Wang *et al.*
European Journal of Medical Research (2025) 30:203
https://doi.org/10.1186/s40001-025-02475-w

European Journal of Medical Research

RESEARCH Open Access

Admission pulse pressure and in-hospital mortality in type A acute aortic dissection: result from a Chinese study in stable patients on admission

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Abstract

Objectives In recent years, several epidemiologic studies have shown that pulse pressure (PP) is a powerful predictor of mortality from many cardiovascular diseases. However, few studies have reported the association between PP and adverse events during hospitalization in patients with type A acute aortic dissection (TAAAD). The aim of this study was to evaluate the relationship between admission PP and in-hospital all-cause mortality, in patients with TAAAD of relatively stable patients.

Methods Patients with TAAAD of relatively stable patients admitted from January 2015 to December 2021 were included and divided into four groups according to the PP values measured at the time of admission: reduced group (PP ≤ 40 mmHg), normal group (40 < PP ≤ 56 mmHg), mildly elevated group (56 < PP ≤ 75 mmHg), and significantly elevated group (PP > 75 mmHg). A multivariate binary logistic regression model was constructed, plotted using nomogram and evaluated with ROC curve.

Results Admission PP and in-hospital all-cause mortality showed a "J-curve" correlation and in-hospital all-cause mortality was significantly higher in the significantly elevated group and reduced group ($P = 0.002$), respectively. Multivariate binary logistic regression analysis showed that significantly elevated PP (PP > 75 mmHg) ($P < 0.001$) and reduced PP ($P = 0.043$), D-dimer ($P < 0.001$), ascending aortic diameter ($P = 0.037$), Abdominal visceral vessels involved ($P = 0.017$), and coronary atherosclerosis ($P = 0.003$) and emergent surgery ($P < 0.001$) were independent predictive factors for in-hospital all-cause mortality. The AUC of ROC plotted was 0.827 (95% CI 0.774–0.880).

Conclusions Our findings demonstrated a "J-curve" association of admission PP with in-hospital all-cause mortality in TAAAD. Significantly elevated and reduced admission PP, D-dimer, ascending aortic diameter and coronary atherosclerosis were independent risk factors for in-hospital all-cause mortality in patients with TAAAD, and emergent surgery was a protective factor.

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Keywords Aortic dissection, Pulse pressure, J-curve, In-hospital all-cause mortality, Multivariate binary logistic regression analysis, ROC curve, Nomogram

Introduction

Type A acute aortic dissection (TAAAD) is widely accepted as a devastating aortic pathology in which aortic lesion involves the ascending aorta. For untreated TAAAD mortality increases to 50% during first 24 h and emergent surgical intervention is indicated by principle for TAAAD for the purpose of timely repair and reconstruction of the impaired aorta. For decades cardiovascular surgeons across the globe have made the painstaking efforts for optimizing the surgical procedures. Although the short- and long-term outcomes of TAAAD have markedly improved worldwide, mortality remains high and is reported between 15 and 30%. Blood pressure control is the highest priority in the treatment of TAAAD. A retrospective study [1] that included 6,238 AAD patients registered in the International Registry of Acute Aortic Dissection (IRAD) from 1999 to 2016 analyzed the relationship between admission systolic blood pressure (SBP) and mortality during hospitalization. The results showed a significant "J-curve" relationship between admission SBP and in-hospital mortality in patients with TAAAD, and a SBP of less than 80 mmHg was an independent risk factor for in-hospital mortality. A similar single-center retrospective study analyzed the relationship between admission SBP and in-hospital mortality in patients with AAD in China [2]. The results showed a non-linear correlation between admission SBP and in-hospital mortality in patients with AAD, and a negative correlation between SBP and in-hospital mortality when SBP was less than 120 mmHg.

In this study, we retrospectively analyzed the clinical data of 488 TAAAD patients, with the aim of evaluating the correlation between admission PP and all-cause in-hospital mortality, in TAAAD patients.

Subjects and methods

Subjects

In this study, patients with TAAAD of relatively stable patients admitted between January 1, 2015, and December 31, 2022, were retrieved through the electronic medical record system of Shandong Provincial Hospital. This retrospective study was supervised by the Ethics Committee of Shandong Provincial Hospital affiliated to First Medical University and informed consent was waived because of its retrospective design. The diagnosis of TAAAD was based on the 2022 ACC/AHA guidelines on the treatment and diagnosis of aortic disease[3]. According to the guidelines, any dissection involving the ascending aorta that occurs within 14 days of symptom onset was defined as TAAAD, and the definitive diagnosis of dissection relied on imaging tests, such as computed tomography (CT) or magnetic resonance imaging (MRI).

488 patients with TAAAD were categorized into four groups based on the initial PP value measured at the time of patient admission: reduced ($PP \leq 40$ mmHg), normal ($40 < PP \leq 56$ mmHg), mildly elevated ($56 < PP \leq 75$ mmHg), and significantly elevated ($PP > 75$ mmHg) [4]. Ethical approval for the study was provided by the hospital institutional review board (NSFC2018-002). As the study was retrospective, informed consent of patients was waived. The study was performed following the Good Clinical Practice (GCP) and principles of the Declaration of Helsinki.

Inclusion and exclusion criteria

Inclusion criteria

(1) Patients presenting to the hospital within ≤ 14 days of symptom onset.
(2) TAAAD clearly diagnosed by imaging examinations, such as CT or MRI.
(3) Patients with complete baseline data (e.g., age, gender, and vital signs, etc.) and complete perioperative data.

Exclusion criteria

(1) Important clinical data was missing (especially whose blood pressure was difficult to measure upon admission).
(2) Variants of typical aortic dissection, such as intermural hematoma, penetrating aortic ulcer, intimal tear without hematoma, medical or traumatic AD, and periaortic hematoma.
(3) Symptoms lasting > 14 days.

Data collection of the study population

The clinical data of the patients included in this study were obtained by searching the electronic medical record system of Shandong Provincial Hospital. The data included: baseline data, past history, clinical

manifestations, imaging and laboratory findings, intraoperative and postoperative conditions, and etc. For patients admitted with aortic dissection, it was routine to measure blood pressure in both arms. If discrepancies were observed, the highest value was retained. Hepatic dysfunction is primarily assessed with the Model for End-Stage Liver Disease Excluding INR (MELD-XI) score. Renal dysfunction is evaluated using the Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury (AKI) classification.

Statistical analysis

Continuous variables were tested for normality and were expressed as mean ± standard deviation (normal distribution) or median (25th percentile, 75th percentile) (skewed distribution). Categorical variables were expressed as number of cases (percentage). One-way analysis of variance (ANOVA) was applied for continuous variables that conformed to a normal distribution, and Kruskal–Wallis test was used for data with a skewed distribution. Chi-square or Fisher's exact test was used to analyze the categorical variables.

Patients were categorized into four groups based on admission pulse pressure (PP) values, following previously established thresholds from prior studies: reduced (PP ≤ 40 mmHg), normal (40 < PP ≤ 56 mmHg), mildly elevated (56 < PP ≤ 75 mmHg), and significantly elevated (PP > 75 mmHg) [4]. For outcome analysis, the total population was divided into two groups: the death group and the survival group, based on whether or not a death event occurred during hospitalization. Initially, study variables were screened using univariate analysis. Then, a multivariate binary logistic regression model was applied, using the backward stepwise method to introduce variables with a *P* value of less than 0.10 from the univariate analysis. In addition to the variables obtained in the univariate regression analysis, important related variables in clinical practice were also included in the multivariate regression analysis. Risk adjustment was performed to determine the correlation between admission PP and in-hospital all-cause mortality, as well as to identify independent predictors of in-hospital all-cause mortality. The obtained results were expressed as odds ratio (OR) with a 95% confidence interval (CI). A disease predictive model for in-hospital all-cause mortality was constructed and presented in the form of a nomogram. The discriminatory power of predictive model was assessed by plotting the receiver operating characteristic (ROC) curve and calculating the area under the curve (AUC). An AUC value greater than 0.75 was considered to indicate good discriminatory power. Based on the level of influence of each predictor variable in the model, i.e., the magnitude of the regression coefficients, a score was assigned to the level of value of each variable, thus obtaining six individual scores, which were then summed up to obtain the total score. Finally, the predicted probability of in-hospital all-cause mortality of TAAAD patients was calculated by the functional transformation relationship between the total score and the probability of death event. The higher the total score, the higher the odds of in-hospital all-cause mortality.

The model's calibration was evaluated using the Hosmer–Lemeshow test of goodness-of-fit. A *P* value greater than 0.05 would suggest a better fit of the model. All statistical tests in this study were performed using two-sided tests, with $P < 0.05$ indicating a statistically significant difference. All statistical analyses were performed using SPSS Statistics 26.0 and RStudio 4.2.2 analysis software.

Results

Characteristics of patients

Inclusion of patients

From January 2015 to December 2021, 488 patients were recruited consecutively with TAAAD. 8 subjects were excluded, as shown in Fig. 1a. The flowchart of the decision-making for the diagnostics and treatment is illustrated in Fig. 1b.

Detailed information of patients

Demographics and past history The mean age of enrolled patients was 51.50 ± 11.01 years, and a total of 124 (25.41%) patients were older than 60 years. 344 (70.49%) were males and 144 (29.51%) were females. 107 patients were treated conservatively and 381 were treated surgically, and 100 (20.49%) died in hospital, of which 52 (48.60%) died after conservative treatment and 48 (12.60%) died after surgical treatment. The difference between the four groups of patients was shown in Table 1. In the reduced group, the patients were older (55.52 ± 9.85 vs. 51.43 ± 10.59 vs. 50.15 ± 11.70 vs. 50.43 ± 10.77, $P < 0.05$), and the proportion of aortic aneurysms was greater [29 (34.52%) vs. 20 (16.13%) vs. 18 (11.54%) vs. 20 (16.95%). $P < 0.001$].

Clinical manifestations Table 1 also shows the differences of clinical manifestation of four groups including abdominal pain [(19.05%) vs. 46 (36.80%) vs. 51 (32.69%) vs. 35 (28.93%), $P = 0.045$] and syncope [19 (22.62%) vs. 11 (8.80%) vs. 6 (3.85%) vs. 7 (5.79%), $P < 0.001$].

Preoperative ultrasound and imaging examination and lab tests Preoperative ultrasound and imaging examination and lab tests are summarized in Table 1. In the reduced group (PP ≤ 40 mmHg), the percentages of bicuspid aortic valve (7.79%), pericardial effusion (53.48%) and cardiac tamponade (9.30%) were highest, and mean diameter of ascending aorta (5.35 ± 1.26 cm) was largest. In the sig-

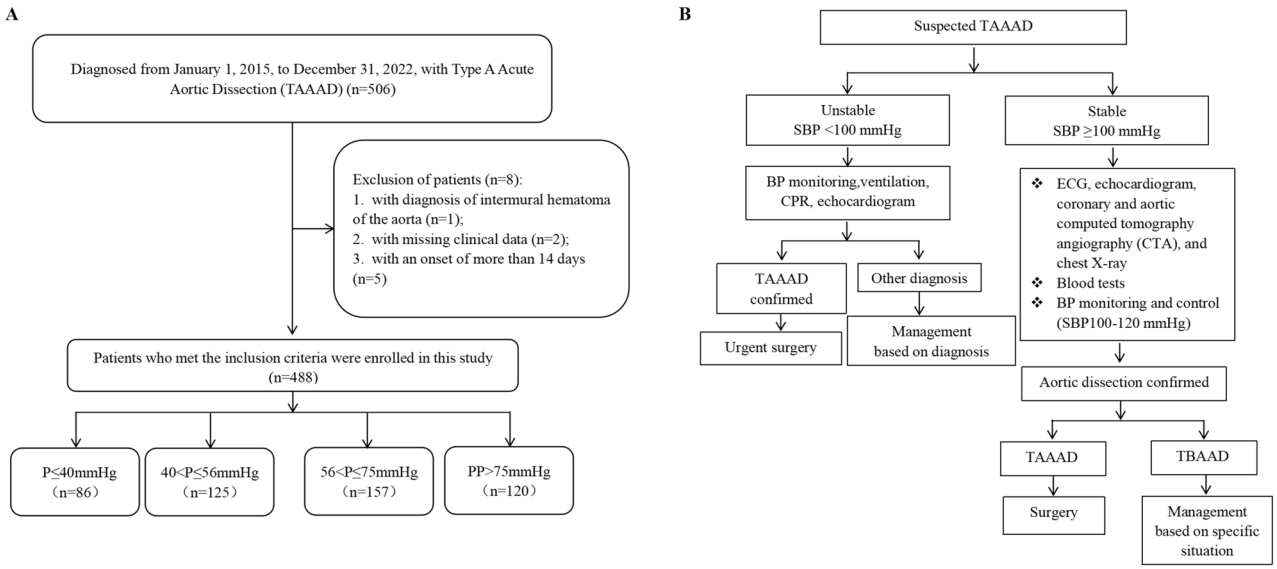

Fig. 1 Flowchart of study patient selection and decision-making. **a** Patient selection. **b** Decision-making. PP: pulse pressure; SBP: systolic blood pressure; ECG: electrocardiogram; CPR: cardiopulmonary resuscitation; TBAAD: type B acute aortic dissection; TAAAD: type A acute aortic dissection

nificantly elevated group (PP > 75 mmHg), involvement of abdominal visceral arteries (61.84%), aortic arch three branches (71.96%), and iliac arteries (53.33%) were more common.

Admission pulse pressure and in-hospital all-cause mortality and other complications

As shown in Fig. 2 and Table 2, PP and in-hospital all-cause mortality showed a "J-curve" correlation, with higher rates in the significantly elevated group and reduced group, which were 28.93% and 27.91%, respectively ($P=0.002$); and rates in the normal group and mildly elevated group were 12.80% and 16.03%, respectively. Besides, the four groups showed significant differences in terms of cerebral infarction ($P=0.032$), hepatic insufficiency ($P=0.039$) and renal insufficiency ($P=0.020$).

Risk factor screening and predictive modeling for in-hospital all-cause mortality

Univariate logistic analysis Emergent surgical treatment was negatively associated with the in-hospital all-cause mortality (0.15, 0.09–0.25). In contrast, variables including age (1.03, 1.01–1.05), age > 60 years (2.03, 1.27–3.25), PP (1.27, 1.04–1.55), coronary atherosclerosis (1.77, 1.10–2.73), syncope (2.74, 1.43–5.26), myocardial infarction/ischemia (4.16, 2.51–6.90) and limb ischemia etc. (2.10, 1.18–3.72), were positively correlated with the in-hospital all-cause mortality ($P<0.05$) (Table 3).

Multivariate logistic analysis The multivariate binary logistic regression analysis (Table 4) showed that significantly elevated PP (PP > 75 mmHg) ($P<0.001$) and reduced PP ($P=0.043$), D-dimer ($P<0.001$), ascending aortic diameter ($P=0.037$), abdominal visceral vessels involved ($P=0.017$), and coronary atherosclerosis ($P=0.003$) and emergent surgery ($P<0.001$) were independent predictive factors for in-hospital all-cause mortality. After plotting the ROC curve, and the AUC of the curve was 0.827 (95% CI 0.774–0.880, $P<0.001$) and the Yoden index of this ROC curve was 0.227, which corresponded to a sensitivity and specificity of 78.7% and 75.3%, respectively (Fig. 3). The Hosmer–Lemeshow test of goodness-of-fit was $\chi=2.285$, $P=0.971>0.05$.

Establishment of nomogram

Subsequently, column line plots were drawn to visualize the results of the prediction model. As shown in Fig. 4, a total of six predictor variables were involved in the composition of the column line plot. We incorporated the ascending aortic diameter, D-dimer levels, pulse pressure, emergency surgery, abdominal vascular involvement, and atherosclerosis into a nomogram. Among these, emergency surgery, abdominal vascular involvement, and atherosclerosis were categorical variables, while ascending aortic diameter, D-dimer levels, and pulse pressure were continuous variables. This nomogram was applied to predict mortality risk among the TAAAD patients.

The calibration plot showed that the calibration curve (Apparent line) closely matched the diagonal

Table 1 Comparing clinical baseline data of four groups

	PP < 40 mmHg	40 < PP < 56 mmHg	56 < PP < 75 mmHg	PP > 75 mmHg	*P* value
Number of cases, *n*	86	125	156	121	
Age (years)	55.52 ± 9.85	51.43 ± 10.59	50.15 ± 11.70	50.43 ± 10.77	0.507
Male, *n* (%)	61 (70.93)	85 (68.00)	110 (70.51)	88 (72.73)	0.880
Age > 60 years, *n* (%)	32 (37.21)	32 (25.60)	33 (21.15)	29 (23.97)	0.050
Hypertension, *n* (%)	59 (70.24)	96 (76.80)	112 (71.79)	91 (76.47)	0.594
Diabetes, *n* (%)	4 (4.76)	4 (3.23)	7 (4.49)	6 (5.08)	0.903
Marfan syndrome, *n* (%)	4 (4.76)	3 (2.42)	8 (5.13)	5 (4.24)	0.708
Atherosclerosis, *n* (%)	35 (41.76)	57 (45.60)	52 (33.33)	43 (36.44)	0.174
Aortic aneurysm, *n* (%)	29 (34.52)	20 (16.13)	18 (11.54)	20 (16.95)	< 0.001
Previous aortic dissection, *n* (%)	2 (2.38)	4 (3.23)	4 (2.56)	4 (3.39)	0.962
History of cardiac surgery, *n* (%)	3 (3.57)	4 (3.23)	3 (1.92)	3 (2.54)	0.863
Heart rate (bpm)	85.0 (72.0, 98.0)	82.0 (70.0, 94.5)	80.5 (68.0, 92.0)	90.0 (75.0, 105.0)	0.123
Systolic blood pressure (mmHg)	126.00 (115.00,136.00)	110.00 (99.75,120.25)	140.00 (128.00,151.75)	165.00 (150.00,180.00)	< 0.001
Diastolic blood pressure (mmHg)	80.00 (69.00,85.50)	76.00 (65.00,84.25)	75.00 (64.25,87.00)	80.00 (65.50,90.00)	0.342
Pulse pressure (mmHg)	33.78 ± 4.91	48.84 ± 4.32	64.67 ± 5.00	87.88 ± 11.78	< 0.001
Mean arterial pressure (mmHg)	86.76 ± 18.58	93.78 ± 14.20	97.09 ± 16.27	107.70 ± 20.71	< 0.001
Markedly elevated (uncontrolled) blood pressure, > 180/110mmHg, *n* (%)	3 (3.49)	2 (1.60)	4 (2.56)	24 (19.83)	< 0.001
Blood pressure, > 140/90mmHg, *n* (%)	9 (10.47)	21 (16.80)	64 (41.03)	85 (70.25)	< 0.001
Hypotension, *n* (%)	12 (13.95)	2 (1.60)	1 (0.64)	0 (0)	< 0.001
Shock, *n* (%)	8 (9.30)	1 (0.80)	1 (0.64)	0 (0)	< 0.001
Antihypertensive Drugs (%)	38 (44.19)	74 (59.20)	107 (68.59)	82 (67.77)	< .001
Antithrombotic Drugs(%)	7 (8.14)	12 (9.60)	14 (8.97)	11 (9.09)	0.988
Inotropic/vasopressive Drugs(%)	23 (26.74)	20 (16.00)	27 (17.31)	22 (18.18)	0.217
Ultrasound and imaging examination					
Bicuspid aortic valve, *n* (%)	6 (7.79)	0 (0)	10 (6.71)	1 (0.85)	0.002
Aortic valve insufficiency, *n* (%)	43 (55.13)	66 (54.55)	83 (55.70)	70 (60.87)	0.758
Pericardial effusion, *n* (%)	46 (53.48)	54 (45.00)	59 (39.86)	37 (32.17)	0.003
Cardiac tamponade, *n* (%)	8 (9.30)	1 (0.80)	1 (0.64)	0 (0)	< 0.001
Pleural effusion, *n* (%)	29 (33.72)	34 (27.20)	39 (25.00)	25 (20.66)	0.150
Coronary artery involved, *n* (%)	8 (12.50)	7 (6.19)	10 (7.35)	9 (8.49)	0.506
Abdominal visceral vessels involved, *n* (%)	34 (39.53)	72 (58.00)	74 (47.32)	75 (61.84)	0.038
Three branch vessel involved, *n* (%)	49 (56.98)	65 (51.79)	104 (66.43)	87 (71.96)	< 0.001
Iliac vessels involved, *n* (%)	24 (27.59)	51 (41.00)	52 (33.33)	64 (53.33)	< 0.001
LVEF (%)	58.81 ± 5.56	59.73 ± 4.89	59.17 ± 5.25	59.42 ± 3.57	0.289
Ascending aorta diameter (cm)	5.00 (4.00,5.00)	5.00 (5.00,6.00)	5.00 (4.00,5.00)	5.00 (4.00,5.00)	0.001
Laboratory tests					
D-dimer (mg/L)	5.91 (1.99,11.63)	5.63 (2.24,12.51)	7.06 (2.47,13.83)	7.74 (2.95,17.33)	0.274
Cr (umol/L)	82.53 (65.52,113.35)	87.20 (65.40,122.91)	78.80 (62.88,117.23)	87.20 (69.36,111.00)	0.513
HS-TnT (pg/ml)	51.69(14.23,322.48)	22.08 (7.39,283.45)	17.17 (8.39,281.66)	29.46 (10.77,239.93)	0.058
CK–MB (ng/ml)	5.64 (1.11,13.63)	3.05 (1.21,10.79)	2.80 (1.31,10.39)	3.78 (1.61,12.01)	0.260
MYO (ng/ml)	82.93 (21.35,452.61)	64.04 (25.62,427.96)	60.70 (24.67,412.05)	121.80 (38.07,427.96)	0.232
Lactates(mmol/L)	3.11 (2.57,3.70)	1.92 (1.56, 2.30)	1.27 (0.99, 1.89)	2.32 (1.76, 2.90)	< .001

Normally distributed data are presented as the mean ± SD; non-normally distributed data are presented as median (IQR), and categorical variables are presented as *n* (%). *P* values were calculated based on *t* test or Mann–Whitney *U* test for continuous variables, and chi-square test or Fisher's exact test for categorical variables

PP: pulse pressure; LVEF: left ventricular ejection fraction; Cr: creatinine; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoproteincholesterol; LDL-C: low-density lipoprotein cholesterol; DBIL: direct bilirubin; IBIL: indirect bilirubin; TBIL: total bilirubin; HS-TnT: hypersensitive troponin; CK–MB: creatine kinase–MB; MYO: myohemoglobin

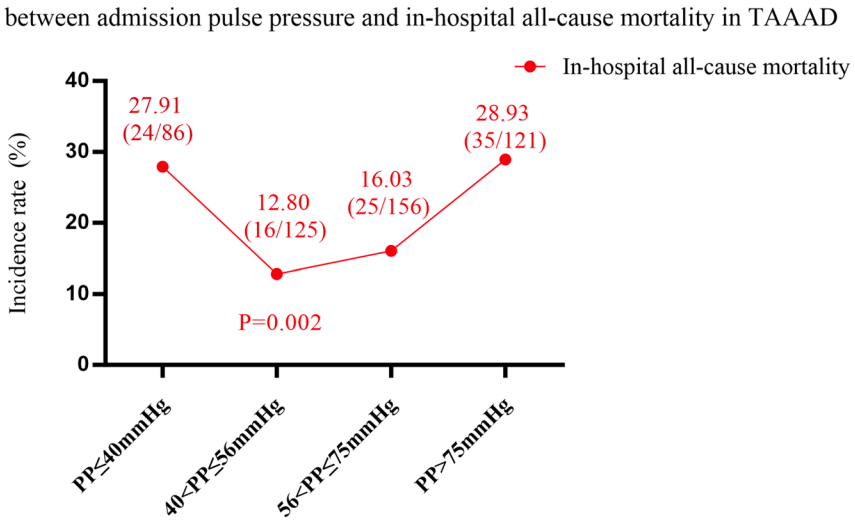

Fig. 2 Relationship between admission pulse pressure and in-hospital all-cause mortality in TAAAD. PP and in-hospital all-cause mortality showed a "J-curve" correlation, with higher rates in the significantly elevated group and reduced group. PP: pulse pressure; TAAAD: type A acute aortic dissection

Table 2 Treatment modalities and complications

	PP < 40 mmHg	40 < PP < 56 mmHg	56 < PP < 75 mmHg	PP > 75 mmHg	*P* value
Treatment					
Drug, *n* (%)	29 (33.72)	26 (20.80)	29 (18.59)	23 (19.01)	0.034
Emergent surgery, *n* (%)	57 (66.28)	99 (79.20)	127 (81.41)	98 (80.99)	0.033
Aortic occlusion (min)	114.58 ± 37.07	111.44 ± 35.19	108.42 ± 26.72	113.49 ± 35.45	0.699
CPB (min)	209.63 ± 60.99	206.25 ± 63.08	207.87 ± 49.54	215.35 ± 62.68	0.570
Complications					
Cerebral infarction, *n* (%)	9 (12.33)	10 (9.01)	21 (14.58)	26 (22.61)	0.032
Hepatic insufficiency, *n* (%)	4 (5.48)	11 (9.91)	15 (10.42)	21 (18.42)	0.039
Renal insufficiency, *n* (%)	9 (12.33)	15 (12.00)	25 (16.03)	31 (27.19)	0.020
In-hospital all-cause mortality, *n* (%)	24 (27.91)	16 (12.80)	25 (16.03)	35 (28.93)	0.002
The length of hospital stay(day)	51.00 (46.00,61.00)	41.00 (38.00,46.00)	38.00 (33.00,45.00)	46.00 (43.00,53.00)	< .001

Normally distributed data are presented as the mean ± SD; non-normally distributed data are presented as median (IQR), and categorical variables are presented as *n* (%). *P* values were calculated based on *t* test or Mann–Whitney *U* test for continuous variables, and chi-square test or Fisher's exact test for categorical variables

PP: pulse pressure; CPB: cardiopulmonary bypass

(Ideal line), indicating good calibration of the predictive model. In addition, internal validation of the predictive model using bootstrap resampling showed that the corrected curve (Bias-corrected line) almost overlapped with the diagonal, suggesting good accuracy of the nomogram model (Fig. 5). Finally, a DCA curve was drawn based on the constructed CPM. The horizontal axis represented the risk threshold, while the vertical axis showed the net benefit (NB) after accounting for benefits and harms. The DCA results indicated that the risk assessment model provided net benefit for patients within a threshold range of 0.01–0.72 (Fig. 6).

Discussion

Systolic and diastolic blood pressure represent the two extremes of blood pressure fluctuations, and the difference between systolic and diastolic blood pressure is defined as pulse pressure (PP), which reflects the magnitude of this fluctuation in a cardiac cycle. In different patient populations, wide PP has been associated with adverse cardiovascular events and all-cause mortality [5–8]. Low admission PP is an independent predictor of mortality in patients with acute coronary syndrome[9]. However, few studies have reported on the relationship

Table 3 Univariate logistic analysis for in-hospital all-cause mortality

	OR value	95% Confidence interval	*P* value
Demographic information and medical history			
Age	1.03	1.01–1.05	0.012
Age > 60 years	2.03	1.27–3.25	0.003
Atherosclerosis	1.77	1.14–2.76	0.011
Clinical manifestations			
PP	1.27	1.04–1.55	0.022
Syncope	2.74	1.43–5.26	0.002
Myocardial ischemia	4.16	2.51–6.90	< 0.001
Heart failure	1.90	0.92–3.91	0.082
Ultrasound and imaging examination			
LVEF	0.96	0.92–0.99	0.042
Limb ischemia	1.95	1.11–3.42	0.019
Bicuspid aortic valve	2.64	1.11–3.42	0.028
Ascending aorta diameter	1.22	1.02–1.47	0.03
Coronary artery involved	2.19	1.15–4.20	0.018
Abdominal visceral vessels involved	1.81	1.14–2.88	0.012
Three branch vessels involved	1.51	0.95–2.42	0.084
Iliac vessel involvement	1.56	1.01–2.43	0.047
Laboratory tests			
D-dimer	1.07	1.04–1.10	< 0.001
Cr	1.01	1.00–1.01	< 0.001
TG	1.41	1.19–1.67	< 0.001
IBIL	1.02	1.00–1.04	0.096
TT	1.00	1.00–1.00	0.017
CKMB	1.01	1.00–1.02	0.018
Lactates	1.28	1.16–1.42	< 0.001
Treatment modalities and complications			
Emergent surgery	0.15	0.09–0.25	< 0.001
Cerebral infarction	5.25	2.96–9.29	< 0.001
Hepatic insufficiency	5.13	2.75–9.55	< 0.001
Renal insufficiency	10.39	5.93–18.20	< 0.001

Study variables were screened using univariate analysis

OR value: odds ratio value; PP: pulse pressure; LVEF: left ventricular ejection fraction; Cr: creatinine; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoproteincholesterol; LDL-C: low-density lipoprotein cholesterol; DBIL: direct bilirubin; IBIL: indirect bilirubin; TBIL: total bilirubin; HS-TnT: hypersensitive troponin; TT: thrombin time; CK–MB: creatine kinase–MB; MYO: myohemoglobi

between admission PP and adverse events during hospitalization in patients with TAAAD.

In this study, we retrospectively analyzed the clinical data of 488 patients with TAAAD and there was a "J-shape" relationship between admission PP and in-hospital all-cause mortality. The mortality in the normal group ($40 < PP \leq 56$ mmHg) was the lowest (12.80%), and in the elevated group ($56 < PP \leq 75$ mmHg, $PP > 75$ mmHg) the mortality increased with the increase of admission PP, which was 16.03% and 28.93%, respectively. When the PP value was lower than normal ($PP \leq 40$ mmHg), the mortality increased inversely (27.91%).

The "J-curve" phenomenon between blood pressure and cardiovascular outcomes is mainly observed in diastolic blood pressure[10]. Interestingly, Stewart et al. reported for the first time that the relationship between DBP and myocardial infarction exists between DBP and myocardial infarction in patients with severe hypertension with a DBP of < 90 mmHg who receive antihypertensive treatment[11]. The study found that the incidence of myocardial infarction was more than five times higher in those whose diastolic blood pressure was reduced to less than 90 mmHg than in those whose diastolic blood pressure was in the range of 100–109 mmHg. This phenomenon has also been confirmed in studies published by

Table 4 Multivariate logistic analysis for in-hospital all-cause mortality

Variables	β	S.E	Z	P	OR (95%CI)
Intercept	−3.44	0.77	−4.48	< .001	0.03 (0.01–0.14)
Ascending aortic diameter	0.26	0.12	2.08	0.037	1.29 (1.02–1.64)
D-dimer	0.08	0.02	4.83	< .001	1.08 (1.05–1.12)
Pulse Pressure Rating					
1					1.00 (Reference)
2	0.87	0.43	2.02	0.043	2.39 (1.03–5.54)
3	0.52	0.39	1.32	0.187	1.68 (0.78–3.63)
4	1.29	0.39	3.32	< .001	3.63 (1.70–7.78)
Coronary atherosclerosis					
0					1.00 (Reference)
1	0.80	0.27	2.96	0.003	2.22 (1.31–3.75)
Emergent surgery					
0					1.00 (Reference)
1	−2.06	0.28	−7.25	< .001	0.13 (0.07–0.22)
Abdominal visceral vessels involved					
0					1.00 (Reference)
1	0.68	0.29	2.38	0.017	1.98 (1.13–3.48)

A multivariate binary logistic regression model was applied, using the backward stepwise method to introduce variables with a *P* value of less than 0.10 from the univariate analysis

S.E.: standard error; OR: odds ratio

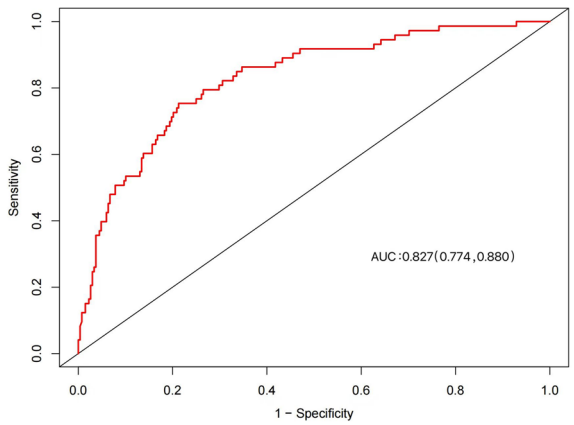

Fig. 3 ROC curve of the model for predicting in-hospital all-cause mortality in TAAAD. After plotting the ROC curve, and the AUC of the curve was 0.827 (95% CI 0.774–0.880, $P < 0.001$). ROC: receiver operator characteristic curve; AUC: area under the curve

Cruickshank and Farnett et al. [12, 13]. All these studies describe an interesting phenomenon, i.e., the incidence of myocardial infarction is increased when diastolic blood pressure falls below a certain level during antihypertensive therapy. The reason for this phenomenon might be that lower diastolic blood pressure reduces myocardial perfusion, which in turn increases the chance of myocardial infarction. Recent studies have found that systolic blood pressure < 130 mmHg or < 120 mmHg is also associated with cardiovascular outcomes and mortality in coronary artery disease, hypertension, or diabetes mellitus in a "J-curve" fashion. McEvoy et al.[14] further showed that in adults with systolic BP ≥ 120 mmHg, elevated PP and low diastolic BP were associated with subclinical myocardial injury and adverse coronary events. When antihypertensive therapy is administered to a systolic BP of less than 140 mmHg, care should be taken to ensure that diastolic BP levels do not fall below 70 mmHg, and in particular do not fall below 60 mmHg.

As mentioned earlier, Eduardo et al. [1] found a significant "J-curve" relationship between admission systolic blood pressure and in-hospital mortality in patients with TAAAD. This study was the first to extend this specific paradigm of association between blood pressure and cardiovascular disease to AAD. In the present study, we for the first time demonstrated the "J-shaped" association between admission PP and in-hospital all-cause mortality in TAAAD. The reason for this "J-shaped" association might be related to the high "co-morbidity" rate in the group with significantly elevated PP and the group with reduced PP. In the group with significantly elevated PP, the proportions of involvement of three branches of the arch, abdominal arteries, and iliac arteries were significantly higher than those of the other three groups, and the proportions of cerebral infarctions, hepatic insufficiency, and renal insufficiency were also the highest in the group with significantly elevated PP. These

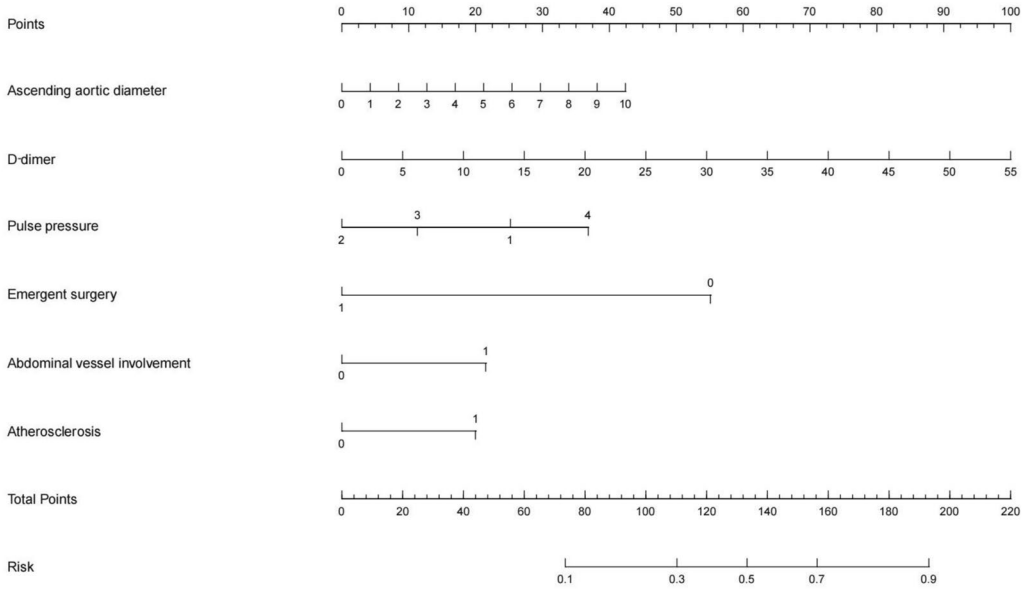

Fig. 4 Nomogram for predicting in-hospital all-cause mortality in TAAAD. Column line plots were drawn to visualize the results of the prediction model. A total of six predictor variables were involved in the composition of the column line plot. Based on the level of influence of each predictor variable in the model, i.e., the magnitude of the regression coefficients, a score was assigned to the level of value of each variable, thus obtaining six individual scores, which were then summed up to obtain the total score. TAAAD: type A acute aortic dissection

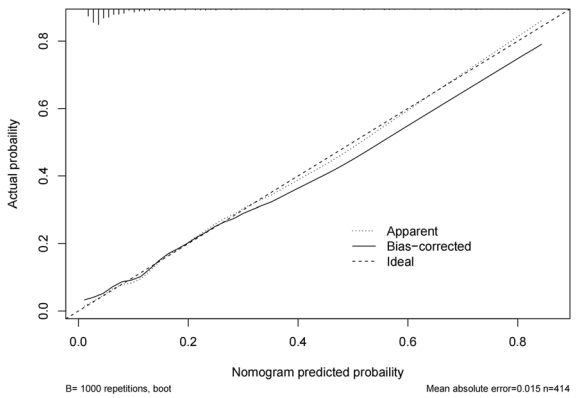

Fig. 5 Calibration curve of the model for predicting in-hospital all-cause mortality in TAAAD. The calibration plot showed that the calibration curve (Apparent line) closely matched the diagonal (Ideal line), indicating good calibration of the predictive model. In addition, internal validation of the predictive model using bootstrap resampling showed that the corrected curve (Bias-corrected line) almost overlapped with the diagonal, suggesting good accuracy of the nomogram model

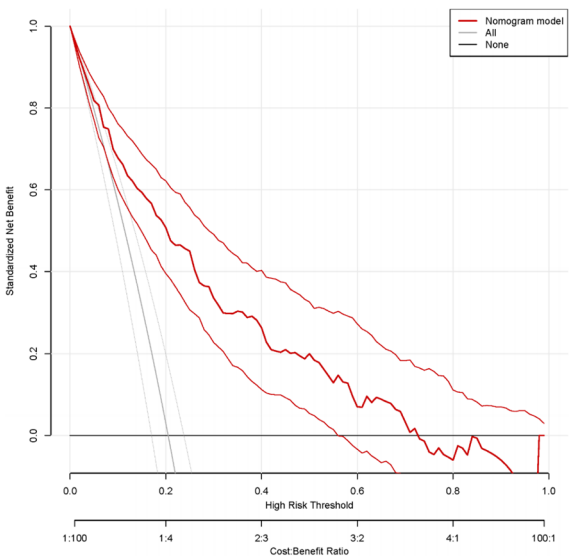

Fig. 6 Clinical decision curve of the model for predicting in-hospital all-cause mortality in TAAAD. A DCA curve was drawn based on the constructed CPM. The horizontal axis represented the risk threshold, while the vertical axis showed the net benefit (NB) after accounting for benefits and harms. The DCA results indicated that the risk assessment model provided net benefit for patients within a threshold range of 0.01–0.72

complications, which were closely related to AAD, might led to fatal events. In the reduced group, the proportions of TAAAD patients with myocardial infarction/ischemia, pericardial effusion, hypotension, shock and cardiac tamponade were significantly higher than those in the other three groups. In addition to this, the reduced group had the lowest percentage of patients undergoing surgical treatment, which might also be an important rationale for its high mortality rate. After adjusting for confounders, multivariate binary logistic regression analysis showed

that significantly elevated PP and reduced PP were independent risk factors for in-hospital all-cause mortality in patients with TAAAD.

In the pharmacological treatment of aortic dissection, the current guideline recommendation is to control systolic blood pressure at 100–120 mmHg and heart rate at 60–80 beats/min, which does not sufficiently emphasize PP. This study found a "J-shaped" relationship between PP and the risk of in-hospital all-cause death in patients with TAAAD, and suggested that a PP > 75 mmHg or PP < 40mmHg is an independent risk factor for in-hospital death. Therefore, we suggested that appropriate antihypertensive medications should be chosen to lower PP while prescribing pharmacological treatment for aortic dissection. However, the traditional evaluation of effect of antihypertensive drugs relies on the detection of systolic as well as diastolic blood pressure, mainly systolic. In addition, studies on the effects of different antihypertensive drugs on PP are extremely limited[15]. Further prospective studies are needed to determine whether PP could be used as a determining factor in the selection of antihypertensive drugs or as an indicator of the effectiveness of treatment.

Currently, there is no sufficient evidence on whether PP elevation increases the incidence of aortic dissection. PP increase might cause intimal damage and elastic fiber rupture, the initial event of aortic dissection. In the present study, the proportion of three-branch vessel involvement in the arch, abdominal vessel involvement, and iliac vessel involvement was significantly higher in the group with significantly elevated PP than in the other three groups. The group with significantly higher PP also had the highest rates of cerebral infarction, hepatic insufficiency, and renal insufficiency during hospitalization. The higher the PP, the more pronounced the pressure fluctuation, the greater the impact and damage to the artery wall, which could lead to an increase in the size and extent of the entrapment tear. Aortic branches could become poorly perfused due to the entrapment, which in turn affects the blood supply to the organs, leading to organ insufficiency and ultimately increasing the incidence of early fatal events [16–18]. The reduction in PP may serve as a significant clinical indicator closely associated with myocardial dysfunction and pericardial tamponade, reflecting the hemodynamic compromise in these pathological conditions. In support of this hypothesis, syncope, myocardial ischemia/infarction, and pericardial effusion were found to be more common in patients with reduced PP in this study.

The AUC of the ROC curve constructed in this study was 0.827 (95% CI 0.774–0.880, $P < 0.001$) > 0.75, which indicated that the prediction model had a relatively good discriminatory degree. The result of the goodness-of-fit test was $\chi = 2.285$, $P = 0.971 > 0.05$, which demonstrated that the model had a relatively good calibration.

Limitations

This study has some limitations. First, this study was a retrospective single-center study with inherent limitations of study design. Thus large, multicenter and prospective cohort studies are needed to validate our results and conclusions. Second, the PP values analyzed in this study were measured at a specific timepoint of admission to the hospital, whereas the PP could change dynamically. Third, some patients died before arriving at the hospital, thus altering the mortality rate of patients with very low/high PP. However, collecting prehospital data in China currently still presents significant challenges and future research is needed to explore this aspect. Fourth, it should be specifically analyzed for patients who received emergency surgery whether the pulse pressure could predict the postoperative mortality rate of patients, which might yield very meaningful results. After excluding the patients who did not undergo surgery, the sample size decreased to some degree in this study. Therefore, this part of the results should be confirmed by larger sample studies or multicenter studies in the future. Fifth, the research results cannot show the association between PP and risk factors, nor have they unraveled the underlying mechanism between PP and the mortality of patients with TAAAD. Sixth, due to the limitations and practical constraints in Chinese emergency medical practice, the majority of patients underwent non-invasive cuff measurements for bilateral upper limb blood pressure. Recognizing that non-invasive blood pressure measurements are less precise and susceptible to various influencing factors, and considering that most patients exhibited minimal differences between bilateral measurements, emergency department typically recorded only the higher value. Consequently, the original bilateral measurements for these patients are unfortunately unavailable for retrospective analysis. For the subset of patients who demonstrated significant differences in bilateral non-invasive blood pressure measurements, both values were documented, and we utilized the higher reading for our analysis. Seventh, as a regional medical center, our cohort primarily included patients transferred after initial stabilization, resulting in a lower proportion of hemodynamically unstable cases at admission (e.g., only ten patients [2.05%] developed shock during hospitalization). While sensitivity analyses excluding these patients yielded consistent results, we retained them as shock events occurred during treatment phases (not at admission), reflecting TAAAD's dynamic course. Nonetheless, this selection bias may limit generalizability to populations with acute instability.

Conclusions

Our results revealed a "J-curve" correlation between admission pulse pressure (PP) and in-hospital all-cause mortality in patients with type A acute aortic dissection (TAAAD). Elevated and reduced admission PP, along with D-dimer levels, ascending aortic diameter, and coronary atherosclerosis, were identified as independent risk factors for in-hospital all-cause mortality in TAAAD patients. In addition, emergent surgery was found to be a protective factor. Large-scale, multicenter, and prospective cohort studies are required to confirm our findings and conclusions in the future.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s40001-025-02475-w.

Additional file 1.

Acknowledgements
None.

Author contributions
Y.L., L. W. and S. Z. wrote the manuscript and performed the statistical analysis. J. L., Y. C. and Y. Y prepared the figures and tables. X. M. and H. Z designed the study and reviewed the manuscript."

Funding
This work was supported by the grants from Young Experts of Taishan Scholar Program of Shandong Province (grant no. tsqn202408359), the National Natural Science Foundation of China (81800255; 82201624), the Nature Science Foundation of Shandong Province (ZR2021MH112; ZR2021QH016; ZR2023MH124; ZR2021QB122) and Jinan Science and Technology Plan Project (202225050).

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

Competing interests
The authors declare no competing interests.

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Received: 16 June 2024 Accepted: 18 March 2025
Published online: 26 March 2025

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