Predicting in-hospital death in patients with type B acute aortic dissection

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Abstract

The outcome of patients with acute type B aortic dissection (BAAD) is largely dictated by whether or not the case is "complicated." The purpose of this study was to investigate the risk factors leading to in-hospital death among patients with BAAD and then to develop a predictive model to estimate individual risk of in-hospital death.

A total of 188 patients with BAAD were enrolled. Risk factors for in-hospital death were investigated with univariate and multivariable logistic regression analysis. Significant risk factors were used to develop a predictive model.

The in-hospital mortality rate was 9% (17 of 188 patients). Univariate analysis revealed 7 risk factors to be statistically significant predictors of in-hospital death (P < .1). In multivariable analysis, the following variables at admission were independently associated with increased in-hospital mortality: hypotension (odds ratio [OR], 4.85; 95% confidence interval [CI], 1.12–18.90; P = .04), ischemic complications (OR, 8.24; 95% CI, 1.25–33.85; P < .001), renal dysfunction (OR, 12.32; 95% CI, 10.63–76.66; P < .001), and neutrophil percentage ≥80% (OR, 5.76; 95% CI, 2.58–12.56; P = .03). Based on these multivariable results, a reliable and simple prediction model was developed, a total score of 4 offered the best point value.

Independent risk factors associated with in-hospital death can be predicted in BAAD patients. The prediction model could be used to identify the prognosis for BAAD patients and assist physicians in their choice of management.

Abbreviations: AAD = acute a ortic dissection, BAAD = acute type B aortic dissection, CI = confidence interval, ESR = erythrocyte sedimentation rate, OR = odds ratio, TEVAR = thoracic endovascular aortic repair.

Keywords: acute, aortic dissection, in-hospital death, predicting, type B

1. Introduction

Acute type B aortic dissection (BAAD) comprises about one-third of all acute aortic dissection (AAD) cases. Although this catastrophic disease was first described in the medical literature over 2 centuries ago, progress on the optimal diagnostic and treatment modalities for BAAD was slow to evolve throughout the latter half of the twentieth century, even as newer diagnostic techniques and management strategies became commonplace. The morbidity and mortality of BAAD are strongly related to the clinical features.^[1,2] Medical treatment is the preferred therapy in all uncomplicated BAAD patients. Surgical and endovascular approaches are reserved for patients with complications such as

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shock, hypotension, limb ischemia, and organ malperfusion. However, these complications are still associated with high mortality rates between 20% and 30% for surgery and 10% and 20% for endovascular repair in the acute setting.^[3–5]

Because of the various clinical features of BAAD, The prognosis of each patient with BAAD remain unpredictable and different.^[6–8] Therefore, more insights into the early prognosis of BAAD based on clinical presentations are needed to optimize treatment strategies and inform patients and their family. However, most studies have focused their attention only on the risk factors without using specific scores.^[1,6,7,9] We sought to provide a simple risk model that could give us better insight into this condition and predict the mortality in patients with BAAD.

2. Methods

2.1. Patient selection

A total of 188 patients with BAAD were enrolled between November 1, 2013 and October 30, 2016 in the second people hospital of He Fei and the First Affiliated Hospital of Anhui Medical University. Aortic dissection was classified according to the Stanford classification. A dissection was considered as an AAD if the time from the onset of the symptoms was within 14 days.^[10] The diagnosis of BAAD was confirmed by symptoms, physical examination, transthoracic echocardiography, or further confirmed by findings from enhanced computed tomography or magnetic resonance imaging studies or visualization at surgery. To minimize bias, patients who had traumatic dissection and those with infectious diseases or chronic kidney disease were

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excluded. This study was approved by the ethic committee of the Second People's Hospital of Hefei and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki. Informed consent was waived by the committee because of the retrospective nature of the study.

2.2. Data collection and measures

Standardized data forms were used to collect data on patient's age, sex, medical history of hypertension, clinical presentations, physical findings, laboratory examination, adverse events, management (medical or surgical), and in-hospital outcomes. Definitions of the following clinical criteria included: hypertension-blood pressure \geq 140/90 mm Hg or the use of antihypertensive agents; hypotension – systolic blood pressure $\leq 100 \text{ mm Hg}$. Ischemic complications were defined as pulse deficit, cerebral ischemia, mesenteric ischemia/infarction, and limb ischemia. Glomerular filtration rate <60 mL/min was defined as renal dysfunction.

2.3. Statistical analysis

The study comprised 2 groups: survived and died. Quantitative variables were presented as mean±standard deviation and categorical variables as percentages. Continuous variables were compared using the t test and categorical data were compared using the Fisher exact or Chi-square tests. Denominators represent only reported cases because missing data were not defaulted to negative. Variables with a marginal association with mortality (P < .10) were entered in stepwise multivariable logistic regression model for in-hospital mortality. Multivariate binary

logistic regression analyses (backward-LR method) were performed to identify the predictors of in-hospital mortality. The odds ratio (OR) and the 95% confidence interval (CI) were also calculated. A value of P < .05 was considered significant. Data analysis was performed with the use of SPSS 19.0 statistical analysis software.

2.4. Development of a risk prediction tool

The variables that were significantly associated with in-hospital mortality in the multivariable analyses were assigned a score equal to their coefficients in the model (P < .05). Each patient would have a sum of score. According to the known prognosis of patients, the sensitivity and specificity of every score to predict inhospital death were evaluated. The appropriate point could then be used to predict in-hospital death. A simple prediction tool would have a threshold value with sensitivity and specificity in predicting in-hospital mortality for patients with BAAD.

3. Results

3.1. Patient characteristics

Among the 188 patients with BAAD, 171 (91%) patients survived and 17 (9%) patients died during hospitalization. The mean age of overall patients was 57.7 ± 12.6 years, with a majority being males (79.1%). About 61.2% of the patients had a medical history of hypertension. Medical therapy was received by 39.9% of the patients compared with 60.1% of patients who received surgery or thoracic endovascular aortic repair (TEVAR) therapy. A small number of patients with BAAD had associated

Table 1

Variable	Overall (n=188)	Survived (n = 171)	Died (n = 17)	Р
Demographics and medical history				
Age, yr	57.7 ± 12.6	57.4±12.3	64.0 ± 14.3	.09
Age ≥65 yr (%)	60 (31.9)	52 (30.4)	8 (47)	<u>.09</u> <u>.06</u> <u>.58</u> .24
Female (%)	43 (22.9)	40 (23.4)	3 (17.6)	.58
Medical history of hypertension (%)	115 (61.2)	105 (61.4)	10 (58.8)	.24
Physical examination findings				
Heart rate, bpm	84.1±13.9	83.4±12.3	87.3±16.6	.64
Heart rate ≥100 bpm (%)	20 (10.6)	17 (10)	3 (17.6)	.57
Mean SBP, mm Hg	144.4±30.9	149.2±29.6	126.7 ± 33.9	<.05
Mean DBP, mm Hg	85.4±19.8	88±19.5	71 ± 16.2	<.05
SBP \leq 100 mm Hg (%)	16 (8.5)	10 (5.8)	6 (35.3)	<.001
Laboratory examinations				
TC, mmol/L	4.3 ± 1.0	1.9 ± 1.6	1.3 ± 0.8	.26
TG, mmol/L	1.3 ± 1.4	1.3 ± 0.8	1.5 ± 1.4	.53
Neutrophil percentage (%)	76.2±8.2	77.1±11.5	83.8±8.27	<.05
Neutrophil percentage ≥80% (%)	93 (49.4)	79 (46.2)	14 (82.3)	<.001
D-dimer, mg/L	8.53 ± 1.8	6.05 ± 1.0	15.6 ± 2.9	<.05
D-dimer \geq 10 mg/L (%)	58 (30.8)	49 (28.6)	9 (52.9)	<.05
C-reactive protein, mg/L	50.4±16.4	51.4±17.6	51.5 ± 11.9	.98
ESR, mm/h	31.2±9.4	30.6 ± 7.2	42.9 ± 11.3	.34
Abnormal ECG (%)	161 (85.5)	146 (85.4)	15 (88.2)	.69
Complications				
Ischemic complications (%)	16 (8.5)	11 (6.4)	5 (29.4)	<.005
Renal dysfunction (%)	34 (18.1)	22 (12.9)	12 (70.6)	<.001
Method of treatment				
Surgery (%)	113 (60.1)	111 (64.9)	2 (11.8)	<.001

AAD = acute aortic dissection, DBP = diastolic blood pressure, ECG = electrocardiogram, ESR = erythrocyte sedimentation rate, SBP = systolic blood pressure, TC = total cholesterol, TG = triglyceride.

Model variables	Coefficient	Wald	Р	OR (95% CI)
Hypotension	1.644	3.57	.04	4.85 (1.124–18.906)
Ischemic complications	2.533	11.54	<.001	8.24 (1.25-33.85)
Renal dysfunction	3.899	14.31	<.001	12.32 (10.628–76.66)
Neutrophil percentage ≥80%	1.685	5.45	.03	5.76 (2.582–12.564)
Surgery	-2.361	10.29	<.001	0.07 (0.017-0.302)

Table 2

Multivariate	loaistic	rearession	for	prediction of	of death.
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CI = confidence interval, OR = odds ratio.

renal dysfunction (18.1%), ischemic complications (8.5%) (Table 1).

3.2. Univariate predictors of in-hospital death for patients with BAAD

Clinical characteristics that showed significant association with in-hospital mortality included systolic/diastolic blood pressure, neutrophil percentage, D-dimer, ischemic complications, and renal dysfunction. There was significant difference in the 2 groups for surgery or TEVAR and medical therapy (64.9% vs 11.8%, P < .05). However, there was no significant difference in age, sex, medical history of hypertension, heart rate, the blood levels of total cholesterol, triglyceride, C-reactive protein, and erythrocyte sedimentation rate. At the same time, the presence of abnormal electrocardiogram was similar in the 2 groups of patients (P > .05) (Table 1).

3.3. Multivariate predictors for in-hospital death in patients with BAAD

In multivariate logistic regression analysis, the following variables at admission were independently associated with increased in-hospital mortality: hypotension (OR, 4.85; 95% CI, 1.12–19.91; P=.04), ischemic complications (OR, 8.24; 95% CI, 1.25–33.85; P<.001), renal dysfunction (OR, 12.32; 95% CI, 10.63–76.66; P<.001), and neutrophil percentage ≥80% (OR, 5.76; 95% CI, 2.58–12.56; P=.03) (Table 2).

3.4. Scores of the variables and the risk prediction model

Based on these multivariable regression results, a simple bedside risk prediction tool was developed. Every corresponding assigned score was equal to the coefficient of each variable. Renal dysfunction was given the highest score (3.9) and hypotension was given the lowest score (1.6), with other variable scores in between these values. The total score for each patient with BAAD was included in the simple risk prediction model. The independent risk variables and their allocated scores were listed in Table 3. The scores for almost all the deaths (93%) were ≥ 2 ,

Table 3

Score assigned in the predictive model.

	Assigned	
Predictive factors	score	Definition
Hypotension	1.6	Systolic blood pressure \leq 100 mm Hg
Ischemic complications	2.5	Pulse deficit, cerebral/limb/mesenteric ischemia
Renal dysfunction	3.9	Glomerular filtration rate \leq 60 mL/min
Neutrophil percentage	1.7	Neutrophil percentage ≥80%

and almost all the survivors (98%) had scores <8. The score of 4 was the best point for the risk prediction model, with appropriate sensitivity (86%) and specificity (78%) (Table 4).

4. Discussion

Despite improvements in diagnosis, management, and surgical or endovascular repair techniques, BAAD remains a catastrophic event that still carries a high in-hospital mortality rate. Similarly, the international registry of acute aortic dissection (IRAD) reported in-hospital mortality rate of 10.6% in 1034 patients between 1996 and 2013,^[11] and a recent Japan nationwide survey of 250 patients with type B AAD between January 2003 and August 2011 found a hospital mortality rate of 10%.^[9] But, the IRAD reported in-hospital mortality rate of 13% in the past 20 years.^[12] In this analysis of 188 consecutive patients during the period from November 2013 to October 2016, the overall hospital mortality rate was 9.0%, which was lower than our previous studies.^[13] In general, there is no much difference in inhospital mortality in these studies.

By univariate and multivariate analysis, the present study showed several clinical variables independently associated with death during in-hospital: hypotension (OR, 4.85; 95% CI, 1.12– 19.91; P=.04), ischemic complications (OR, 8.24; 95% CI, 1.25–33.85; P<.001), renal dysfunction (OR, 12.32; 95% CI, 10.63–76.66; P<.001), and neutrophil percentage ≥80% (OR, 5.76; 95% CI, 2.58–12.56; P=.03). The similarities of variables predicting in-hospital death in other prior reports and the present study support the clinical relevancy of the current predictive model that can be used to make a decision regarding management and patient counseling.

A more recent publication including 1035 patients with BAAD identified the following as predictors of in-hospital mortality in the IRAD: mesenteric ischemia (OR, 9.03), hypotension/shock (OR, 6.43), descending diameter \geq 5.5 cm (OR, 6.04), renal failure (OR, 3.61), periaortic hematoma (OR, 3.06), acute limb ischemia (OR, 3.02), and age (OR, 1.03). Based on these multivariable results, a reliable and simple bedside risk prediction tool was developed by IRAD.^[11] Suzuki et al analyzed 384 patients with BAAD enrolled in the IRAD, the total in-hospital

Table 4 The risk prediction model.				
≥2	93	61	<.01	
≥4	86	78	<.01	
≥6	62	91	<.01	
≥8	34	98	<.01	

mortality rate was 13% with most deaths occurring within the first week, independent predictors of in-hospital mortality were hypotension/shock (OR, 23.8, P < .0001), absence of chest/back pain (OR, 3.5, P = .01), and branch vessel involvement (OR, 2.9, P = .02).^[14] The most common causes of hypotension are AAD rupture, hypotension may lead to the complications of organ/ tissue malperfusion, syncope and acute renal dysfunction. Meanwhile, recent study shows that AAD patients with hypotension may have longer in-hospital stay and higher mortality rate.^[15]

The present study confirmed that acute renal dysfunction was an independent risk factor for in-hospital death and it had the highest score in multivariate analysis. Occurrence of acute renal dysfunction is often associated with BAAD. The possible reasons for acute renal dysfunction might be the contrast medium used in imaging examination, aggressive blood pressure lowering, and the BAAD teared to renal artery branch. A recent study by Mitsuoka et al, which enrolled 250 patients in Japan, demonstrated that renal dysfunction is a predictor for in-hospital mortality with BAAD (OR, 2.782, P = .037).^[9] Patel et al enrolled 3018 patients in IRAD, 348 (11.5%) presented with acute renal dysfunction, acute renal dysfunction is associated with an increased risk for in-hospital death and persistence of renal dysfunction at midterm follow-up in type B but not type A AAD. Despite its early implications, presentation with renal dysfunction is not associated with late mortality after AAD.^[16] Nardi et al analyzed 214 patients who underwent AAAD surgery, patients with postoperative malperfusion have a higher mortality rate, independent predictors for in-hospital mortality and reduced long-term survival included renal postoperative malperfusion.^[17] In the present study, we have not studied the relationship between acute renal dysfunction and prognosis for BAAD in the medium and long term. At the same time, the study found that the percentage of neutrophils ($\geq 80\%$) was not only an inflammatory marker but also predicted poorer prognosis for BAAD patients (OR, 5.76; 95% CI, 2.58–12.56; P=.03). The count of white blood cell (WBC) and neutrophils could indicate the severity of the inflammatory reaction. Recent studies have shown that AAD is associated with systemic inflammation and fibrinolytic. D-dimer and inflammatory markers such as WBC and C-reactive protein are known to increase in the acute phase and are independently associated with in-hospital death in patients with AAD.^[18,19] In addition, a large number of studies have demonstrated the high neutrophil to lymphocyte ratio is an independent predictor of in-hospital mortality for type A AAD.^[20,21] Zhao et al indicate that elevated WBC count upon admission might be used as a predictor for increased risk of inhospital death in uncomplicated BAAD. WBC count was a significant predictor for in-hospital death as both a continuous variable and a categorical variable using a cut off of 11.0×10^9 /L (hazard ratio, 2.056, 95% CI: 1.673–5.253, P=.034). However, there was no relationship observed between WBC count on admission and long term death.^[22] In this study, not D-dimer but WBC was the independent risk factors for in-hospital death in patients with BAAD by the multivariate analysis. However, each study has different cut-off level of the D-dimer and WBC. The present study used 10 mg/L as the cut-off point of D-dimer and 80% as the point of percentage of neutrophils. If the critical cutoff level of variables was changed, there may get a different result.

TEVAR treatment has emerged over the past decade and now exceeds open surgery as the preferred treatment modality for most of these cases. In this study, surgery or TEVAR therapy was identified as a predictive factor (OR=0.07, P<.001). This has been confirmed by previous studies, which have demonstrated that emergency TEVAR has dramatically improved the outcome of BAAD, especially in patients with complications.^[23] However, previous study demonstrated that in-hospital mortality was significantly higher after open surgery than after endovascular treatment.^[24] This study did not distinguish TEVAR treatment and surgery. Ahmad et al indicate that endovascular treatment was associated with better early, midterm and long-term outcomes in terms of mortality and associated complications than open surgery and medicine.^[4]

Similarly, the INSTEAD-XL trial showed a 5-year all-cause mortality benefit with TEVAR compared to optimal medical management alone, driven primarily by aorta-related mortality.^[25] It appears that TEVAR therapy can modify the natural history of aortic disease without carrying an unacceptably higher procedure-related mortality risk in complicated type B dissection. The impact of TEVAR on uncomplicated type B dissection remains to be investigated. IRAD data suggests that TEVAR therapy may be a promising therapy for appropriately selected patients.^[26]

Finally, the present study developed a simple risk prediction model that is relatively accurate in predicting the risk of death in patients with BAAD. The model should help physicians and patients to estimate the risk of in-hospital death with percentage and quickly decide to optimize treatment strategies. Furthermore, the prediction model should be useful in evaluating the effects of new diagnostic and treatment methods for patients with BAAD.

4.1. Limitations of the study

This retrospective study was performed at 2 centers, thus posing a risk for possible patient selection bias. Although the number of patients enrolled in this study was not low, it might have been underpowered in identifying other predictive factors. The analyses of patient outcomes were based on the results from the initial admission. Finally, the end points of the study are restricted to the in-hospital period, as full access to the follow-up clinical data after discharge was not available; thus, longer-term results are not available.

5. Conclusions

The results of the present study show multiple variables as predictors of in-hospital death for BAAD. Furthermore, it develops a useful and simple prediction model that could be used in the prognosis and to quickly determine the treatment strategies for patients with BAAD.

Author contributions

Conceptualization: Jing Zhang, Ziping Cheng.

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