Impact of high-risk features on outcome of acute type B aortic dissection

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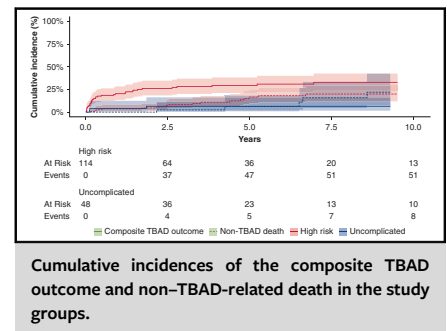
ABSTRACT

Background: Acute type B aortic dissection (TBAD) is a severe condition associated with significant morbidity and mortality. The optimal classification and treatment strategy of TBAD remain controversial and inconsistent.

Methods: This analysis includes patients treated for acute TBAD at the Helsinki University Hospital, Finland between 2007 and 2019. The endpoints were early and late mortality, intervention of the aorta, and a composite of death and aortic intervention in uncomplicated patients and high-risk patients.

Results: This study included 162 consecutive TBAD patients (27.8% females), 114 in the high-risk group and 48 in the uncomplicated group, with a mean age of 67.6 ± 13.9 years. Intramural hematoma was reported in 63 cases (38.9%). The mean follow-up was 5.1 ± 3.9 years. In-hospital/30-day mortality (n = 4; 3.5%) occurred solely in the high-risk group (P = .32). Additionally, TBAD-related adverse events (n = 23; 20.2%) were observed only in the high-risk group (P < .001). The cumulative incidences of the composite TBAD outcome with non–TBAD-related death as a competing risk were 6.6% (95% CI, 1.7%-16.5%) in the uncomplicated group and 29.5% (95% CI, 21.1%-38.3%) in the high-risk group at 5 years and 6.6% (95% CI, 1.7%-16.5%) and 33.0% (95% CI, 23.7%-42.6%) at 10 years (P = .001, Gray test). Extracardiac arteriopathy (subdistribution hazard ratio [SHR], 2.61; 95% CI, 1.08-6.27) and coronary artery disease (SHR, 2.24; 95% CI, 1.07-4.71) were risk factors for adverse aortic-related events in univariable competing-risk regression analysis.

Conclusions: Recognition of risk factors underlying adverse events related to TBAD is essential because the disease progression impacts both early and late outcomes. Early aortic repair in high-risk TBAD may reduce long-term morbidity and mortality. (JTCVS Open 2023;13:20-31)

Cumulative incidences of the composite TBAD outcome and non–TBAD-related death in the study groups.

CENTRAL MESSAGE

Disease progression in patients with high-risk type B aortic dissection (TBAD) impacts both early and late outcomes. Recognition of risk factors of TBAD may prompt early subacute interventions.

PERSPECTIVE

Acute type B aortic dissection (TBAD) is a severe condition associated with significant early and late morbidity and mortality. Traditionally TBAD patients are classified as complicated and uncomplicated patients. A new classification of high-risk TBAD patients recognizes risk factors that may prompt early subacute interventions.

Aortic dissection is an emergency condition, that in one-third of cases present as type B aortic dissection (TBAD) with an intimal tear sited distal to the left subclavian artery, directing blood flow to the true and false lumens.[1] Traditionally, TBAD is classified as uncomplicated or complicated dissection[2]; however, a new classification of TBAD includes uncomplicated, complicated, and high-risk groups.[3] The complicated group includes aortic wall rupture or malperfusion. High-risk TBAD is associated with such clinical features as refractory pain, hypertension, and/or hospital readmission. The presence of bloody pleural effusion, aortic diameter >40 mm, aortic false lumen diameter >22 mm, radiographic-only malperfusion, or entry tear

Abbreviations and Acronyms
HR = hazard ratio
ICU = intensive care unit
IMH = intramural hematoma
PAU = penetrating aortic ulcer
STS = Society of Thoracic Surgeons
SVS = Society for Vascular Surgery
TBAD = type B aortic dissection
TEVAR = thoracic endovascular aortic repair

in the lesser curve are features of high-risk TBAD as suggested by the recent reporting standards by the Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS).[3] In addition, an entry tear longer than 10 mm has been associated with poor late outcome.[4,5] A patent or partially thrombosed false lumen and age >60 years are other features to be noted and further assessed in high-risk patients.[6]

Although an invasive treatment strategy is indicated in patients with complicated TBAD, the potential benefit of early intervention in high-risk TBAD is controversial. We investigated this issue in the present institutional series.

METHODS

A total of 205 consecutive patients were treated for acute TBAD at the Helsinki University Hospital, Finland between January 2007 and December 2019. Forty-three patients with complicated TBAD, rupture, or malperfusion were excluded from this analysis. Thus, the present study comprised 162 TBAD patients, including 114 with high-risk TBAD and 48 with uncomplicated TBAD. The classification of TBAD was based on recent reporting standards of the SVS and STS.[3]

TBAD patients were classified as high risk if they had refractory arterial hypertension, visceral malperfusion on imaging, aortic diameter >40 mm in Ishimaru zones 3 to 8, an aortic false lumen diameter >22 mm, or an entry tear in the lesser curvature. The Helsinki University Hospital Institutional Review Board gave permission to conduct this study (HUS/144/2020; October 21, 2020). The Helsinki University Hospital is the largest tertiary care center in Finland, providing hospital care for more than 2.2 million residents. It is the only hospital providing care for patients with aortic diseases. Therefore, the present study is representative of all TBAD cases occurring in our hospital area. Index or local hospitals organized computed tomography follow-up and patients telephoned by index hospital.

Data were collected retrospectively into an electronic datasheet with prespecified variables and underwent review for completeness and consistency. Data on the dates and causes of death were retrieved from the Statistics Finland national registry. The most recent causes of death were classified as unknown when they were not available from this registry. TBAD-related deaths included cases in which the main cause of death was dissection or in which dissection was a contributing factor to the process. The primary endpoints of this analysis were early and late mortality. Early mortality included in-hospital and 30-day mortality, whereas late mortality was specified as death during follow-up. The secondary endpoints were any intervention on the aorta during follow-up and a composite endpoint defined as TBAD-related death or a TBAD-related intervention or any aortic reintervention during follow-up.

Statistical Analysis

Statistical analyses were performed using a SPSS version 26.0 (IBM) and R 4.2.2 (packages survminer, ggplot2, and tidycmprsk). Categorical data are reported as count and percentage, and continuous variables are reported as mean and standard deviation. Categorical data was evaluated using the Pearson χ^2 test or Fisher exact test. The Shapiro–Wilk test of normality was used to assess normality. Continuous variables were analyzed using the Student t test. Survival was estimated using Kaplan–Meier methodology with the log-rank test. Cox proportional hazards analysis with a backward stepwise method was used to identify risk factors affecting survival. Competing risk analysis with the Gray test was performed for TBAD-related adverse events, because non–TBAD-related death might be a confounding factor with traditional methods. Fine and Gray competing-risks regression was used to identify risk factors affecting TBAD-related adverse events. Adjusted risk estimates were reported as hazard ratio (HR) or subdistribution hazard ratio (SHR) with 95% confidence interval (CI). Statistical significance was set at $P < .05$.

RESULTS

Patient Characteristics

A total of 205 patients were treated for TBAD during the study period. After careful radiological and clinical evaluations, 162 patients (mean age, 67.6 ± 13.9 years; 27.8% females) were diagnosed with uncomplicated TBAD (n = 48) or high-risk TBAD (n = 114) and included in the present analysis. The most common feature of high-risk TBAD was an aortic diameter >40 mm (n = 97; 59.9%) followed by a false lumen diameter >22 mm (n = 33; 20.4%). Most of the high-risk patients had 1 feature of high-risk TBAD (n = 76; 66.7%) (Table 1).

Aortic aneurysm was known in 5 patients (10.4%) with uncomplicated TBAD and in 30 (26.3%) with high-risk TBAD ($P = .04$). Previous aortic surgery, mainly on the abdominal aorta, had been performed in 11 patients (9.6%) in the high-risk group but in no patients in the uncomplicated

TABLE 1. High-risk classification of acute TBAD patients (N = 162)

High-risk TBAD feature	n (%)
Visceral malperfusion at imaging*	5 (3.1)
Celiac trunk	1 (0.6)
Superior mesenteric artery	2 (1.2)
Inferior mesenteric artery	0 (0)
Left renal artery	3 (1.9)
Right renal artery	2 (1.2)
Left lower limb arteries	0 (0)
Right lower limb arteries	0 (0)
Refractory arterial hypertension	10 (6.2)
Aortic diameter >40 mm in Ishimaru zone 3-8	97 (59.9)
Entry tear in the lesser curve	13 (8.0)
Aortic false lumen diameter >22 mm	33 (20.4)
Number of TBAD-related features	
1	76 (46.9)
2	33 (20.3)
3	5 (3.1)

TBAD, Type B aortic dissection. *Some patients had multiples sites involved in visceral malperfusion at imaging.

TABLE 2. Demographics of high-risk and uncomplicated acute TBAD patients

Characteristic	Overall series (N = 162)	Uncomplicated TBAD (N = 48)	High-risk TBAD (N = 114)	*P* value	Missing data, n
Age, y. mean (SD)	67.6 (13.9)	67.2 (12.6)	67.8 (14.4)	.79	
Body mass index, mean (SD)	27.9 (5.7)	28.2 (4.6)	27.7 (6.2)	.72	47
Female sex, n (%)	45 (27.8)	14 (29.2)	31 (27.2)	.85	
Hypertension, n (%)	100 (61.7)	29 (60.4)	71 (62.3)	.86	
Coronary artery disease, n (%)	24 (14.8)	5 (10.4)	19 (16.7)	.35	
Extracardiac arteriopathy, n (%)	14 (8.6)	2 (4.2)	12 (10.5)	.24	
Diabetes mellitus, n (%)				.13	
Non–insulin-dependent diabetes	10 (6.2)	1 (2.1)	9 (7.9)		
Insulin-dependent diabetes	1 (0.6)	1 (2.1)	0 (0)		
Pulmonary disease, n (%)	20 (12.3)	5 (10.4)	15 (13.2)	.80	
Smoking, n (%)				.17	
Current smoker	52 (32.1)	21 (43.8)	31 (27.2)		
Ex-smoker	23 (14.2)	6 (12.5)	17 (14.9)		
Nonsmoker	87 (53.7)	21 (43.8)	66 (57.9)		
Bicuspic aortic valve, n (%)	4 (2.5)	0 (0)	4 (3.5)	.32	
Connective tissue disease, n (%)					
Marfan disease	11 (6.8)	2 (4.2)	9 (7.9)	.51	
Ehlers–Danlos disease	0 (0)	0 (0)	0 (0)	N/A	
Loeys–Dietz syndrome	0 (0)	0 (0)	0 (0)	N/A	
Preoperative cerebrovascular accident, n (%)					
Prior stroke	10 (6.2)	4 (8.3)	6 (5.3)	.48	
Prior transient ischemic attack	4 (2.5)	3 (6.3)	1 (0.9)	.78	
Laboratory parameters, mean (SD)					
Creatinine, μmol/L	80 (23)	80 (24)	80 (23)	.89	4
eGFR, mL/min/1.73^2	90 (27)	90 (29)	90 (27)	.98	4
Hemoglobin, mg/dL	132 (15)	131 (15)	133 (15)	.64	4
Platelets, $\times 10^9$/L	211 (81)	216 (64)	209 (88)	.62	4
C-reactive protein, mg/L	43 (65)	31 (57)	48 (68)	.10	4
Leukocytes, $\times 10^9$/L	9.8 (3.3)	10.0 (3.1)	9.7 (3.3)	.53	4
Prior aortic aneurysm, n (%)					
Ascending aorta	35 (21.6)	5 (10.4)	30 (26.3)	**.04**	
Aortic arch	13 (8.0)	2 (4.2)	11 (9.6)	.35	
Descending aorta	1 (0.6)	0 (0)	1 (0.9)	1.00	
Abdominal aorta	3 (1.9)	1 (2.1)	2 (1.8)	1.00	
Combination of aortic segments	9 (5.6)	1 (2.1)	8 (7.0)	.28	
Prior aortic surgery, n (%)	19 (11.7)	2 (4.2)	17 (14.9)	.06	
Ascending aorta	5 (3.1)	1 (2.1)	4 (3.5)	1.00	
Abdominal aorta	11 (6.8)	0 (0)	11 (9.6)	**.04**	
Ascending and abdominal aorta	3 (1.9)	1 (2.1)	2 (1.8)	1.00	
Prior aortic stent grafting, n (%)	1 (0.6)	0 (0)	1 (0.9)	1.00	
Prior cardiac surgery, n (%)	11 (6.8)	2 (4.2)	9 (7.9)	.51	
Prior PCI, TAVI, n (%)	9 (5.6)	2 (4.2)	7 (6.1)	.73	

Significant *P* values are in bold type. *TBAD*, Type B aortic dissection; *SD*, standard deviation; *N/A*, not applicable; *eGFR*, estimated glomerular filtration rate; *PCI*, percutaneous coronary intervention, *TAVI*, transcatheter aortic valve implantation.

group (P = .04). Only one patient in the high-risk group had a prior aortic stent grafting (Tables 2 and 3).

In univariable analysis with Fine and Gray competing-risks regression with non–TBAD-related death as a competing risk, the presence of 2 high-risk features (subdistribution hazard ratio [SHR], 3.17; 95% CI, 1.64-5.89) and an aortic diameter >40 mm (hazard ratio [HR], 6.36; 95% CI, 2.24-18.1) were identified as risk factors for TBAD-related late adverse

TABLE 3. Clinical characteristics of high-risk and uncomplicated acute TBAD patients

Characteristic	Overall series (N = 162)	Uncomplicated TBAD (N = 48)	High-risk TBAD (N = 114)	*P* value
Clinical characteristics, n (%)				
Chest/back pain	145 (89.5)	44 (91.7)	101 (88.6)	.60
Hypotension/shock	0 (0)	0 (0)	0 (0)	N/A
Neurologic deficit	0 (0)	0 (0)	0 (0)	N/A
Clinical malperfusion	0 (0)	0 (0)	0 (0)	N/A
Iatrogenic dissection	2 (1.2)	1 (2.1)	1 (0.9)	.51
Pseudoaneurysm, n (%)	0 (0)	0 (0)	0 (0)	N/A
Penetrating aortic ulcer, n (%)	15 (9.3)	2 (4.2)	13 (11.4)	.23
Atherosclerosis, n (%)	132 (81.5)	43 (91.5)	89 (79.5)	.10
Intramural hematoma, n (%)	63 (38.9)	25 (53.2)	38 (33.9)	**.03**
Aortic rupture, n (%)	0 (0)	0 (0)	0 (0)	N/A
Contained rupture	0 (0)	0 (0)	0 (0)	N/A
Free rupture	0 (0)	0 (0)	0 (0)	N/A

Significant *P* values are in bold type. *TBAD*, Type B aortic dissection; *N/A*, not applicable.

events, whereas an aortic diameter >40 mm (HR, 2.37; 95% CI, 1.20-4.68) was the sole risk factor associated with increased mortality after TBAD (Tables E1 and E2).

Early Outcome

All patients in the uncomplicated group received conservative treatment during their initial hospital stay. Aortic intervention was performed in 13 patients (11.4%) of the high-risk group at a mean of 8.5 days after admission. Progression of the dissection, when the aortic diameter exceeded 45 mm, was the main cause of aortic intervention (n = 10; 8.8%). Only 2 patients were treated due to a penetrating aortic ulcer (PAU), and 1 patient had visceral malperfusion detected on imaging. Three patients (2.6%) underwent surgery of the descending thoracic aorta, whereas thoracic endovascular aortic repair (TEVAR) was performed in 9 patients (7.9%), with concomitant carotid-subclavian bypass in 3 patients. Only 1 patient underwent endovascular treatment involving the visceral arteries (Table 4). After 2015, TEVAR was the most common procedure in this series, possibly decreasing the rate of postoperative complications.

During the initial hospital stay, some patients in both study groups suffered from renal ischemia. Aortic rupture, renal failure necessitating dialysis, and spinal ischemia were detected only in the high-risk group, whereas bowel ischemia was detected solely in the uncomplicated group. There was no between-group difference in the length of hospital stay (mean, 12.0 ± 6.5 days in the high-risk group vs 14.0 ± 8.0 days in the uncomplicated group; *P* = .13) or in intensive care unit (ICU) stay (mean, 0.5 ± 3.0 days vs 0.5 ± 2.5 days; *P* = .52). Overall, 4 patients (3.5%) of the high-risk group died during their initial hospital stay, all from TBAD-related causes (*P* = .32), whereas none of the uncomplicated TBAD patients died (Tables 5 and E3).

Late Survival

The mean follow-up of the overall series was 5.1 ± 3.9 years. TBAD-related death (ie, TBAD as the main cause of death or dissection as a contributing factor) was the most common cause of death in both study groups, followed by neurologic and cardiovascular causes (Table 6). Survival at 10 years was 71% in the uncomplicated group and 60% in the high-risk group (*P* = .05, log-rank test) (Figure 1).

In univariable analysis, age >65 years (HR, 2.95; 95% CI, 1.41-6.17), coronary artery disease (HR, 2.27; 95% CI, 1.12-4.59), hypertension (HR, 2.11; 95% CI, 1.07-4.18), extracardiac arteriopathy (HR, 4.87; 95% CI, 2.09-11.35), and previous aortic surgery (HR, 2.19; 95% CI, 1.05-4.57) were associated with increased early and late mortality after TBAD. In multivariable analysis, age (HR, 1.07; 95% CI, 1.04-1.11) and extracardiac arteriopathy (HR, 3.01; 95% CI, 1.28-7.08) were identified as independent predictors of mortality (Table E4).

TBAD-Related Adverse Events

Patients with connective tissue disorders were classified using the same criteria as all patients in this series. Eleven patients with connective tissue disease with Marfan syndrome were included in the study group. One patient was classified as a complicated acute TBAD patient requiring surgical intervention during the initial stay. Eight patients were classified as high-risk TBAD patients, 7 of whom required surgical intervention during follow-up.

Sixty-three patients (38.9%) had an intramural hematoma (IMH) at presentation, with a higher prevalence in the uncomplicated TBAD group compared with the high-risk group (53.2% vs 33.9%; *P* = .03). Six patients in both study groups had IMH and dissection findings, whereas solely IMH findings were detected in 19 patients

TABLE 4. Treatment strategies and indications of high-risk and uncomplicated acute TBAD patients

Strategy/indication	Overall series (N = 162)	Uncomplicated TBAD (N = 48)	High-risk TBAD (N = 114)	P value
Conservative treatment, n (%)	149 (92.0)	48 (100)	101 (88.6)	**.02**
Indication for intervention, n (%)				
Malperfusion at imaging	1 (0.6)	0 (0)	1 (0.9)	N/A
Progression of dissection	10 (6.2)	0 (0)	10 (8.8)	N/A
Aortic rupture	0 (0)	0 (0)	0 (0)	N/A
Penetrating aortic ulcer	2 (1.2)	0 (0)	2 (1.8)	N/A
Time to intervention, d, mean (SD)	8.5 (7.9)	N/A	8.5 (7.9)	N/A
Number of aortic procedures, n (%)				
1	13 (8.0)	0 (0)	13 (11.4)	**.02**
2	0 (0)	0 (0)	0 (0)	N/A
3	0 (0)	0 (0)	0 (0)	N/A
Aortic intervention, n (%)	13 (8.0)	0 (0)	13 (11.4)	**.02**
Thoracic aorta	12 (7.4)	0 (0)	12 (10.5)	**.02**
Open aortic surgery	3 (1.9)	0 (0)	3 (2.6)	.56
DTA surgery	3 (1.9)	0 (0)	3 (2.6)	.56
Abdominal aorta surgery	0 (0)	0 (0)	0 (0)	N/A
Abdominal fenestration	0 (0)	0 (0)	0 (0)	N/A
Extra-anatomic bypass	0 (0)	0 (0)	0 (0)	N/A
Visceral artery bypass	0 (0)	0 (0)	0 (0)	N/A
Renal artery bypass	0 (0)	0 (0)	0 (0)	N/A
TEVAR, n (%)	9 (5.6)	0 (0)	9 (7.9)	.06
TEVAR proximal landing, n (%)				
Ishimaru zone 1	0 (0)	N/A	0 (0)	N/A
Ishimaru zone 2	3 (1.9)	N/A	3 (2.6)	N/A
Ishimaru zone 3	6 (3.7)	N/A	6 (5.3)	N/A
Carotid-subclavian bypass, n (%)	3 (1.9)	0 (0)	3 (2.6)	.56
Endovascular treatment of visceral arteries, n (%)	1 (0.6)	0 (0)	1 (0.9)	1.00

Significant *P* values are in bold type. *TBAD*, Type B aortic dissection; *N/A*, not applicable; *SD*, standard deviation; *DTA*, descending thoracic aorta; *TEVAR*, thoracic endovascular aortic repair.

(39.6%) in the uncomplicated group and 32 patients (28.1%) in the high-risk group. In addition, 15 patients (9.2%) had a PAU, including 2 (4.2%) in the uncomplicated group and 13 (11.4%) in the high-risk group. Patients with IMH and PAU were treated according to the guidelines of that time.

During follow-up, 35 patients (21.6%) developed aneurysmal degeneration, including 4 (8.3%) in the uncomplicated group and 31 (27.2%) in the high-risk group, with a statistically significant difference (*P* = .01). One new dissection and 1 antegrade extension of a dissection were detected during the follow-up, both in the high-risk group. One fifth of the high-risk patients (n = 23; 20.2%) required a TBAD-related aortic intervention. Surgical thoracic aortic intervention was the most common procedure (n = 16; 14.0%). TEVAR was performed in 7 patients (6.1%), and surgery for abdominal aortic aneurysm was performed in 9 patients (7.9%). In addition, 1 endovascular abdominal aorta procedure and 1 fenestration procedure were performed during follow-up. The mean interval for the first

TBAD-related intervention was 0.6 ± 0.8 years (range, 0.3-31.5 months) (Table 6). The estimated cumulative incidence for TBAD-related adverse events was 29.5% (95% CI 21.1%-38.3%) at 5 years and 33.0% (95% CI 23.7%-42.6%) at 10 years in the high-risk group and significantly lower at 6.6% (95% CI, 1.7%-16.5%) at 5 years and 6.6% (95% CI, 1.7%-16.5%) at 10 years in the uncomplicated group. Competing-risk analysis with non TBAD death as a competing risk was performed according to the method of Fine and Gray (*P* = .001, Gray test) (Figure 2). Cumulative incidence estimates for non–TBAD-related death in the high-risk and uncomplicated groups were 16.6% (95% CI, 9.6%-25.2%) and 6.2% (95% CI, 1.0%-18.4%), respectively, at 5 years and 19.9% (95% CI, 11.9%-29.4%) and 22.3% (95% CI, 7.2%-42.6%) at 10 years (*P* = .39, Gray test) (Figure 2).

Extracardiac arteriopathy (SHR, 2.61; 95% CI, 1.08-6.27) and coronary artery disease (SHR, 2.24; 95% CI 1.07-4.71) were risk factors in univariable analysis for TBAD-related adverse events in Fine and Gray

TABLE 5. In-hospital outcomes of high-risk and uncomplicated acute TBAD patients

In-hospital outcome	Overall series (N = 162)	Uncomplicated TBAD (N = 48)	High-risk TBAD (N = 114)	*P* value	Missing data, n
RBC transfusion, n (%)	8 (4.9)	1 (2.1)	7 (6.3)	.44	
Aortic rupture, n (%)	1 (0.6)	0 (0)	1 (0.9)	1.00	2
Bowel ischemia, n (%)	1 (0.6)	1 (2.1)	0 (0)	.30	2
Renal ischemia, n (%)	5 (3.1)	1 (2.1)	4 (3.5)	1.00	2
Renal failure (dialysis), n (%)	2 (1.2)	0 (0)	2 (1.8)	.58	2
Spinal ischemia, n (%)	1 (0.6)	0 (0)	1 (0.9)	1.00	2
Limb ischemia, n (%)	0 (0)	0 (0)	0 (0)	N/A	2
Stroke, n (%)	6 (3.7)	3 (6.3)	3 (2.7)	.37	2
Myocardial infarction, n (%)	0 (0)	0 (0)	0 (0)	N/A	2
Hospital stay, d, mean (SD)	13.5 (7.5)	12.0 (6.5)	14.0 (8.0)	.13	2
Intensive care unit stay, d, mean (SD)	0.5 (2.5)	0.5 (3.0)	0.5 (2.5)	.52	-
In-hospital mortality, n (%)	4 (2.5)	0 (0)	4 (3.5)	.32	-
30-d mortality, n (%)	4 (2.5)	0 (0)	4 (3.5)	.32	-

TBAD, Type B aortic dissection; *RBC*, red blood cell; *N/A*, not applicable; *SD*, standard deviation.

competing-risks regression analysis, with non–TBAD-related death as a competing risk (Table E5).

DISCUSSION

The findings of the present analysis can be summarized as follows: (1) high-risk TBAD patients had worse survival and freedom from TBAD-related aortic events compared to uncomplicated TBAD patients; (2) 20.2% of high-risk TBAD patients required a TBAD-related aortic intervention after an average of 6 months; and (3) uncomplicated TBAD patients were free from aortic interventions during the entire follow-up. During the study period, clinical decisions were made according to guidelines of that time.[2] The recent reporting standards from the SVS and STS introduced the definition of high-risk TBAD patients whose characteristics and prognosis differ substantially from those of uncomplicated and complicated acute TBAD.[3]

The optimal treatment strategy for TBAD is under debate. After an era of a surgical approach to TBAD followed by an era of medical treatment dominating the optimal treatment strategy debate, endovascular treatment has established its value in the treatment of TBAD patients, decreasing their risk of morbidity and mortality.[6-8] Endovascular treatment was first introduced in 1994 for the treatment of descending aorta aneurysms and in 1999 for aortic dissections.[9,10] Aortic intervention is required for complicated TBAD, rupture, and malperfusion. TEVAR is guideline-recommended to limit mortality in these patients.[11,12]

Accordingly, in our study, only 13 high-risk patients underwent aortic intervention during their initial hospital stay. Disease progression was followed by computed tomography scan while under optimal medical treatment, and,

consequently, aortic procedures were deemed necessary at 1 week after hospital arrival (mean time, 8.5 ± 7.9 days). Moreover, within 6 months, one-fourth of the high-risk patients required an aortic procedure for a TBAD-related event. The high number of surgical thoracic approaches reflects the previous trend of waiting for a dissection aneurysm to grow and reach 5.5 to 6.0 cm in diameter before performing an operation in the chronic phase. Several late abdominal aortic operations might be explained by a previous aortic aneurysm detected prior to dissection. Of note, no aortic interventions or TBAD-related aortic interventions were performed in the uncomplicated group.

Recently, interest has increased in the management of uncomplicated acute TBAD patients and their follow-up outcomes. The INSTEAD, INSTEAD XL, and ADSORB trials, which focused on the optimal treatment strategy of uncomplicated TBAD patients in acute and chronic settings, reported favorable aortic remodeling and aortic-specific mortality during follow-up in TEVAR-treated groups.[13-15] Typically, favorable aortic remodeling includes expansion of the true lumen, regression of the false lumen, stabilization of the transaortic diameter, and complete thrombosis of the false lumen.[7] Additionally, early TEVAR therapy for uncomplicated TBAD patients lowers intervention-free survival during follow-up, providing a long-term benefit.[8] Unfortunately, data on aortic remodeling after TEVAR was not available in our study.

TBAD can be classified based on the interval from the onset of symptoms as hyperacute (<24 hours), acute (1-14 days), subacute (15-90 days), or chronic (>90 days).[3,16] The subacute phase seems to be the optimal therapeutic window for TEVAR in high-risk TBAD patients[6]; however, the optimal time at which to perform TEVAR in high-risk

TABLE 6. Outcomes of high-risk and uncomplicated acute TBAD patients

Outcome	Overall series (N = 162)	Uncomplicated TBAD (N = 48)	High-risk TBAD (N = 114)	P value
10-y mortality, %	63.0	71.0	60.0	**.05**
Cause of death, n (%)				
TBAD-related death	16 (35.5)	3 (37.5)	13 (35.1)	1.00
Cardiovascular	8 (17.8)	1 (12.5)	7 (18.9)	1.00
Cancer	6 (13.3)	1 (12.5)	5 (13.5)	1.00
Neurologic	8 (18.2)	1 (12.5)	7 (19.4)	.55
Pulmonary	2 (4.4)	1 (12.5)	1 (2.7)	.33
Unknown or other	4 (8.9)	1 (12.5)	3 (8.1)	1.00
Other TBAD-related events, n (%)				
New aortic dissection	1 (0.6)	0 (0)	1 (0.9)	1.00
Aneurysm degeneration	35 (21.6)	4 (8.3)	31 (27.2)	**.01**
Antegrade extension of dissection	1 (0.6)	0 (0)	1 (0.9)	1.00
Stroke	5 (3.1)	3 (6.3)	2 (1.8)	.16
Myocardial infarction	3 (1.9)	1 (2.1)	2 (1.8)	1.00
Aortic intervention, n (%)	28 (17.3)	0 (0)	28 (24.6)	**<.001**
TEVAR	7 (4.3)	0 (0)	7 (6.1)	.11
Surgical DTA repair	16 (9.9)	0 (0)	16 (14.0)	**.01**
EVAR	1 (0.6)	0 (0)	1 (0.9)	1.00
Surgical repair of AAA	9 (5.6)	0 (0)	9 (7.9)	.06
Aortic fenestration	1 (0.6)	0 (0)	1 (0.9)	1.00
TBAD-related intervention, n (%)	23 (14.2)	0 (0)	23 (20.2)	**<.001**
TBAD-related intervention, y, mean (SD)	0.6 (0.8)	N/A	0.6 (0.8)	N/A
TBAD-related composite outcome, n (%)	38 (23.5)	3 (6.4)	35 (31.3)	**<.001**
Length of follow-up, y, mean (SD)	5.1 (3.9)	5.4 (3.9)	4.9 (3.8)	.40

Significant *P* values are in bold type. *TBAD*, Type B aortic dissection; *TEVAR*, thoracic endovascular aortic repair; *DTA*, descending thoracic aorta, *EVAR*, endovascular aortic repair; *AAA*, abdominal aortic aneurysm; *SD*, standard deviation; *N/A*, not applicable.

TBAD patients has not been well studied. The subacute interval is extrapolated from the results of the Virtue registry and the STABLE trial, which included 100 and 86 patients with complicated TBAD, respectively. Furthermore, the subacute group of complicated TBAD patients included only 24 patients in Virtue and 31 patients in STABLE. However,
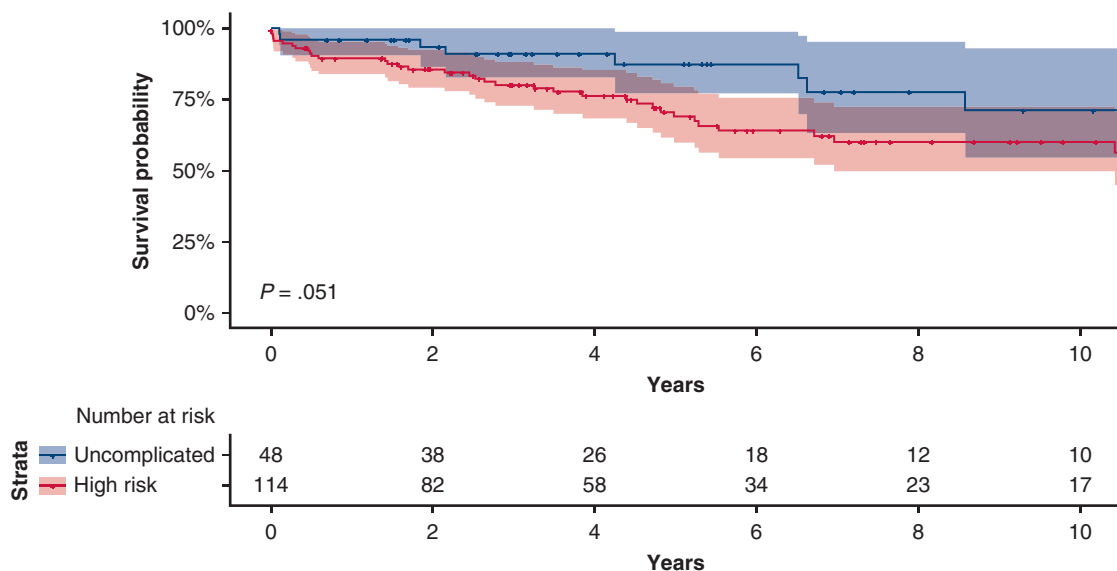

FIGURE 1. Survival in patients with high-risk and uncomplicated acute type B aortic dissection (TBAD), reported with 95% CI (*P* = .051, log-rank test).
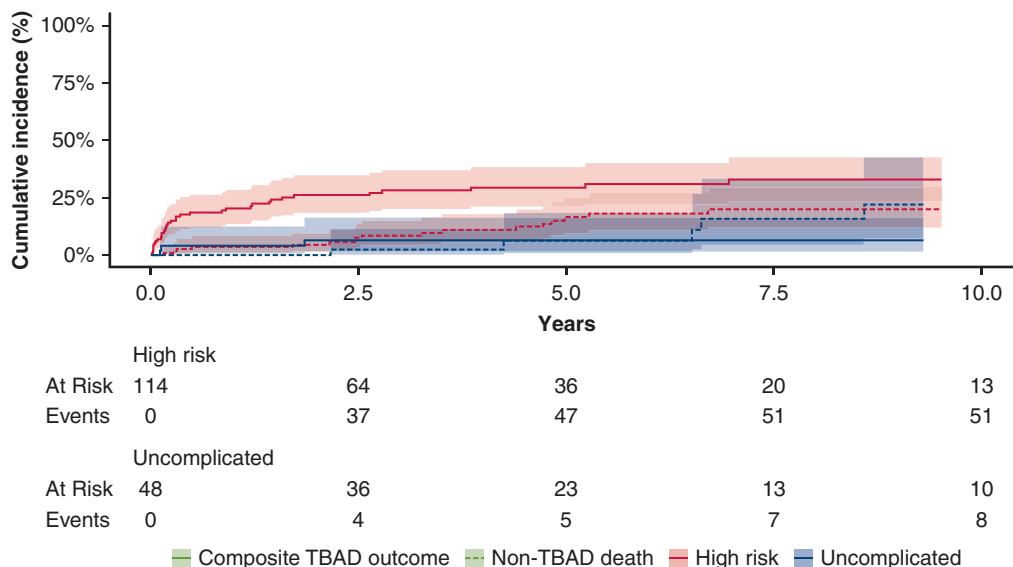
FIGURE 2. Cumulative incidences of composite type B aortic dissection (*TBAD*) outcome and non-TBAD death in high-risk and uncomplicated TBAD patients, reported with 95% CI (*P* = .001, Gray test, with non-TBAD death as a competing risk).

TEVAR treatment during the subacute phase showed better survival and favorable aortic remodeling in the 2 studies.[7,17,18] TEVAR performed in the acute phase was associated with early aortic rupture, retrograde type A dissection, and disabling stroke in one study supporting the optimal subacute phase for interventions.[19] In addition, life expectancy >5 years should be considered in the decision making of early TEVAR in high-risk patients.[20] This study supports the importance of recognizing TBAD high-risk features and providing early subacute interventions.

Study Limitations

The main limitation of this study is its retrospective nature. Second, systematic collection of data on the optimal medical treatment of these patients was not feasible. Third,

identification of patients with refractory pain and hypertension during the initial hospital stay was difficult, as data on subjective estimation of pain severity were not available. Fourth, we did not encounter any patient who was readmitted for refractory pain in our study population, but we cannot exclude the possibility that some of them required medical attention because of pain or discomfort related to TBAD. Finally, the limited sample of patients prevented any matched comparative analysis of the study groups.

CONCLUSIONS

Overall survival and freedom from adverse aortic intervention were significantly better in the uncomplicated TBAD group compared with high-risk group. High-risk TBAD patients are exposed to lifelong risk for
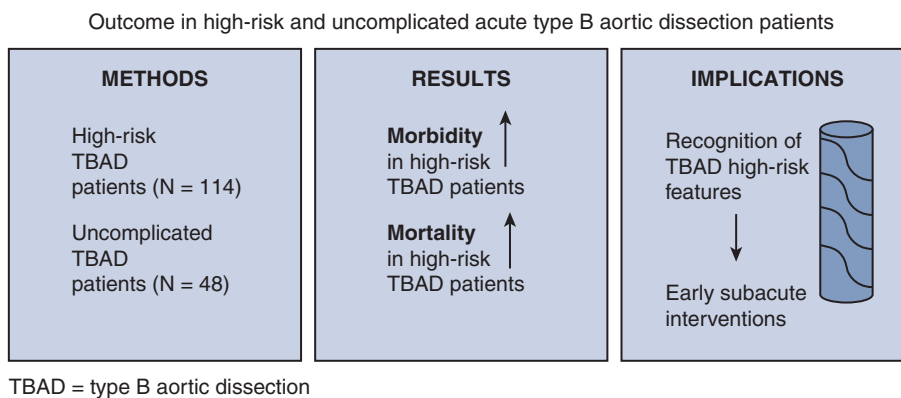

FIGURE 3. Recognition of high-risk features of acute type B aortic dissection (*TBAD*) would favor early subacute interventions, given the increased morbidity and mortality in high-risk acute TBAD patients.

TBAD-related death and aortic intervention with possible TBAD-related morbidity. Thus, recognition of high-risk features of TBAD may prompt early subacute interventions for these patients ([Figure 3](#)). Prospective, randomized clinical trials are warranted on to define the benefits and optimal timing of TEVAR for high-risk TBAD.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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TABLE E1. Aortic characteristics predicting TBAD-related aortic operation or TBAD-related death in Fine and Gray competing-risk regression univariable analysis with non-TBAD death as a competing risk

Variable	SHR (95% CI)	*P* value
High-risk TBAD features		
1 feature	Reference	—
2 features	3.17 (1.64-5.89)	**<.001**
3 features	1.12 (0.37-8.78)	.910
Refractory arterial hypertension	1.34 (0.38-4.76)	.650
Malperfusion at imaging	1.18 (0.42-8.33)	.750
Aortic diameter >40 mm	6.36 (2.24-18.1)	**<.001**
Entry tear in the lesser curve	1.79 (0.76-4.23)	.180
False lumen >22 mm	1.24 (0.59-2.60)	.570

Significant *P* values are in bold type. *TBAD*, Type B aortic dissection; *SHR*, subdistribution hazard ratio; *CI*, confidence interval.

TABLE E2. Aortic characteristics predicting survival in univariable analysis

Variable	HR (95% CI)	*P* value
High-risk TBAD features		
1 feature	Reference	—
2 features	1.57 (0.80-3.08)	.186
3 features	0.59 (0.08-4.37)	.603
Refractory hypertension	0.38 (0.09-1.59)	.186
Malperfusion at imaging	1.14 (0.16-8.33)	.899
Aortic diameter >40 mm	2.37 (1.20-4.68)	**.013**
Entry tear in the lesser curve	0.87 (0.27-2.80)	.809
False lumen >22 mm	1.85 (0.97-3.52)	.063

Significant *P* values are in bold type. *HR*, Hazard ratio; *CI*, confidence interval; *TBAD*, type B aortic dissection.

TABLE E3. Variables included in univariable and multivariable Cox regression analyses

Age
Sex
Body mass index
Coronary artery disease
Preoperative cerebrovascular accident
Hypertension
Extracardiac arteriopathy
Pulmonary disease
Smoking
Diabetes mellitus
Connective tissue disorder
Bicuspid aortic valve
Previous aortic aneurysm
Previous aortic surgery
Intramural hematoma

TABLE E4. Predictors of survival

Clinical variables	HR (95% CI)	*P* value
Age	1.08 (1.04-1.11)	**<.001**
Age >60 y	3.63 (1.43-9.19)	**.007**
Age >65 y	2.95 (1.41-6.17)	**.004**
Male sex	1.80 (0.97-3.32)	.061
Body mass index	0.95 (0.87-1.04)	.295
Coronary artery disease	2.27 (1.12-4.59)	.023
Preoperative cerebrovascular event	2.26 (0.80-6.38)	.125
Hypertension	2.11 (1.07-4.18)	**.032**
Extracardiac arteriopathy	4.87 (2.09-11.35)	**<.001**
Pulmonary disease	1.15 (0.48-2.72)	.754
Smoking habit		
Current smoker	0.80 (0.42-1.53)	.505
Ex-smoker	0.41 (0.14-1.19)	.101
Diabetes mellitus		
Non–insulin-dependent diabetes	2.12 (0.83-5.39)	.114
Connective tissue disorder	0.28 (0.04-2.05)	.212
Bicuspid aortic valve	0.05 (0.00-87.80)	.427
Prior aortic aneurysm	1.26 (0.65-2.44)	.495
Prior aortic surgery	2.19 (1.05-4.57)	**.037**
Intramural hematoma	0.82 (0.56-1.16)	.253
Multivariable analysis		
Age	1.07 (1.04-1.11)	**<.001**
Extracardiac arteriopathy	3.01 (1.28-7.08)	**.012**

Significant *P* values are in bold type. *HR*, Hazard ratio; *CI*, confidence interval.

TABLE E5. Fine and Gray competing-risk regression analysis for TBAD-related aortic operation or TBAD-related death with non–TBAD-related death as a competing risk

Clinical variables	SHR (95% CI)	*P* value
Univariable analysis		
Age	1.00 (0.97-1.03)	.920
Age >60 y	1.27 (0.61-2.68)	.520
Age >65 y	1.38 (0.69-2.77)	.370
Male sex	0.77 (0.37-1.59)	.480
Body mass index	0.96 (0.86-1.07)	.430
Coronary artery disease	2.24 (1.07-4.71)	**.033**
Preoperative cerebrovascular event	1.53 (0.46-5.03)	.480
Hypertension	1.51 (0.75-3.04)	.250
Extracardiac arteriopathy	2.61 (1.08-6.27)	**.033**
Pulmonary disease	0.85 (0.30-2.43)	.760
Smoking habit		
Current smoker	1.20 (0.79-1.83)	.400
Ex-smoker	1.14 (0.66-1.71)	.490
Diabetes mellitus		
Non–insulin-dependent diabetes	1.04 (0.39-2.78)	.940
Connective tissue disorder	1.80 (0.66-4.91)	.250
Bicuspid aortic valve	1.06 (0.16-7.07)	.950
Previous aneurysm	1.50 (0.75-2.97)	.250
Previous aortic surgery	1.42 (0.61-3.30)	.410
Intramural hematoma	0.81 (0.39-1.69)	.570

Significant *P* values are in bold type. *SHR*, Subdistribution hazard ratio; *CI*, confidence interval.