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Original Article

Endovascular Therapy for Chronic Type B Aortic Dissection

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Objectives: Endovascular repair of the thoracic aorta (TEVAR) represents a therapeutic option for type B aortic dissection. However, the optimal timing for TEVAR is controversial. We examined the outcomes of TEVAR for chronic type B dissection and reviewed aortic morphology using pre- and postoperative CT scan images.
Methods: Between 2012 and 2017, 12 patients underwent TEVAR for chronic type B dissection at our institution. We retrospectively reviewed the clinical and operative data including CT scan images, comparing the values between early group (5 cases, 3 months to 1 year from initial dissection) and late group (7 cases, more than 1 year from initial dissection).
Results: There were no paraplegia, stroke, and death in our cohort. There was no difference in degree of the aortic remodeling between two groups.
Conclusions: Outcomes after TEVAR for chronic type B aortic dissection were favorable. Aortic remodeling could be obtained in selected patients by closing an entry with TEVAR procedure. (This is a translation of Jpn J Vasc Surg 2018; 27: 281–287.)

Keywords: aortic dissection, endovascular surgery, operative indication

Introduction

Recently, the INSTEAD-XL trial has shown that entry occlusion with early thoracic stent grafting/thoracic endovascular aortic repair (TEVAR) for uncomplicated type B aortic dissection improves long-term outcomes.[1,2] Treatment can be challenging as the expansion of the true lumen can be difficult to obtain even if a stent graft is placed at the chronic stage as intima hardens and thickens over time in aortic dissection.[3] However, effective remodeling with entry occlusion with TEVAR has been reported in patients who have received dissection ≥ 1 year previously.[4]

Nevertheless, the optimal timing for TEVAR in chronic-stage type B aortic dissection is controversial. In addition, considering the risk of new onset of stent edge dissection as a complication of TEVAR for aortic dissection,[5–7] the indications for TEVAR in uncomplicated type B aortic dissection, for which conservative treatment in the acute stage produces favorable outcomes, have not yet been defined.

In our department, the policy is to prefer conservative treatment for uncomplicated type B acute aortic dissections and to perform TEVAR only for patients with large (≥45-mm diameter) or rapidly enlarging (≥5 mm growth per year) dissecting aortic aneurysms. In this study, we report our treatment outcomes with TEVAR.

Methods

Among 25 patients who underwent TEVAR for type B aortic dissection in our department between November 2012 and December 2017, 12 patients who underwent the procedure for extensive communicating DeBakey type IIIb dissecting aortic aneurysms from the site of entry to the site of re-entry below the abdominal aortic branch were included in this study.

Among the 12 target patients, 5 who underwent TEVAR at 3 months to 1 year after the onset of dissection were categorized into the early chronic group and 7 who underwent TEVAR at >1 year after the onset were categorized into the late chronic group. Clinical outcomes were compared between the groups using preoperative and postoperative computed tomography (CT) scans. Thirteen patients, including one with rapidly enlarging DeBakey

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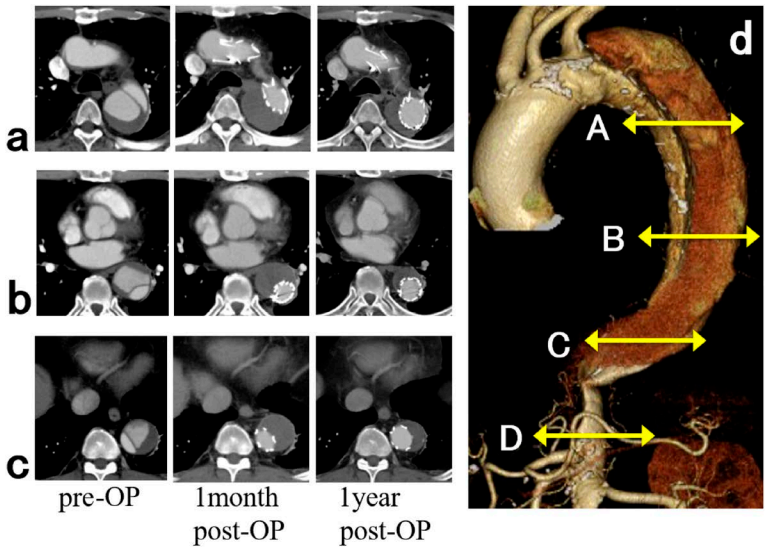

Fig. 1 Three-dimensional CTA reconstruction demonstrated type B dissection. A: Carina level, B: aortic valve level, C: posterior descending level, D: thoracoabdominal level. (**a**) Change of the aorta after TEVAR at A, (**b**) change of the aorta after TEVAR at B, (**c**) change of the aorta after TEVAR at C.

type IIIa disease at 1 month, and 12 with dissecting aortic aneurysms containing localized enlarging ulcer-like projections (ULP), were excluded.

Contrast CT scans were performed 1 month, 6 months, and 1 year after TEVAR, and then annually. The presence or absence of endoleaks and false lumen thrombosis was noted, and the aortic diameter was measured in the arterial and delayed phases. The maximum short axis diameter of the aorta in the axial plane was measured at the following levels: (A) upper descending (tracheal bifurcation level), (B) central descending (aortic valve level), (C) lower descending (level of the mid-cardiac vein in the posterior ventricular sulcus), and (D) thoracoabdominal (level immediately above the celiac artery) (**Fig. 1**). The false lumen diameter (maximum short axis diameter of the aorta excluding the minor diameter of the true lumen in the same plane) was also measured at the same levels (**Figs. 1a–1d**).

For statistical analysis, the t-test (continuous data) and χ^2 test (categorical data) were used. Numerical values were expressed as mean and standard deviation. Statistical significance was defined as $p<0.05$.

Surgical Indications and Procedure

Patients with a maximum short diameter of the aorta of ≥ 45 mm and/or enlargement of ≥ 5 mm per year during radiographic follow-up underwent TEVAR since 2012.

As per the TEVAR policy, the basis was to secure the central landing by clearing a 20-mm region in the non-dissected area, concomitantly using the debranching method when there was a need to occlude the left common carotid and left subclavian arteries. Commercially available stent grafts were used with specific model selection based on the characteristics and size of each device. The diameter of the blood vessel distal to the dissection was calculated as the mean of the short and long diameters of the true lumen. Graft size was selected according to the instructions for use (IFU) for both proximal and distal sides. The shortest diameter was selected when multiple devices could be selected based on IFU. Two stent grafts were often used owing to the narrowing of the true lumen on the peripheral side; device caliber differences were compensated for by prolonging the overlap between the central device and peripheral device. In addition, when deciding the device length, at least one of the paired lower intercostal arteries was always preserved.

Results

There were no significant differences in patient background between the chronic early and chronic late groups. Period from dissection onset to TEVAR was 235 ± 90 days in the chronic early group and 917 ± 385 days in the chronic late group (**Table 1**). The devices used in the chronic early group were CTAG (W. L. Gore & Associates, Flagstaff, AZ, USA) in 2 patients, Zenith TX2 (Cook Medical Incorporated, Bloomington, IN, USA) in 1 patient, and RELAY Plus (Bolton Medical, Barcelona, Spain) in 2 patients. In the chronic late group, the devices used were CTAG in 4 patients and RELAY Plus in 3 patients. In the chronic early group, 2 patients underwent 1-debranch TEVAR. In the chronic late group, 5 patients underwent 1-debranch TEVAR and 1 underwent 2-debranch TEVAR. The mean duration of surgery was significantly longer in

Table 1 Characteristics

	Early chronic (n=5)	Late chronic (n=7)
Age (year)	67.8±10.5	64.7±10.3
Male (%)	3 (60)	6 (86)
Co-morbidities (%)		
Smoking	2 (40)	3 (43)
DM	1 (20)	0
CKD	2 (40)	0
COPD	1 (20)	3 (43)
Hypertention	5 (100)	6 (86)
Coronary artery disease	1 (20)	1 (14)
TEVAR from onset (day)	234±89	917±385

DM: diabetes mellitus; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease

the chronic late group (270 ± 48 min) than in the chronic early group (148 ± 74 min), which included more patients who underwent debranching. Stroke and paraplegia were not observed in either group, and all patients were discharged without perioperative complications. The mean duration of follow-up after surgery was 27.8 ± 6.6 months in the chronic early group and 23.1 ± 5.8 months in the chronic late group. There were no aortic dissection-related deaths or re-interventions in either group; however, 1 patient in the chronic late group died 3 years after surgery because of lung cancer.

At the time of onset, the maximum short axis diameter of the aorta was 39.7 mm in the chronic early group and 36.7 mm in the chronic late group; however, the difference was not statistically significant ($p = 0.39$). No significant differences were found between the chronic early and late groups in terms of true lumen diameter (17.7 mm vs. 15.4 mm; $p = 0.52$) and false lumen diameter (19 mm vs. 20.9 mm; $p = 0.63$). The rate of expansion of the aorta diameter was 14.4 mm/year in the chronic early group and 5.3 mm/year in the chronic late group.

On postoperative follow-up CT, no endoleaks were noted in any patient. However, extensive thrombosis of the false lumen was observed 1 month after surgery in all the patients and it continued to progress to the descending distal side. One year after surgery, the level of thrombosis was almost unchanged. Thrombosis extended over the entire length of the false lumen to just above the site of re-entry in 5 patients with re-entry surrounding the abdominal segmental branches (2 in the chronic early group and 3 in the chronic late group), except for locally patent false lumina that remained as ULP consistent with the re-entry. One patient in the chronic early group showed all false lumina thrombosed without a re-entry. There were 7 patients with re-entry surrounding the abdominal branches and in the iliac artery region (3 in the early group and 4 in the late group); 1 of these patients in the chronic early group showed all false lumina thrombosed, including the site of re-entry. In 3 patients, the false lumen was patent from the site of re-entry of the abdominal segmental branch to the site of re-entry of the iliac arterial region (1 in the chronic early group and 2 in the chronic late group), while the thoracic false lumen showed thrombosis just above the site of abdominal re-entry. In the remaining 3 patients (1 in the chronic early group and 2 in the chronic late group), the thrombosis stopped in the descending thoracic aorta.

Changes in the diameter of the aortic false lumen after TEVAR were measured in 3 thoracic areas: (A) upper descending, (B) middle descending, and (C) lower descending. Compared with measurements before surgery (**Fig. 2**), a reduction over time was observed in both the chronic early and late groups, providing evidence of sufficient aortic remodeling in both the groups. However, in 2 of the 3 patients in whom the false lumen thrombosis stopped in the descending thoracic area (both in the chronic late group), the false lumen diameter remained unchanged at C; slight enlargement was observed in the remaining patient (chronic early group). Subsequently, changes in the maximum short axis diameter of the aorta after TEVAR were then measured and compared in four regions: (A) upper descending, (B) middle descending, (C) lower descending, and (D) thoracoabdominal areas (**Fig. 3**). In A, B, and C, adequate reduction in the maximum short axis diameter of the aorta was observed in both the chronic early and late groups over time. In D, the diameter remained unchanged in both the groups. In addition, 2 years after surgery, the reduction in aortic diameter in the chronic early group in B and C improved further. In one patient whose thrombosis had stopped in the descending thoracic aorta, the aortic diameter slightly enlarged; the diameters before and 24 months after surgery were 41 and 47 mm, and 37 and 43 mm, respectively. An increased aortic diameter and enlarged false lumen were observed in the same patient in the chronic early group. Entry was present in the descending thoracic aorta and re-entry in the Celiac artery, superior mesenteric artery (SMA), and bilateral common iliac arteries. TEVAR was performed using CTAG at 71 days after the onset of dissection; the false lumen was thrombosed in the descending thoracic aorta, but was patent distally from this site to the common iliac arteries on both sides.

Discussion

Uncomplicated type B acute aortic dissection has a relatively good prognosis with conservative treatment, as recommended in the 2011 revised guidelines for the treatment of aortic aneurysms/dissection.[8] The incidence

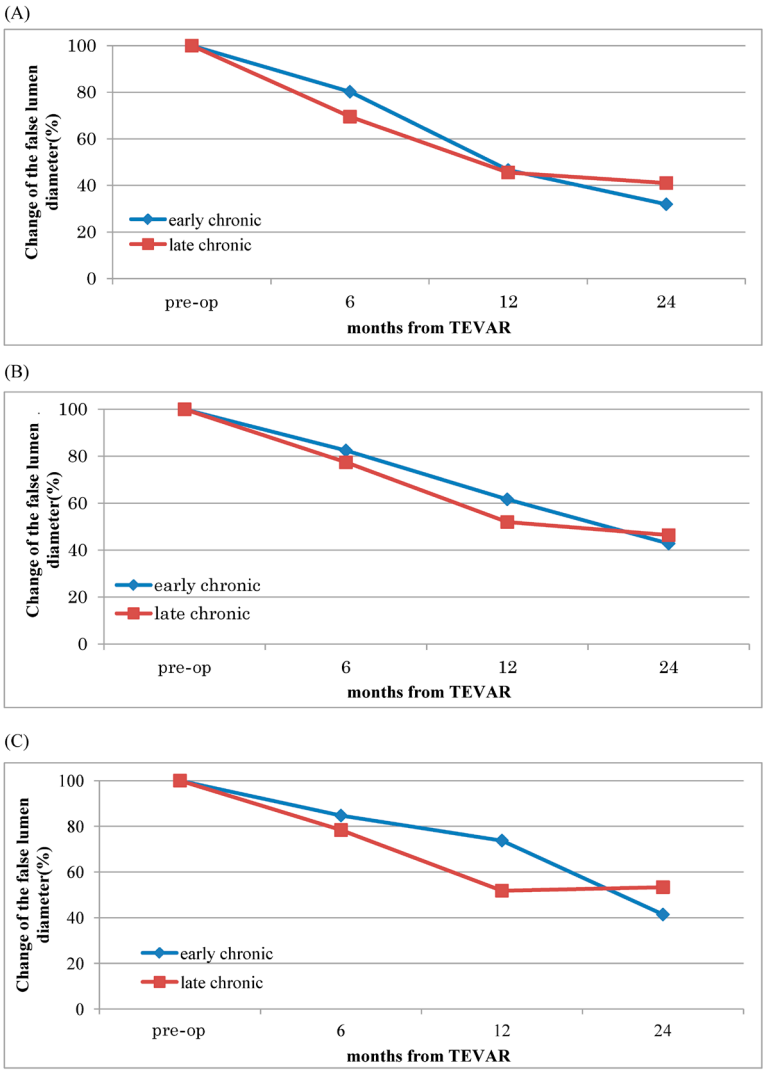

Fig. 2 (**A**) Change of the false lumen diameter at carina level. (**B**) Change of the false lumen diameter at aortic valve level. (**C**) Change of the false lumen diameter at posterior descending level.

of aneurysm enlargement and events such as recurrent dissection in the chronic stage is not low, and the outcome has been reported to be poor in patients with communicating aortic dissection or aortic diameters of >40 mm at the time of dissection in particular (**Table 2**).[9]

Recently, the INSTEAD-XL trial has shown that early TEVAR for uncomplicated type B acute aortic dissection resulted in improvements in long-term outcome.[1,2] In addition, obtaining a therapeutic effect with aortic dissection is difficult since the intima hardens and thickens with time and, even if a stent graft is placed in the chronic stage, sufficient expansion of the true lumen cannot be obtained[3]; however, there have been reports that effective remodeling can be attained with entry occlusion with TEVAR even in patients for whom more than 1 year has passed after the onset.[4] However, the optimal timing for TEVAR for type B aortic dissection in the chronic stage remains controversial.

Currently, only emergency cases such as patients with a ruptured aorta and/or malperfusion are regarded as candidates for TEVAR. The indications and optimal timing for TEVAR in uncomplicated type B acute aortic dissection have not been established. In addition, TEVAR-associated complications may occur, including stent edge dissection.[5–7] Additional stent grafts may be placed for iatrogenic distal dissection; however, new proximal tears can result in retrograde type A aortic dissection, requiring surgical replacement of the ascending arch, or death.[1,7] It is easy to predict that earlier after the onset is, the more fragile the intimal flap becomes, which carries a higher risk of progressive or new dissection. Based on this premise, our department policy prefers conservative treatment for uncomplicated type B acute aortic dissection; however, therapeutic intervention is only delivered to patients with

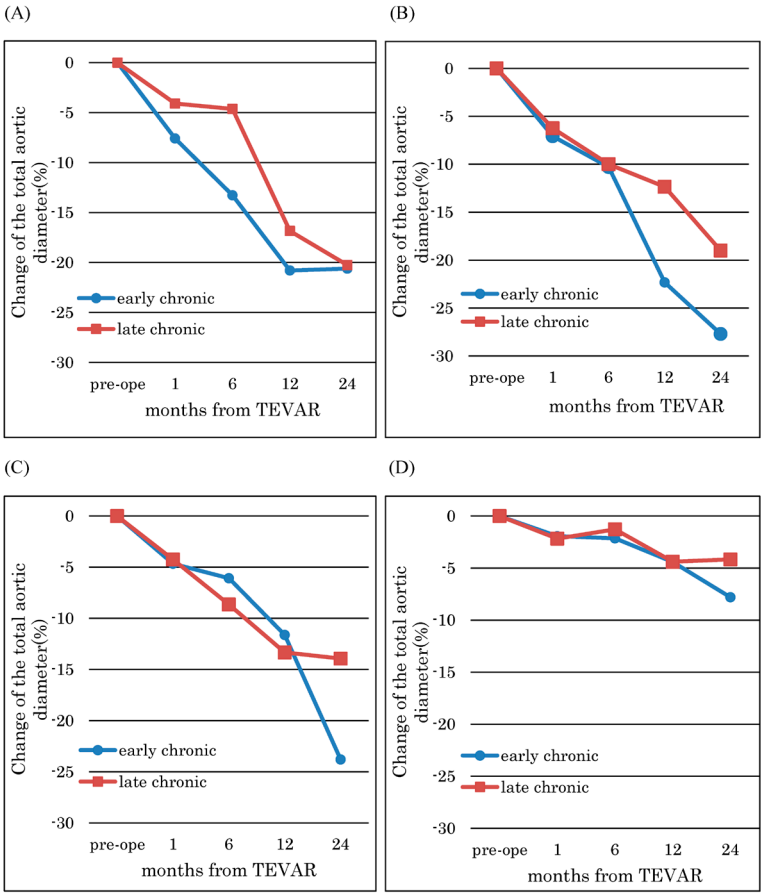

Fig. 3 (**A**) Change of the total aortic diameter at carina level. (**B**) Change of the total aortic diameter at aortic valve level. (**C**) Change of the total aortic diameter at posterior descending level. (**D**) Change of the total aortic diameter at thoracoabdominal level.

Table 2 Early outcomes

	Early chronic (n=5)	Late chronic (n=7)	
Stent-graft			
CTAG	2	4	
TX2	1	0	
RELAY Plus	2	3	
Zone			
3	3	1	
2	2	5	
1	0	1	
Op duration (minute)	148±74	270±48	P<0.05
ICU stay (day)	1±0	1±0	
Hospital stay (day)	10±2	14±8	
Stroke	0	0	
Spinal cord ischemia	0	0	
Aortic event	0	0	
Early death	0	0	
Late death	0	1	
F/U period (month)	27.8±6.6	23.1±5.8	

an obvious expansion of the dissection during the chronic stage (≥3 months or later after onset), i.e., patients with the expansion of the maximum short axis diameter of the aorta to ≥45 mm or a diameter growth rate of ≥5 mm per year.

Although conservative treatment is generally selected for uncomplicated type B acute aortic dissection, approximately 40% of the patients develop dissecting aortic aneurysms during follow-up, and many of these are difficult to treat. In the INSTEAD-XL trial, Nienaber et al. have reported outcomes of patients with uncomplicated disease and aortic diameter of ≤60 mm who were treated with TEVAR during the subacute to early chronic stages (2 weeks to 1 year after dissection onset); no difference in survival or incidence of adverse events in the first 2 years after surgery was noted when TEVAR and conservative treatment were compared.[1)]

However, long-term reductions in aortic dissection-related death and aneurysm enlargement were observed.[2)] In addition, Thompson et al. have reported that aortic remodeling with the reduction of the false lumen and enlargement of the true lumen was similar in the VIRTUE

registry patients who underwent TEVAR in the subacute stage (2 weeks to 3 months after onset) and those who underwent TEVAR in the acute stage; however, there were fewer complications such as retrograde dissection.[10–12] Poor prognostic factors for chronic-stage uncomplicated type B acute aortic dissection included maximum short axis diameter of the aorta of ≥40 mm at the time of onset and patent false lumen.[9,13] Taken together, these reports suggest that performing TEVAR is beneficial in patients having subacute-stage uncomplicated type B aortic dissection associated with poor prognostic factors. In our department also, the mean maximum short axis diameter of the aorta in the chronic early group who underwent TEVAR for aortic diameter enlargement within 1 year of dissection onset was 39.8 mm, which is considered to be consistent with the above prognostic factors, and the rate of expansion of the maximum short axis diameter of the aorta was 14.4 mm/year.

Other studies have reported that TEVAR for dissecting aortic aneurysms in the chronic stage (>1 year after onset) resulted in significant remodeling.[4,14] Patients in our department were followed up for an average of 700±465 days after dissection onset. Extensive false lumen reductions were observed in all patients, and there were patients who underwent TEVAR in the third year after the onset who experienced complete resolution of the false lumen. In addition, there were no significant differences between the chronic early and chronic late groups in terms of the extent of false lumen thrombosis, extent of aortic remodeling, and incidence of adverse events, which is consistent with previous reports. Thus, TEVAR appears to be an effective treatment for chronic-stage dissecting aortic aneurysms.

In the INSTEAD-XL trial, aortic diameters at baseline in patients with dissection and aortic diameter of ≤60 mm in the TEVAR and conservative treatment groups were 44.1 and 43.6 mm, respectively. After 5 years, expansion was noted in the conservatively treated group, with measured diameters of 44.5 and 56.4 mm, respectively. Based on rupture or sudden death after 3 years, TEVAR was concluded useful; however, according to our criteria, the surgery was indicated for most cases already at the baseline. In chronic type B aortic dissection, aortic diameter of ≥40 mm at the time of diagnosis is a risk factor for enlargement, which mostly occurs within 1 year.[15] Cumulatively, these reports suggest that TEVAR is indicated after the subacute stage in patients with uncomplicated type B acute aortic dissection and aortic diameter enlargement to ≥45 mm.

The thoracoabdominal measurement (D) in this study corresponds to the diaphragm level measurement in the INSTEAD-XL trial. That trial showed no significant changes in the aortic diameter at the diaphragm level after TEVAR, with measured diameters of 44.3, 41.2, and 42.0 mm at baseline, 2 years, and 5 years, respectively; however, patients who received conservative treatment alone showed clear enlargement with measured diameters of 41.3, 43.4, and 48.1, respectively; our results are comparable with these findings. If the thoracolumbar/diaphragm level aorta enlarges during follow-up, these aneurysms also require surgical intervention; however, this can be avoided in the medium to long term with TEVAR, as evidenced in the INSTEAD-XL trial and our study. In addition, prioritizing TEVAR in these aneurysms is advantageous if there is enlargement as these can be managed as Crawford type 4 rather than type 2 disease. However, in one patient in this study, the false lumen thrombosis was incomplete, and some patients showed a slight tendency for enlargement. In the chronic early group, there were 2 patients in whom complete thrombosis without re-entry was observed, suggesting that remodeling improved in the early group if the observation period was longer. However, the long-term effects and prognosis remain unclear. Future studies involving larger numbers of patients are warranted.

Conclusion

TEVAR for chronic type B aortic dissection resulted in sufficient aortic remodeling and reductions of the false lumen diameter and thoracic descending aortic diameter and prevented the obvious enlargement of the aortic diameter surrounding the site of abdominal re-entry, demonstrating its effectiveness.

Disclosure Statement

The authors have no conflicts of interest to disclose.

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