

# Early and midterm outcomes of thoracic endovascular aortic repair (TEVAR) for acute and chronic complicated type B aortic dissection

Min-Hong Zhang, MD, Xin Du, MD, Wei Guo, MD\*, Xiao-Ping Liu, MD, Xin Jia, MD, Yang-Yang Ge, PhD

## Abstract

Thoracic endovascular aortic repair (TEVAR) in the current era has been chosen as a dominant and minimally invasive treatment for complicated aorta dissection. This study aimed to assess safety and feasibility of TEVAR in acute and chronic type B aortic dissection.

Between January 2011 and December 2013, 85 patients with complicated type B aortic dissection undergoing TEVAR were divided into acute aortic dissection (AAD) (n=60) group and chronic aortic dissection (CAD) group (n=25). Computed tomography was used to evaluate postoperative changes in maximal aortic diameter and true and false lumen diameters at 3 levels during a mean follow-up period of  $26.4 \pm 15.6$  months.

The technical success rate was 100%. In-hospital and 30-day rates of death were 3.3% in acute group and 0 in chronic group. Postdischarge rates of type I leak, type II leak, and retrograde type A dissection were 6.7%, 5.2%, and 3.4% (acute) and 0%, 4.0%, and 4.0% (chronic), respectively. The maximal aorta diameter remained stable in all the 3 levels in both acute and chronic group. The cumulative freedom from all-cause mortality at 3 years was similar in acute and chronic groups (89.5% vs 95.5%,  $P = .308$ ). The cumulative freedom from aortic-related mortality was also not significantly different in the acute and chronic groups (92.8% vs 95.2%,  $P = .531$ ). In the thoracic aorta, TEVAR treatment resulted in a significant increase in true lumen (TL) diameter and decrease in false lumen (FL). However, in the abdominal aorta, TEVAR did not lead to significant change in TL and FL diameters. The rates of complete thrombosis thoracic false lumens were better than that in the abdominal false lumen.

TEVAR was a safe and effect therapy for complicated acute and chronic type B dissection with low early and mid-term mortality and morbidity.

**Abbreviations:** AAD = acute aortic dissection, CAD = chronic aortic dissection, CTA = computed tomography angiography, FD = maximal false diameter, MD = maximal aorta diameter, RTAD = retrograde type A dissection, TD = true diameter, TEVAR = thoracic endovascular aortic repair.

**Keywords:** aortic remodeling, endoleak, retrograde type A dissection, thoracic endovascular aortic repair, type B aortic dissection

## 1. Introduction

Aortic dissection is the most common life-threatening disorder of the aorta, and the incidence has been reported approximately 3 cases per 100,000 people per year, with higher rates in men.<sup>[1,2]</sup> According to the International Registry of Acute Aortic Dissection (IRAD), about one third of all the acute aortic dissections (AADs) are of type B.<sup>[1]</sup> Among all the acute type B aortic dissection, about 25% of patients are complicated with malperfusion syndrome or hemodynamic instability at admission and untreated complicated dissection will result in irreversible

end organ damage or death.<sup>[3]</sup> Similarly, recurrence of symptoms, aneurysmal dilation (55 mm), or an aortic yearly increase of 4 mm was indicative of complicated chronic type B aortic dissection, and these factors are indicative of higher risk.<sup>[3]</sup> Untreated complicated dissection will result in irreversible end organ damage or death. Therefore, efficient and timely measures should be adopted to save patients' lives.<sup>[4]</sup>

Although great advances have been made over the years, surgical mortality was reported to exceed more than 30%<sup>[5,6]</sup> and considerable morbidity including spinal cord ischemia, cerebrovascular accident, and renal failure.<sup>[7]</sup> Thoracic endovascular aortic repair (TEVAR) in the current era has become the dominant and minimally invasive treatment for type B complicated aorta dissection.<sup>[8,9]</sup> The first report of TEVAR repair was firstly introduced by Dake et al 1994<sup>[10,11]</sup> and the first commercial device gained US Food and Drug Administration approval for this indication in 2005.<sup>[12]</sup> The aim of the treatment for the complicated aortic dissection is to deliver the blood flow through true lumen by sealing the proximal entry tear, abrogation of frank or impending aortic rupture, and relief of dynamic malperfusion. This process is termed as aortic remodeling, during which the false lumen is gradually thrombosed and true lumen is enlarged but without enlargement of the total aortic diameter.<sup>[13]</sup> Furthermore, aortic remodeling after TEVAR has been reported to be a significant prognostic factor for better outcome.<sup>[14]</sup> However, there are few studies on aortic remodeling for patients undergoing TEVAR in China, and early

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Department of Vascular Surgery, General Hospital of People's Liberation Army, Beijing, China.

\*Correspondence: Wei Guo, Department of Vascular Surgery, General Hospital of People's Liberation Army, 28# Fuxing Road, Beijing 100853, China (e-mail: weiguo\_2015@163.com).

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and midterm clinical efficiency of TEVAR in complicated type B aortic dissection required further investigations. Therefore, we performed this retrospective clinical study to evaluate the clinical outcomes and aortic remodeling of acute and chronic aortic dissection (CAD) in a Chinese population.

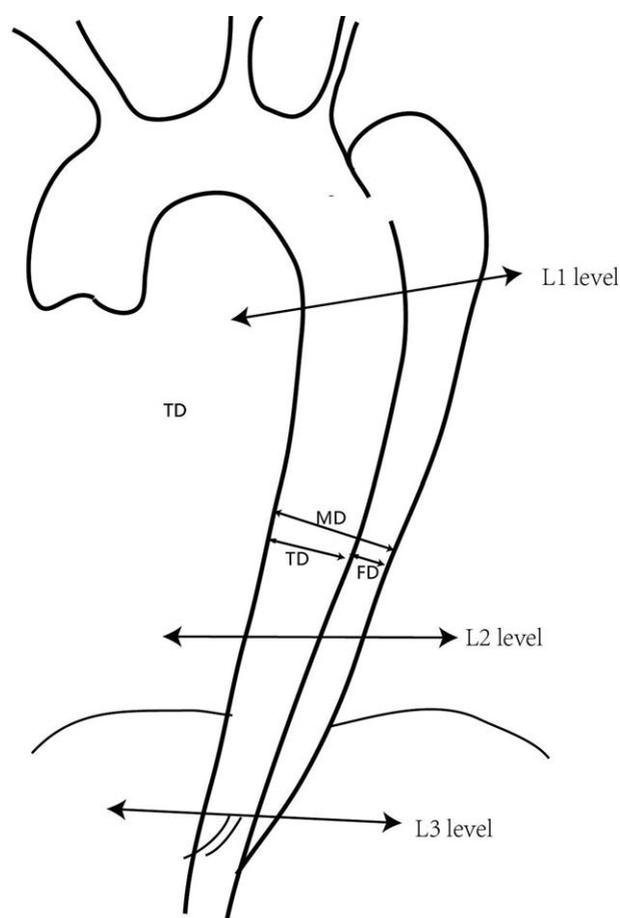
## 2. Materials and methods

### 2.1. Patient population

The study was performed according to the principles of the Declaration of Helsinki and approved by the ethics committee of our institution. Written informed consent was provided by all patients who participated in the study. Between January 2011 and December 2013, 85 consecutive patients with type B aortic dissection undergoing TEVAR in the Department of Vascular Surgery of the General Hospital of the People's Liberation Army were recruited in this study. Indications for complicated AAD included one or more followed clinical symptoms or anatomical characteristics: free or contained rupture; clinical or radiographic evidence of malperfusion; and refractory pain/impending rupture. Indications for complicated CAD included maximum aortic diameter  $>55$  mm, an aortic increase of  $>5$  mm within 3 months, detection of organ ischemia, and recurrence of other symptoms (pleural effusion, refractory pain, and resistant hypertension). Refractory pain was defined as ongoing symptoms of back and/or chest pain requiring narcotic medications in case of excellent blood pressure control. Patients with connective tissue disease (eg, Marfan syndrome), atypical aortic dissection (including intramural hematoma and penetrating atherosclerotic ulcer), residual type A aortic dissection, and trauma patients were excluded. Aortic dissection was defined as acute within 14 days from dissection onset and chronic 14 days after onset of acute symptoms, respectively. The whole 85 patients were divided into AAD group ( $n=25$ ) and CAD group ( $n=60$ ).

### 2.2. Surgical technique

All the procedures were performed under general anesthesia in the operation room. Patients should be evaluated by a team of cardiothoracic and vascular surgeons, interventional radiologists, and anesthesiologists before operation and only the qualified patients could receive TEVAR procedure. Detailed descriptions of procedural and technical information have been described previously.<sup>[15,16]</sup> In brief, arterial access was established and the stent grafts were deployed over a stiff wire. A proximal sealing zone of at least 15 mm was required. Oversizing by 10% to 20% was achieved according to the operator. Stent graft was deployed to prevent displacement of the stent graft with systolic pressure titrated to 80 to 90 mmHg with sodium nitroprusside. Since the first description of TEVAR, it has been the 1st-line therapeutic option for acute complicated type B aortic dissection and increasingly used in the treatment of chronic complicated type B aortic dissection due to it provides relative low morbidity and mortality and satisfactory clinical outcome compared to open surgery. Therefore, the standard clinical practice should be performed.<sup>[17]</sup> Angiography was applied to evaluate the reestablishment of distal perfusion after the stent-graft was deployed in the hybrid operation room. Computed tomography angiography (CTA) was performed again to assess the reestablishment of distal perfusion during the follow-ups. Subsequent deployment of the bare metal stent component was recommended if branch vessel obstruction or false lumen perfusion persisted, and was performed at the discretion of each implanting physician.



**Figure 1.** Levels on which the maximal aorta diameter, true diameter, and maximal false diameter were measured; the S1, S2, and S3 segments were indicated.

### 2.3. Postprocedural imaging and follow-up

All patients underwent preoperative CTA of the entire aorta, including the bilateral carotid and pelvic arteries. Delayed scans were used for the detection of endoleaks. The Digital Imaging and Communications in Medicine data were transferred to 3mensio v6.1 (Pie Medical Imaging B.V. Inc, Maastricht, The Netherlands) for measurement and calculation of maximal aorta diameter (MD), true diameter (TD), and maximal false diameter (FD) using multiple plane reconstruction technique at 3 different levels.<sup>[14]</sup> The MD, TD, and FD were measured directly at the levels of the bronchial bifurcation (L1), the lower edge of left atrium (L2), and the celiac trunk (L3) (Fig. 1). Status of the false lumen was qualitatively assessed using CTA as patent (evidence of contrast without evidence of thrombus), partially thrombosed (evidence of both contrast and thrombus), or completely thrombosed (evidence of thrombus without evidence of contrast) in the descending thoracic and abdominal aorta.<sup>[18]</sup> The typical images in the same patient before and after TEVAR were shown in Fig. 2.

Postoperative CT was routinely performed at 3, 6, and 12 months, and yearly thereafter to measure the MD, TD, and FD. The entire descending aorta was divided into 3 segments distally from the stent graft to the celiac trunk: S1, stent covered aorta; S2, from the stent to the start of the celiac trunk; and S3, from the start of the celiac trunk to iliac branch arteries. The patency and remodeling of the false lumen were assessed by postoperative



**Figure 2.** Three-dimensional CTA reconstruction demonstrated an acute type B aortic dissection (DeBakey III) (A) and the same patient 1 year after TEVAR procedure. No sign of dissection was seen. CTA=computed tomography angiography, TEVAR=thoracic endovascular aortic repair.

CTA. The diameter of the S1 segment was measured using curved planar reconstruction and the presence of distal perfusion in false lumen at the S1 segment, secondary entry tear of the end of the stent, and re-entries distal to the stent were recorded.

#### 2.4. Statistical analysis

All data were analyzed using SPSS statistical software version 17.0 (SPSS Inc., Chicago, IL). Continuous variables were expressed as mean  $\pm$  standard deviation (SD) or median (interquartile range), according to whether they exhibited a normal distribution, and were compared by *t* test or a Wilcoxon rank-sum test. Categorical variables were expressed as percentages, and analyzed by chi-square and Fisher exact tests. Survival analyses were performed using the Kaplan–Meier method, and between-group comparisons were assessed by log-rank tests. Longitudinal data of total lumen, true lumen, and false lumen in acute and chronic group were compared with preoperative assessments.  $P < .05$  was considered statistically significant.

### 3. Results

#### 3.1. Patient demographic data and aortic dissection characteristics

The demographics of the patient cohort are illustrated in Table 1. There were 43 and 16 male patients in the acute and chronic group, respectively. There was no significant difference between

the 2 groups. More DeBakey type IIIb patients in the acute group compared to that in chronic group (96.7% vs 80.0%,  $P = .021$ ). The mean onset to procedure time was 8 and 39 days in the acute and chronic group. Other detailed demographic information was also shown in Table 1. The FL was patent without evidence of thrombus formation in all patients. Beta-blocker and calcium channel blocker (CCB) were more prevalent in chronic group, and the other parameters were similar in the 2 groups.

The indications for all patients receiving TEVAR were summarized in Table 2. A subset of patients in both acute and chronic group has 2 or more indications for TEVAR. The mean length of aorta covered in chronic group was significantly longer than that in the acute group (215.8 vs 269.7 mm,  $P = .041$ ).

#### 3.2. Clinical complications and secondary intervention

The technical success of TEVAR was achieved in all patients with correct deployment of the stent grafts and complete exclusion of the primary entry tear. All proximal landing zones were  $>2$  cm. There were no conversions to open surgery and no type I endoleaks. Four patients (6.7%) in the acute group and 1 patient (4.0%) in the chronic group were implanted with 2 stents (Table 3). In addition, 3 (3/60, 5%) patients were implanted with a bare stent at the superior mesenteric artery, right renal artery, and right common iliac artery to provide a blood supply. The average postoperative intensive care unit (ICU) stay was 4.2 and 3.7 days in acute and chronic group, and no significant difference

**Table 1****Demographic information for patients with complicated acute and chronic aortic dissection.**

	Acute (n=60)	Chronic (n=25)	P
Age, y (mean±SD)	63.2±11.3	65.5±10.0	.683
Male	43 (71.7%)	16 (64.0%)	.458
DeBakey type			.021
Illa	2 (3.3%)	5 (20.0%)	
IIlb	58 (96.7%)	20 (80.0%)	
Onset to procedure time, d	8 (4–11)	39 (24–98)	<.001
Comorbidities (n, %)			
Hypertension SVS 2/3	49 (81.7%)	22 (88.0%)	.749
Smoking SVS 2/3	36 (60%)	10 (40%)	.092
Diabetes SVS 2/3	7 (11.7%)	4 (16.0%)	.724
Hyperlipemia SVS 2/3	5 (8.3%)	2 (8.0%)	1.000
Cardiac disease SVS 2/3	5 (8.3%)	1 (4.0%)	.666
Carotid disease SVS 2/3	2 (3.3%)	1 (4.0%)	1.000
Renal impairment SVS 2/3	2 (3.3%)	2 (8.0%)	.577
Pulmonary disease SVS 2/3	3 (5.0%)	0 (0%)	.552
Medication			
Antiplatelet	5 (8.3%)	4 (16.0%)	.439
Anticoagulant	2 (3.3%)	3 (12.0%)	.149
Beta-blocker	19 (31.7%)	16 (64.0%)	.006
ACE inhibitor	15 (25.0%)	11 (44.0%)	.083
ARB	4 (6.7%)	2 (8%)	1.000
CCB	6 (10.0%)	7 (28.0%)	.049
Diuretics	13 (21.7%)	10 (40.0%)	.083

ACE inhibitor = angiotensin-converting enzyme inhibitor, ARB = angiotensin receptor blocker, CCB = calcium channel blocker, SD = standard deviation, SVS = society for vascular surgery grading system for common risk factors.

was found ( $P = .867$ ). Hospital stay was potentially longer in the acute group, although the statistical significance was not strong ( $P = .072$ ).

Type I endoleak only occurred in 4 acute dissections. Of them, 3 cases were successfully repaired by TEVAR, and the other case with small distal endoleaks experienced spontaneous resolution. Type II endoleak was found in 3 acute dissections that were treated with transcatheter embolization with coils and 1 chronic dissection that experienced spontaneous thrombosis. Ten cases in the acute group and 3 in the chronic group presented with distal perfusion. Five cases in the acute group and 3 cases in the chronic

**Table 2****Dissection characteristics.**

	Acute (n=60)	Chronic (n=25)	P
Indications for treatment			
Pleural effusion	14 (14/60)	3 (3/25)	.234
Periaortic hematoma	16 (16/60)	3 (3/25)	.139
Limb or visceral ischemia	15 (15/60)	7 (7/25)	.774
Refractory pain	28 (28/60)	9 (9/25)	.366
Resistant hypertension	5 (5/60)	3 (3/25)	.688
Aortic growth ≥5 mm within 3 mo	0 (0/60)	4 (4/25)	.006
Total diameter of L1 >55 mm	11 (11/60)	11 (11/25)	.014
Length of dissected aorta, mm	215.8±75.7	269.7±128.2	.041
Primary tear location			
Descending thoracic aorta			.504
S1	59 (59/60)	24 (24/25)	
S2	1 (1/60)	1 (1/25)	
Maximal diameter at L1	36.7±5.1	41.3±10.0	.008
Maximal diameter at L2	31.8±5.9	33.2±5.5	.194
Maximal diameter at L3	28.9±3.5	30.5±4.3	.103

**Table 3****Operation data related with TEVAR for acute and chronic type B aortic dissection.**

	Acute (n=60)	Chronic (n=25)	P
Emergent (n, %)	7 (11.7%)	0 (0)	.100
ASA class (n, %)			.592
II	36 (60.0%)	17 (72.0%)	
III	22 (36.7%)	7 (28.0%)	
IV	2 (3.3%)	1 (0)	
Operation time, min (mean±SD)	179.2±75.8	183.7±98.4	.586
Contrast medium, mL (mean±SD)	141.2±39.6	132.0±34.7	.323
Blood loss, mL (mean±SD)	84.6±34.2	79.4±30.2	.507
ICU stay, d (mean±SD)	4.2±4.4	3.7±3.9	.867
Device number (n, %)			1.000
One stent	56 (93.3%)	24 (96.0%)	
Two stents	4 (6.7%)	1 (4.0%)	
Technical success (n, %)	60 (100.0%)	25 (100.0%)	1.000
Hospital stay, d (mean±SD)	17.6±9.6	13.4±6.2	.072

ASA = American Society of Anesthesiology, ICU = intensive care unit, SD = standard deviation, TEVAR = thoracic endovascular aneurysm repair.

group were found to have secondary entry tears. Secondary endovascular treatment was performed in 1 case in the acute group and 2 in the chronic group. Two cases in the acute group and 1 in the chronic group presented with retrograde type A dissection (RTAD) at postoperative months 1, 8, and 21. All primary entry tears were successfully closed, and all proximal landing zones were >2 cm (Table 4).

**3.3. Survival analysis**

There were 2 early deaths in the acute group and no death in the chronic group. One died from massive pleural effusion 3 hours after TEVAR and 1 died from intestinal ischemia on day 14. Thus, total 30-day mortality was 2.4%. In addition, 3 cases in the acute group died at 10, 12, and 14 months after TEVAR procedure, and 1 patient in the chronic group died at 19 months (Table 5). In the S1 level, 2 mortalities within 30 days after surgery without sign of false lumen thrombosis and another 5 cases died during the longer follow-up. Of the 5 cases, 2 cases died with partial false lumen thrombosis and 3 cases died with patent false lumen, indicating aortic remodeling was essential for successful TEVAR.

The mean follow-up duration was 26.4 (interquartile range, 14.6–32.1) months. The cumulative freedom from all-cause mortality at 1 and 3 year were 93.3% and 89.5% (acute) and 100% and 95.2% (chronic) for the 2 groups. The patients in the acute group experienced similar all-cause mortality compared with patients in the chronic setting ( $P = .308$ ) (Fig. 3A). The freedom from aortic-related mortality at 1 and 3 year was 96.7% and 92.8% (acute) and 100% and 95.2% (chronic) for the 2 groups. Log-rank tests did not reveal a significantly different mortality rate in the acute group versus the chronic group ( $P = .531$ ) (Fig. 3B).

**3.4. Aortic remodeling**

Changes in the average diameters of the true lumen, false lumen, and total aortic lumen were demonstrated in Fig. 4. In the acute group, the TEVAR treatment resulted in a significant increase in TL diameter over time at the level of L1 (from a mean of 16.2–36.7 mm after 36 months,  $P < .001$ ) and L2 (from a mean of 12.1–26.2 mm after 36 months,  $P < .001$ ). The FL diameter also

**Table 4**  
**Outcomes after TEVAR.**

	Acute	Chronic	P
Mean follow-up time, mo (mean ± SD)	24.3 ± 13.5	28.7 ± 17.8	.323
30-d mortality	2/60 (3.3%)	0/25 (0%)	1.000
In-hospital complications			
Aorta rupture	4/60 (6.7%)	0/25 (0%)	.315
Stroke	1/60 (1.7%)	1/25 (4%)	.504
Spinal cord ischemia/paraplegia	3/60 (5%)	1/25 (4%)	1.000
Renal insufficiency	2/60 (3.3%)	2/25 (8%)	.577
Pulmonary infection	2/60 (3.3%)	0/25 (0%)	1.000
Postdischarge complications			
Endoleak			
Type I	4/58 (6.7%)	0/25 (0%)	.310
Type II	3/58 (5.2%)	1/25 (4.0%)	1.000
Distal perfusion	7/58 (12.1%)	3/25 (12.0%)	1.000
Device distal injury	5/58 (8.6%)	3/25 (12.0%)	.692
Distal injury required intervention	2/58 (3.4%)	2/25 (8.0%)	.580
Retrograde type A dissection	2/58 (3.4%)	1/25 (4.0%)	1.000
False lumen status of S1			1.000
Patent	0/58 (0%)	0/25 (0%)	
Partial thrombosis	5/58 (8.6%)	2/25 (8%)	
Complete thrombosis	53/58 (91.4%)	23/25 (92%)	
False lumen status of S2			.052
Patent	4/58 (6.9%)	2/25 (8%)	
Partial thrombosis	19/58 (32.8%)	15/25 (60%)	
Complete thrombosis	35/58 (60.3%)	8/25 (32%)	
False lumen status of S3			.899
Patent	9/58 (15.5%)	3/25 (12%)	
Partial thrombosis	41/58 (70.7%)	18/25 (72%)	
Complete thrombosis	8/58 (13.8%)	4/25 (16%)	

n, 58 because of 2 in-hospital/30-d mortality cases. SD=standard deviation, TEVAR=thoracic endovascular aneurysm repair.

significantly decreased at the level of L1 ( $P < .001$ ) and L2 ( $P < .001$ ) at 3 years. However, at the level of L3, both TL and FL diameters did not change significantly during the follow-up. In addition, the MD remained stable in all the 3 levels.

In chronic group, TEVAR significantly increases the aortic diameter of TL ( $P < .01$ ) and decrease the dimension of FL ( $P < .001$ ) at the level of L1. However, the diameter of TL and FL at the level of both L2 and L3 did not change significantly. The total aortic caliber was also not significantly modified by TEVAR in all the 3 levels. Notably, such aortic remodeling occurred early after TEVAR treatment and subsequently changes occurred mostly within 18 months.

Although a patent false lumen was seen more frequently in acute dissections before TEVAR procedure, there was no significant difference in the false lumen status at L1, L2, and L3 levels between acute and chronic dissections during follow-up.

All thoracic false lumens were either partially or completely thrombosed after the 30-day follow-ups. In comparison, the rates of complete thrombosis in the abdominal false lumen were lower. Complete thrombosis of the false lumen at the level of L1 was observed in 53 patients (91.4%) in the AD group and 23 patients (92%) in the CD. However, the complete thrombosis rate in the distal abdominal false lumen (L3 level) decreased to 13.4% (8/58) in acute dissection and 16% in chronic dissection, respectively.

#### 4. Discussion

This study demonstrated the feasibility of TEVAR in complicated type B aortic dissection, with acceptable midterm survival rates and relative low complications. All stent grafts were successfully deployed and technical success was as high as 100%. The 30-day mortality was low in acute dissection (3.3%) and no perioperative deaths occurred in chronic group. The overall survival was 93.3% and 89.5% (acute) and 100% and 95.5% (chronic) at 1 and 3 years, respectively. FL thrombosis was more evident in AAD within the 1st 12 months after treatment compared with CAD.

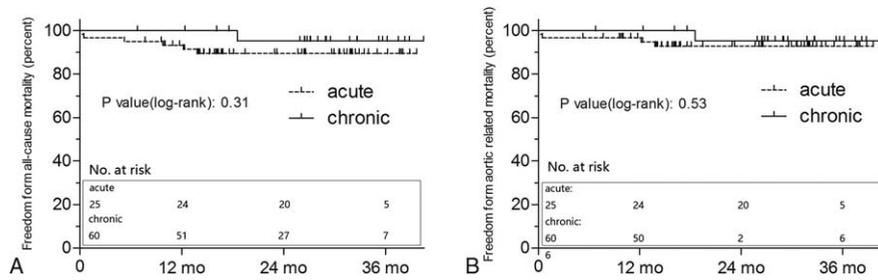
TEVAR appears to offer a substantially option for complicated aortic dissection in both acute and chronic setting, and several reports have confirmed this conclusion. Sayer et al<sup>[5]</sup> reported that the cumulative survival was superior in AAD compared to CAD (93% vs 66.5%,  $P = .015$ ). In contrast, Böckler et al<sup>[19]</sup> reported that overall survival rate was significantly better in patients with chronic CAD compared with these with AAD ( $P = .038$ ). However, some studies did not find significance between the 2 groups.<sup>[18,20]</sup> Chen et al<sup>[20]</sup> reported that patients in both acute phase and chronic phase have similar rates of freedom from all-cause mortality (92.4% vs 96.4%,  $P = .293$ ) and freedom of aortic-specific mortality (89.0% vs 96.4%,  $P = .102$ ). Our results also revealed similar survival in acute group and chronic group, which was consistent with this study. The discrepancy among various studies was suspected to derive from the fact that the enrolled patients were heterogeneous and from different medical centers. With more accumulated experience and properly selected patients, the 30-day postoperative mortality has decreased from initial 21%<sup>[21]</sup> to 0%–7.6%.<sup>[22–24]</sup> Therefore, TEVAR has been adopted as 1st-line therapy for complicated aortic dissection, and European Society of Cardiology recommended TEVAR as a class I treatment for complicated type B dissections.<sup>[25]</sup>

As TEVAR has become a popular therapy, deep insights into the related complications will help to alleviate the perioperative morbidity. The most commonly reported complication following TEVAR is endoleak.<sup>[26]</sup> Type I and type III endoleaks require correction, such as proximal cuff or extension, while type II endoleaks may seal spontaneously in about 50% of cases.<sup>[25]</sup> Fortunately, most type I endoleaks can be successfully treated by

**Table 5**  
**Causes of all deaths during the follow-up time.**

Case	Group	Age	Sex	Time to death	Aorta-related	Cause of death
1	Acute	49	M	3 h	Yes	Massive pleural effusion
2	Acute	35	F	14 d	Yes	Intestinal ischemia
3	Acute	73	M	5 mo	No	Cerebral infarction
4	Acute	58	M	10 mo	No	Myocardial infarction
5	Acute	62	F	12 mo	Yes	Aorto-esophageal fistula
6	Acute	35	M	14 mo	Yes	Rupture of enlarging thoracic false lumen
7	Chronic	38	M	19 mo	Yes	Retrograde type A dissection

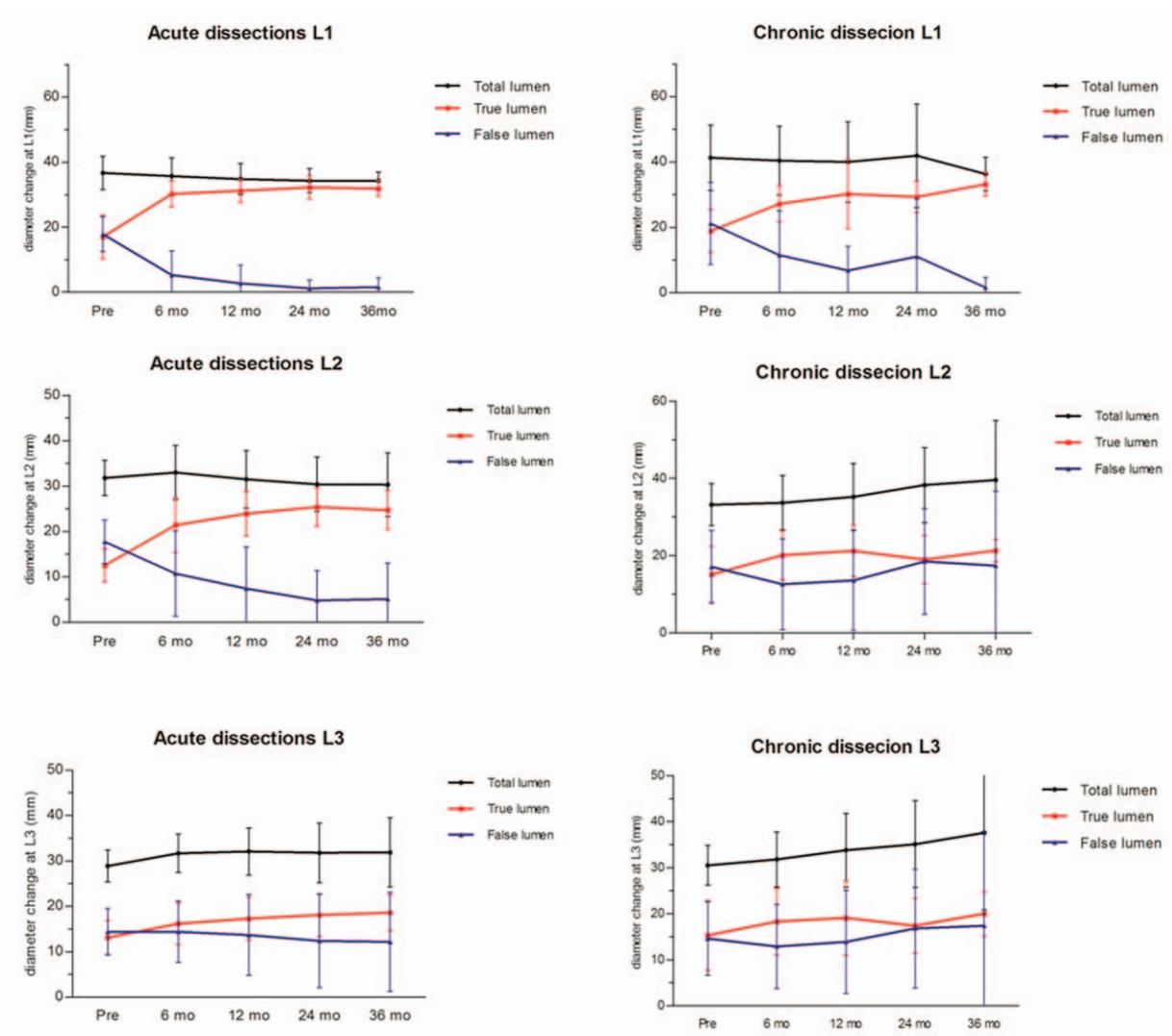
F=female, M=male.



**Figure 3.** Kaplan-Meier analysis of freedom from all-cause mortality (A) and freedom from aortic-related mortality (B). Vertical bar represents censored data. The number of patients at risk at various time points was given.

secondary endovascular techniques. Moreover, some type 1 endoleaks may eventually seal without any intervention. Sze et al<sup>[27]</sup> revealed that coverage of the left subclavian artery, a small radius of aortic arch curvature, and incomplete proximal apposition of the stent graft were significant portending factors for endoleaks. Therefore, coverage of LSA is often performed to achieve a proximal seal, and this proportion of patients was

reported to be as high as 40%.<sup>[28]</sup> Meanwhile, some studies have demonstrated intentional coverage of the LSA was associated with a higher overall stroke rate (13% vs 2%) and posterior circulation stroke rate (5.5% vs 1.2%) compared with LSA revascularization.<sup>[29,30]</sup> Therefore, the reconstruction of LSA prior to or during procedure should be carefully evaluated and performed in elective patients.



**Figure 4.** Total aortic diameter, true lumen, and false lumen regression trends at different measured levels, along with the time after thoracic endovascular aortic repair (TEVAR).

RTAD during or after TEVAR is a potentially lethal complication unique to thoracic endografting. The incidence of RTAD after TEVAR for acute type B aortic dissection was 8.4% and 3% for chronic dissection<sup>[31]</sup>; however, the overall mortality of this catastrophic complication could be as high as 33.6% to 57%.<sup>[31,32]</sup> The occurrence proved to be related with incomplete design of stent-graft system, rough handling, and presence of vascular wall lesions.<sup>[33]</sup> Indeed, the extent of graft oversizing was associated with an increased risk of RTAD. Increased graft oversizing (>9%) appeared to translate to an increased relative risk of RTAD by 14% for each percent oversize, so many clinicians consider a 10% oversize was appropriate.<sup>[31,32,34]</sup> Inappropriate endograft oversizing should be avoided, a close surveillance program is recommended and immediately surgical procedure should be performed in case of retrograde type A aortic dissection.<sup>[35]</sup>

In the last decade, TEVAR has been widely accepted for in clinic for its safety, convenience, and efficiency, but the TEVAR-associated mortality has been a troublesome issue for many surgeons. In this cohort, a total of 7 patients died during the follow-ups, including 6 (10%) acute AD patients and 1 (2.5%) chronic AD patient. Mortality tended to occur more frequently in the acute AD patients without statistical significance due to limited samples ( $P = .668$ , Fisher exact test). Two reasons may account for this result. On the one hand, as shown in Table 1, DeBakey IIIA was more frequent in the chronic AD patients compared to acute AD patients. It has been reported that thrombosis of false lumen was more common in DeBakey IIIA, and the thrombosis of false lumen is a protective factor for the better prognosis.<sup>[36,37]</sup> On the other hand, beta-blocker and calcium channel blocker were more frequently used in the chronic group (Table 1). Previous studies have confirmed that improved survivals were shown in patients both in acute and chronic phase of AD and calcium channel blocker also proved to have a protective effect for acute AD patients.<sup>[17,38]</sup> Therefore, medical therapy including blood pressure control is an essential part for the management of acute AD patients. Other technology, such as rapid pacing, as an efficient and safe method for lowering blood pressure in selected patients, also facilitates accurate deployment of stent-graft.<sup>[39]</sup>

TEVAR aims to stable the dissected aorta and prevent late complications by inducing aortic remodeling process. In the INSTEAD trial, the maximum aortic diameter of the patients treated with TEVAR was stable but significantly increased in patients who were medically treated during 5 years.<sup>[40]</sup> In the present study, TEVAR lead to aortic remodeling by stabling the maximal aortic diameter of 3 levels during an average follow-up of 26.4 months. The complete false lumen thrombosis rate in the thoracic aorta was more than 90% in the acute and chronic group but was relatively low in the abdominal false lumen (Table 4). It has been reported that higher false lumen thrombosis predicted better long-term outcome.<sup>[41,42]</sup> This finding implied that long-term surveillance after TEVAR is vital, and distal arterial tree should be monitored by imaging examination during follow-ups. Besides, the false lumen complete thrombosis rate tends to be significantly higher in acute group compared to chronic group, which suggests that the capacity for aortic remodeling in CAD is weaker.<sup>[43,44]</sup> During the chronic phase, the adventitial and intimal flaps become more stable and rigid, thus reducing the capacity of prominent remodeling ability to open up the distal true lumen. However, our results and several other reports indicated that this phenomenon seems to have no effect on the postoperative outcomes.<sup>[5,45]</sup> Since aortic dissection is a systematic disease with entire aorta and its branches, systemic hypertension, older age, aortic size, and

presence of patent false lumen essential predictors of late complications. Therefore, basic anti-impulse drugs, including  $\beta$  blockers, are required to decrease aortic wall, and regular surveillance is necessary to improve prognosis.<sup>[34,35]</sup> Aortic diameter was also reported to be a risk factor for mortality. In the IRAD study, 18.4% of patients with acute type B aortic dissection present with a descending aortic diameter of 5.5 cm or greater, in-hospital mortality could reach as high as 23.0%, which was significantly higher than these with an aortic diameter less than 5.5 cm ( $P < .001$ ).<sup>[46]</sup> In our study, 2 patients died within 30 days after surgery and these patients had aortic diameter of 5.5 cm or greater; therefore, aortic diameter of 55 mm or greater is an indicator for mortality and more attention should be paid.

False lumen (FL) diameter was also an important factor impacting morbidity and mortality. Song et al reported a 22 mm initial false lumen diameter in the upper thoracic descending aorta was a predictor for late aneurysm with a sensitivity of 100% and a specificity of 76% and also demonstrated higher aneurysm or death rate than others.<sup>[47]</sup> Chang et al<sup>[48]</sup> reported that false lumen size was an indicator for in-hospital complications after acute type B aortic dissection, and patients with maximal false lumen area (MFLA)  $\geq 922 \text{ mm}^2$  showed higher complications than other patients. Therefore, more aggressive procedures should be applied for patients with larger false lumen diameter.

However, there exist some limitations in this study. First, it represents a retrospective analysis of collected data and selection biases may exist. Second, the conclusions were drawn from a single institution and the number of patients was limited, a larger patient cohort with long-term outcomes is still required. Finally, several types of stent grafts were utilized, and this difference may influence the final clinical results to some extent.

In conclusion, TEVAR procedure is an effective alternative option for complicated type B aortic dissection, which gains a sufficient proximal landing zone with a healthy aorta for the stent graft to fix and seal. The technical success rate and early and mid-term mortality and morbidity are acceptable in both acute and chronic dissection. However, monitoring of longer-term clinical outcomes and continuous careful surveillance of the entire aorta, especially the distal end of the stent graft, are required, particularly for stainless steel-based stent grafts or any other distal high radial force design.

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