 ± 52.76	217.70 ± 71.25	202.63 ± 50.11	0.160
Circulatory arrest time (min)	18.04 ± 10.51	23.78 ± 11.40	13.37 ± 10.2	0.004
Total operation time (h)	6.5 (6.0–7.5)	7.5 (6.8–9.0)	6.5 (5.5–7.5)	0.002
Intraoperative valve implantation	51 (19.84)	3 (11.11)	48 (20.87)	0.343
Concomitant CABG	30 (11.67)	3 (11.11)	27 (11.74)	1.000
Surgical methods				
Surgical methods S	186 (72.66)	25 (96.15)	161 (70.00)	0.009
Surgical methods B	11 (4.2)	0 (0)	11 (4.30)	0.529
Surgical methods C	7 (2.73)	0 (0)	7 (3.04)	0.789
Surgical methods D	3 (1.17)	0 (0)	3 (1.30)	1.000
Surgical methods W	1 (0.39)	0 (0)	1 (0.43)	1.000
Surgical methods E	11 (4.30)	0 (0)	11 (4.78)	0.529
Surgical methods F	4 (1.56)	0 (0)	4 (1.74)	1.000
Surgical methods G	1 (0.39)	0 (0)	1 (0.43)	1.000
Surgical methods B+S	20 (7.81)	0 (0)	20 (8.70)	0.238
Surgical methods C+S	9 (3.12)	2 (3.85)	7 (3.04)	1.000
Surgical methods D+S	4 (1.56)	0 (0)	4 (1.74)	1.000

Footnote:

Based on the intraoperative factor data in Table 3, a comparative analysis was conducted on the intraoperative conditions of the positive group (N = 27) and the negative group (N = 230).

CPB time, Cardiopulmonary Bypass time; Concomitant CABG, Concomitant coronary artery bypass surgery.

infection was statistically significant (*p*-value was 0.009), which suggested that there was a certain correlation between Sun's procedure and bloodstream infection. However, for the other procedures, the *p*-values were relatively high, meaning that the association with bloodstream infection was not statistically significant, and we cannot conclude that they have a clear association with bloodstream infection. Ultimately, we found that prolonged procedure times were strongly associated with BSI. The BSI-positive group had significantly longer total operation time and circulatory arrest time (all $p < 0.05$) (Table 3).

3.4 Postoperative Course and Outcomes

BSI was associated with greater levels of postoperative care, including: longer duration of pericardial mediastinal drainage, tracheal intubation, arterial and central venous catheterization, and nasogastric intubation, higher reintubation rate, increased use of CRRT, and more frequent invasive procedures such as reintubation, thoracentesis, tracheostomy, exploratory sternotomy, and chest wound debridement (all $p < 0.001$). Patients with BSI received significantly more transfusions of red blood cells, plasma, and platelets ($p < 0.05$). Major complications were more common in the BSI group, including cerebral infarction, acute renal insufficiency, and pulmonary infection ($p < 0.001$). A trend toward higher rates of pleural effusion was also observed ($p = 0.120$). Patients with BSI experienced significantly worse outcomes, including prolonged ICU ($p < 0.001$) and hospital stay (>30 days) ($p < 0.001$), postoperative maximum body temperature ($p < 0.001$), and significantly increased in-hospital mortality ($p < 0.001$) (29.63% vs. 2.17%) (Table 4).

3.5 Analysis of Risk Factors and Development of a Prediction Model

Variables with significant univariate associations were subsequently entered into a multivariate binary logistic regression analysis using the forward likelihood ratio method. This identified five independent predictors for postoperative bloodstream infection: preoperative C-reactive protein (OR = 1.010, 95% CI: 1.002–1.019, $p = 0.020$), tracheostomy (OR = 9.186, 95% CI: 2.463–34.266, $p = 0.001$), infectious pneumonia (OR = 32.872, 95% CI: 4.186–258.174, $p = 0.001$), circulatory arrest time (min) (OR = 1.048, 95% CI: 1.004–1.093, $p = 0.033$), and age (OR = 1.055, 95% CI: 1.010–1.103, $p = 0.016$). These factors were incorporated to establish the final prediction model.

Based on the multivariate logistic regression analysis, a prediction model for postoperative bloodstream infection was established using five significant independent factors: preoperative C-reactive protein, tracheostomy, infectious pneumonia, circulatory arrest time, and age. The

Table 4. Postoperative course and outcomes of the included subjects.

	Whole cohort	Positive group	Negative group	*p*-value
	N = 257	N = 27	N = 230	
Duration of pericardial mediastinal drainage	240 (144.0–312.0)	360 (192.0–360.0)	237.0 (69.0–237.0)	<0.001
Duration of tracheal intubation (h)	43.0 (19.0–110.0)	216.0 (110.5)	38.0 (18.0–95)	<0.001
Duration of arterial catheterization	6.0 (4.0–10.0)	21.0 (13.5–36.0)	6.0 (4.0–9.0)	<0.001
Duration of central venous catheterization	10.0 (6.0–15.0)	27.0 (18.5–36.0)	9.0 (6.0–13.0)	<0.001
Duration of nasogastric tube placement	2 (0–6.0)	14 (7.0–26.5)	2 (0–6.0)	<0.001
Postoperative invasive procedures				
Thoracentesis	65 (25.29)	12 (44.44)	53 (23.04)	<0.001
Tracheostomy	15 (5.84)	8 (29.63)	7 (3.04)	<0.001
Reintubation	27 (10.51)	12 (44.44)	15 (6.52)	<0.001
Exploratory sternotomy	16 (6.23)	6 (44.44)	10 (6.52)	<0.001
Chest wound debridement	5 (1.95)	3 (11.11)	2 (0.87)	<0.001
Postoperative transfusion volume				
RBC (U)	2 (0–4)	21 (8–30)	2 (0–4)	<0.001
Plt (Unit)	0 (0–1)	2 (0–19)	0 (0–1)	<0.001
Postoperative complications				
Pleural effusion	116 (414)	16 (59.26)	100 (43.48)	0.120
Liver failure	29 (11.28)	15 (55.56)	14 (6.09)	<0.001
Acute renal insufficiency	32 (12.45)	15 (55.56)	17 (7.39)	<0.001
Cerebral infarction	35 (13.62)	11 (40.74)	24 (10.43)	<0.001
Infectious pneumonia	111 (43.19)	26 (96.30)	85 (39.96)	<0.001
Outcome				
Hospital stay >30 Days	16 (6.22)	12 (44.44)	4 (1.74)	<0.001
Postoperative maximum body temperature	38.32 ± 0.62 °C	39.03 ± 0.66 °C	38.24 ± 0.55 °C	<0.001
Postoperative ICU length of stay	6 (4–11)	27 (13.5–38)	6 (4–9)	<0.001
In-hospital mortality	13 (5.06)	8 (29.63)	5 (2.17)	<0.001

Footnote:

Significant differences between the two groups of patients in terms of postoperative course and outcomes of the included subjects. RBC, Red blood cell; Plt, Platelet; ICU, Intensive care unit.

Table 5. Multivariate analysis of the included subjects.

	Odds ratio	95% confidence interval	*p*-value
Age	1.055	1.010, 1.013	0.016
Infectious Pneumonia	32.872	4.186, 258.174	0.001
Tracheostomy	9.186	2.463, 34.266	0.001
Preoperative C-Reactive Protein	1.010	1.004, 1.093	0.020
Circulatory Arrest Time	1.048	1.018, 1.095	0.003

Footnote:

Multivariate analysis showed that age, preoperative C-reactive protein (CRP), time of circulatory arrest, tracheotomy, and infectious pneumonia were independent predictors of BSI.

Wald test confirmed the statistical significance of all included variables. The model's goodness-of-fit was evaluated using the Hosmer-Lemeshow test, which showed good calibration with a chi-square of 9.873 ($p = 0.274$). The likelihood ratio test further supported model significance with $p < 0.001$. Discrimination ability was assessed by ROC analysis, revealing an AUC of 0.897 (95% CI: 0.843–0.950, $p < 0.001$). At the optimal cutoff corresponding to the maximum Youden's index of 0.896, the model demonstrated sensitivity of 0.850 and specificity of 0.900, indicating strong predictive performance (Fig. 2, Table 5). The model's discrimination ability was assessed by ROC analysis, revealing an AUC of 0.897 (95% CI: 0.843–0.950, $p < 0.001$). Bootstrap internal validation (1000 repetitions) yielded an optimism-corrected AUC of 0.872 (95% CI: 0.815–0.929), indicating robust internal performance.

The above variables were then assigned appropriate scores based on their relative importance in the nomogram (Fig. 3). Finally, we constructed the decision curve analysis (DCA) curve (Fig. 4). These analyses demonstrated that the

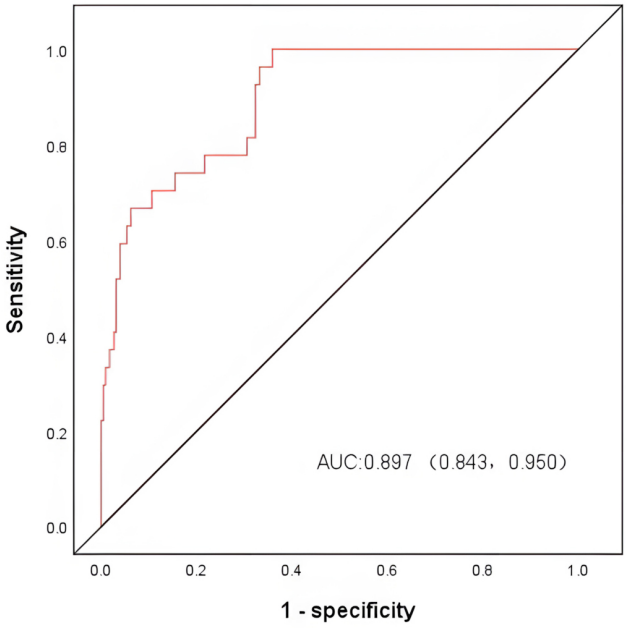

Fig. 2. ROC curves for the model to predict the occurrence of bloodstream infection after admission in the SAAD. After plotting the ROC curve, the area under the (AUC) was 0.897 (95% CI: 0.843–0.950, $p < 0.001$). ROC, receiver operating characteristic; AUC, area under the curve.

developed nomogram performed well across all threshold probabilities, confirming that it would provide the highest clinical net benefit across a wide range of clinical scenarios.

4. Discussion

This retrospective study of 257 patients undergoing surgical repair for Stanford type A aortic dissection provides a comprehensive analysis of the incidence, risk factors, and clinical impact of postoperative bloodstream infection. Our key findings indicate that BSI is a frequent (10.5%) and devastating complication, associated with a significantly prolonged hospital course, a higher incidence of multi-organ failure, and a significant increase in in-hospital mortality (29.63% vs. 2.17%). Furthermore, we developed and internally validated a predictive model incorporating five independent risk factors—age, preoperative C-reactive protein, circulatory arrest time, tracheostomy, and infectious pneumonia—which demonstrated strong discriminatory power (AUC 0.897) for identifying high-risk patients.

The observed BSI incidence of 10.5% aligns with the higher end of rates reported for major cardiac surgery, a finding likely attributable to the extreme acuity and complexity of SAAD surgery [13]. The profound clinical consequences of BSI in our cohort underscore its role as a critical determinant for poor outcomes. Patients with BSI had a more invasive postoperative course, characterized by prolonged dependence on mechanical ventilation and invasive catheters, a greater need for renal replacement therapy, and higher rates of re-operation. The cascade of complications, including cerebral infarction, acute renal insufficiency, and liver failure, suggests that BSI acts as a potent trigger for exacerbation of the systemic inflammatory response syndrome and subsequent multi-organ dysfunction syndrome [14]. The dramatic 13-fold increase in mortality in the BSI group illustrates that BSI is not merely a comorbid condition but a pivotal event that can tip the precarious balance of a post-dissection patient towards a fatal outcome. Therefore, strategies aimed at preventing and aggressively managing BSI are paramount to improving overall survival in SAAD repair.

The five independent risk factors identified in our multivariate analysis provide crucial insights into the pathophysiology of BSI. Elevated preoperative CRP emerged as a significant predictor, highlighting the importance of the pre-existing inflammatory state [15]. SAAD itself is now recognized as a highly inflammatory condition [16], with recent studies elucidating the role of damage-associated molecular patterns released from the dissected aorta in triggering a fulminant innate immune response [17]. A high CRP level upon admission is a biomarker of this intense systemic inflammation, which is often coupled with a state of compensatory immunoparalysis [18]. This early immunodysfunctional state, characterized by impaired neutrophil function and monocyte suppression, renders the patient highly susceptible to secondary postoperative infections [19]. Thus, preoperative CRP serves as a quantifiable measure of a patient's baseline vulnerability to infection even before the surgical insult [20].

The highly significant associations of tracheostomy and infectious pneumonia with BSI underscore the central role of the respiratory tract as a primary source for bloodstream invasion [21]. Tracheostomy, while sometimes necessary for prolonged ventilation, disrupts the anatomical barrier of the airway, facilitating colonization with hospital-acquired pathogens [22]. Infectious pneumonia, particularly ventilator-associated pneumonia, then establishes a persistent nidus of infection from which bacteria can readily translocate into the pulmonary circulation and cause BSI [23,24]. The predominance of Gram-negative bacilli in our blood culture analysis is consistent with this pathway and strongly suggests that rigorous adherence to VAP prevention bundles is not only a pulmonary protective strategy but a critical defense against life-threatening BSI [25].

Prolonged circulatory arrest time, a marker of surgical complexity, implicates gut mucosal injury and bacterial translocation as another key pathophysiological pathway [26]. Deep hypothermic circulatory arrest induces splanchnic hypoperfusion and ischemia-reperfusion injury, which can compromise the integrity of the intestinal barrier [27]. This allows commensal Gram-negative bacteria and their endotoxins to leak into the systemic circulation. The role of gut-derived infection in critical illness is a topic of renewed interest, and our data provide strong clinical evi-

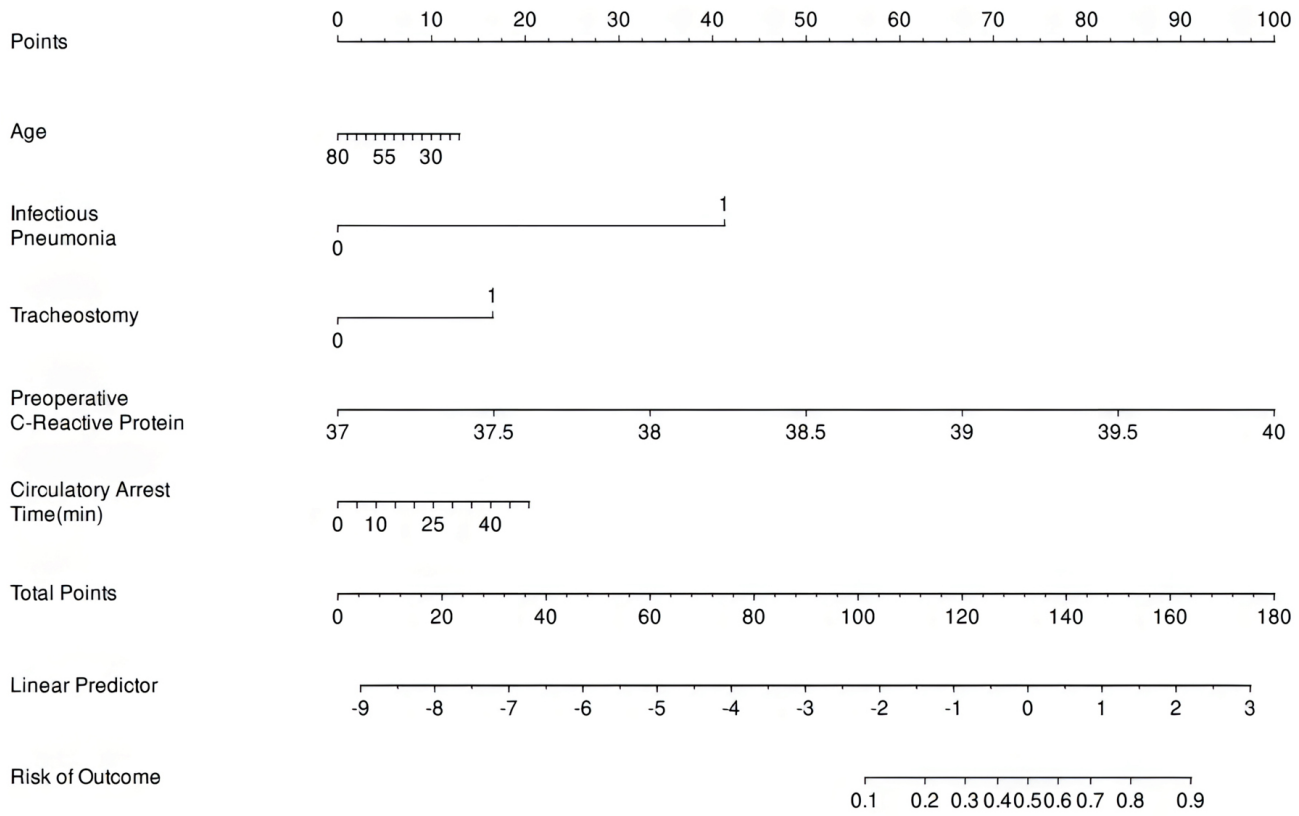

Fig. 3. Prediction chart for all-cause in-hospital mortality of TAAAD. A bar plot was drawn to visualize the results of the prediction model. A total of five prediction variables were involved in the composition of the bar plot. According to the extent of influence of each prediction variable in the model, i.e., the magnitude of the regression coefficient, a score was assigned to each value level of the variable, resulting in five separate scores, and then these scores were added together to obtain the total score. TAAAD, Type A acute aortic dissection.

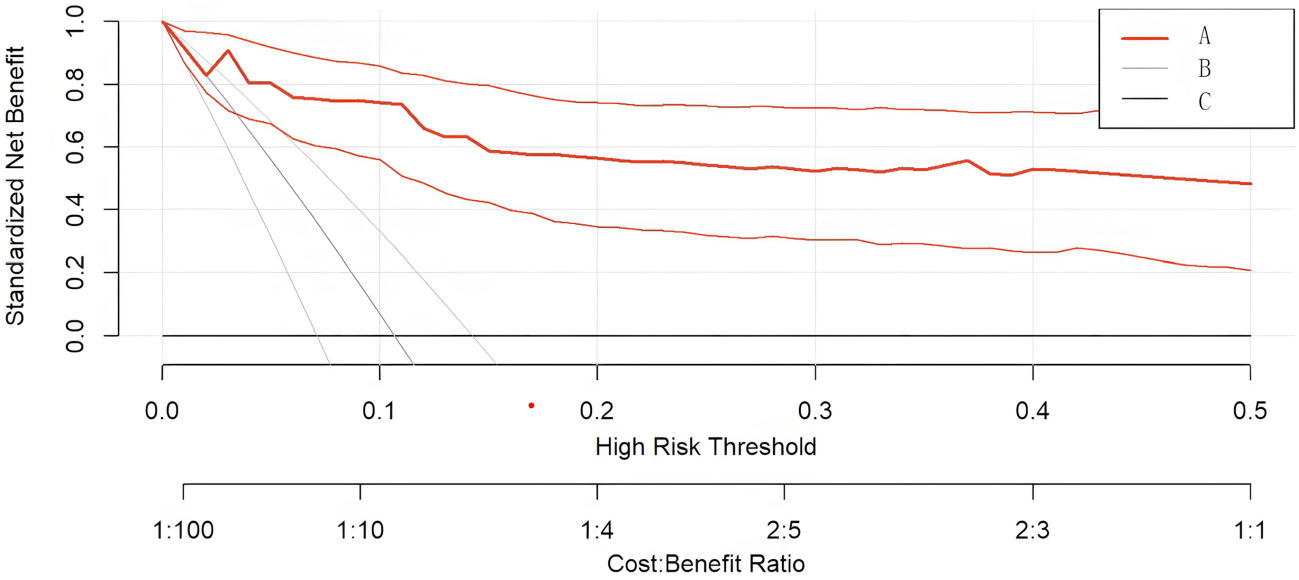

Fig. 4. Curve A represents model construction, Curve B represents full intervention, and Curve C represents no intervention. The clinical decision curve for predicting all-cause in-hospital mortality in TAAAD. The DCA curve was plotted based on the constructed CPM. The horizontal axis indicates the risk threshold, and the vertical axis indicates the net benefit (NB) after considering benefits and harms. The DCA results show that the risk assessment model brings net benefit to patients over a risk threshold range of 0.01–0.7.

dence supporting this pathway in SAAD surgery, linking the technical demands of the procedure directly to the risk of a subsequent septic complication. Furthermore, advanced age, a non-modifiable risk factor, contributes through the well-documented decline in immune competence known as immunosenescence, which reduces the patient's ability to withstand the cumulative insults of dissection, surgery, and infection [28].

The predictive model developed from these factors offers a practical tool for early risk stratification. Its strength lies in the integration of readily available preoperative and immediate postoperative variables, allowing clinicians to identify high-risk patients at the conclusion of the operation or upon ICU admission. This risk stratification enables a proactive, targeted management approach. For high-risk patients, clinicians can institute intensified surveillance, such as enhanced microbiological monitoring, have a lower threshold for initiating prudent empiric antibiotic therapy guided by local antibiograms, aggressively pursue source control, and consider adjunct supportive measures such as immunonutrition. By focusing resources on the patients who need them most, this model facilitates a paradigm shift from reactive treatment to proactive prevention, potentially reducing the incidence and mortality of BSI.

5. Limitations

Several limitations of our study must be acknowledged. Its single-center, retrospective design allows for the potential for selection and information bias. The number of BSI events, while substantial for this patient cohort, is relatively small for a multivariate model with five predictors, raising a potential risk of overfitting. Most critically, the model requires external validation in independent, multi-center cohorts to confirm its generalizability before widespread clinical adoption. Moreover, the number of BSI events, while substantial for this patient cohort, results in a low events-per-variable ratio in our multivariate model, which remains a limitation. Although bootstrap internal validation suggested acceptable optimism, external validation in independent, multi-center cohorts is crucial to confirm generalizability before any clinical application. Furthermore, the predictors identified in our model—advanced age, elevated preoperative inflammation, prolonged circulatory arrest, tracheostomy, and infectious pneumonia—are based on strong biological plausibility regarding immune function, procedural invasiveness, and infection pathways. As such, they are likely to remain relevant risk factors in other settings. However, the magnitude of their association (i.e., their specific odds ratios) may vary across institutions employing different surgical techniques (e.g., strategies to shorten circulatory arrest) or postoperative care bundles (e.g., rigorous ventilator-associated pneumonia prevention protocols that reduce the need for tracheostomy). Consequently, while the core predictors are expected to be generalizable, the model's absolute risk estimates may require recalibration during external validation in centers with distinct clinical protocols before it can be applied for local risk stratification. Finally, the inclusion of "infectious pneumonia" as a predictor means the model is most accurately applied in the early postoperative period for dynamic risk assessment, rather than as a pure preoperative prediction tool. While the sample size provided sufficient statistical power to identify the strong independent predictors included in our final model, it may have been underpowered to detect weaker associations (e.g., odds ratios <2.0) or to definitively evaluate risk factors with very low baseline prevalence in this surgical population.

6. Conclusion

In conclusion, this study confirms the critical impact of postoperative BSI following type A aortic dissection surgery. We established a predictive model incorporating five independent risk factors: age, preoperative CRP, circulatory arrest time, tracheostomy, and infectious pneumonia. The model shows strong predictive ability, offering a practical tool for early risk stratification. This enables timely intervention for high-risk patients, potentially improving outcomes. External validation in future studies is recommended to generalize its application.

Availability of Data and Materials

Data will be made available from the corresponding author upon reasonable request.

Author Contributions

Conception and design of the study: ZFX, FYL, FHS, LYW, HZZ and XCM. Acquisition of data: FHS, ZFX, FYL, LYW, MQW, and XZL. Data analysis and interpretation: ZFX, FYL, FHS, LYW, and MQW. Drafting of manuscript: ZFX, FYL, FHS, LYW, MQW, and XZL. Critical revision: ZFX, FYL, FHS, LYW, MQW, XZL, HZZ, and XCM. Final approval and supervision: HZZ and XCM. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This retrospective study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Shandong Provincial Hospital Affiliated to Shandong First Medical University (Number: NSFC2018-002). Informed consent was exempted due to the retrospective nature of the study.

Acknowledgment

Not applicable.

Funding

This study was supported by the fund with an additional project number. Add content as follows: This work was supported by the Young Experts of Taishan Scholar Program of Shandong Province (grant no. tsqn202408359), the Nature Science Foundation of Shandong Province (ZR2023MH124), and Jinan Science and Technology Plan Project (202225050).

Conflict of Interest

The authors declare no conflict of interest.

References

[1] Kovoor JG, Glynatsis JM, Glynatsis NC, Perrotta D, Chan E, Daniell T, *et al*. Factors affecting acute aortic dissection mortality: A multicentre cohort study. Surgery in Practice and Science. 2025; 23: 100311. https://doi.org/10.1016/j.sipas.2025.100311.

[2] Clouse WD, Hallett JW, Jr, Schaff HV, Spittell PC, Rowland CM, Ilstrup DM, *et al*. Acute aortic dissection: population-based incidence compared with degenerative aortic aneurysm rupture. Mayo Clinic Proceedings. 2004; 79: 176–180. https://doi.org/10.4065/79.2.176.

[3] Trimarchi S, Nienaber CA, Rampoldi V, Myrmel T, Suzuki T, Mehta RH, *et al*. Contemporary results of surgery in acute type A aortic dissection: The International Registry of Acute Aortic Dissection experience. The Journal of Thoracic and Cardiovascular Surgery. 2005; 129: 112–122. https://doi.org/10.1016/j.jtcvs.2004.09.005.

[4] Tang GHL, Malekan R, Yu CJ, Kai M, Lansman SL, Spielvogel D. Surgery for acute type A aortic dissection in octogenarians is justified. The Journal of Thoracic and Cardiovascular Surgery. 2013; 145: S186–S190. https://doi.org/10.1016/j.jtcvs.2012.11.060.

[5] Lin L, Chen Y, Zhou N, Wu Y, Yu Y, Liu J, *et al*. Clinical Evaluation of Digital PCR for Rapid Pathogen Detection in Suspected Bloodstream Infections. British Journal of Hospital Medicine. 2025; 86: 1–22. https://doi.org/10.12968/hmed.2025.0561.

[6] Isselbacher EM, Preventza O, Hamilton Black J, 3rd, 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.

[7] Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, *et al*. Diagnosis and Treatment of Adults with Community-acquired Pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America. American Journal of Respiratory and Critical Care Medicine. 2019; 200: e45–e67. https://doi.org/10.1164/rccm.201908-1581ST.

[8] Kalantar M, Rezayan AH, Hajghassem H, Farazkish M, Moghadam RA. Point-of-care testing for early detection of sepsis: a systematic literature review. Clinica Chimica Acta; International Journal of Clinical Chemistry. 2026; 578: 120569. https://doi.org/10.1016/j.cca.2025.120569.

[9] Haghighatpanah MA, Mazaheri-Tehrani S, Adibzadeh A, Ostadsharif N, Moradi P, Vali M, *et al*. Predictability of kinetic estimated glomerular filtration rate for acute kidney injury in patients undergoing coronary artery bypass surgery. BMC Nephrology. 2025; 26: 409. https://doi.org/10.1186/s12882-025-04266-1.

[10] Ede J, Teurneau-Hermansson K, Ramgren B, Moseby-Knappe M, Larsson M, Sjögren J, *et al*. Outcomes following repair of acute type A aortic dissection in patients with cerebral malperfusion. Scandinavian Cardiovascular Journal. 2025; 59: 2514742. https://doi.org/10.1080/14017431.2025.2514742.

[11] Ludusanu A, Ciuntu BM, Tanevski A, Bernic V, Tinica G. The correlation between the European System for Cardiac Operative Risk Evaluation and the Model for End-Stage Liver Disease in patients with coronary artery bypass graft surgery. Journal of Medicine and Life. 2024; 17: 926–933. https://doi.org/10.25122/jml-2024-0311.

[12] Barnett NM, Liesman DR, Strobel RJ, Wu X, Paone G, DeLucia A, 3rd, *et al*. The association of intraoperative and early postoperative events with risk of pneumonia following cardiac surgery. The Journal of Thoracic and Cardiovascular Surgery. 2024; 168: 1144–1154.e3. https://doi.org/10.1016/j.jtcvs.2023.09.056.

[13] Zhou L, Chen C, Gong W, Shang L, Liu L, Duan W, *et al*. Integrated clinical and imaging predictors of 60-day mortality in acute aortic dissection: emphasizing innominate and carotid artery involvement. Clinical Radiology. 2025; 90: 107078. https://doi.org/10.1016/j.crad.2025.107078.

[14] Zukowska A, Kaczmarczyk M, Listewnik M, Zukowski M. Impact of Post-Operative Infection after CABG on Long-Term Survival. Journal of Clinical Medicine. 2023; 12: 3125. https://doi.org/10.3390/jcm12093125.

[15] Poredos P, Komadina R. Inflammation and Perioperative Cardiovascular Events. Cells. 2025; 14: 1362. https://doi.org/10.3390/cells14171362.

[16] Xu C, Chen W, Nie X, Xu R, Feng X, Chen Z, *et al*. The innate immune axis drives aortic dissection pathogenesis through inflammation and presents novel therapeutic targets. Frontiers in Immunology. 2025; 16: 1654622. https://doi.org/10.3389/fimmu.2025.1654622.

[17] Kimura N, Machii Y, Hori D, Mieno M, Eguchi N, Shiraishi M, *et al*. Influence of false lumen status on systemic inflammatory response triggered by acute aortic dissection. Scientific Reports. 2025; 15: 475. https://doi.org/10.1038/s41598-024-84117-5.

[18] Silva EE, Skon-Hegg C, Badovinac VP, Griffith TS. The Calm after the Storm: Implications of Sepsis Immunoparalysis on Host Immunity. Journal of Immunology. 2023; 211: 711–719. https://doi.org/10.4049/jimmunol.2300171.

[19] Santacroce E, D'Angerio M, Ciobanu AL, Masini L, Lo Tartaro D, Coloretti I, *et al*. Advances and Challenges in Sepsis Management: Modern Tools and Future Directions. Cells. 2024; 13: 439. https://doi.org/10.3390/cells13050439.

[20] Mouliou DS. C-Reactive Protein: Pathophysiology, Diagnosis, False Test Results and a Novel Diagnostic Algorithm for Clinicians. Diseases. 2023; 11: 132. https://doi.org/10.3390/diseases11040132.

[21] Cui Y, Yi C, Zhang C, Yang C, Wang X, Chen W, *et al*. Risk factors for bloodstream infection among patients admitted to an intensive care unit of a tertiary hospital of Shanghai, China. Scientific Reports. 2024; 14: 12765. https://doi.org/10.1038/s41598-024-63594-8.

[22] Schuderer JG, Reider L, Wunschel M, Spanier G, Spoerl S, Gottsauner MJ, *et al*. Elective Tracheotomy in Patients Receiving Mandibular Reconstructions: Reduced Postoperative Ventilation Time and Lower Incidence of Hospital-Acquired Pneumonia. Journal of Clinical Medicine. 2023; 12: 883. https://doi.org/10.3390/jcm12030883.

[23] Fumagalli J, Panigada M, Klompas M, Berra L. Ventilator-associated pneumonia among SARS-CoV-2 acute respiratory distress syndrome patients. Current Opinion in Critical Care. 2022; 28: 74–82. https://doi.org/10.1097/MCC.0000000000000908.

[24] Massart N, Dupin C, Legris E, Fedun Y, Barbarot N, Legay F, *et al*. Multiple-site decontamination in mechanically ventilated

ICU patients: A real-life study. Infectious Diseases now. 2023; 53: 104666. https://doi.org/10.1016/j.idnow.2023.104666.

[25] Azkarate I, Salas E, Cabarcos E, Ganzarain M, Quevedo I, Elósegui I, *et al*. Respiratory sepsis: a 12-year prospective observational study in critically ill patients. Respiratory Medicine. 2025; 249: 108476. https://doi.org/10.1016/j.rmed.2025.108476.

[26] Mu J, Chen Q, Zhu L, Wu Y, Liu S, Zhao Y, *et al*. Influence of gut microbiota and intestinal barrier on enterogenic infection after liver transplantation. Current Medical Research and Opinion. 2019; 35: 241–248. https://doi.org/10.1080/03007995.2018.1470085.

[27] Khailova L, Robison J, Jaggers J, Ing R, Lawson S, Treece A, *et al*. Tissue alkaline phosphatase activity and expression in an experimental infant swine model of cardiopulmonary bypass with deep hypothermic circulatory arrest. Journal of Inflammation. 2020; 17: 27. https://doi.org/10.1186/s12950-020-00256-2.

[28] Hernández-Quiles R, Merino-Lucas E, Boix V, Fernández-Gil A, Rodríguez-Díaz JC, Gimeno A, *et al*. Bacteraemia and quick Sepsis Related Organ Failure Assessment (qSOFA) are independent risk factors for long-term mortality in very elderly patients with suspected infection: retrospective cohort study. BMC Infectious Diseases. 2022; 22: 248. https://doi.org/10.1186/s12879-022-07242-4.